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Estimated Human Health Risk from Exposure to Diesel Exhaust in Toronto

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1. Executive Summary

Dr. Pavel Muller of ToxProbe Inc. prepared this report in response to a Request for Proposals (RFP) expressed by the Health Promotion and Environmental Protection Office of Toronto Public Health (TPH). The Board of Health recommended that the Medical Officer of Health submit a report on the current literature with regard to the relationship between exposure to diesel exhaust and cancer. TPH expanded the Board's goal to include a quantitative assessment of human health risk from exposure to diesel emissions.

Diesel exhaust is emitted from both on-road and off-road vehicles. According to the analysis of the United States Environmental Protection Agency (USEPA), off-road vehicles are more important sources of diesel exhaust than on-road vehicles. Since no data were available for the off-road vehicles, this study focused on the on-road vehicles only. Diesel exhaust is considered to be carcinogenic to humans and many of its components have both cancer-causing effects and other adverse effects on human health. Furthermore, diesel exhaust is released at ground level and close to where people live and work, and thus the chances for exposure are high. In this report, the term "diesel exhaust" refers to that emitted from all diesel fuel-powered vehicles unless otherwise noted.

The data to estimate exposure to diesel exhaust in Toronto are poor and lacking as compared to those available for US urban communities. It was therefore necessary to estimate the Toronto exposure to diesel exhaust indirectly from the total ambient levels of contaminants in Toronto air and the estimated contribution of diesel exhaust to these levels based on the US data.

Assessment of exposure and risk from diesel exhaust was conducted using two approaches. In the first approach (Method 1), exposure and risk were calculated as an aggregate risk from several contaminants present in the exhaust. Method 2 estimated the exposure and risk based on the air concentration of diesel particulate matter (DPM). DPM served as an indicator of exposure and risk for all contaminants present in the diesel exhaust. Thus as the level of DPM increases, the assumption is that all components of the exhaust would also increase.

The assessment was performed for the Toronto residents who also work in Toronto either in indoor or outdoor jobs as adults. Both indoor and outdoor workers are assumed to have been born and raised in Toronto. The outdoor worker's job is assumed to be more strenuous resulting in a higher respiration rate during work-related activities. During the rest of the time, the pattern of indoor and outdoor activities is similar for both the adult outdoor and indoor workers. For the outdoor and indoor workers, three exposure scenarios were assessed:

- Exposures for 9 years to the estimated average Toronto ambient levels of contaminants. Nine years is an average residency time at one address.
- Exposures for 30 years to the estimated average Toronto ambient levels of contaminants. Thirty years is considered to be a reasonable maximum residency at one address. This

scenario was included because many people may move within the Toronto area. This scenario was considered to likely represent the exposures of most Torontonians reasonably well.

- Lifetime exposures to the 90th percentile of ambient levels of selected contaminants. This is assumed to be the reasonable upper bound on the exposure.

On-road diesel is an outdoor source, but the atmospheric contaminants released were assumed to penetrate indoors. The indoor air levels (concentrations) of on-road diesel exhaust contaminants were assumed to be half of those measured outdoors. Since risk assessment assumes continuous exposure to a particular level of a contaminant, the weighted average air levels were calculated taking into consideration time spent indoors and outdoors, age-specific respiration rates and higher respiration rates for outdoor labourers during working hours. The weighted average air levels of contaminants are presented in the top rows of Table 1.1.

Cancer risks were calculated as a product of contaminant levels (as shown in Table 1.1) and the potency factors (unit risks) that are tabulated in Table 5.1. Exposure ratios (ERs) were calculated as ratios of the estimated exposure levels and the permissible exposure level. For non-carcinogens, the permissible exposure level corresponds to the Reference Concentration (RfC, the level below which adverse effects are not expected). In the case of carcinogens, the permissible exposure level corresponds to cancer risk of one in a million (1E-6). In general, ER values of one or less are below the level of regulatory concern. Small exceedances of ER equal to one, such as ER of 1.12 seen for Method 1 (cancer), are not a cause for concern because there is considerable uncertainty associated with any risk assessment, and a difference of less than an order of magnitude in cancer risk may well represent the same actual level of risk. The ERs for cancer effects obtained using Method 2 were substantially elevated. The results are listed in Table 1.1.

Overall, the health risk from diesel exhaust cannot be assessed reliably at this point, partly because of limitations in the monitoring and modeling results for contaminants in Toronto's air. However, based on the available data, the air contamination in Toronto is similar to the typical levels found in US urban areas.

Diesel exhaust is recognized as being carcinogenic, and a number of contaminants present in the diesel exhaust are carcinogenic. Furthermore, diesel exhaust is released near ground level and close to where people live and work, thus the exposure can be expected to be significant.

On the other hand, there is considerable uncertainty associated with the assessment of the toxicity of diesel emissions. The approach used by the California EPA, based on the use of diesel particulate as an indicator for the whole exhaust (Method 2), appears promising as a concept. However, this estimate of potency has not been universally accepted and it should be seen as controversial.

Table 1.1. Exposure-weighted levels of contaminants to which indoor and outdoor workers are exposed and the exposure ratios for the indoor and outdoor workers. Exposure ratios > 1 are bolded and shaded. The non-cancer endpoint is assumed to be independent of the residency duration. See text for further explanation.

	Indoor worker			Outdoor worker		
	9 year exposure, average air levels	30 year exposure, average air levels	Lifetime exposure, maximum air levels	9 year exposure, average air levels	30 year exposure, average air levels	Lifetime exposure, maximum air levels
Levels (µg/m^3)						
1,3-Butadiene	1.03E-03	3.44E-03	1.61E-02	1.24E-03	4.13E-03	1.94E-02
Acetaldehyde	1.96E-02	6.52E-02	3.11E-01	2.35E-02	7.84E-02	3.73E-01
Acrolein	2.44E-04	8.13E-04	3.97E-03	2.93E-04	9.77E-04	4.77E-03
PAHs	5.21E-08	1.74E-07	6.93E-07	6.26E-08	2.09E-07	8.32E-07
Benzene	1.11E-03	3.71E-03	1.49E-02	1.34E-03	4.46E-03	1.78E-02
Formaldehyde	1.74E-02	5.79E-02	2.45E-01	2.09E-02	6.95E-02	2.94E-01
Diesel PM	4.04E-02	1.35E-01	5.70E-01	4.85E-02	1.62E-01	6.84E-01
Exposure ratios						
Method 1 cancer	6.02E-2	2.01E-1	9.30E-1	7.23E-2	2.41E-1	**1.12E+0**
Method 1 non-cancer	1.66E-1		3.20E-1	1.98E-1		3.83E-1
Method 2 cancer	**2.42E+1**	**8.07E+1**	**3.42E+2**	**2.91E+1**	**9.69E+1**	**4.11E+2**
Method 2 non-cancer	2.73E-2		4.63E-2	3.26E-2		5.52E-2

An alternative approach based on the summation of risk attributable to selected individual contaminants found in the exhaust (Method 1) led to substantially lower estimates of risk than the estimates using diesel particulate as an indicator. The risks calculated by Method 1 are below the level of regulatory concern (a risk of 1 in a million or 1E-6). Occasional minor exceedances of risk of 1E-6 are not significant. Since diesel exhaust contains many more contaminants than the ones tested in this assessment, the risk calculated by summing the risk from selective individual contaminants is considered to be the lower bound on the actual risk.

The cancer risk calculated using Method 2 (diesel particulate as an indicator for diesel exhaust) resulted in higher risk levels. For typical Toronto residents working indoors, and for those working outdoors, the risk was between 1 in 10 000 and 1 in 100 000 (1E-5 to 1E-4). It should be stressed again that calculations based on Method 2 utilize a potency estimate that is controversial. It is therefore strongly recommended not to over-interpret the risk estimates. It is likely not appropriate to use them for estimating the number of people in Toronto that are likely to be affected by diesel emissions or for estimating the percentage of cancer risk from exposure to Toronto air that can be attributed to diesel. Rather, it may be prudent to use the risk estimates as an indication that diesel emissions may have a significant impact on Toronto air and to develop policies to minimize the impact.

It is also important to note that studies such as California's MATES II study or Environmental Defense Fund's analysis probably utilize the California potency factor for diesel exhaust. Other problems are likely and are described in Sections 9 and 10. It is therefore recommended that the conclusions from these two studies not be assumed to be correct or applicable to Toronto.

This report attempted to estimate the risk from exposure to diesel exhaust due to on-road vehicles. Although health risk due to off-road vehicular diesel exhaust cannot be reliably calculated at this time, since off-road vehicles are more important emitters of diesel exhaust relative to on-road vehicles, the cancer risk due to diesel exhaust is expected to be higher than the estimated risk due to on-road vehicles alone. Therefore, it is prudent to assume that diesel emissions could have an impact on Toronto air. There are considerable uncertainties about the magnitude of risk posed by diesel, but there is good evidence that diesel exhaust and its components are carcinogenic. Current controversies about the diesel potency need to be resolved before a more reliable assessment is undertaken. The impact on Toronto could be determined more reliably, if data describing the levels of contaminants attributable to various types of diesel engines were available.

2. Background

Dr. Pavel Muller of ToxProbe Inc. prepared this report in response to a Request for Proposals (RFP) expressed by the Health Promotion and Environmental Protection Office of Toronto Public Health (TPH).

The Board of Health recommended that the Medical Officer of Health submit a report on the current literature with regard to the relationship between exposure to diesel exhaust and cancer. TPH extended the Board's goal to include a quantitative assessment of human health risk from exposure to diesel emissions. This report will be the basis for a report to Toronto's Board of Health.

This report explores hazard identification, dose-response, exposure assessment, risk characterization and uncertainty as they pertain to the human health risk of diesel exhaust in Toronto. Comments are also provided on California's MATES II study and on an analysis by the Environmental Defense Fund. Brief descriptions of the properties, fate and toxicokinetics of diesel exhaust are provided in Appendix A, and an introduction to basic risk assessment concepts and methods is given below.

3. Risk, Risk Assessment Concepts and Methodology Overview

This part of the report is intended for readers who are not familiar with risk assessment. Please skip this section if you are well versed with this process.

3.1. Description of Risk

Risk is defined as the probability of the occurrence of an adverse event. For example, the likelihood of having a fatal accident on a commercial flight is 1 in 400,000 (Laudan, 1994)**.** Based on past experience, it is expected that one out of 400,000 passengers will have a fatal accident on a commercial flight. The adverse event in this case is death and the probability of this event (= risk) is one in 400,000. Note that risk indicates a probability, rather than a certainty that the event will take place. For example, the risk of an average person dying of heart disease is roughly 3 in 10. This does not mean that if the cause of death were examined at random for 100 people, exactly 30 would have died of heart disease. The actual number may be higher or lower, but based on past experience, 30 "on average" would be expected to have died of heart disease.

Most lifetime risk from environmental exposure to toxic chemicals is very small (typically 1 in 100,000 or less), and it is sometimes difficult to imagine such a low probability. Table 3.1.1 shows the probability of drawing a certain number of spades in a 13-card hand drawn from a deck of 52 cards. This example is not intended to trivialize the potential risk from environmental exposure. Rather, it is meant to give readers a yardstick that could be useful in visualising very low probabilities.

Table 3.1.1. The probability of being dealt spades in a hand of thirteen cards (from a 52-card deck)

Spades/deal	Likelihood (scientific notation)	Likelihood
5 spades	1.3 E-1	close to 1 in 10
7 spades	8.8 E-3	close to 1 in 100
8 spades	1.2 E-3	close to 1 in 1000
9 spades	9.3 E-5	close to 1 in 10 000
10 spades	6.1 E-6	over 1 in a million
13 spades	1.6 E-12	close to 1 in a trillion

3.2. Risk Assessment

In this context, risk assessment is a scientific activity undertaken to determine the nature and the likelihood of potential adverse effects that may occur in humans and in environments exposed to toxic chemicals. The activities involve evaluating the toxic properties of the chemicals and the conditions of exposure.

As the definition implies, risk assessment requires an understanding of the general properties of chemicals, including their behaviour in the environment and their toxicity, as well as an understanding of how and to what extent humans and non-human living organisms are exposed to these chemicals. In general, risk assessment consists of the following five components:

- Hazard Identification
- Dose-Response Assessment
- Exposure Assessment
- Risk Characterization
- Uncertainty Assessment

Discussion of the individual elements is provided in the sections below.

3.2.1. Hazard Identification

In general, hazard identification provides the first qualitative look at the issue from the perspective of impact on health. The purpose is to gather all relevant information derived from laboratory experimentation and epidemiology to identify the presence of human health hazards in the environment. Subsequent steps of the risk assessment are dependent upon the findings during hazard identification.

The following are some of the issues addressed in hazard identification:

- List of chemicals to be included in the risk assessment and a rationale for selecting them;
- Evaluation of the relevant physical, chemical and toxicological properties of the selected chemicals, as well as their fate in the environment and in humans. Data quality is reviewed and evidence is evaluated using a weight-of-evidence approach.
- Identification of sub-populations (*receptors*) at particular risk because of their special vulnerability to a given contaminant or because of greater exposure;
- Choice of the appropriate pathway and route of exposure for each chemical; and
- Selection of the most sensitive *endpoints* (affected body tissues and the type of effect such as “liver cancer”) and receptors (classes of individuals, such as children or office workers).

3.2.2. Dose-response Assessment

The dose-response assessment identifies the relationship between the exposure level and the magnitude of risk. The toxicity of a chemical is often expressed in terms of lifetime risk per unit exposure. It may also be expressed as a threshold level of exposure below which no adverse effect is expected. In order to get a better understanding of dose-response assessment, it is therefore useful to understand the concept of *threshold*.

Different toxicants can induce their effects by different mechanisms, and this is often reflected in the shape of the dose-effect relationship. Based on the shape of the dose-response curve, the toxicants and their effects are generally classified into two general categories.

- Threshold
- Non-threshold

The difference between the two categories and its significance in the outcome of the assessment are explained below.

Threshold versus non-threshold dose-response effects

Some chemicals are believed to induce adverse effects even at very minute doses, though with low probability. There is no safe level for these chemicals. As the level of exposure increases, so does the risk. At low dose levels, the increase is linear. This means that if the level of exposure increases by two-fold, the risk is also expected to increase proportionately. Many cancer-inducing compounds and some other toxicants fall into this category and they are referred to as non-threshold toxicants.

In contrast, most non-cancer-inducing chemicals and some carcinogens are thought not to induce an adverse effect until a certain minimal exposure (threshold exposure) is reached. Above the threshold, the severity of the effect increases in proportion to the exposure level. For example, atropine will cause widening of the pupil of the eye at a certain concentration. Below that concentration, atropine is thought to have no effect on the pupil. These toxicants are referred to as threshold toxicants.

Some toxicants may illicit both threshold and non-threshold effects. For example, benzo[*a*]pyrene may cause skin irritation at some relatively high dose, but this effect will not be observed at lower doses. At the same time, benzo[*a*]pyrene is a cancer-causing compound and a threshold is not expected for the cancer effect. Under such circumstances, it is possible to assess the chemical as a threshold toxicant, non-threshold toxicant or both. For those chemicals for which both threshold and non-threshold effects have been reported, the usual practice is to focus on non-threshold effects. This approach is used because the levels of toxic chemicals are generally low in the environment. An exposure level could be well below the threshold with respect to the threshold toxic effects, but nevertheless the chemical could still pose a (low) risk

because of its non-threshold effects. In most cases, considering the non-threshold effects of a chemical rather than its threshold effects in the risk assessment process is a more conservative approach and is more protective of human health. If it is not clear which approach is going to be more protective of human health, it is prudent to assess both threshold and non-threshold effects.

Methods for assessment of threshold and non-threshold effects

The distinction between threshold and non-threshold effects is needed because the approach to assessing the risk for the two groups of chemicals is different. For chemicals with a threshold, the purpose of the dose-response assessment is to identify this threshold below which no adverse effect is expected. The *no observable adverse effect level* (NOAEL), or benchmark dose or concentration, is determined either experimentally or from human epidemiological studies. For threshold toxicants NOAEL is a measure of toxic potency. The more potent the threshold toxicant is, the lower the dose level at which no adverse effect is detected. By applying an appropriate *safety factor* that accounts for the uncertainties in the estimation of the threshold, the *reference dose* (RfD) that is also called *tolerable daily intake* (TDI) is determined. The RfD is defined as an estimate of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

Since there is no "safe" level for the non-threshold chemicals, it is necessary to establish a level of exposure for each chemical that is deemed operationally as "tolerable". Such a level is called the risk-specific dose (RsD).

The decision on a "tolerable" risk level differs not only from chemical to chemical but also from organization to organization and circumstance to circumstance, and it is often controversial. What is "tolerable" depends usually on an individual's perspective. However, generally for environmental exposures, a tolerable risk has been operationally defined as the probability of the occurrence of an adverse event ranging from one in ten thousand to one in a million. For the purposes of this report, the risk level of one in a million for human health will be used as the benchmark in this assessment.

The RsD is affected not only by the level of risk deemed tolerable, but also by the potency of the non-threshold effect of the toxicant, generally expressed as the initial slope of the dose-response curve. This slope estimates the additional risk as the exposure is incrementally increased. The higher the potency, the greater the increment of risk resulting from a given increase in exposure, and as a result, the higher the slope of the dose-response curves. The RsD is derived from this slope.

The result of the dose-response assessment provides either an RfD below which the threshold toxicant is expected to pose little or no hazard or an RsD below which a non-threshold toxicant poses a tolerably low probability of an adverse effect. Similarly, in the case of inhalation exposure, the dose-response assessment derives a reference concentration (RfC) for a threshold

effect or a risk specific concentration (RsC) for a non-threshold effect. These values (RfD, RfC, RsD, and RsC) are referred to as exposure limits.

Development of reliable estimates of potencies of individual toxicants for humans is a difficult, labour-intensive, multidisciplinary process. This report follows the general practice of not developing project-specific values, but rather selecting appropriate potency estimates developed by the Ontario Ministry of the Environment (MOE), Health Canada, the United States Environmental Protection Agency (USEPA), the World Health Organization (WHO), the California Environmental Protection Agency (CalEPA) and other credible agencies. The potency estimates developed by the above agencies have undergone an extensive peer review process that serves as an assurance of the quality of the values developed.

3.2.3. Exposure Assessment

Exposure assessment estimates the level (dose) of chemicals in the environment to which different groups of people (subpopulations) are exposed. The degree of exposure depends in part on the environmental levels, but also on the location and activity of individuals. Thus the risk will probably be different for city residents than for farmers living on isolated farms. Levels of physical activity may also affect risk. For example, individuals involved in heavy physical activity will inhale larger volumes of air and thus are exposed to a higher level of an airborne toxicant than individuals involved in light or sedentary activities. Finally, lifestyle characteristics also affect risk. For example, individuals living with a smoker will be exposed to higher levels of the chemicals present in cigarette smoke than individuals living in a home without a smoker.

The goal is to identify the magnitude of exposure for those subpopulations (receptors) that are either particularly vulnerable to a contaminant (e.g. children to lead and methyl mercury) or that are expected, as a result of their lifestyle or other factors, to be exposed to higher levels than the remainder of the population. T*ypical* exposure is also usually determined.

In conducting exposure assessments, it is important to distinguish between *intake* and *uptake* of contaminants. Uptake represents the amount of a contaminant that crosses body barriers (lungs, skin, and intestines) and enters body tissues. Intake is the amount of a contaminant that comes into contact with body barriers but which may or may not be taken up. The potencies provided by the world's leading agencies are typically expressed in terms of intake and thus under most circumstances the exposure is expressed as intake. Sometimes, contaminants are present in a matrix (such as soil), which retards uptake in comparison to a pure compound. It may then be necessary to correct for the reduced bioavailability by a suitable factor.

3.2.4. Risk Characterization

At this stage of the assessment, information about toxicity of the chemicals from the dose-response assessment, and the exposure information for different subgroups from the exposure assessment, are integrated to predict the health impact on various subpopulations. For non-threshold carcinogens, cancer risk can be estimated for the subpopulations identified under selected exposure scenarios. The exposure ratio (ER), a ratio of the estimated exposure and exposure limit, will then be calculated for all chemicals. The exposure limit may be a RfD or a RsD determined in the dose-response assessment section or an environmental guideline. If the ER is approximately 1, it signifies that the exposure is approximately equal to the RfD, RsD or an environmental guideline. A large ER indicates a possible health concern because the exposure level exceeds the desirable maximum level. A low ER suggests a low or negligible impact on human health.

Although risk characterization involves a significant computational component, it is essential that risk characterization includes a thorough discussion that aims to assist risk managers in making safe and pragmatic decisions to mitigate risk. This aspect is stressed in the assessment. Reaching sensible risk management decisions also requires an understanding of the uncertainty involved in any risk assessment, as discussed below.

3.2.5. Uncertainty in Risk Assessment

Ideally environmental risk assessment should be based on human toxicological data where:

- Subjects were exposed to low known environmental levels of a specific toxicant in the absence of any other toxicants;
- Exposure of the assessed population could be accurately determined;
- Individuals did not differ in their response to the same level of exposure; and
- Interaction among toxicants in the environment is well understood and readily quantifiable.

Risk assessments are generally conducted under much less favourable circumstances, and uncertainty is present due to gaps in knowledge from incomplete and/or imperfect available data, from variability among individuals and from measurement errors. It is useful to distinguish between the two types of uncertainty.

- Mathematical uncertainty associated with variability in the data and numerical processing of the data. This type of uncertainty has long been recognised and described in risk assessments.
- Uncertainty due to missing, incomplete or unreliable data and the necessity to use personal professional judgement to compensate for and to fill data gaps.

It is the second type of uncertainty, the uncertainty associated with professional judgement, that is usually the largest source of error. Unlike the uncertainty related to data variability, the judgement-related uncertainty is much harder to express numerically. For example, it is difficult to predict how reliable the estimation of potency in humans is when it has been extrapolated from potency in rodents. Humans and rodents often differ in metabolism and in sensitivity to a given chemical, but how much these differences will affect the potency is often hard to estimate.

Although uncertainty is common in every step of the risk assessment process, dose-response assessment by far contributes the most uncertainty. This is in part because of the frequent need to extrapolate from animal data to humans. It is also always necessary to estimate risk at low environmental levels from known risk at high occupational exposure levels or from animal data obtained at high exposure levels. These extrapolations are difficult to undertake and are potential sources for large errors. This is why the estimates of potency prepared by different agencies for a given toxicant often vary substantially even if they have used the same sets of data to derive their estimates. It is therefore reasonable to assume that the uncertainty of the RfD or RsD of a typical chemical ranges from ten- to a hundred-fold.

Exposure assessment is associated with less uncertainty. The sources of uncertainty include environmental levels at the points of exposure and the lifestyle/activity characteristics of potentially exposed populations. In general, however, the range of uncertainty is much less than ten-fold. The uncertainty associated with exposure assessment is to a large extent captured in the range of exposures estimated for different exposure scenarios for different subpopulations.

4. Hazard Identification

Diesel exhaust is a complex mixture of gases and fine particles that are emitted by internal combustion engines using diesel oil as fuel. Several agencies have classified diesel exhaust and its key components as carcinogens, and carcinogenicity is the main cause for concern with diesel exhaust.

In North America, the diesel engine is used mainly in trucks, buses, agricultural and other off-road equipment, locomotives and ships. The chief advantages of the diesel engine over the gasoline engine are its fuel economy and durability. Diesel engines however, emit more particulate matter per mile driven than gasoline engines of a similar weight class. Over the past decade, modifications of diesel engine components have substantially reduced particle emissions. Further details about diesel emissions are contained in Appendix A.

4.1. Characterization of Diesel Exhaust for the Purposes of the Assessment

Diesel emissions contain thousands of contaminants. Some are volatile and are normally present in the gaseous phase while others tend to become associated with diesel exhaust particles. Some of the classes of contaminants associated with diesel exhaust are listed in Table 4.1.1. The table is based on a USEPA report (USEPA, 2000a, Table 2-19).

This range of contaminants is far too large to assess each compound individually. Data are available only for a handful of these contaminants. In this report, the focus is on the contaminants listed in Table 4.1.2. The selection was done to some degree iteratively and it largely reflects the availability of data. The starting point was a report by the USEPA (1999a and b). This report identified benzene, formaldehyde, 1,3-butadiene, acetaldehyde and diesel particulate matter (DPM) as the key components of diesel emissions. Acrolein and PAH were added later because these were also considered key constituents of diesel emissions and because data for the assessment were available.

Table 4.1.1. Classes of compounds present in diesel exhaust

	Major classes	Minor classes and individual compounds
Particulate phase	Heterocyclics, hydrocarbons (C_{14}-C_{35}), polycyclic aromatic hydrocarbons (PAHs) and derivatives	Acids, Cycloalkanes, Alcohols, Esters, Alkanoic acids, Halogenated compounds, *n*-Alkanes, Ketones, Anhydrides, Nitrated compounds, Aromatic acids, Sulphonates, Quinones
	Inorganics	Elemental carbon, Inorganic sulfates and nitrates, Metals, Water
Gaseous phase	Heterocyclics, hydrocarbons (C_1-C_{10}), and derivatives	Acids, Cycloalkanes, Cycloalkenes, Aldehydes, Dicarbonyls, Alkanoic acids, Ethyne, *n*-Alkanes, Halogenated compounds, *n*-Alkenes, Ketones, Anhydrides, Nitrated compounds, Aromatic acids, Sulphonates, Quinones
	Others	Acrolein, Ammonia, Carbon dioxide, Carbon monoxide, Benzene, 1,3-Butadiene, Formaldehyde, Formic acid, Hydrogen cyanide, Hydrogen sulphide, Methane, Methanol, Nitric and nitrous acids, Nitrogen oxides, Nitrous oxide, Sulphur dioxide, Toluene, Water

4.1.1. Assessing Complex Mixtures

Fundamentally, there are two approaches to the assessment of complex mixtures. The assessment can either be conducted on individual components of the mixture or on the mixture as a whole. Both of these approaches have advantages and disadvantages. The chief disadvantage with conducting the assessment on individual components is that there are many components of complex mixtures, but typically only a handful can be assessed. The result is that the assessment is likely to underestimate the potency of a mixture as a whole. Such underestimates have been reported, for example, for PAHs. The sum of the risk attributable to a handful of routinely measured PAHs underestimates the risk attributable to the chemically isolated *PAH fraction* of a mixture by one to two orders of magnitude (MOE, 1997). Another issue often cited as a source of uncertainty is the assumption that the risk attributable to individual components of the mixture is additive. For these reasons assessing the impact of a mixture as a whole would conceptually be preferable. However, assessing the mixture as a whole can include technical problems, which makes the assessment based on individual components more robust and reliable. These issues are discussed below.

Table 4.1.2. Contaminants considered in the current assessment

Gaseous phase	1,3-butadiene Acetaldehyde Acrolein Benzene Formaldehyde
Particulate phase	PAHs (B[*a*]P as the surrogate) DPM

Assessing a mixture as a whole generally calls for selecting a component of the mixture that can serve as an indicator of the concentration of that mixture (*indicator*). The implied assumption is that the concentration of the indicator in the mixture is representative of the concentration of at least the key contaminants in the mixture. In the case of diesel emissions, diesel particulate matter (DPM) has been selected as an indicator in epidemiological studies, in some monitoring and modeling studies and in the regulatory attempts to develop a potency factor for this complex mixture (for references see USEPA, 2000a; CalEPA, 1998a and b). Recent advances in chemical analytical techniques have facilitated the development of sophisticated molecular source profiles, including detailed speciation of PM-associated organic compounds that allow the apportionment of PM to gasoline and diesel sources with increased confidence.

In this report, both approaches have been used for the assessment. The first approach (*Method 1*) is based on the aggregated impact of selected diesel exhaust components, in this case 1,3-butadiene, acetaldehyde, acrolein, benzene, formaldehyde and PAHs. The air concentration of the PAHs, a group of contaminants, is estimated as the concentration of benzo[*a*]pyrene (B[*a*]P). However the potency used to estimate the impact of PAHs is not that of B[*a*]P, but higher. The potency used is that of the whole PAH fraction of a typical mixture (including B[*a*]P), as shown in Table 5.1. As a result, B[*a*]P serves as an indicator for the whole PAH mixture and it is believed that the risk calculated in this manner estimates reasonably accurately the potency of the PAH fraction of the environmental mixture. In contrast, the potency of the diesel mixture in the air is likely to be underestimated by this method because the mixture contains many other contaminants not assessed in this report.

The second approach (*Method 2*) uses DPM as an indicator for all the contaminants in diesel emissions in a way analogous to B[*a*]P serving as an indicator for PAHs. This approach should in theory reflect the health impact from diesel emissions better than the previous method. However, as discussed in the dose–response section of this report, the estimate of potency using DPM is controversial. The differences between the two approaches are further discussed in the risk characterization section in the context of the results of the assessment.

4.1.2. Estimating Toronto Exposure

One of the major problems with assessing health impacts of diesel emissions in the ambient air in a city like Toronto is that in urban environments, many sources contribute to the ambient levels of each contaminant present in diesel emissions. Although it was stated that modern analytical methods might be used to estimate the source of PM, such analysis is not available for Toronto. The most desirable method for quantifying the amounts of diesel exhaust-derived contaminants would be based on the known number of kilometers diesel vehicles traveled in Toronto in one year and the emission factors for these vehicles established separately for each of the contaminants in diesel exhaust. This information is not available for Toronto and therefore an alternative indirect method was used to derive the ambient concentrations resulting from diesel exhaust alone.

First, the ambient air levels of the contaminants in Toronto were estimated. These levels are a result of emissions from all sources that impact on Toronto, and not solely due to diesel exhaust from vehicular traffic (see Table 4.1.2.1).

Table 4.1.2.1. Ambient air levels of selected contaminants in Toronto

	Toronto measured levels (µg/m^3)	References
1,3-Butadiene	3.20E-01	CEPA , 2000a
Acetaldehyde	2.00E+00	CEPA, 2000b
Acrolein	1.00E-01	CEPA, 2000c
Benzene	2.20E+00	Dann, 1999
B[*a*]P	2.40E-04	CEPA, 1994
Formaldehyde	3.30E+00	CEPA, 2000d
PM	2.40E+01	CEPA/FPAC, 1998

As described below, next it was assumed that the levels of contaminants in ambient air attributable to on-road vehicles in Toronto are a fraction of the ambient air levels and that this fraction is the same as the fraction attributable to on-road vehicles in the USA. Finally, the proportion of ambient levels that is attributable to on-road diesel emissions was estimated.

4.1.3. Levels of Contaminants in Toronto Air Originating from On-road Vehicles

The USEPA (1999a) has estimated the average ambient air levels for a range of contaminants and the levels attributable to on-road emissions, as shown in Table 4.1.3.1. Assuming that the on-road emissions contribute to the same proportion of the overall contaminant emissions in Toronto as they do in the USA, the levels of contaminants in Toronto air attributable to on-road vehicles can be estimated as shown in the following equation.

$$TO = TC \times USO/USC \qquad \textbf{Equation 1}$$

Where:

TO is the level of a contaminant in Toronto air originating from on-road vehicle emissions ($\mu g/m^3$);
TC is the level of contaminant in Toronto air ($\mu g/m^3$);
USO is the average level of a contaminant in US (urban county) air originating from on-road vehicle emissions ($\mu g/m^3$); and
USC is the average level of a contaminant in US (urban county) air ($\mu g/m^3$).

It should be noted that the USEPA does not recommend that this model be used in areas smaller than a state. USEPA states: *EPA strongly cautions that these modeling results should not be used to draw conclusions about local concentrations or risk. The results are most meaningful when viewed at the state or national level; for smaller areas, the modeling becomes less certain. In addition, these results represent conditions in 1996 rather than current conditions.*

The warning is certainly justified in principle. However, examination of the range of estimates contained in Table 4.1.3.1 suggests that the range of values between the 10^{th} and 90^{th} percentile is less than an order of magnitude for all contaminants selected for this study (see Table 4.1.3.2). The range between the 5^{th} and 95^{th} percentiles is only slightly larger. Given the uncertainties associated with the estimates of potency, which may often exceed an order of magnitude, an uncertainty of this magnitude associated with the estimated levels of contaminants is tolerable for this type of assessment.

The estimated levels of the selected contaminants attributable to on-road vehicles in Toronto are presented in Table 4.1.3.3. The levels were calculated as a product of Toronto levels of these contaminants (Table 4.1.2.1) and the proportion of the levels that is attributed to the on-road sources of these contaminants in the urban counties in the USA (based on values in Table 4.1.3.1).

Table 4.1.3.1. US (urban counties) levels of selected contaminants and estimated contributions from various types of sources (µg/m^3)

	Ambient concentration								Source of contaminant				
Contaminant	5th %tile	10th %tile	25th %tile	Median	Average	75th %tile	90th %tile	95th %tile	Major stationary sources	Area & other sources	On-road mobile sources	Off-road sources	Back-ground
1,3-Butadiene	1.29E-2	2.03E-2	4.09E-2	7.34E-2	9.33E-2	1.16E-1	1.75E-1	2.24E-1	3.09E-3	9.49E-3	6.05E-2	2.02E-2	0.00E+0
7-PAH	1.37E-4	2.24E-4	3.71E-4	5.71E-4	1.26E-3	9.80E-4	2.01E-3	3.29E-3	2.86E-4	8.09E-4	1.39E-4	2.91E-5	0.00E+0
Acetaldehyde	1.13E-1	1.84E-1	3.66E-1	6.80E-1	8.92E-1	1.09E+0	1.70E+0	2.41E+0	7.18E-3	7.55E-2	4.83E-1	3.26E-1	0.00E+0
Acrolein	2.13E-2	3.25E-2	5.77E-2	9.59E-2	1.32E-1	1.54E-1	2.58E-1	3.75E-1	4.52E-3	2.40E-2	5.45E-2	4.91E-2	0.00E+0
B[*a*]P	8.73E-6	1.43E-5	2.36E-5	3.64E-5	8.03E-5	6.24E-5	1.28E-4	2.10E-4	1.82E-5	5.15E-5	8.85E-6	1.85E-6	0.00E+0
Benzene	6.48E-1	7.48E-1	1.00E+0	1.41E+0	1.56E+0	1.92E+0	2.50E+0	2.96E+0	3.60E-2	9.25E-2	6.68E-1	2.83E-1	4.80E-1
Formaldehyde	4.16E-1	5.06E-1	7.16E-1	1.07E+0	1.45E+0	1.62E+0	2.45E+0	3.71E+0	1.31E-2	1.45E-1	4.55E-1	5.91E-1	2.50E-1
Diesel PM	5.13E-1	7.18E-1	1.17E+0	1.86E+0	2.41E+0	2.70E+0	4.09E+0	6.06E+0	0.00E+0	0.00E+0	7.25E-1	1.69E+0	

Table 4.1.3.2. Ranges of values found in the US urban counties (ratios of percentile values)

Contaminant	95th percentile/ 5th percentile	90th percentile/10th percentile
1,3-Butadiene	17	9
7-PAH	24	9
Acetaldehyde	21	9
Acrolein	18	8
B[*a*]P	24	9
Benzene	5	3
Formaldehyde	9	5
Diesel PM	12	6

Table 4.1.3.3. Toronto levels of selected contaminants and estimated contribution from on-road sources (µg/m^3)

	Ambient concentration			Source of contaminant		
Contaminant	Toronto outdoor air	US average outdoor air	US 90th %tile outdoor air	US on-road	Toronto average on-road	Toronto on-road 90th %tile
1,3-Butadiene	3.20E-01	9.33E-02	1.75E-01	6.05E-2	2.08E-01	3.89E-01
Acetaldehyde	2.00E+00	8.92E-01	1.70E+00	4.83E-1	1.08E+00	2.06E+00
Acrolein	1.00E-01	1.32E-01	2.58E-01	5.45E-2	4.13E-02	8.07E-02
B[*a*]P	2.40E-04	8.03E-05	1.28E-04	8.85E-6	2.65E-05	4.22E-05
Benzene	2.20E+00	1.56E+00	2.50E+00	6.68E-1	9.42E-01	1.51E+00
Formaldehyde	3.30E+00	1.45E+00	2.45E+00	4.55E-1	1.04E+00	1.75E+00
Diesel PM	2.00E+00	2.41E+00	4.09E+00	7.25E-1	6.02E-01	1.02E+00

4.1.4. Levels of Contaminants in Toronto Air Originating from On-road Diesel Vehicles

The levels of selected contaminants attributable to diesel traffic in Toronto are listed in Table 4.1.4.1. These levels were calculated as a product of the estimated on-road fraction of the concentration of the selected contaminants in Toronto air (Table 4.1.3.3) and the relative proportion of annual emissions from diesel engines relative to gasoline engines in the US (Table 4.1.4.3, from USEPA, 1999b).

US annual emissions of B[*a*]P were not available in the USEPA report. They were calculated using the annual *vehicle miles traveled* (USEPA, 1999b) and B[*a*]P emission factors (µg/mile – see Eastern Research Group, 2000). The estimated annual emissions of B[*a*]P for each vehicle type in the US are presented in Table 4.1.4.2. The proportion of acrolein emissions attributable to diesel was calculated from the annual emissions from diesel vehicles in the USA (1,092 tons; Eastern Research Group, 2000) and the annual emissions attributable to all vehicles (12,400 tons; USEPA, 2000b).

The values in Table 4.1.4.1 are the estimated levels of selected contaminants due to on-road diesel vehicles operating in Toronto. These are the values that were used to estimate risk from on-road diesel emissions in Toronto.

Table 4.1.4.1. Estimated levels of selected contaminants derived solely from on-road diesel vehicles in Toronto air (µg/m^3), and the estimated on-road diesel contribution relative to all vehicles.

Contaminant	Annual emissions: diesel/all vehicle	Toronto on-road diesel	Toronto 90th%tile diesel
1,3-Butadiene	0.074349	1.54E-02	2.89E-02
Acetaldehyde	0.269235	2.92E-01	5.56E-01
Acrolein	0.088054	3.64E-03	7.11E-03
B[*a*]P	0.028513	7.55E-07	1.20E-06
Benzene	0.017604	1.66E-02	2.66E-02
Formaldehyde	0.250579	2.59E-01	4.38E-01
Diesel PM	1	6.02E-01	1.02E+00

Table 4.1.4.2. US B[*a*]P annual emissions by on-road vehicle type

Vehicle	VMT (1E+6 miles/ year)	B[*a*]P EF (μg/mile)	B[*a*]P emissions (μg/year)	Fraction of B[*a*]P annual vehicular emissions
LDGV	1376014	0.565	777447	
LDGT1	665733	0.341	227015	
LDGT2	220261	0.366	80615	
HDGV	51972	0.37	19229	
MC	12374	0.026	322	
Total gasoline vehicles			1104630	0.971487
LDDV	7425	0.002	15	
LDDT	4950	0.002	10	
HDDV	136116	0.238	32396	
Total diesel vehicles			32420	0.028513
Total			1137050	

(USEPA, 1993)

Table 4.1.4.3. Emissions of contaminants from vehicles by vehicle type. Data from USEPA (1999b) unless otherwise indicated. Emissions expressed in US tonnes/year, unless otherwise indicated.

	LDGV	LDGT1	LDGT2	HDGV	LDDV	LDDT	HDDV	MC	Total vehicles	Total diesel vehicles	Diesel vehicles/ all vehicles
1,3-Butadiene	9592	5705	3190	1710	61	63	1527	358	22206	1651	0.074
Acetaldehyde	9149	5759	2995	1871	84	86	7210	256	27411	7380	0.269
Acrolein									12400[1]	1092[2]	0.088
B[a]P[3]	777447	227015	80615	19229	15	10	32396	322	1137050	32420	0.029
Benzene	81864	47830	21965	9246	136	140	2629	1212	165023	2905	0.018
Formaldehyde	24662	16573	9111	8868	263	271	19578	936	80262	20112	0.251
Diesel PM	0	0	0	0	1755	1384	108390	0	111530	111529	1.000

[1] *from Eastern Research group (2000)*
[2] *from USEPA (2000b)*
[3] *from Table 4.1.4.2 (above), values in μg/year*

LDGV - Light duty gasoline-powered vehicles
LDGT1 - Light duty gasoline-powered trucks up to 6,000 lbs gross vehicle weight
LDGT2 - Light duty gasoline-powered trucks from 6,000 lbs to 8,500 gross vehicle weight
HDGV - Heavy duty gasoline-powered vehicles
MC - Motorcycles
LDDV - Light duty diesel-powered vehicles
LDDT - Light duty diesel-powered trucks
HDDV - Heavy duty diesel-powered vehicles.
VMT - Vehicle miles travelled
EF - Emission factor

4.2. Uncertainty in Extrapolating from US to Toronto

There is a measure of uncertainty associated with the estimate of the contribution of on-road diesel vehicles to contamination in Toronto air. That is normal with any measured or modeled data. Nevertheless, if the modeling is to be useful, it needs to predict the measurement reasonably well. Table 4.2.1 is based on the data extracted from USEPA (1999a). The modeled data are for contaminants released by on-road vehicles, while the ambient data reflect the levels of contaminants derived from all sources. On the other hand, mobile sources are the major contributor for all of the contaminants listed in Table 4.2.1. It is therefore expected that modeled levels will be consistently lower, but not vastly different from the ambient measurements. This is true for most of the data presented in Table 4.2.1. The conclusion of the USEPA (1999a) on the reliability of the modeling is as follows.

…despite limited data, the … motor vehicle-related modeled exposure estimates appear to correspond reasonably well with ambient data. With the possible exception of 1,3-butadiene for Houston (where stationary source emissions for this compound may be high), the comparison shows that the modeled exposure and measured concentration are not orders of magnitude different. (From USEPA, 1999a)

Table 4.2.1. Comparison of measured (for all sources) and modeled (for on-road vehicles only) levels of contaminants in outdoor air (µg/m^3)

Pollutant	Urban area	Modelled levels	Measured levels	Modelled/ measured
Benzene	Chicago	0.482	1.4	0.3443
	Houston	0.536	2.9	0.1848
	Minneapolis	1.108	1.9	0.5832
	New York	0.787	1.9	0.4142
Acetaldehyde	Minneapolis	0.269	1.4	0.1921
	New York	0.146	2.5	0.0584
Formaldehyde	Minneapolis	0.459	2	0.23
	New York	0.508	3.1	0.1639
1,3-Butadiene	Houston	0.057	1	0.057

Another important source of uncertainty arises as a result of extrapolation from the generic modeling results obtained for the *urban US counties* to Toronto. Although the USEPA warns that application of these generic data to a specific location may lead to larger errors (see Section

4.1.3), the US data summarized in Table 4.1.3.2 suggest that for most urban areas, the ranges of values are within an order of magnitude. Given the uncertainties, which are often a part of dose-response assessment, such uncertainties in estimating exposure parameters are tolerable. Furthermore, Toronto has few point sources that would distort the relationship between the ambient levels and levels attributable to mobile sources. In cities with major point sources of the selected contaminants, the actual levels attributable to mobile sources may be lower than those predicted from the ambient levels.

The relative contribution of emissions from gasoline vehicles and diesel on-road vehicles in the US urban areas and Toronto could also be different. Nevertheless, the differences in the mix of gasoline and diesel vehicles between the US urban areas and Toronto are not expected to be large enough to be a major source of uncertainty.

Overall, ToxProbe Inc. would expect the estimates of ambient levels of selected contaminants attributable to diesel on-road vehicular traffic to be accurate well within an order of magnitude. This assessment does not assess exposures from off-road diesel vehicles (for example construction vehicles) or from stationary diesel engines. ToxProbe Inc. concluded that extrapolation from US data for these sources may not be reliable. Off-road vehicles can contribute considerably to diesel emissions, as can be seen from the comparison of environmental levels attributable to on-road and off-road vehicle emissions (see Table 4.1.3.1).

4.3. Selection of Exposure Pathways and Receptors

The assessment was conducted for two subpopulations of Toronto, referred to in this report as Local Indoor Worker (LIW) and a Local Outdoor Worker (LOW). Both LIW and LOW were born and raised from childhood in Toronto. The difference is that LIW has an indoor job, while LOW works outdoors. In addition, LIW's job is not physically demanding while the job held by LOW is. As a result, the respiration rate of LOW during business hours was assumed to be higher than that of LIW. The indoor worker represents a large subpopulation of Torontonians who both live and work in the City. People who only work in the City but live in an area with lower traffic density would be expected to receive lower exposure from on-road diesel vehicles. Similarly City residents working in low-traffic areas will be expected to have lower exposures to the exhaust from on-road diesel engines than LIW. LOW is expected to be more exposed to diesel fumes than the indoor worker, although it is assumed that the selected contaminants penetrate into the indoor air and that the level of penetration for the selected contaminants is about 50% (see USEPA, 1996 for discussion and references). This assessment assumes that exposure takes place only via inhalation. Smaller exposures by other routes are possible, but for the selected contaminants these exposures would be negligible and would not be expected to affect the outcome of the assessment.

5. Dose-Response Assessment

The potency estimates of selected contaminants were obtained by reviewing and selecting assessments from Ontario Ministry of the Environment (MOE), Health Canada, the USEPA, California EPA (CalEPA), and the World Health Organisation (WHO). The selected estimates of potency are listed in Table 5.1. The assessments on diesel particulates by the three agencies are controversial and are discussed in some detail in the following section.

Table 5.1. Potency estimates for the selected contaminants and routes of exposure. Bolded diesel particulate values were used in the assessment

Contaminant	Unit risk $(\mu g/m^3)^{-1}$	RfC (mg/m^3)	Reference
1,3-Butadiene	6.3E-6	3.3E-4	USEPA, 1998
Acetaldehyde	2.2E-6	9E-3	USEPA, 1997d
Acrolein		2E-5	USEPA, 1997e
PAH*	2.3E-2		MOE, 1997
Benzene	4.1E-6 (geometric mean)		USEPA, 2000c
Formaldehyde	8.0E-7		USEPA, 1991
Diesel PM	**6E-4** (1.3E-4 and 2.4E-3)[a] 3.4E-5 (1.6E-5 to 7.1E-5)[b]	5E-3[a] 2.3E-3 to 5.6E-3[b] **1.4E-2**[c]	[a]CalEPA, 1998b [b]WHO, 1999 [c]USEPA, 2000a
* *PAH (as measured by B[a]P)*			

5.1. WHO

5.1.1. Non-cancer

WHO (1996, 1999) has developed guidelines for both cancer and non-cancer endpoints for diesel particulates. WHO used several approaches to estimate the Guidance Value for diesel exhaust in 1996, and has retained two of these approaches in 1999. In both cases, the study by Ishinishi *et al.* (1988) forms the basis of the assessment. In this study, the male and female rats (strain F344/Jcl) were exposed to diesel particles by inhalation 16 hours a day, 6 days a week for 130 weeks. The particle concentration for the different exposure groups varied from 0.1 to 1.84 mg/m^3. The observed effects included inflammation, hyperplasia, lung tumors and shortening or loss of cilia in the trachea and bronchi. The no observed adverse effect level (NOAEL) in rats was identified to be 0.41 mg/m^3. This value was converted to an equivalent continuous exposure of 0.23 mg/m^3.

Two general approaches were used for risk characterization. The no-effect level of diesel exhaust particles in humans was calculated using a dosimetric model of Yu & Yoon (1990) to be 0.139 mg/m^3. An uncertainty factor of 25 was applied to calculate the Guidance Value for the general population of 5.6 $\mu g/m^3$. Another approach eschewed the use of the dosimetric model, but applied an uncertainty factor of 100 (instead of 25, which was used with the dosimetric model), producing a Guidance Value of 2.3 $\mu g/m^3$. The description of the guidance value in Table 3.2 of the 1999 WHO report is either incorrect or confusing. This description is based on the 1996 WHO report, which is the source for the Guidance Value calculations reported in the 1999 WHO report. WHO defined the Guideline Value as a level of exposure at which the great majority of people, even among sensitive groups, would be unlikely to experience any adverse effects.

5.1.2. Cancer

WHO (1996, 1999) also developed a unit risk for diesel particles based on cancer potency in rats. The estimated unit risk was based on the rat studies by Mauderly *et al*. (1987), Heinrich *et al*. (1995), Ishinishi *et al.* (1986) and Brightwell *et al*. (1986). In the Mauderly study, male and female Fischer 344 rats were exposed by inhalation 7 hours/day, 5 days a week. The concentration in the treatment groups ranged from 0.35 mg/m^3 to 7.08 mg/m^3. In the study of Heinrich *et al*., female Wistar rats were treated 18 hours/day, 5 days a week. The concentration in the treatment groups ranged from 0.84 mg/m^3 to 6.89 mg/m^3. In the study of Ishinishi *et al*., male and female Fischer 344 rats were treated 16 hours/day, 6 days a week. The concentration in the treatment groups ranged from 0.46 mg/m^3 to 3.72 mg/m^3. In the study of Brightwell *et al*., male and female Fischer 344 rats were treated 16 hours/day, 5 days a week. The concentration in the treatment groups ranged from 0.7 mg/m^3 to 6.6 mg/m^3. The lung tissue concentrations were estimated from the air concentrations using the dosimetric model of Yu *et al.* (1991). It was assumed that the sensitivity of rats and humans is the same for every mg of particulate in contact with 1 cm^2 of lung surface. The model of Yu *et al.* was used to estimate the pollutant air concentrations (experienced by rats), which would produce comparable lung tissue loading in humans. The unit risks were calculated separately for each of the four studies using the linearised multistage model. The calculated unit risks ranged between 1.6E-5 and 7.1E-5 $(\mu g/m^3)^{-1}$.

5.2. USEPA

5.2.1. Non-cancer

USEPA (2000a) also developed an RfC for diesel particles based on rat data. Pulmonary effects, histopathology and inflammation were determined to be the critical non-cancer effects. The rat data were corrected for species differences in deposition efficiency, normal and particle overload lung clearance rates, respiratory exchange rates, and particle transport to lung-associated lymph nodes. Note that although the data manipulation is intended to correct for the differences between

the rat and humans in terms of the delivered dose, it does not address the issue of possible difference between rats and humans in terms of their vulnerability to the effects of diesel emissions. The assessment was based on the study by Ishinishi *et al.* (1988). The NOAEL in rats was identified to be 0.46 mg/m^3. The corresponding NOAEL in humans was estimated using the dosimetric model of Yu *et al*. (1991) to be 0.144 mg/m^3. An uncertainty factor of 10 was applied to the human NOAEL to account for intraspecies variability yielding an RfC of 0.014 mg/m^3.

The USEPA's RfC is based on a large number of animal studies. The studies tend to be of high quality and are in general agreement with each other. The toxicity of diesel exhaust to the organism as a whole and to different tissues and organs is generally well described in the literature. The relative contribution of the carbonaceous particle core of the diesel particle and the adsorbed organic compounds, including PAHs and various heterocyclic and substituted organics, cannot be distinguished at this time. The USEPA considers the uncertainty associated with the RfC to be about one order of magnitude.

5.2.2. Cancer

USEPA had originally derived a cancer unit risk factor for the cancer effects of diesel particles in humans, but the USEPA's Science Advisory Board (see SAB, 2000) did not support the USEPA estimate. The current draft reflects the SAB position and does not include a unit risk for diesel particles, because according to the USEPA (2000a), neither human nor rat data are sufficient to estimate risk from diesel emissions.

5.2.2.1. Human data

In general, the USEPA (2000a) concludes that human data are not suitable for developing a dose response relationship for diesel particulates for the following reasons:

- Matching the toxicological data with past chronic exposures to diesel particulates has not yet been done reliably.
- The impact of smoking on the outcome of the assessment cannot be fully controlled.
- It is difficult to extrapolate from occupational worker data to cancer hazard to the general population and sensitive subgroups.

Railroad studies

Garshick and colleagues (1987, 1988) have conducted both cohort and case-control studies of lung cancer mortalities among U.S. railroad workers who had registered with the U.S. Railroad Retirement Board (RRB).

Garshick *et al.* (1988) cohort study

In the cohort study, lung cancer mortality was determined for 55,407 railroad workers (Garshick *et al.*, 1988). The cohort was selected on the basis of job titles. Based on the description of job activities, workers were classified as exposed or unexposed to diesel emissions (DE). Workers with recognized asbestos exposure were excluded from the study, but a few jobs with some potential for asbestos exposure were included in the cohort. The author reported statistically significant relative risk increases of 1.57 for the 40-44 year age group and 1.34 for the 45-49 year age group, after exclusion of workers exposed to asbestos and controls for smoking.

"A main strength of the cohort study is the large sample size (55,407), which allowed sufficient power to detect small risks. This study also permitted the exclusion of workers with potential past exposure to asbestos. The stability of job career paths in the cohort ensured that of the workers 40 to 64 years of age in 1959 classified as DE-exposed, 94% of the cases were still in DE-exposed jobs 20 years later.

The main limitation of the cohort study is the lack of quantitative data on exposure to DE. The number of years exposed to DE was used as a surrogate for dose. The dose, based on duration of employment, has inaccuracies because individuals were working on both steam and diesel locomotives during the transition period. It should be noted that the investigators included only exposures after 1959; the duration of exposure prior to 1959 was not known. Other limitations of this study include its inability to examine the effect of years of exposure prior to 1959 and the less-than-optimal latency period for lung cancer expression. No adjustment for smoking was made in this study." (From USEPA, 2000a)

Garshick *et al*. (1987) case control study

Garshick and colleagues also conducted a case-control study of railroad workers who died of lung cancer between 1981 and 1982 (Garshick *et al.,* 1987). The author reported statistically significant increased odds ratios for the group with over 20 years of exposure when compared to the group exposed for 0-4 year. The study was based on approximately 650,000 male U.S. railroad workers with 10 years or more of railroad service. The cases were selected from deaths with primary lung cancer. Each case was matched to two deceased age-matched controls. Controls were selected randomly from workers who did not have cancer and who did not die of suicide or accidental or unknown causes. A total of 1,256 cases and 2,385 controls were selected for the study. Smoking history information was obtained from the next of kin.

"The strengths of the case control study are consideration of confounding factors such as asbestos exposure and smoking; classification of DE exposures by job titles and industrial hygiene sampling; and exploration of interactions between smoking, asbestos exposure, and DE exposure. Major limitations of this study include: (a) possible overestimation of cigarette consumption by surrogate respondents; (b) use of the Interstate Commerce Commission (ICC)

job classification as a surrogate for exposure, which may have led to misclassification of DE exposure jobs with low intensity and intermittent exposure, such as railroad police and bus drivers, as unexposed; (c) lack of data on the contribution of unknown occupational or environmental exposures and passive smoking; and (d) a suboptimal latency period of 22 years, which may not be long enough to observe a full expression of lung cancer." (From USEPA, 2000a)

The biggest issue with the railroad studies is the difficulty in demonstrating a dose-effect relationship. Garshick *et al.* (1988) reported a positive relationship of relative risk and duration of exposure by modeling age in 1959 as a covariate in an exposure-response model. However other investigators and Garshick himself reported a flat or negative dose-effect relationship when the exposure is expressed differently. Given the equivocal evidence for positive exposure-response, EPA has not derived a unit risk on the basis of the available railroad worker data.

Teamsters Union Trucking Industry Studies

Steenland *et al.* (1990) conducted a case-control study of lung cancer deaths among different trucking industry occupations. The study found statistically significant increased odds ratios for lung cancer, depending on years of employment. Cases comprised all deaths from lung cancer (1,288). The 1,452 controls comprised every sixth death from the entire file, excluding deaths from lung cancer, bladder cancer, and motor vehicle accidents. Individuals were required to have 20 years tenure in the union to be eligible to claim benefits. Detailed information on work history and potential confounders such as smoking, diet, and asbestos exposure was obtained using a questionnaire. On the basis of interview data and the 1980 census occupation and industry codes, subjects were classified either as non-exposed or as having held other jobs with potential DE exposure. The information on whether teamsters drove diesel or gasoline trucks was not available.

Steenland *et al.* (1998) supplemented the earlier study with exposure estimates based on a 1990 industrial hygiene survey of elemental carbon (EC) exposure (Zaebst *et al.*, 1991) by assuming that the exposure for workers in different job categories is a function of highway mileages traveled by heavy-duty vehicles.

According to the USEPA (2000a), the studies of Steenland *et al.* have the following strengths:

- The studies demonstrated a significant positive trend in lung cancer risk with increasing cumulative exposure to diesel exhaust (DE).
- The smoking histories and asbestos exposure of workers were obtained to the extent possible.
- Exposures of Teamsters are closer to ambient exposures than are those of railroad workers.

The apparent weakness is as follows:

- Both case and control could be exposed to DE.

Reanalysis of DE exposure for this study is underway. Given the ongoing reanalysis of exposure, EPA has not at this time used the Steenland occupational risk assessment findings to derive cancer unit risk estimates.

5.2.2.2. Rat data

The following are the USEPA's (2000a) conclusions regarding the suitability of rat data for deriving a human cancer unit risk factor from exposure to diesel particulates.

- There is an adequate understanding of how DE causes lung tumors in the rat under experimental exposure conditions.
- Prolonged exposure to high concentrations of a variety of insoluble particles including diesel particulate matter (DPM and its carbon core, devoid of organics) causes lung tumors in rats. This occurs through a mode of action that involves impairment of lung clearance mechanisms (referred to as "lung overload response"), leading to persistent chronic inflammation, cell proliferation, metaplasia, and ultimately the development of lung tumors.
- Because this mode of action is not expected to be operative at environmental exposure conditions, the rat lung tumor dose-response data are not considered suitable for predicting human risk at low environmental exposure concentrations.

5.3. Health Effects Institute

The Health Effects Institute (HEI) is an independent source of information on the health effects of motor vehicle emissions. HEI supports research on major pollutants and engages in special review and evaluation activities. Typically, HEI receives half of its funds from the USEPA and half from 28 manufacturers and marketers of motor vehicles and engines in the United States. It occasionally receives funds from other public or private organizations.

An HEI panel of epidemiologists reviewed the two key studies on railroad workers and teamsters reviewed above and concluded that neither study is suitable at the present time for the development of diesel exhaust potency (HEI, 1999). The main reason was the inadequate assessment of exposure, particularly in the railroad worker study. HEI also noted a negative correlation between the duration of employment and lung cancer risk within a given job category in the railroad worker study. The panel recommended against the use of the railroad study in the present form and recommended further work on the teamster study before using it to develop an estimate of carcinogenic potency for diesel exhaust. HEI did not review the rat carcinogenicity studies.

5.4. CalEPA

5.4.1. Non-cancer

5.4.1.1. Human data

CalEPA concluded that the available data from studies of humans exposed to diesel exhaust are not sufficient for deriving a non-cancer health risk guidance value. The lung is the major target organ for diesel exhaust non-cancer effects, but CalEPA concluded that occupational studies have not provided sufficient exposure information to establish a non-cancer health risk guidance value for respiratory effects.

5.4.1.2. Rat data

CalEPA's assessment is based on an earlier USEPA assessment, which has since been withdrawn. However, the CalEPA used the same experimental study of Ishinishi *et al.* (1988) that was used by the USEPA (2000a) and WHO (1999). The derivation process is also similar and thus the estimated RfC is similar to those of USEPA and WHO (see Table 5.1).

5.4.2. Cancer

5.4.2.1. Human data

CalEPA (1998b) estimated the cancer risk from diesel exposure based on the two studies on railroad workers by Garshick *et al*. A range of estimated unit risks was calculated based on different assumptions. As noted above, both USEPA (2000a) and HEI (1999) expressed strong reservations about using these studies for risk assessment.

5.4.2.2. Rat data

CalEPA (1998b) did calculate the cancer risk from rat studies, but concluded that the uncertainty associated with this assessment was too high and set the estimates aside. Instead, CalEPA based its assessment on human data.

5.5. Dose-response Assessment Conclusions

5.5.1. Non-cancer Effects

There has been a general consensus that human data are not suitable for development of RfC for diesel particulate matter. USEPA, WHO and CalEPA all used the same rat data and a similar process to derive the RfC by extrapolation from animal data. The estimates of the RfC derived by the three jurisdictions are within an order of magnitude. Given the estimated magnitude of the uncertainty, these estimates are not significantly different from each other. The assessment by the USEPA is recommended because of the thoroughness with which the process used to develop the RfC was described.

5.5.2. Cancer Effects

There is little agreement among the agencies with regards to the estimation of cancer potency for diesel particulate matter. As can be seen in Table 5.5.2.1, WHO has developed a cancer unit risk using the rat data, while both USEPA and CalEPA considered the rat data unsuitable for extrapolation to humans at environmental levels of exposure. In contrast, CalEPA developed the unit risk based on a human study which HEI, USEPA and the USEPA Science Advisory Board considered unsuitable in its present form for the development of potency. Furthermore, the median estimates based on the human and rodent data differ by more than 1.5 order of magnitude and the extremes of the two ranges differ by more than 2 orders of magnitude (see Table 5.1). There is thus little consensus on what the carcinogenic potency is and even on whether or not currently available data are suitable for the derivation of the cancer potency. Under these conditions, ToxProbe recommends extreme caution in using the WHO or CalEPA potency estimates or in relying on the results of assessments based on these estimates. At the same time, diesel exhaust contains known and suspected human carcinogens and diesel emissions themselves are a probable human carcinogen. Furthermore, there is likely a high volume of diesel vehicular traffic in Toronto and the exhaust is emitted near ground level and close to people's residences and workplaces. The precautionary principle would recommend that effort be made to minimize the release and impact of diesel exhaust when possible, even if the carcinogenic impact cannot be quantified reliably. For these reasons, ToxProbe Inc. recommends considering the use of the WHO and CalEPA estimates, but only as a range-finding estimate for planning and prioritizing purposes. Until there is a reasonable consensus it is strongly recommended that the calculations of cancer risk based on WHO or CalEPA DPM unit risk estimates not be interpreted as reliable estimates of risk to Toronto residents and/or workers.

Table 5.5.2.1. Data used or approved by agencies for developing cancer and non-cancer potencies

		USEPA	WHO	HEI	CalEPA
Non-cancer	Rat data	y	y	NA	y
	Human data	n	n	NA	n
Cancer	Rat data	n	y	NA	n
	Human data	n	n	n	y

y - yes
n - no
NA - not available

6. Exposure Assessment

6.1. Definition of Receptors and Pathways

As described in Section 4.3, the assessment was conducted for two subpopulations of Toronto referred to in this report as Local Indoor Worker (LIW) and Local Outdoor Worker (LOW). Both receptors are Toronto residents and also work in Toronto. LIW holds a physically undemanding indoor job, while LOW is assumed to have a physically demanding job outdoors. LIW represents a large subpopulation of Torontonians who both live and work in the City. People who only work in the City but live in an area with lower traffic density would be expected to receive lower exposure from on-road diesel vehicles. Similarly City residents working in low-traffic areas will be expected to have lower exposures to the exhaust from on-road diesel engines than LIW. LOW is expected to be more exposed to diesel fumes than the indoor worker, although it is assumed that the selected contaminants penetrate into the indoor air and that the level of penetrance for the selected contaminants is about 50%.

In the absence of indoor sources the ambient levels tend to be higher than indoor levels, but because people spend more time indoors than outdoors, it is the indoor exposure to the air contaminants that contributes more to the overall cancer risk. The penetrance of gases and fine particles (such as diesel and other PAH-rich particles) into private homes has been assessed by several investigators and has been estimated to be roughly 50% (for extensive discussion, see USEPA, 1996). However, in Toronto many residents live in apartments with forced ventilation. The penetrance into this type of building has not been adequately studied. It is expected however, that it will be less than in private homes because modern apartment buildings tend to be air-tight and depend on forced air rather than window-opening for ventilation. Thus the assumption of 50% penetrance is probably conservative.

This assessment assumes that exposure takes place only by inhalation. Lower exposures by other routes are possible, but for the selected contaminants these exposures would be negligible and would not be expected to affect the outcome of the assessment.

6.2. Receptor Exposure Parameters

Summaries of the relevant exposure parameters for Local Indoor Worker (LIW) and Local Outdoor Worker (LOW) are included in Table 6.2.1. For the most part, the estimates were obtained from USEPA (1997a, b and c). Table 6.2.2 contains parameters common to both receptors.

Table 6.2.1. Summary of lifestyle and biological factors for different life stages of a *Local Indoor Worker* receptor and a *Local Outdoor Worker* receptor

Parameter	Infant	Pre-school	School	Teen	Adult - LIW	Adult-LOW	Retirement age
Age (yrs) (ED)	0-2	3-5	6-11	12-17	18-64	18-64	65-75
Hours indoor (ET)	21.6	21.4	21.3	20.7	21.3	13.3*	20.8
Hours outdoor (ET)	2.4	2.6	2.7	3.3	2.7	10.7*	3.2
Respiration rate (m^3/h) (IR)	0.235	0.35	0.5	0.59	0.55	0.55/1.3**	0.55
Body weight (kg) (BW)	7.8	17.5	30.8	55.9	72.4	72.4	70.7

* *Hours indoor were calculated by subtracting a 40-hour working week from the weekly time spent indoor by LIW. Hours outdoor were increased by the same number of hours*

** *1.3 m^3/h assumed for outdoor during working hours*

Table 6.2.2. Summary of lifestyle and biological factors common to both receptors

Parameter	Value
Exposure frequency (EF)	52 weeks
Averaging time (AT)	3910 hours
Average respiration (AR)	0.53 m^3/h

6.3. Estimation of Exposure

This section estimates the weighted average exposure of workers from inhalation of contaminants outdoors, and contaminants that may have migrated into the indoor air from outdoors. The weighted lifetime average exposure was calculated using Equation 2,

$$\text{Lifetime average air levels } (\mu g/m^3) = \sum_{1}^{j}\sum_{1}^{i}((C_i)_j \times IR_{i,j} \times ET_{i,j} \times ED_{i,j})/(AR \times AT) \quad \textbf{Equation 2}$$

Where:

C is the concentration of a given contaminant;
i_{th} element refers to the i_{th} life-stage; and
j_{th} element refers to the j_{th} microenvironment (such as residential indoor air).

The parameters are defined in Tables 6.2.1. and 6.2.2. The results are presented in Tables 6.3.1. and 6.3.2.

The lifetime average air levels were calculated using estimated Toronto average pollutant air concentrations (Table 4.1.4.1) as well as the 90th percentile pollutant air concentrations (described as reasonable maximum air concentration in Table 6.3.1. The calculations were conducted for three types of residents, those who reside in Toronto all of their lives (*lifetime*), those with average residency in Toronto (*9 years*) and those with reasonable maximum residency in Toronto (*30 years*).

Table 6.3.1. Estimated lifetime average inhalation exposure of Local Indoor Worker receptor to selected contaminants derived from on-road diesel only (µg/m^3)

	Based on Average air concentration			Based on Reasonable maximum air concentration		
	Lifetime residency	9 year residency	30 year residency	Lifetime residency	9 year residency	30 year residency
1,3-Butadiene	8.60E-03	1.03E-03	3.44E-03	1.61E-02	1.94E-03	6.46E-03
Acetaldehyde	1.63E-01	1.96E-02	6.52E-02	3.11E-01	3.73E-02	1.24E-01
Acrolein	2.03E-03	2.44E-04	8.13E-04	3.97E-03	4.77E-04	1.59E-03
Benzo[*a*]pyrene	4.22E-07	5.06E-08	1.69E-07	6.73E-07	8.07E-08	2.69E-07
Benzene	9.27E-03	1.11E-03	3.71E-03	1.49E-02	1.78E-03	5.94E-03
Formaldehyde	1.45E-01	1.74E-02	5.79E-02	2.45E-01	2.94E-02	9.79E-02
Diesel PM	3.36E-01	4.04E-02	1.35E-01	5.70E-01	6.84E-02	2.28E-01

Table 6.3.2. Estimated lifetime average inhalation exposure of Local Outdoor Worker receptor to selected contaminants derived from on-road diesel only (µg/m^3)

	Based on Average air concentration			Based on Reasonable maximum air concentration		
	Lifetime residency	9 year residency	30 year residency	Lifetime residency	9 year residency	30 year residency
1,3-Butadiene	1.03E-02	1.24E-03	4.13E-03	1.94E-02	2.33E-03	7.76E-03
Acetaldehyde	1.96E-01	2.35E-02	7.84E-02	3.73E-01	4.48E-02	1.49E-01
Acrolein	2.44E-03	2.93E-04	9.77E-04	4.77E-03	5.73E-04	1.91E-03
Benzo[*a*]pyrene	5.07E-07	6.08E-08	2.03E-07	8.08E-07	9.70E-08	3.23E-07
Benzene	1.11E-02	1.34E-03	4.46E-03	1.78E-02	2.14E-03	7.14E-03
Formaldehyde	1.74E-01	2.09E-02	6.95E-02	2.94E-01	3.53E-02	1.18E-01
Diesel PM	4.04E-01	4.85E-02	1.62E-01	6.84E-01	8.21E-02	2.74E-01

7. Risk Characterization

Table 7.1 summarizes the estimated human health risk in Toronto from the exposure to on-road diesel emissions for the Local Indoor Worker. Table 7.3 contains comparable data for Local Outdoor Worker. Each table is divided into two parts. Method 1 estimates the health impact of on-road diesel vehicle emissions as an aggregated impact of selected contaminants present in the diesel exhaust. The aggregated impact is displayed in the row labeled *Total*. Method 2 uses DPM as a surrogate for the entire diesel exhaust. DPM levels are estimated as an indicator of the levels of the diesel exhaust as a whole. Accordingly, the DPM is assigned the potency of the entire diesel exhaust mixture.

In Tables 7.1 and 7.3, cancer risks are shown, and these values have been converted into exposure ratios (ER) in Tables 7.2 and 7.4. The exposure ratio was calculated as a ratio of the estimated risk and a risk of 10^{-6} (one in a million), which most regulatory agencies consider tolerable. Non-cancer ER values are in columns 8 and 9 of Tables 7.2 and 7.4. Non-cancer ERs were calculated as ratios of weekly average levels (see Tables 6.3.1 and 6.3.2) and the corresponding RfCs.

Tables 7.1 and 7.3 show that Method 1 leads to a lower cancer risk estimate than Method 2 by about two orders of magnitude for both LIW and LOW. This is not an unexpected finding. Method 1 estimates the risk from only a few key components of the mixture, yet diesel exhaust contains many other contaminants not included in the assessment. In contrast, Method 2 is intended to estimate the health impact of the exhaust as a whole, thus taking into account all the contaminants in the mixture as well as any possible interactions among them.

It is recommended to regard the Method 1 estimate as a lower bound on the actual risk for diesel exhaust. Method 1 estimates of cancer risk are too low by themselves to be considered a cause for health concern. However, since the risk marginally exceeds one in a million level and since this estimate is treated as a lower bound on the actual risk, it is reasonable to conclude that diesel emissions pose cancer risk to Torontonians, although the risk may be relatively low. This recommendation is supported by the estimates based on Method 2 which suggest that diesel exhaust has a significant health impact on Torontonians although the potency estimate used in Method 2 is highly controversial (refer to the dose-response section of this report). It is not recommended to rely on the Method 2 results as the actual risk to Torontonians. It is recommended that these values be used for prioritizing or planning purposes only.

It is surprising that Method 1 produced higher estimates of non-cancer risk than Method 2 (Tables 7.2 and 7.4). However, the differences are not large. This was true for both LIW and LOW. Summation of ER values is not scientifically appropriate, even though it is often used in a regulatory setting in order to gain an overall picture of the magnitude of the health impact. As both methods lead to non-cancer risk estimates that are below the level of regulatory concern, it is recommended that the City focus its attention on cancer risk from diesel exhaust.

Table 7.1. Estimated human health risk from on-road diesel vehicle exhaust in Toronto – Local Indoor Worker

	Based on Average air concentration			Based on Reasonable maximum air concentration		
	Lifetime residency	9 year residency	30 year residency	Lifetime residency	9 year residency	30 year residency
Method 1: Aggregated health impact from selected components of diesel exhaust						
1,3-Butadiene	5.42E-08	6.50E-09	2.17E-08	1.02E-07	1.22E-08	4.07E-08
Acetaldehyde	3.59E-07	4.31E-08	1.44E-07	6.83E-07	8.20E-08	2.73E-07
Acrolein						
PAHs	9.70E-09	1.16E-09	3.88E-09	1.55E-08	1.86E-09	6.19E-09
Benzene	3.80E-08	4.56E-09	1.52E-08	6.09E-08	7.31E-09	2.44E-08
Formaldehyde	4.05E-08	4.86E-09	1.62E-08	6.85E-08	8.22E-09	2.74E-08
Total	**5.01E-07**	**6.02E-08**	**2.01E-07**	**9.30E-07**	**1.12E-07**	**3.72E-07**
Method 2: Impact using DPM as a surrogate for all contaminants in diesel exhaust						
Diesel PM	2.02E-04	2.42E-05	8.07E-05	3.42E-04	4.10E-05	1.37E-04

Table 7.2. Estimated exposure ratios – Local Indoor Worker

	Cancer – Based on Average air concentration			Cancer – Based on Reasonable maximum air concentration			Non-cancer	
	Lifetime residency	9 year residency	30 year residency	Lifetime residency	9 year residency	30 year residency	Mean air levels	Reason. max. air levels
Method 1: Aggregated health impact from selected components of diesel exhaust								
1,3-Butadiene	5.42E-2	6.50E-3	2.17E-2	1.02E-1	1.22E-2	4.07E-2	2.96E-2	5.56E-2
Acetaldehyde	3.59E-1	4.31E-2	1.44E-1	6.83E-1	8.20E-2	2.73E-1	2.06E-2	3.92E-2
Acrolein							1.16E-1	2.26E-1
PAHs	9.70E-3	1.16E-3	3.88E-3	1.55E-2	1.86E-3	6.19E-3		
Benzene	3.80E-2	4.56E-3	1.52E-2	6.09E-2	7.31E-3	2.44E-2		
Formaldehyde	4.05E-2	4.86E-3	1.62E-2	6.85E-2	8.22E-3	2.74E-2		
Total	**5.01E-1**	**6.02E-2**	**2.01E-1**	**9.30E-1**	**1.12E-1**	**3.72E-1**	**1.66E-1**	**3.20E-1**
Method 2 : An impact using DPM as a surrogate for all contaminants in diesel exhaust								
Diesel PM	2.02E+2	2.42E+1	8.07E+1	3.42E+2	4.10E+1	1.37E+2	2.73E-2	4.63E-2

Table 7.3. Estimated human health risk from on-road diesel vehicle exhaust in Toronto – Local Outdoor Worker

	Based on Average air concentration			Based on Reasonable maximum air concentration		
	Lifetime residency	9 year residency	30 year residency	Lifetime residency	9 year residency	30 year residency
Method 1: Aggregated health impact from selected components of diesel exhaust						
1,3-Butadiene	6.51E-08	7.81E-09	2.60E-08	1.22E-07	1.47E-08	4.89E-08
Acetaldehyde	4.31E-07	5.17E-08	1.72E-07	8.21E-07	9.85E-08	3.28E-07
Acrolein						
PAHs	1.17E-08	1.40E-09	4.66E-09	1.86E-08	2.23E-09	7.43E-09
Benzene	4.57E-08	5.48E-09	1.83E-08	7.32E-08	8.78E-09	2.93E-08
Formaldehyde	4.87E-08	5.84E-09	1.95E-08	8.23E-08	9.88E-09	3.29E-08
Total	**6.02E-07**	**7.22E-08**	**2.40E-07**	**1.12E-06**	**1.34E-07**	**4.47E-07**
Method 2: Impact using DPM as a surrogate for all contaminants in diesel exhaust						
Diesel PM	2.42E-04	2.91E-05	9.69E-05	4.11E-04	4.93E-05	1.64E-04

Table 7.4. Estimated exposure ratios – Local Outdoor Worker

	Cancer – Based on Average air concentration			Cancer – Based on Reasonable maximum air concentration			Non-cancer	
	Lifetime residency	9 year residency	30 year residency	Lifetime residency	9 year residency	30 year residency	Mean air levels	Reason. max. air levels
Method 1: Aggregated health impact from selected components of diesel exhaust								
1,3-Butadiene	6.51E-2	7.81E-3	2.60E-2	1.22E-1	1.47E-2	4.89E-2	3.54E-2	6.64E-2
Acetaldehyde	4.31E-1	5.17E-2	1.72E-1	8.21E-1	9.85E-2	3.28E-1	2.46E-2	4.68E-2
Acrolein							1.38E-1	2.70E-1
PAHs	1.17E-2	1.40E-3	4.66E-3	1.86E-2	2.23E-3	7.43E-3		
Benzene	4.57E-2	5.48E-3	1.83E-2	7.32E-2	8.78E-3	2.93E-2		
Formaldehyde	4.87E-2	5.84E-3	1.95E-2	8.23E-2	9.88E-3	3.29E-2		
Total	**6.02E-1**	**7.22E-2**	**2.40E-1**	**1.12E+0**	**1.34E-1**	**4.47E-1**	**1.98E-1**	**3.83E-1**
Method 2 : An impact using DPM as a surrogate for all contaminants in diesel exhaust								
Diesel PM	2.42E+2	2.91E+1	9.69E+1	4.11E+2	4.93E+1	1.64E+2	3.26E-2	5.52E-2

The results of Method 1 suggest that the aldehydes, especially acetaldehyde, account for a greater proportion of the overall health risk than other high-profile contaminants (benzene, PAHs, 1,3-butadiene). There is good agreement between the Toronto ambient contaminant levels and the average levels in US urban counties (see Table 4.1.3.3), and thus the findings are not specific to Toronto. PAHs are the most potent carcinogen among those considered in this study (see Table 5.1), but they contribute relatively little to the overall health risk due to diesel exhaust. In contrast, acetaldehyde is present in diesel exhaust in relatively high concentrations and thus it appears to have higher impact than other contaminants. Despite these apparent results, it is not recommended to place great weight on this relative contribution to health risk, because a relatively small change in the potency estimates could substantially alter the relative contribution from the various components of the exhaust mixture. Differences in potency of less than an order of magnitude are quite common among major regulatory agencies, and would alter substantially the relative contribution of individual contaminants to the overall risk of the whole mixture.

8. Uncertainty Assessment

General concepts of uncertainty were discussed in Section 3.2.5. The overall uncertainty of this assessment is the aggregate of the uncertainties associated with each step of the assessment. One source of uncertainty is related to the estimate of the proportion of Toronto ambient air concentration that is attributable to diesel exhaust. As discussed in Section 4.2, the data which are available for many parts of the US and which would improve the reliability of the assessment are not available for Toronto. As a result, this assessment had to rely on making a comparison with the US data, in order to estimate the contribution of diesel exhaust to the level of selected contaminants in Toronto air. Another important source of uncertainty is associated with the estimation of diesel exhaust potency, which is discussed in detail in Section 5. At present the potency estimate used in this assessment should be seen as controversial. Method 1, which involves the summation of health risk of individual components present in diesel exhaust is also associated with uncertainty, because only a subset of contaminants present in the exhaust has been included in the study.

9. Comments on California's Mates II Study

One of the requirements of the request for proposals was to review the MATES II (Multiple Air Toxics Exposure Study II) study (AQMD, 1999) and its implications for Toronto. The available report does not contain detailed technical information, which would allow full reconstruction of the risk assessment process. Section 9.1 provides a general summary of the study and Section 9.2 discusses some key strengths and limitations of the study and its implication for Toronto.

9.1. Summary of the Mates Study

The MATES II study was undertaken to identify the air toxics of most concern in the Greater Los Angeles Area. Monitoring was based on two monitoring programs: 10 fixed sites which monitored over a one-year period and a complementary microscale study in which samples were collected for one month at each of 14 additional locations. The microscale sites were selected to reflect potential localized influences of toxic-emitting sources near residential neighborhoods. The MATES study thus built a comprehensive detailed emissions inventory, which spatially allocated emissions from gasoline service stations, perchloroethylene dry cleaning operations and chrome-plating operations, and diesel emissions from on-road and off-road sources. The monitoring data were augmented by and compared to the results obtained by dispersion modeling.

The carcinogenic potency of selected compounds was based on the estimates of both the USEPA and the California Environmental Protection Agency (CalEPA). The contaminants selected for the assessment are listed in Table 9.1.1. The actual potencies (unit risks) selected were not provided.

The authors of MATES II made the following conclusions:

- The average carcinogenic risk in the Basin (Los Angeles area) is about 1.4E-3. Mobile sources (e.g., cars, trucks, trains, ships, aircraft, etc.) represent the greatest contributor. About 70% of all risk is attributed to diesel particulate emissions; about 20% to other toxics associated with mobile sources (including benzene, butadiene, and formaldehyde); about 10% of all risk is attributed to stationary sources (which include industries and other certain businesses such as dry cleaners and chrome-plating operations).
- There are strong seasonal variations in the levels of toxic air contaminants, primarily for those pollutants associated with mobile sources. Elemental carbon (a surrogate for diesel particles), benzene, and butadiene all have seasonal peaks in the late fall and winter months. Lowest levels are observed during the spring and summer months.
- Modeled results are similar to the results of the monitoring studies, but the modeled levels generally underestimate measured values.

Table 9.1.1. Contaminants monitored in the MATES II study

Benzene	Formaldehyde
1,3-Butadiene	Acetaldehyde
Dichlorobenzene (*o*- & *p*-)	Acetone
Vinyl chloride	Arsenic
Ethyl benzene	Chromium
Toluene	Lead
Xylene (*m*-, *p*-, *o*-)	Nickel
Styrene	Cobalt
Carbon tetrachloride	Copper
Chloroform	Manganese
Dichloroethane [1,1]	Phosphorous
Dichloroethylene [1,1]	Selenium
Methylene chloride	Silica
Perchloroethylene	Silver
Trichloroethylene	Zinc
Chloromethane	PAHs
Organic carbon	Elemental carbon

9.2. Discussion of MATES II Study

There are many reasons why the MATES study is not directly comparable to this study for Toronto.

- Geographic and climatic conditions in the Los Angeles area and Toronto are quite different.
- MATES II is based on and includes extensive monitoring and modeling components and similar data are not available for Toronto. With an emissions inventory similar to the one available to MATES II, a more sophisticated and reliable assessment could have been possible for Toronto.
- For the assessment of the impact of diesel emissions, MATES II relies on the California potency estimate of DPM. As discussed in the dose-response section of the report, this assessment is highly controversial.
- It appears that there is an error in the MATES II study in the assessment of the cancer risk from diesel emissions. The exposure of railroad workers in the epidemiological study, on the basis of which the diesel potency was estimated, was expressed in terms of DPM as an indicator. It is important to remember, however, that the exposure was also to other contaminants, including benzene, 1,3-butadiene and formaldehyde. The contribution of these contaminants to the overall carcinogenic potency of diesel exhaust in the railroad study was not separated from the contribution of DPM. As a result, the DPM serves as an indicator for all other components of diesel exhaust and assumes

aggregate potency of the whole mixture. However, in the MATES II study, the risk from diesel exhaust was determined by adding risk represented by DPM indicator to the potency of individual quantified components of diesel exhaust. This double counting has raised the risk from diesel exhaust considerably.

- It is very problematic to assign a specific percentage of health risk to a particular contaminant, because even relatively small differences in potency (such as 5 fold) would completely alter the percentage of the risk attributable to particular contaminants. Such differences in potency estimates are common among regulatory agencies and reflect uncertainties in the estimation of potencies. For example, the USEPA estimates of cancer potency for benzene exposure span almost a 4-fold range. Thus the estimated contribution of benzene to the cancer risk of the mixture would change significantly if one uses the two extremes of potency range in the calculation.

10. Environmental Defense Fund (EDF) Ranking

In it's July 12, 2001 news release, EDF estimated that in the US, "*exhaust from diesel engines accounts for 78% of the total added cancer risk in outdoor air from all hazardous air pollutants combined, based on U.S. Environmental Protection Agency (EPA) data…The analysis is based on a massive EPA study, which provides detailed estimates of the levels of 41 top hazardous air pollutants in every community in the U.S. EPA's previous version did not include information on diesel particulate emissions.*"

No details about the process by which EDF calculated the risk could be found on the Scorecard site (http://www.scorecard.org) or the EDF site (http://www.environmentaldefense.org). As there are no details with regards to how the results were obtained, it is difficult to comment. However, it must be assumed that EDF used the California EPA's estimate of potency of diesel particulate in order to estimate risk. As discussed earlier in this report, the California potency estimate may be based on unsatisfactory epidemiological data and the estimate has not gained general acceptance in the scientific community. As discussed in the context of the Mates II study, small differences in the estimates of potency could have a large effect on the estimated contribution to the overall risk.

11. Conclusions

Overall, the risk from diesel exhaust cannot be assessed reliably at this point. This is in part due to limitations in the monitoring and modeling of contaminants in Toronto air. Based on the available data, the levels of air contaminants in Toronto are similar to the typical levels found in the US urban areas.

Diesel exhaust is recognized as a carcinogen and a number of contaminants present in the diesel exhaust are carcinogenic. Furthermore, diesel exhaust is released at ground level and close to where people live and work, thus the exposure is expected to be significant.

There is considerable uncertainty associated with the assessment of the toxicity of diesel emissions. The approach used by the California EPA, based on the use of diesel particulate as an indicator for the whole exhaust appears promising as a concept, but the estimate of potency has not been universally accepted and it should be seen as controversial. An alternate approach, based on summation of risk attributable to selected individual contaminants found in the exhaust, led to significantly lower estimates of risk than the estimates based on the diesel particulate as an indicator.

The risks calculated by Method 1 (summation of individual risks) are below the level of concern (a risk of 1 in a million, 1E-6). Occasional minor exceedances of risk of 1E-6 are not significant. The non-cancer effects were also estimated to be below the level of concern using each of the two methods. Since diesel exhaust contains many more contaminants than those tested in this assessment, the risk calculated by summing the risk from individual contaminants (Method 1) is considered to be the lower bound on the actual risk.

The cancer risk calculated using Method 2 (diesel particulate as an indicator for diesel exhaust) resulted in higher risk levels. For both typical residents of Toronto (LIW) and the outdoor workers (LOW), the risk was between 1 in 10 000 to 1 in 100 000 (1E-5 to 1E-4). It should be stressed again, that the latter calculations based on Method 2 utilize a potency estimate that is controversial. It is therefore strongly recommended not to over-interpret these results. It may not be appropriate to use them to estimate the number of people in Toronto likely to be affected by diesel or to estimate the percentage of cancer risk from Toronto air that is attributable to diesel vehicles. Rather, it may be prudent to use the data as an indication that diesel vehicles may have a significant impact on Toronto air and to develop policies to minimize the impact.

It is also important to note that some studies, such as California's MATES II or Environmental Defense Fund's analysis, probably utilize the California potency factor for diesel exhaust. Other problems are likely and are described above. It is therefore recommended not to assume that the conclusions from these studies are necessarily applicable to Toronto.

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13. Definition of Terms (from USEPA)

Diesel Particulate Matter:

Diesel Particulate Matter (DPM) is a mixture of particles that is a component of diesel exhaust. EPA has recently proposed listing diesel exhaust as a mobile source air toxic due to the cancer and non-cancer health effects associated with exposure to whole diesel exhaust. The USEPA believes that exposure to whole diesel exhaust is best described, as many researchers have done over the years, by diesel particulate concentrations.

Major sources:

As defined in the US Clean Air Act, major sources are those stationary facilities that emit or have the potential to emit 10 tons of any one toxic air pollutant, or 25 tons of more than one toxic air pollutant, per year.

Non-road mobile sources:

Mobile sources not found on roads and highways (e.g., airplanes, trains, lawn mowers, construction vehicles, farm machinery).

On-road mobile sources:

Vehicles found on roads and highways (e.g., cars, trucks, buses).

Urban:

According to the USEPA in its analysis supporting the Integrated Urban Air Toxics Strategy, a county was considered "urban" if, based on 1996 data, it either includes a metropolitan statistical area with a population greater than 250,000 or the U.S. Census Bureau designates more than 50 percent of the population as "urban". This definition does not necessarily apply for any regulatory or implementation purpose.

Area and other sources:

Include sources that generally have smaller emissions on an individual basis than "major sources" and are often too small or ubiquitous in nature to be inventoried as individual sources. "Area sources" include facilities that have air toxics emissions below the major source threshold as defined in the air toxics sections of the US Clean Air Act and thus emit less than 10 tons of a single toxic air pollutant or less than 25 tons of multiple toxic air pollutants in any one year. Area sources include smaller facilities, such as dry cleaners. "Other sources" include sources such as wildfires and prescribed burns that may be more appropriately addressed by other programs rather than through regulations developed under certain air toxics provisions (section 112 or 129) in the Clean Air Act. For example, wildfires and prescribed burning are being addressed through the burning policy agreed to by the Interim Federal Wildland Policy.

Background concentrations:

In this context, the USEPA uses background concentrations to mean the contributions to outdoor air toxics concentrations resulting from natural sources, persistence in the environment of past years' emissions and long-range transport from distant sources. Background concentrations could be levels of pollutants that would be found in 1996 even if there had been no recent manmade emissions. To accurately estimate outdoor concentrations, it is necessary to account for the background concentrations by adding them to the modeled concentrations. In this assessment, except for diesel PM, background concentrations are based on values identified in the Cumulative Exposure Project (a study which estimated 1990 ambient concentrations of air toxics). From that study, EPA used background concentration values reported in the technical literature for 13 of the air toxic pollutants, and for the rest the EPA assumed a value of zero. For diesel PM, instead of using monitored air quality data to estimate background concentrations, a modeling-based approach was used.

Appendix – Properties of Diesel Emissions

A fact sheet prepared by ToxProbe

Diesel exhaust is a complex mixture of gases and fine particles that are emitted by internal combustion engines using diesel oil as fuel. The gaseous component of diesel exhaust is similar to the combustion products of other fuels. Although the adverse effects of diesel emissions are due both to the gaseous and particulate components, the toxicity of diesel exhaust is often expressed in relation to its particulate component. Several agencies have classified diesel exhaust as a carcinogen.

In North America, the diesel engine is used mainly in trucks, buses, agricultural and other off-road equipment, locomotives, and ships. The chief advantages of the diesel engine over the gasoline engine are its fuel economy and durability. Diesel engines, however, emit more particulate matter per mile driven compared with gasoline engines of a similar weight class. Over the past decade, modifications of diesel engine components have substantially reduced particle emissions.

Appendix A refers to diesel exhaust only, and not to diesel fuel oil, which also needs to be considered when estimating the risk from the use of diesel vehicles and other diesel engines.

Physico-Chemical Properties

Complete and incomplete combustion of fuel in diesel engines results in a complex mixture of gases and particles composed of hundreds of organic and inorganic compounds. The physical and chemical characteristics of diesel exhaust are dependent on many factors such as the composition of the fuel, the characteristics of the engine and the conditions under which the diesel is burned. This section provides an overview of the different components of diesel exhaust. Table A1 lists the major constituents of diesel exhaust.

There are several toxic gaseous components in diesel exhaust. The primary one is formaldehyde, which makes up 65%-80% of the aldehyde emissions. The other main aldehydes present are acetaldehyde and acrolein. The gaseous portion also includes benzene, 1,3-butadiene, carbon monoxide, polyaromatic hydrocarbons (PAHs), and nitro-PAHs. Dioxin compounds have also been detected in trace quantities. Dioxins from diesel exhaust account for 1.2% of total annual dioxin emissions in the US.

Diesel particulate matter (DPM) is the particle-phase of substances emitted in diesel exhaust. It refers to both the primary emissions and the secondary particles that are formed by atmospheric processes. Primary diesel particles are considered fresh after being emitted and undergo ageing (oxidation, nitration, or other chemical and physical changes) in the atmosphere.

Table A1. Percent Composition (by weight) of light-duty diesel engine exhaust (IPCS, 1996)

Pollutant	*Percent Composition*
Carbon dioxide	*7.1*
Water vapour	*2.6*
Oxygen	*15.0*
Nitrogen	*75.2*
Carbon monoxide	*0.03*
Hydrocarbons	*0.0007*
Nitrogen oxides	*0.03*
Hydrogen	*0.002*
Sulphur dioxide	*0.01*
Sulphates	*0.00016*
Aldehydes	*0.0014*
Ammonia	*0.00005*
Particles	*0.006*

Diesel exhaust particles are aggregates of primary spherical particles that consist of solid carbonaceous material and ash with associated adsorbed material. The particle portion of diesel exhaust contains elemental carbon (EC), organic carbon (OC), and small amounts of sulphate, nitrate, metals, trace elements, water, and other unidentified compounds. Elemental carbon usually makes up 50%-75% of the particles. Organic carbon makes up 19%-43% of the exhaust. It is composed of unburned fuel, engine oil, and small amounts of partial combustion and pyrolysis products. Polyaromatic hydrocarbons make up less than 1% of diesel exhaust particle mass.

Carbonaceous matter refers to all carbon-containing compounds in diesel particles, and includes the elemental and organic carbon. Organic carbon is made up of compounds containing carbon and hydrogen. The soluble organic fraction (SOF) is the portion of diesel particulate matter that can be extracted into solution. About one quarter of SOF is unburned fuel and three quarters is unburned engine lubrication oil. Partial combustion and pyrolysis products represent a very small fraction of the mass of SOF. Soot is the insoluble portion of diesel particle matter formed by clusters of elemental carbon and organic carbon particles.

The soluble organic fraction of diesel exhaust varies with many factors but has generally decreased since 1975. At present, exhaust particles from light-duty diesel engines have a higher proportion of soluble organic fraction than particles from heavy-duty engines. However, even with newer engines, some driving modes may produce a soluble organic fraction as high as 50% of the particulate matter.

A large number of elements and metals have been detected in diesel exhaust. They include barium, calcium, chlorine, chromium, copper, iron, lead, manganese, mercury, nickel, phosphorus, sodium, silicon, and zinc. These make up less than 1% of particle mass.

Most of the sulphur in the fuel is oxidized to sulphur dioxide (SO_2), but about 1-4% is oxidized and then converted to sulphate and sulphuric acid in the exhaust. The amount of SO_2 emitted is related to the sulphur content of the fuel. Non-road equipment uses fuel containing more sulphur than on-road diesel engines. The maximum allowable sulphur content in diesel is being reduced. Vehicles tested using low-sulphur fuel were found to have a sulphate content of only about 1%. Water content is about 1.3 times the amount of sulphate.

About 1-20% of total particle mass in diesel exhaust is in the ultra-fine size range ($PM_{2.5}$). The majority of these ultra-fine particles have an average size of 0.02 microns (range of 0.005-0.05 microns). They account for 50%-90% of the total number of particles. These very small particles are largely composed of sulphate and/or sulphate with condensed organic carbon. The composition of the ultra-fine particle component in the eastern United States differs from that in the west. In the east it is mostly composed of sulfates, and in the west, of nitrate, ammonium or organic carbon.

Approximately 80%-95% of the mass of particles in diesel exhaust is made up of fine particles (PM_{10}) with an average diameter of about 0.2 microns size range (range from 0.05-1.0 microns). The particles in this range are composed of spherical elemental carbon cores on which are adsorbed organic compounds, sulphate, nitrate and trace elements. Their large surface area makes them excellent carriers for the adsorbed compounds, which can effectively reach the lowest parts of the lung.

PAH and nitro-PAH make up about 1% of the particulate component of diesel exhaust. Differences in engine type and make, general engine condition, fuel composition and test conditions can influence the emissions levels of PAH. Increasing the aromatic content of the fuel may also increase PAH emissions.

The chemical composition of diesel particles to which people are currently exposed is a product of old and new technology and on-road and non-road engines. Although it is not possible to accurately characterize the mix, available data indicate that toxicologically significant organic components of diesel exhaust (e.g., PAHs, PAH derivatives, nitro-PAHs) that were present in the 1970s are still present.

Environmental Fate

The effects of diesel exhaust in the environment are similar to the effects of emissions from burning other fossil fuels. Diesel exhaust contributes to acid deposition (acid rain), the formation of ground-level ozone and global warming. Knowledge concerning the products of chemical transformation of diesel exhaust in the air is still limited. Secondary aerosols such as nitoarenes, nitrates and sulphates from diesel exhaust may also exhibit different biological reactivity than the primary particles. There is evidence that reaction of PAH in the exhaust with nitrogen oxides will form nitroarenes that are often more mutagenic than their precursors. A recent study has suggested that reaction with ground-level ozone increases the inflammatory effect of diesel particles in the lung of the rat.

After being emitted, diesel particles undergo ageing (oxidation, nitration or other chemical and physical changes) in the atmosphere. The atmospheric lifetime of the various compounds found in diesel exhaust varies and ranges from hours to days. Particles that are smaller than 1 micron can remain in the atmosphere for up to 15 days.

Primary diesel emissions are a complex mixture containing hundreds of organic and inorganic constituents in the gas and particle phases. The more reactive compounds with short atmospheric lifetimes will undergo rapid transformation in the presence of the appropriate reactants, whereas more stable pollutants can be transported over greater distances.

The particulate portion of diesel exhaust can be either primary (emitted directly) or secondary (formed from the transformation of the gaseous component). There is little or no hygroscopic growth of primary diesel particles, however products of oxidation are more hygroscopic. Since the products of oxidation are more soluble they are more readily removed from the air. Particles are removed from the atmosphere through accretion of the particles and dry or wet deposition. Particles of small diameter (<1 µm) are removed less efficiently and thus have longer atmospheric residence times. Because of their small size, diesel exhaust particles have residence times in air of several days, and they may be transported over long distances. Ultimately, they may be removed by wet deposition if they serve as condensation nuclei for water vapour deposition or are scavenged by precipitation in or below cloud.

Atmospheric lifetimes for several gas-phase components of diesel exhaust are on the order of hours or days, during which time atmospheric turbulence and advection can disperse these pollutants widely. Inorganic species such as sulphur dioxide (SO_2) and nitric acid have relatively fast deposition rates and remain in the air for less time than the organic components. Dry deposition of organic species is typically on the order of weeks to months.

Gaseous diesel exhaust will primarily react with sunlight, the hydroxyl (OH) radical, ozone, the hydroperoxyl (HO_2) radical, various nitrogen oxides and sulphuric acid. Reaction with the OH radical is the major removal route for PAHs in the gas phase and occurs within a few hours in daylight. In the presence of nitrogen oxides, this oxidation reaction can lead to the formation of nitroarenes or nitro-PAHs.

Oxides of nitrogen (primarily NO) that are emitted in diesel exhaust are also oxidized in the atmosphere to form nitrogen dioxide (NO_2) and particulate nitrate.

About 98% of sulphur emitted from diesel engines is in the form of SO_2. This is readily oxidized by the OH radical in the atmosphere and then rapidly transformed into sulphuric acid aerosols (H_2SO_4) through the reaction of the HO_2 radical and HSO_3 with water. Because SO_2 is soluble in water, it is scavenged by fog, cloud water and raindrops.

The particle matter of diesel exhaust is primarily composed of carbonaceous material (organic and elemental carbon) with a very small fraction composed of inorganic compounds and metals. The elemental carbon component of diesel exhaust is inert to atmospheric degradation.

High-molecular-weight PAHs in particulate matter are generally more resistant to atmospheric reactions than PAHs in the gas phase, leading to an anticipated half-life of 1 or more days. PAHs undergo photolysis, nitration, and oxidation. They react with sunlight, ozone, hydroxyl radicals, nitrogen oxides, nitrates and sulphates.

Ultra-fine particles emitted by diesel engines undergo nucleation, coagulation and condensation to form fine particles.

Toxicokinetics

Absorption
The primary route of human exposure to diesel exhaust is through inhalation. The properties and composition of an individual particle influence the biological fate of the various components of diesel exhaust. About 10% of diesel particles are deposited in the alveolar region of the lung. The half-time for clearance of particles in the alveolar region in humans is several months. Particles that are not cleared are absorbed by macrophages.

Distribution
The lung is the major target organ for diesel exhaust. Diesel particles absorbed by macrophages remain mostly in the lung. Elevated levels of DNA adducts in the lymphocytes of workers, and the presence of radio-labelled organic compounds in biological tissue and fluids of animals exposed by inhalation, suggest that some components of diesel exhaust are bioavailable.

Metabolism
The metabolism of diesel exhaust particles is similar to that of other insoluble foreign bodies. The particles are taken in by macrophages. This is followed by inflammation, cell death, impaired clearance and eventually deposition of collagen.

Excretion
Lung clearance mechanisms will remove diesel particles. At high concentrations, an overload of the removal mechanisms can occur. Macrophages that are laden with particles show decreased movement and lessened removal ability.

Human Health Effects

The main target organ of diesel exhaust is the lung. Available evidence indicates that current exposure levels are high enough to lead to adverse health effects. Diesel exhaust may cause cancer and may affect the immune system.

Death
Diesel exhaust is of low acute toxicity, however exposure can result in death from carbon monoxide, a component of diesel exhaust.

Respiratory effects

Acute exposure to diesel exhaust has been associated with irritation of the eye, nose, and throat, and with respiratory symptoms such as cough and phlegm. Diesel exhaust is known to contain various irritants in both the gaseous phase and particulate phase (for example, sulphur oxides (SO_x), nitrogen oxides (NO_x) and aldehydes). The evidence for potential chronic non-cancer health effects of diesel exhaust is based primarily on findings from chronic animal inhalation studies showing chronic inflammation and tissue changes in the lung in rats, mice, hamsters and monkeys. A few studies of workers have noted some respiratory symptoms, but overall, available studies have not shown significant chronic non-cancer health effects associated with diesel exhaust exposure in humans. Several epidemiological studies have demonstrated an association between air pollution and day-to-day changes in mortality, hospital emergency visits, and changes in lung function. The specific contribution of diesel exhausts to these effects is not known, however.

Immunological effects

Some studies in animals have shown decreased immune function after exposure to diesel exhaust, but others have not. Recent human and animal studies have shown that short exposures to diesel exhaust can produce allergic reactions and exacerbate symptoms to other allergens. Given the increases in allergic hypersensitivity in the U.S. population, the USEPA has indicated that this endpoint is of potential public health concern.

Neurological effects

Some reports of individuals in the workplace and in clinical studies exposed acutely to high concentrations of diesel exhaust have shown neurophysiological symptoms such as headache, light-headedness, nausea, vomiting, and numbness or tingling of the extremities. There has been some evidence from animal studies indicating possible neurological and behavioural effects. However, these have been observed at exposures higher than those have that caused respiratory effects.

Developmental and reproductive effects

There have been a few studies in animals showing sperm abnormalities, neurobehavioural effects in pups and other effects on reproduction.

Genotoxic effects and cancer

Diesel particulate matter and extracts of its organic components have induced gene mutations and chromosomal changes in a variety of bacterial and mammalian cell test systems. Both the particle core and the associated organic compounds have demonstrated carcinogenic properties. The particle component appears to contribute the most to carcinogenic effects, at least at high exposure levels. It is possible that the absorbed organic compounds, such as PAHs, play a more important role at lower exposure levels. The role of the gaseous components is still unclear.

The mechanism by which diesel exhaust causes tumours is not well understood. The carcinogenic effects may be related to the small size of diesel exhaust particles. It has been suggested that this could be the result of the genotoxicity of the compounds that condense on

the particles. Others suggest the exhaust causes DNA damage or that the particles cause an inflammation that then leads to increased cell multiplication.

Many studies in both humans and animals have shown the potential for diesel exhaust to cause or contribute to the development of cancer in the lung. The evidence linking diesel exposure to bladder cancer is weak. Increased lung cancer risk has been observed in railroad workers, truck drivers, heavy equipment operators, and professional drivers. Several well-conducted studies in the rat have demonstrated that chronic inhalation exposure produced dose-related increases in lung tumours (benign and malignant). However, in other species the evidence is less clear. The consistent findings of carcinogenic activity by the organic extracts of diesel particle matter in non-inhalation studies (intratracheal instillation, lung implantation and skin painting) further contribute to the overall animal evidence.

It is biologically plausible for the mutagenic and carcinogenic components of diesel exhaust to increase the risk of lung cancer. This supports a causal relation between the association observed between exposures and cancers. Overall, the human evidence that diesel exhaust is carcinogenic is judged to be strong but not sufficient to consider diesel exhaust a human carcinogen. There is a lack of consensus about whether the effects of smoking have been adequately accounted for in various studies. The USEPA has concluded that chronic inhalation exposure to diesel exhaust has the potential to induce lung cancer in humans and has classed diesel exhaust particles in Group B1 – probable human carcinogen. In the 9th Report on Carcinogens (2000), diesel exhaust particles were listed as reasonably anticipated to be a human carcinogen. The International Agency for Research on Cancer (IARC, 1989) classified diesel exhaust in group 2A – probably carcinogenic to humans. There is insufficient information for an evaluation of the potential cancer hazard posed by the oral or dermal route of exposure.

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