

Appendix E

Diesel Engine Exhaust

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Appendix E

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Introduction

The MPCA is concerned about the potential for health effects from diesel exhaust and intends to collect more information to better understand the seriousness of the concern for Minnesotans.

The scientific evidence collected to date indicates that diesel exhaust concentrations higher than current ambient (outdoor) levels can cause lung cancer and other adverse effects in humans (i.e., diesel exhaust is a hazard). The potential for noncancer effects is also a concern. Recent evidence that diesel particulate matter may exacerbate asthma symptoms in asthmatic individuals is of concern and is an area of active research.

As is the case with many environmental pollutants, researchers cannot say whether current ambient diesel exhaust concentrations are causing lung cancer (i.e., the actual risk at ambient concentrations). However, the range in diesel exhaust concentrations believed to be associated with lung cancer is much closer to ambient levels than many other chemicals for which scientists have estimated risks at ambient concentrations. For this reason, and because many people are regularly exposed, the MPCA is concerned about ambient concentrations of diesel exhaust.

In addition to the diesel-specific information summarized in this appendix, diesel particles constitute a sizeable amount of fine particulate matter concentrations, and thus contribute to the premature deaths, hospital admissions, asthma attacks, and other adverse health impacts described in Appendix B, Particulate Matter.

This appendix summarizes the MPCA's current understanding of diesel exhaust including scientific information about the sources of diesel exhaust, ambient and workplace concentrations, the health hazards, and areas of uncertainty where additional information is needed. Key questions remain regarding potential health effects of environmental levels of diesel exhaust.

1.0 Definitions

1.1 What is Diesel Exhaust?

Diesel exhaust is a complex mixture containing hundreds of organic and inorganic materials, in gaseous and particulate forms, from diesel engine combustion processes. Diesel engines include light- and heavy-duty engines in trucks, buses, some automobiles, train locomotives, marine vessels, industrial generators and farm and construction equipment. Diesel exhaust includes both gases and particles. The particles are typically described as diesel particulate matter (DPM). The gaseous fraction contains nitrogen, oxygen, carbon dioxide, water vapor, and many toxic substances including aldehydes. The particles consist of an elemental carbon core with hundreds of organic compounds, sulfates, nitrogen oxides, heavy metals, trace elements, and irritants (such as acrolein, ammonia and

acids) adsorbed to the surface. Specific toxic chemicals of concern include polycyclic aromatic hydrocarbons (PAHs) and nitroarenes (CAL EPA, 1998) which are concentrated in the particle phase. Diesel particulate matter is a subset of ambient particulate matter. EPA approximated that 90% of the diesel particles have diameters less than 1 micron (HEI, 1995) and at least 94% are less than 2.5 microns (CAL EPA, 1998).

1.2 Diesel Engine Exhaust Composition is Variable and Changes Over Time

The particle size distribution and chemical composition of diesel exhaust emissions can vary greatly depending on the engine type (light vs. heavy duty), the speed and load at which it is run, the fuel composition, the lubricating oil, and the emission control technology (CAL EPA, 1998, NTP, 2000). The mass, composition, and particle size distribution of diesel exhaust have also changed over time. For example, by the early 1990's the emissions (by mass) of nitrogen oxides and particulate matter from on-road diesel engines were much lower than in prior years (HEI, 1995). No information relating to possible changes in off-road diesel engine emissions was obtained by EPA (2000a). These changes in diesel engine technology complicate the discovery of possible health effects from current emissions, because most toxicological effect data was obtained from historic diesel emissions.

Despite the lower particulate mass emissions from newer engines, it is not yet clear whether the hazard from diesel particulates has similarly decreased, in other words whether the diesel emissions are less toxic (CAL EPA, 1998). Total mass may not be the best descriptor of health effects. If technology improvements lead to a larger number of smaller particles, with greater surface areas and depositional efficiency, then it is conceivable the hazards could increase with technology improvements. The health implications of the potentially higher numbers of ultrafine diesel particles (less than 0.01 μm) are not yet known (Mauderly, 2000). With respect to the overall fleet of diesel engines in use, diesel engines are well known for their durability and many older models are still in use today. In addition, diesel fuel consumption in Minnesota is expected to continue to increase. EPA concluded it is not clear if the risk of diesel emissions has decreased over time (EPA, 2000a).

2.0 Sources and Emissions

Emission inventories estimate the mass of diesel emissions from various source categories. "Mobile" sources, such as on-road or off-road large trucks, diesel powered passenger vehicles, and some farm and construction vehicles, are the main sources of diesel exhaust emissions in Minnesota. Other sources include diesel generators used for emergency electricity and peak demand periods.

2.1 Diesel Exhaust Emissions in Minnesota

Fine particles emitted from combustion sources, such as diesel engines, are emitted directly. They also from secondarily, as gases (such as nitrogen oxides, sulfur oxides and volatile organic compounds) emitted into the atmosphere condense and undergo chemical reactions. The following emission inventory information includes only the primary (direct) emissions of diesel particulate matter. The mass of secondary diesel particulate matter formation in Minnesota is not known at this time.

Table 1 illustrates that the majority of the inventoried diesel emissions in Minnesota result from on-road and off-road mobile sources rather than from large point sources. Based on the Minnesota inventory, the off-road mobile source diesel particulate emissions are about 50 percent higher than the on-road sources.

Table 1. 1996 Total diesel particulate emissions

Source Category	Emissions (lb)	Percentage (%) of Direct Estimated Emissions
Point	173,580.00	1.31
On-road	5,363,511.21	40.61
Off-road	7,668,920.18	58.07
Total	13,206,011.39	100.00

Small diesel engines were not included in this inventory, except those within the off-road mobile source category. However, the contribution of these engines is not significant.

Table 2 shows the diesel particle emissions contribution from the various sources that are part of the off-road mobile source category, based on the 1996 Minnesota inventory.

Table 2. 1996 Diesel particulate emissions from off-road mobile sources

Source Category	Emissions (lb)	Percentage (%) of Direct Estimated Emissions
Construction	4,513,678.54	58.86
Farm	1,558,507.60	20.32
Railroads	778,014.59	10.15
Industrial	441,041.32	5.75
Airport Services	244,371.38	3.19
Light Commercial	86,418.03	1.13
Logging	28,163.32	0.37
Lawn & Garden	18,725.40	0.24
Total	7,668,920.18	

EPA estimated that mobile sources contribute 98 percent of all diesel particulate matter emissions and that on-road heavy duty diesel vehicles contribute a third of that and off-road equipment contributes the remainder (EPA, 2000b).

EPA also reported that on a national scale on-road diesel emissions (trucks, some cars) have been decreasing, while there is limited evidence suggesting off-road emissions (locomotives, ships, heavy-duty equipment) may be slightly increasing (EPA, 2000a).

In comparison with gasoline-powered vehicles, diesel engines are superior in fuel economy and durability. They emit less carbon dioxide, carbon monoxide and hydrocarbons than gasoline engines, but they emit relatively more nitrogen oxides and particulate matter per mile traveled (HEI, 1999a). Thus the use of diesel rather than gasoline is advantageous in lessening global climate change gases (CO₂) but disadvantageous in terms of generating higher fine particulate (PM_{2.5}) emissions.

3.0 Concentrations and Trends

The MPCA does not measure of the ambient diesel exhaust concentrations or Minnesotans' personal exposures. A concentration is the mass of material in a cubic meter of air (e.g., µg/m³). A personal exposure is the overall exposure an individual experiences in their daily lives from breathing air indoors, outdoors, and while commuting, working and during all daily activities. Personal exposure concentrations can differ from the concentrations in outdoor air. For diesel exhaust, indoor residential concentrations have been estimated to be lower than the outdoor air concentrations (EPA, 2000b).

3.1 Estimates and Direct Measurements from Occupational Settings

Most of the direct human evidence of the adverse health effects of diesel stems from findings in occupational settings where diesel exhaust exposures were higher than in typical outdoor or residential settings. Overall, the Health Effects Institute estimated average diesel particulate matter air concentrations in workplace settings to range from 4 µg/m³ to 1,700 µg/m³ (EPA, 2000a).

In 1989, the National Institute of Occupational Safety and Health (NIOSH) estimated that 1.35 million workers were exposed to diesel particles in about 80,000 U.S. workplaces. (NTP, 2000). Estimates of workplace eight-hour average diesel particulate matter exposures range from 1 to 100 µg/m³ diesel particulate matter in trucking or transportation occupational settings and from 100 to 1,700 µg/m³ for underground mining with diesel equipment (HEI, 1995). Underground miners have been identified as the workers with the highest occupational exposures to diesel particulate matter (66 Federal Register 5706). Exposures of U.S. miners are further described in the January 19, 2001 Federal Register (Volume 66, beginning on page 5561). Woskie et al. (1988) estimated railroad workers' personal exposures to respirable particulate matter, reported as geometric means, to be 17 µg/m³ for clerks and 134 µg/m³ for locomotive shop workers.

In another study, diesel exposures were estimated by using a surrogate measurement, namely that of very fine elemental carbon ≤ 1 µm (EC₁). EC₁ measurements for workers of the trucking industry were measured in 1990 and found to average 1.6 µg/m³ for dock workers, 26.6 µg/m³ for mechanics, 5.4 µg/m³ for short-haul drivers, 5.1 µg/m³ for long

haul drivers, 3.4 $\mu\text{g}/\text{m}^3$ for roadside area samples and 1.4 $\mu\text{g}/\text{m}^3$ for off-roadway area samples (HEI, 1999a). Using an estimated conversion factor of 1.04 to convert from EC_1 to diesel, the California Air Resources Board estimated an overall diesel exhaust concentration range between 5.3 $\mu\text{g}/\text{m}^3$ to 27.8 $\mu\text{g}/\text{m}^3$ diesel particulate matter in these occupational settings (CAL EPA, 1998).

3.2 Estimates and Direct Measurements of Diesel Particles in Ambient Air

Currently methods don't exist to directly measure diesel particulate matter in air. Ambient air concentrations of diesel particulate matter have been estimated using at least three general approaches.

1. Ambient air concentrations can be estimated from emission inventory data (lb. emitted per year) and air dispersion modeling.
2. Elemental carbon, a major component of diesel particulate matter, (contributing approximately 50 to 85 percent of diesel particulate matter in most ambient environments (EPA, 2000a)), has been used as a surrogate to estimate diesel particulate matter concentrations. Other sources of elemental carbon include gasoline particulate matter, combustion of coal, oil, and wood, charbroiling, cigarette smoke, and road dust. More than one method for measuring elemental carbon exists and can lead to different results (CASAC, 2000b).
3. Chemical mass balance source apportionment models use a chemical-specific fingerprinting approach to identify sources of chemicals in mixtures.

On a national scale, average ambient exposures of the general public to diesel exhaust are generally believed to fall in the range of 1 $\mu\text{g}/\text{m}^3$ to 10 $\mu\text{g}/\text{m}^3$ (HEI, 1995). Urban DIESEL PARTICULATE MATTER concentrations are generally higher than concentrations in rural areas. EPA estimated the U.S. annual average airborne diesel soot concentration in 1990 was 1.80 $\mu\text{g}/\text{m}^3$, with urban and rural averages of 2.03 $\mu\text{g}/\text{m}^3$ and 1.10 $\mu\text{g}/\text{m}^3$, respectively (EPA, 1993).

3.2.1 Ambient Concentrations and Exposure Estimates

In outdoor air, Los Angeles' 1982 average monthly diesel concentrations diesel were estimated to range from 1.7 $\mu\text{g}/\text{m}^3$ to 3.3 $\mu\text{g}/\text{m}^3$ in low pollution areas, and EPA estimated that the highest monthly average concentrations might be 10 $\mu\text{g}/\text{m}^3$ in the most polluted areas during winter (HEI, 1995). It is likely that short exposures in street canyons (roads with high buildings on either side) of urban areas would be higher than 10 $\mu\text{g}/\text{m}^3$ (HEI, 1995). Numerous methods to measure or predict diesel particulate matter in a number of ambient locations during the 1980's and early 1990's estimated average concentrations ranging from 0.2 to 23 $\mu\text{g}/\text{m}^3$ for 24-hour measurement periods (CARB, 1998).

The California Air Resources Board (CARB) used the 1990 PM_{10} inventory and air dispersion modeling to calculate the statewide exposure to diesel exhaust PM_{10} . Stratified by air basin, the estimated outdoor population-weighted concentration of diesel exhaust PM_{10} ranged from 0.2 $\mu\text{g}/\text{m}^3$ in the Great Basin Valley to 3.5 $\mu\text{g}/\text{m}^3$ in the South Coast Air

Basin. The population-weighted average outdoor diesel exhaust PM₁₀ concentration in California for 1990 was 3.0 µg/m³. As would be expected, the population-weighted overall average is more reflective of the higher concentrations breathed in the densely populated South Coast Air Basin. CARB predicted declining diesel particle concentrations for 1995, 2000, and 2010 if CARB's policies were implemented. These estimated concentrations were 2.2 µg/m³, 1.8 µg/m³, 1.7 µg/m³, respectively (CARB, 1998).

Using measurements of EC, average annual diesel particulate concentrations ranged from approximately 2.5 µg/m³ to 4.5 µg/m³ across a number of communities in the vicinity of Los Angeles (South Coast Air Quality Management District, 1999).

Rough estimates of ambient diesel particulate matter concentrations in Minnesota can be surmised by considering information from other states that have done monitoring. Ambient diesel particulate matter monitoring information is available from Phoenix, AZ and Denver, CO. Measured ambient concentrations in two Colorado cities during 1996 were 1.7 µg/m³ and 1.2 µg/m³. Sampling in Phoenix, AZ during 1994 and 1995, measured an average concentration of 2.4 µg/m³ (EPA, 2000b).

3.2.2 Evidence of Higher Outdoor Concentrations

Short-term higher exposures may occur near diesel sources. For example, on busy urban streets, street level breathing zone concentrations of diesel exhaust particulate matter have been estimated to be as high as 30 µg/m³ (HEI, 1995). Diesel soot is considered a minor fraction of the overall fine particulate matter emitted in most urban settings, but it constitutes the majority of the particulate matter emitted from on-road vehicles (HEI, 1995).

One study measured PM_{2.5} and elemental carbon in an urban neighborhood (Harlem, New York City) with high diesel traffic (Kinney et al., 2000). In this study, 8-hour mid-day samples were taken at 4 locations during July 1996. The average PM_{2.5} ranged from 37 µg/m³ – 47 µg/m³, while elemental carbon, which was used as a surrogate measure of diesel exhaust particulate, ranged from 1.5 µg/m³ to 6 µg/m³.

Evidence based on the elemental carbon surrogate indicates that diesel particulate matter concentrations are measurably higher near common sources of diesel exhaust. For example, in the Netherlands a study of elemental carbon concentrations in the vicinity of schools reported average concentrations of 3.4 µg/m³ at schools within 400 meters of the freeway, compared to 1.4 µg/m³ at schools measured farther away (EPA, 2000b). Concentrations of elemental carbon measured in vehicles on California roads ranged from 2.8 µg/m³ to 36.6 µg/m³, with the higher concentrations occurring when the vehicles were following large diesel vehicles (EPA, 2000b).

A number of studies have assessed the increase in diesel particulate levels near trafficked areas. California Air Resources Board measured elemental carbon and organic carbon to estimate diesel PM₁₀ exhaust and found diesel PM₁₀ concentrations near roadways up to 8 µg/m³ for 24-hour samples (CARB, 1998). One study found diesel particulate

concentrations of 7.1 $\mu\text{g}/\text{m}^3$ at distances of four meters from the road and 8.8 $\mu\text{g}/\text{m}^3$ at one meter from the curb near light-duty diesel traffic (CARB, 1998).

Minnesota-Specific Concentration Information

Currently the MPCA lacks specific information regarding measured diesel exhaust air concentrations for Minnesota. University of Minnesota researchers are studying diesel exhaust exposures within the Metro Transit system. (See this website for more information http://www.healtheffects.org/program_summaries.htm.) Personal and area samples were collected to better identify the contribution of diesel exhaust within ambient urban air. The results of this study will be available shortly.

EPA conducted personal exposure modeling for 1990 diesel particulate matter exposures in Minneapolis and nine other U.S. cities. As described in a recent summary of this effort (EPA, 2000b), the Hazardous Air Pollutant Emissions Modeling (HAPEM-MS3) model was used. The following estimates only reflect on-road diesel particulate matter sources. Nationally, 99 percent of the diesel particulate matter exposures from on-road vehicles were from heavy-duty diesel vehicles (e.g., large trucks used for hauling freight), and one percent were from light-duty diesel vehicles (e.g., diesel powered pick-up trucks). The general population exposure was estimated to be 0.84 $\mu\text{g}/\text{m}^3$ diesel particulate matter. The Minneapolis-specific estimate of public exposure was 1.0 $\mu\text{g}/\text{m}^3$ (EPA, 2000b).

As discussed in Appendix D, Air Toxics of this report, EPA is in the process of conducting a National Air Toxics Assessment (NATA) of the 33 Urban Air Toxics and diesel particulate matter (more info at: <http://www.epa.gov/ttn/uatw/nata/>). Preliminary Minnesota information suggests that county average diesel particulate matter concentrations are approximately 1.7 $\mu\text{g}/\text{m}^3$ in the more densely populated urban counties and about 0.47 $\mu\text{g}/\text{m}^3$ in the rural counties, average county diesel particulate matter concentrations range from a low of 0.068 $\mu\text{g}/\text{m}^3$ for Cook County to a high of 2.5 $\mu\text{g}/\text{m}^3$ for Ramsey County. The statewide county average is approximately 1.4 $\mu\text{g}/\text{m}^3$.

EPA (2000b) compared the exposure concentrations which have been shown to cause lung cancer, with estimated daily exposures experienced by the public, to calculate an approximate exposure margin (Table 3). In this case, the exposure margin is an estimate of the ratio of the study participants' average exposure (a worker) to the average exposure of the general public. EPA estimated the range in annual average diesel particulate matter exposure concentrations to be between 0.84 $\mu\text{g}/\text{m}^3$ and 4.0 $\mu\text{g}/\text{m}^3$ and so calculated an exposure margin for both estimates.

Relatively small exposure margins indicate that ambient levels are fairly close to levels of concern. EPA considers an exposure margin of one or two orders of magnitude to be fairly small (EPA, 2000a). The accuracy of the exposure margin depends on the accuracy of the concentration estimates. In this case, the occupational concentrations in particular are uncertain. The details of this quantitative analysis is described in the report, Regulatory Impact Analysis (EPA420-R-00-026) Chapter II: Health and Welfare, which is accessible at: <http://www.epa.gov/otaq/diesel.htm#documents>.

Table 3. Occupational and Population Exposure to Diesel Exhaust, Environmental Equivalent Exposures and Exposure Margins

Occupational Group	Estimated Occupational Exposure ($\mu\text{g}/\text{m}^3$)	Environmental Equivalent Exposure ($\mu\text{g}/\text{m}^3$)	Exposure Margin Ratio – based on a 0.84 $\mu\text{g}/\text{m}^3$ ambient exposure	Exposure Margin Ratio – based on a 4.0 $\mu\text{g}/\text{m}^3$ ambient exposure
Non-coal Miners	38 – 1,280	8 – 269	10 – 320	2 – 67
U.S. Railroad Workers	39 – 191	8 – 40	10 – 48	2 – 10
Firefighters	4 – 748	0.8 – 157	1 – 187	0.2 – 39
Public Transit Workers, Dockworkers	2 – 98	0.4 – 21	0.5 – 25	0.1 – 5

Excerpt from Table II.A-23 (EPA, 2000b)

Environmental Equivalent Exposure - Occupational exposure (40 hours per week) are recalculated as a continuous exposure (24 hours each day). The equation is environmental equivalent occupational exposure = 0.21 x occupational exposure.

For this example, the exposure margin (EM) is the ratio of the adjusted occupational exposures (those which caused effects) to the lower estimated ambient exposures. Exposure margins are calculated using both high (4.0 $\mu\text{g}/\text{m}^3$) and low (0.84 $\mu\text{g}/\text{m}^3$) estimated exposure concentrations.

3.3 Diesel Particulate as a Portion of $\text{PM}_{2.5}$

Because of their small size, diesel exhaust particles are primarily associated with particulate matter smaller than 2.5 microns ($\text{PM}_{2.5}$). At this time it is not known precisely what fraction of $\text{PM}_{2.5}$ is diesel particulate matter in Minnesota, or how this varies temporally and spatially across the state. It is expected that diesel sources may contribute a significant amount of $\text{PM}_{2.5}$ to the ambient air, particularly in urban areas. EPA estimated that the fraction of diesel particulate matter in $\text{PM}_{2.5}$ is typically in the range of 10 percent, though it may exceed 30 percent in some urban settings (EPA, 2000a). As a first approximation, if the Twin Cities has about 2.5 $\mu\text{g}/\text{m}^3$ diesel particulate matter, and an average of 12.3 $\mu\text{g}/\text{m}^3$ $\text{PM}_{2.5}$ (see Table 12 of the Particulate Matter Appendix), then diesel particulate matter would be about 20 percent of the fine particulate matter by mass. This estimate is in line with estimates from the other cities. Additional information will be needed to characterize the contributions from other sources, such as power plants and woodburning, to Minnesota levels of $\text{PM}_{2.5}$.

Appendix B, Particulate Matter, provides more details on emissions, concentrations, and composition of fine particles.

4.0 Health Information

Diesel exhaust is just one of multiple sources of fine particulate matter and gaseous air pollution, so it is difficult to distinguish the specific health effects of ambient diesel exposures from those of other pollutants (HEI, 1995). Often diesel exhaust health effects studies in laboratory studies have focused on the particulate fraction and reported concentrations as $\mu\text{g}/\text{m}^3$ diesel particulate matter. The lung is the primary organ adversely affected by diesel exhaust. Because of their small particle diameters (most are less than 1 μm), diesel particles are readily inhaled and deposited deeply in the lung.

Information from past occupational exposures to diesel exhaust suggests that workers exposed to elevated diesel concentrations have been harmed by those exposures, suffering from bronchitis, lung function changes, and lung cancer (EPA, 2000a). Although the general public is usually exposed to lower amounts of diesel than those found in occupational settings, diesel exhaust is widely present in urban and rural areas.

In contrast to its potential for causing lung cancer, the noncancer effects of diesel exposures may have at least as great, if not greater, public health impacts, in part because more people may be affected (CAL EPA, 1998). Potential allergenic effects of diesel exhaust are of growing interest. The major noncancer effects of diesel exhaust that the Health Effects Institute (HEI) identified as needing additional research include asthma, respiratory airway inflammation, and allergic responses (HEI, 1999b).

Urban particulate air pollution has been demonstrated to cause a range of adverse effects on the respiratory and cardiovascular systems (HEI, 1999a). The scientific evidence specifically relating to diesel exhaust health effects is summarized below. It is described in terms of effects measured following acute exposures (to short term high levels) and chronic exposures (to lower levels for longer time periods). Both lung cancer and noncancer effects from chronic exposures are discussed.

4.1 Health Effects from Short-term, Elevated Exposures

Based on evidence derived from animal and human data, short-term exposures to high levels of diesel exhaust are believed to irritate the eyes, nose, throat, and bronchi (EPA, 2000a). Neurophysiological symptoms of exposures have included lightheadedness, dizziness, headache, nausea, vomiting, and tingling and numbness of the extremities (EPA, 2000a). Short-term animal studies found diesel exhaust can cause increased susceptibility to lung infection, chronic lung tissue inflammation, and decreased lung function (CAL EPA, 1998). In addition to the fine particles which may cause irritation, diesel exhaust contains respiratory irritants including sulfur oxides, nitrogen oxides, and aldehydes (e.g., formaldehyde and acetaldehyde).

In sensitized individuals who are already allergic to certain allergens (such as pollen), it can worsen their allergic reactions. For example, human exposure to both diesel exhaust particles and ragweed pollen resulted in an immune response greater than that following either alone (Diaz-Sanchez et al., 1997). Similar studies with different fine particle sources have shown somewhat similar results (Mauderly, 2000).

The EPA Clean Air Science Advisory Committee concluded that animal studies have shown cellular and chemical changes that are biological markers consistent with asthma (e.g., increased mast cell influx, increases in immunoglobulin E (IgE), goblet cell hyperplasia and cytokine changes), but that studies have not shown that the condition of asthma was caused by exposure to diesel exhaust (CASAC, 2000b). One expert reviewer noted that 2/3 of the studies in the last 10 years have assessed the immunological changes associated with diesel exhaust and that acute exposures may be of more relevance than

lifetime exposures (CASAC, 2000b). California (CAL EPA, 1998) considers it possible that diesel exhaust particles may increase the prevalence of asthma and other allergic respiratory disease such as hay fever (allergic rhinitis). The possible effects of diesel in causing asthma or in exacerbating asthma attacks (increasing the symptoms in asthmatics) are areas of active research.

Human exposure studies at concentrations in the range of 1,000 $\mu\text{g}/\text{m}^3$ diesel particulate matter for one hour caused slight lung inflammatory responses and altered macrophage function, but did not cause significant changes in lung function (Mauderly, 2000).

In its draft Health Assessment Document, EPA concluded that short-term high diesel exposures can cause reversible changes in human lung function (EPA, 2000a). Most studies found no significant decreases in lung function. However, the respiratory symptoms (cough, phlegm, chest tightness, wheezing) were observed sooner and at lower diesel exhaust concentrations than decreased lung function (EPA, 2000a). Some evidence suggests that smokers may be more sensitive to the effects of diesel exposures than nonsmokers (CAL EPA, 1998).

Animal studies (with rats, mice, hamsters, cats, guinea pigs) found inflammation of the airways but mild to no decreased lung function at concentrations of 6,000 $\mu\text{g}/\text{m}^3$ diesel particulate matter (EPA, 2000a). A number of animal species were assessed for possible reproductive and teratogenic effects (birth defects) but no adverse effects were identified (EPA, 2000a).

4.2 Health Effects from Long-term Exposures

The specific evidence for cancer and noncancer effects from long-term diesel particulate matter exposures is described in the next two sections.

4.2.1 Cancer Health Effects from Long-term Exposures

Is Diesel Exhaust a Human Carcinogen?

The scientific community is in general agreement that exposures to relatively high levels of diesel exhaust are likely to cause cancer in humans. Over 30 epidemiology studies have assessed the potential human carcinogenicity from occupational exposures to diesel exhaust. Diesel particulate matter exposure estimates for these epidemiology studies were based on the workers' job classifications, employment duration, etc., because more specific exposure information was unavailable. Two studies in particular have been used extensively in evaluating lung cancer effects; these were studies of teamsters and of railroad workers (HEI, 1999a). Although none of these studies alone would point to diesel exhaust as a cause of lung cancer, as a group they consistently suggest a weak association between diesel exhaust exposure and human lung cancer (EPA, 2000a, HEI, 1999, CAL

EPA, 1998). Additional information considered in judging whether diesel exhaust could cause cancer in humans includes animal studies and studies of the effects of diesel exhaust on genetic material. The scientific consensus of the organizations listed in Table 4 is that diesel exhaust is a likely human carcinogen at the elevated concentrations found in some occupational settings, but that it is not known whether it can cause human cancer at current ambient concentrations.

In defining the cause of a disease, epidemiologists consider causal associations to be weak if the incidence of the disease (e.g., cancer) isn't at least found to be doubled in the exposed population compared to the non-exposed population. Weaker associations suggest that unrecognized other factors (i.e., confounding factors) may actually cause the disease or that the statistical finding may be influenced by unrecognized biases in study design. In contrast, an example of a strong association is that between smoking cigarettes and dying of lung cancer. In the Six Cities study (Dockery et al., 1993) current smokers who smoked an average of one pack of cigarettes daily for 25 years were found to have an 800% higher greater chance of death from lung cancer than non-smokers (i.e., an 8 times higher chance).

Table 4 summarizes the findings of organizations that have evaluated the diesel exhaust evidence for human carcinogenicity.

Table 4. Health Organizations' Judgements on Diesel Exhaust Carcinogenicity

Organization	Characterization	Comments by Organization
National Institute for Occupational Safety and Health (NIOSH)	potential occupational carcinogen	Studied whole diesel engine exhaust, based on confirmatory animal and limited human evidence
International Agency for Research on Cancer (IARC) (1989)	Group 2A – Probably carcinogenic to humans	Studied whole diesel engine exhaust – sufficient evidence in experimental animals and limited evidence in humans
National Toxicology Program (NTP) (2000)	Reasonably anticipated to be human carcinogens	Studied diesel engine exhaust particulates – limited findings of elevated lung cancer rates in occupational groups exposed in occupational settings (railroad, mine, bus garage, and trucking company workers)
July 2000 Draft EPA Health Assessment Document for Diesel Emissions (2000a)	Diesel exhaust is a probable human carcinogen (Group B1) - Likely to be carcinogenic to humans by inhalation at any exposure concentration	Studied whole diesel engine exhaust

World Health Organization (WHO) (1996)	Diesel exhaust is probably carcinogenic to humans	Rat data support carcinogenicity, human data suggests probably a carcinogen but inadequate information for quantitative risk assessment
American Conference of Governmental Industrial Hygienists (ACGIH) (2001)	Suspected human carcinogen (2001 notice of intended changes)	Studied diesel exhaust particulate matter
California Environmental Protection Agency, Office of Environmental Health Hazard Assessment (OEHHA) (1998)	Reasonable and likely explanation for causing human lung cancer and rat data demonstrating carcinogenicity	Studied diesel exhaust particulate matter
Health Effects Institute (HEI) found a weak association (HEI, 1999b)	Epidemiological data consistently showed a weak association between exposure and lung cancer	Studied whole diesel engine exhaust

Note that although there remains substantial uncertainty regarding the potency for causing human cancer, the EPA weight of evidence rating of B1, indicates EPA found stronger evidence that diesel exhaust is a human carcinogen than a number of other air toxics such as polychlorinated biphenyls (PCBs), chloroform, tetrachloroethylene, methylene chloride, 1,3-butadiene, etc. (refer to the Air Toxics Appendix for a description of EPA's weight of evidence scheme and the ratings for other air toxics).

How potent a carcinogen is diesel exhaust ?

The potency with which diesel exhaust may cause human cancer is highly uncertain. The Health Effects Institute Diesel Work Group, reviewed the epidemiology studies of diesel exposures of workers exposed to higher than ambient levels of diesel exhaust during the 1950's through the 1980's and found a 20% to a 50% increased incidence of lung cancer in the exposed groups compared to workers with minimal exposures (i.e., overall, the exposed people of the study had a 20% to 50% higher chance of lung cancer than the non-exposed people) (HEI, 1999b). This difference in risk represents the difference in risk between the groups, though the individuals are also at risk from other factors such as smoking. California EPA conducted a similar analysis and reported a 40% increase in the risk of lung cancer from certain occupational exposure levels. EPA concluded there is strong but not definitive evidence demonstrating an association between diesel exhaust exposure in workers and increased incidence of lung cancer (EPA, 2000a) and the Science Advisory Board reviewers agreed (CASAC, 2000b).

Some of the diesel epidemiology studies accounted for the confounding effect of smoking which, overall, is clearly the most important contributor to lung cancer incidence. Other potential confounding factors, such as asbestos exposure, diet, socioeconomic factors, environmental tobacco smoke, and nondiesel particles were considered less often, if ever, in these studies (HEI, 1995). Although EPA has identified radon as a significant contributor to the risk of lung cancer, the diesel epidemiology studies also did not control for the possible confounding effect of radon exposure. Despite this source of uncertainty, the consistency of the statistical association has lead most authoritative organizations to judge diesel exhaust to be a likely human carcinogen.

Does a Threshold Exist and What is the Mode of Action for Cancer?

Direct measurements of the relationship (dose response curve) between the amount of exposure and the harm diesel exhaust may cause at typical ambient concentration levels is not currently possible. Health risk estimates are based on extrapolations of effects measured from high exposures to estimated effects at ambient exposure levels.

A threshold is a concentration below which diesel exhaust will not cause health effects. Most scientists believe it is likely that diesel exhaust causes cancer in humans, but are not sure how this may occur or whether there is a threshold for cancer effects. The mode of action by which a chemical causes cancer is relevant to understanding its hazard and whether it is a material that can cause cancer at very low doses or not. Two of the possible methods by which diesel exhaust might cause cancer are listed below, along with an explanation for why it makes a difference.

- DNA-Damaging (Genotoxic) - It is well known that many chemicals present on diesel particles (e.g., benzo[a]pyrene) are likely human carcinogens. As diesel particles are inhaled, if these chemicals are absorbed into the lung cells, and they chemically react with and alter the DNA, and the immune system is unable to destroy the DNA-altered cells, then cancer may result. If diesel exhaust chemicals cause cancer by altering DNA, then inhaling a very small amount of diesel exhaust may cause cancer.
- A Particle Overload Mode of Action Implies a Threshold for Cancer Effects Animal cancer effects also may result from the sheer presence of small particles. The rat appears to be the species most sensitive to the carcinogenic effects of diesel exhaust particles. Rats are well known to have difficulty clearing excessively high amounts of inhaled particles from their respiratory tracts. Studies in rats have shown that several types of particles sized similar to diesel particles, but which lack the cancer-causing chemicals, can cause cancer. It is believed that for these particles, the rat lungs don't clear the particles well, inflammation and immune reactions occur, some cells are damaged and others grow quickly. This chain of events can lead to cancer. This may only occur following exposures to excessively high particle concentrations, at levels greater than those typically found in ambient environments. HEI estimates suggest that in humans, the particle overload threshold may be at approximately 100 to 200 $\mu\text{g}/\text{m}^3$ for continuous exposures or at 500 to 1,000 $\mu\text{g}/\text{m}^3$ for 8 hours per day, 5 days per week

(HEI, 1995). For particles which cause cancer due to this biological mechanism, at low enough concentrations, no cancer would occur. Stated another way, there might be a threshold exposure below which cancer would not occur.

Given the uncertainty regarding a possible threshold for potential cancer effects, and in the absence of clear data to the contrary, regulatory agencies such as EPA typically conclude it is prudent to assume that cancer may occur at low exposure levels.

4.2.2 Non-cancer Health Effects from Long-term Exposures

Heavy exposure to diesel exhaust is clearly associated with upper airway pulmonary inflammation (CASAC, 2000a). The California Air Resources Board (CARB) concluded that long term chronic effects of diesel exhaust exposures in occupational workers may include a greater incidence of cough, phlegm, chronic bronchitis and reduced pulmonary function (CARB, 1998).

Most epidemiology studies have not found diesel to cause increased chronic respiratory disease (CAL EPA, 1998, EPA, 2000a). EPA's Clean Air Scientific Advisory Committee (CASAC) found that fibrosis, emphysema, pulmonary hypertension and associated heart disease occurred in heavily exposed animal studies, but did not consider these likely to occur in humans under environmental or most occupational exposure settings (CASAC, 2000a).

Particles from diesel exhaust can induce immunological allergic reactions and localized inflammatory responses in humans. At some level of exposure, diesel exhaust can cause systemic and pulmonary inflammatory responses in healthy humans (EPA, 2000a).

Recent evidence suggests that heavy-duty truck traffic and environmental levels of diesel air pollution may cause respiratory symptoms, decreased pulmonary function and allergic symptoms in children and adults (CAL EPA, 1998).

Several studies have examined the relationship between various measures of traffic density and health effects in communities. One example is a study in Holland which identified children living closer to major freeways to have more coughs, wheezing, runny noses and more frequent doctor-diagnosed asthma than those children living farther away (EPA, 2000a). Additional studies by Brunkreef et al. have suggested possible associations between traffic density and children's lung function, bronchitis, and allergy to pets and dust (EPA, 2000a).

These traffic studies did not measure diesel exposures, nor did they specifically assess most other factors that may provide alternate explanations for the apparent relationship. These preliminary findings provide suggestive, but not conclusive, evidence that traffic is related to these illnesses. They are useful to generate theories about possible causative relationships that can be further tested with more rigorous epidemiological studies to better assess the possible disease causes.

4.3 What Measures are Used to Assess Whether Exposures are Too High?

The traditional regulatory approach for managing exposures to hazardous chemicals in air is to estimate concentrations of the chemicals in air that are judged to be acceptable. These may be developed for ambient air exposures by the public or for occupational exposures by workers. This report focuses on the measures designed for the public, but provides some information on occupational air concentrations for comparison. Because the occupational concentrations are developed assuming the workers are healthy, and that workday exposures only occur during working hours, they generally result in higher, less restrictive, concentrations than those developed for the general public.

Both for the general public and for workers there may be regulatory standards and/or various types of guideline concentrations. Regulations may provide specific enforcement authority for these concentrations (i.e., they are standards such as the National Ambient Air Quality Standard for particulate matter or the Occupational Safety and Health Administration's Permissible Exposure Limits). Alternately these recommended guideline concentrations may lack enforcement status (i.e., health benchmarks such as Minnesota Department of Health's proposed Health Risk Value for benzene or the American Conference of Governmental Industrial Hygienist Threshold Limit Values). In either case, they may apply to either short-term (e.g., 1 hour, 3 hr, etc.) or longer-term (e.g., a year or a lifetime) exposure periods.

4.3.1 Measures for Short-term Exposures and Non-cancer Effects

There are no specific short-term ambient air federal standards for the public addressing short-term exposures to diesel engine exhaust. EPA didn't propose to develop a health benchmark for short-term high level exposures to diesel exhaust because the dose response information for acute effects was inadequate (EPA, 2000a). Similarly, the Minnesota Department of Health (MDH) has not proposed a short term, acute health risk value (HRV) for diesel exhaust.

4.3.2 Measures For Long-term Exposures for Protection from Non-cancer Effects

Several organizations have reviewed the toxicological literature on the health effects of diesel exhaust and proposed various guidelines (inhalation health benchmarks) for protection from the noncarcinogenic effects of inhalation exposures. The derivation and use of health benchmarks are described in some detail in Appendix D, Air Toxics, of this report. These health benchmarks are used to evaluate the concern about chemical concentrations in air the public breathes.

Available inhalation health benchmarks for protection from the non-cancer health effects of diesel exhaust are summarized in Table 5 along with exposure concentrations currently considered acceptable for workplace exposure. This table illustrates the range in concentrations currently considered acceptable by the following organizations. Note that

with the exception of the mining industry, there is no enforceable workplace standard for diesel exhaust.

Table 5. Diesel Exhaust Benchmark Concentrations for Non-cancer Effects

Organization	Benchmark	Comment
Inhalation Health Benchmark Concentrations for Public Exposures		
Minnesota Department of Health – Proposed Health Risk Value (HRV)	5 µg/m ³	Developed using EPA’s Integrated Risk Information System (IRIS) toxicity information. Developed for lower respiratory system protection and are based on animal studies.
EPA Reference Concentration (RfC) (EPA, 2000a)	5 µg/m ³	4 chronic inhalation rat studies – rats, mice, hamsters, monkeys all showed dose dependent increases in chronic inflammation and lung tissue changes- the RfC will be based on lung tissue changes - Developed for lower respiratory system protection and are based on animal studies.
World Health Organization – 1996	2 µg/m ³ to 21 µg/m ³	Used same data as EPA and California – Developed for lower respiratory system protection and are based on animal studies.
CAL EPA Recommended Exposure Level (REL) (1998)	5 µg/m ³	Same toxicology data as EPA used and a benchmark dose calculation method – Developed for lower respiratory system protection and are based on animal studies.
Occupational Concentration Limits for Worker Exposure		
American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value (TLV) (2001)	20 µg/m ³ elemental carbon (2001 notice of intended changes)	Recommended guideline, not strictly enforceable
Mine Safety and Health Administration (MSHA) 66 Federal Register 5706 (January 19, 2001)	Interim – 400 µg/m ³ total carbon; Final – 160 µg/m ³ total carbon	Enforceable standard. Developed for exposures of workers in the underground metal and nonmetal mining industry -- Currently there is no concentration limit. The interim limit applies as of July 19, 2002 and the final limit will be applicable after January 19, 2006.

Developed for lower respiratory system protection and are based on animal studies

RfC - An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

Threshold Limit Values - refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse health effects. Because of

wide variation in individual susceptibility, however, a small percentage of workers may experience discomfort from some substances at concentrations at or below the threshold limit; a smaller percentage may be affected more seriously by aggravation of a pre-existing condition or by development of an occupational illness.
<https://www.acgih.org/Products/tlvintro.htm>

4.3.3 Measures for Long-term Exposures - Protection from Possible Cancer Effects

To address cancer risks, regulatory scientists traditionally use an inhalation “unit risk factor” to describe how effectively a substance might cause cancer. This unit risk factor is used to characterize the highest amount of cancer that could occur in a population of people who breathe the substance for 70 years. Technically, the unit risk usually represents an upper bound estimate of cancer risk per million people exposed to 1 µg/m³ (a microgram of diesel exhaust particulate in a cubic meter of air) over a 70-year lifetime. The actual risk of cancer may be less than this upper bound estimate, possibly zero. The cancer risk is traditionally assumed to be directly proportional to the long-term average exposure concentration. Inhalation health benchmarks, based on protection from potential cancer effects, are developed from unit risk estimates by dividing the selected risk level (e.g., 1 in 100,000 or 10⁻⁵) by the unit risk estimate.

The scientific regulatory community has a range of opinions about an appropriate unit risk estimate for diesel exhaust. These are summarized in Table 6.

Table 6. Summary of Diesel Exhaust Unit Risk Factors for Carcinogenic Potency

Organization	Unit Risk factors^a	Comments by Organization
MDH	no value available	due to the uncertainty wait for better information before considering potential cancer effects in –HRV development
HEI	no value available	Garshick data is inconclusive; wait for better information
EPA SAB review of EPA 1999 Health Assessment of Diesel Emissions Document	no value should be listed	Too much uncertainty – Don’t use rat data - Wait for better information
EPA’s Scientific Advisory Board	no value should be listed	Too much uncertainty – Don’t use rat data -- Wait for better information

EPA Health Assessment for Diesel (2000a) and CASAC (2000b)	No value selected. Instead for illustration, noted a range of possible upper bound cancer risks from diesel exposure to be 10^{-3} to 10^{-5} , and possibly as low as zero	Range from animal and human data using various calculation methods:
California Air Resources Board Scientific Review Panel	Use 3×10^{-4} as the best point estimate	Selected a point estimate from CAL OHEEA range
California OHEEA Toxic Air Contaminant Documentation	1.3×10^{-4} m ³ /μg to 2.4×10^{-3} m ³ /μg is a range of unit risk estimates	Based on Garshick et al. Case control (1987) and cohort railroad worker studies (1988); Woskei, et al. (1988) exposure estimates

^a Higher unit risk values indicate a higher potency to cause cancer

Clearly the appropriate unit risk value for diesel exhaust is uncertain. A human study that specifically measures the exposures to diesel exhaust in relation to lung cancer effects is needed to help confirm the potency with which diesel exhaust can cause cancer in humans. Ongoing studies, including a joint study by the National Cancer Institute and the National Institute of Occupational Safety and Health, should allow a more accurate estimate of the unit risk value. It is hoped that several ongoing studies will provide better exposure response estimates. The results are expected within a few years.

Although, based on the uncertainty, most organizations have chosen not to calculate cancer risk estimates from the available carcinogenic potency data, California adopted a unit risk value and is using it to report upper bound diesel particulate matter cancer risks in the state.

California used these measurements estimating diesel particulate matter of 2.5 μg/m³ to 4.5 μg/m³ described in Section 3.2 along with the California Scientific Review Panel unit risk value of 3×10^{-4} m³/μg to estimate that diesel particulate matter accounted for the majority of the cancer risk in the air at all long-term monitoring sites. The reported upper bound excess cancer risk estimates ranged from 1,120 to 1,740 per million, based on average annual concentrations (South Coast Air Quality Management District, 1999).

4.4 Issues for Additional Research

Although most scientists agree diesel exhaust most likely can cause lung cancer in humans, defining how potently it causes lung cancer at typical environmental levels is a matter of greater debate. A better understanding the following issues would improve the characterization of the risks Minnesotans face from diesel exhaust and inform effective reduction strategies.

4.4.1 Exposure Estimates in Epidemiology Studies

Most regulatory and health organizations have not identified a cancer unit risk estimate for diesel exhaust primarily because the exposure concentrations in the epidemiology studies were uncertain. Better exposure estimates in future epidemiology studies will allow for better understanding of the potency for causing cancer and the development of a unit risk toxicity value. Cancer unit risk values are used in risk assessment to derive upper bound cancer risk estimates for breathing toxic air pollutants for a lifetime. They serve to provide a frame of reference for identifying pollutants are greater concern.

4.4.2 Cancer Risks at Ambient Exposure Concentrations

Direct measurements of the relationship (dose response curve) between the amount of exposure and the harm diesel exhaust may cause at typical ambient concentration levels is not currently possible. A better understanding of the mode of action for cancer and whether a threshold exists will help scientists to estimate actual risks at ambient exposure levels.

4.4.3 Characterization of Potential Allergic and Asthma Effects at Ambient Concentrations

The potential hazard that diesel exhaust may pose due to effects related to the immune response and asthma need to be better understood. Due the ubiquitous nature of diesel exhaust particulate matter, and the importance of asthma and allergies as a public health problem, a better understanding of these potential effects at ambient concentrations is critical.

4.4.4 Ambient Concentrations and Exposures to Diesel Exhaust and Diesel Particulate Matter

Minimal Minnesota-specific measurements of diesel particulate matter are available. Monitoring data defining the ambient diesel particulate matter levels, areas of higher concentrations, and actual personal exposures are needed to better understand potential Minnesota health risks.

4.4.5 Sources of Exposure

The relative risks from different diesel exhaust emission sources should be better understood, particularly as the technology changes over time. Important issues include understanding which sources are more important in personal exposures and whether the health hazards are best estimated based on measured mass or by some other measure (such as specific chemical composition, size distribution, surface area, etc.).

4.5 Other Sources of Health Information

See Research on Diesel Exhaust at: http://www.healtheffects.org/program_summaries.htm

California EPA Office of Environmental Health Hazard Assessment (OEHHA)
http://www.oehha.org/air/diesel_exhaust/index.html

EPA Integrated Risk Information System Database
<http://www.epa.gov/iris/subst/0642.htm>

Health Effects Institute
<http://www.healtheffects.org/>

EPA Science Advisory Board Reports
<http://www.epa.gov/sab>

Mine Safety and Health Administration (MSHA) <http://www.msha.gov/REGSFINL.HTM>

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