

Estimating the Health Impact of Exposure to Diesel Exhaust in Toronto

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Introduction

Diesel exhaust (DE) has been classified by various agencies as a probable human carcinogen (IPCS, 1996; US NTP, 2000). In 1999, the South Coast Air Quality Management District in California released the results of its Multiple Air Toxics Exposure Study (MATES-II). This study concluded that about 70 percent of the carcinogenic potential of air contaminants in the Los Angeles and surrounding area was due to the levels of diesel particulate matter or DPM (AQMD, 1999). A recent study estimated that each $10 \mu\text{g}/\text{m}^3$ of $\text{PM}_{2.5}$ was associated with an eight-percent increase in death from lung cancer (Pope, et al., 2002). Toronto Public Health therefore reviewed the data available to assess the degree to which DE exposure could be of concern in Toronto.

DE is a complex mixture of hundreds of different compounds. These are found as gases or particulate matter (PM). The gaseous components of DE include carbon dioxide, oxygen, nitrogen, water vapour, carbon monoxide, nitrogen compounds, sulphur compounds, and low-molecular-weight hydrocarbons. The particles in DE, or diesel particulate matter (DPM), consist of inhalable (PM_{10}) and respirable ($\text{PM}_{2.5}$) particles. These particles are inhaled deep into the lung and have a very large surface area, which make them an excellent carrier for adsorbed inorganic and organic compounds. DPM is made up of elemental carbon, adsorbed organic compounds, and small amounts of sulphate, nitrate, metals, and other trace elements (US EPA, 2000).

Toxic substances in the gaseous phase of DE include aldehydes (e.g., formaldehyde, acetaldehyde, acrolein), benzene, 1,3-butadiene, and polyaromatic hydrocarbons (PAHs) and nitro-PAHs. Organic compounds that are adsorbed onto the particles include PAHs, nitro-PAHs, and oxidized PAH derivatives. PAHs and their derivatives comprise about 1% or less of the DPM mass.

This report summarises the available data on the levels of DE in the air and the health effects of DE to assess if current exposures in Toronto are of potential health concern.

Estimating Exposures to Diesel Exhaust in Toronto

In the ambient environment, DE is emitted from “on-road” diesel engines (e.g. buses, cars, and trucks) and “off-road” engines (e.g. heavy-construction equipment, locomotives, marine vessels, etc.). DPM mass (expressed as $\mu\text{g}/\text{m}^3$ of DPM) has historically been measured as a surrogate for whole DE. There are no data available on levels of DE or DPM in Toronto.

DE emissions vary significantly in chemical composition and particle sizes with different engine types (heavy-duty, light-duty), engine operating conditions (idling, acceleration, deceleration) and fuel formulations. Available data indicate that toxicologically relevant organic components of DE present in DPM and DE emitted from older vehicle engines are still present in emissions from newer engines. Once emitted from the exhaust of the engine, DE undergoes dilution and chemical and physical transformations in the atmosphere, as well as dispersion and transport in the atmosphere. It is not clear what is the overall health consequence of DE ageing because some compounds in the DE mixture are altered during ageing to more toxic forms while others are made less toxic (US EPA, 2000). The type of pollutants emitted by gasoline and diesel

engines is similar. One of the major differences in the exhaust is the number of particles emitted. Diesel engines emit about 20 times the number of particles as do gasoline engines (IPCS, 1996).

A large percentage of the population is exposed to ambient PM_{2.5}, of which DE is a part. Data from the U.S. suggest that DE contributes to between 10% and 36% of the total ambient PM_{2.5} in urban centres. In the early to mid-1990s, annual average DE exposure from on-road engines in the U.S. was about 0.5 to 1.0 µg /m³ of inhaled air in many rural and urban areas, respectively. Where people spend a large portion of their time outdoors, in urban areas, the exposures may range up to 4.0 µg /m³ of inhaled air. These estimates take into account the proportion of time spent outdoors as opposed to indoors. Some people such as workers in the transportation sector may be more at risk to high exposures to DE. As well, studies in the U.S. have highlighted the potential of high exposure to children in school buses (US EPA, 2000). Over the years, there has been significant reduction in DPM emissions from the exhaust of on-road diesel engines in the U.S.

Exposure from On-road Sources

Although there are no data on levels of DE in Toronto's air, data are available on the levels of specific contaminants. The contribution of on-road DE to these levels (Table 1) was estimated using the fraction of the total level of a contaminant that is attributed to on-road diesel based on a U.S. Environmental Protection Agency (EPA) model, except for benzo[a]pyrene (B[a]P) where the annual vehicle miles travelled and B[a]P emission factors were used (ERG, 2000; US EPA, 1999). There are several limitations with these estimates. The U.S. EPA model was developed to estimate levels in large areas such as a state or province. As well, it assumes that the relative contribution of DE to the pollutant mix in Toronto is the same as in the U.S. However, overall these values are within the normal margin of error for this type of estimate. More details on the derivation of these estimates are available in the report "Estimated Human Health Risk from Exposure to Diesel Exhaust in Toronto" prepared by ToxProbe Inc. for Toronto Public Health (TPH, 2002a). Toronto Public Health estimates that workers in the transportation sector are among those with a high potential for exposures to PAHs, a component of DE (TPH, 2002b).

Table 1: Estimated Levels of Selected Pollutants Due to On-road Diesel Engines in Toronto

Substance	Average (µg/m ³)	90 th percentile (µg/m ³)
Acetaldehyde	0.292	0.556
Acrolein	0.00364	0.00711
Benzene	0.0166	0.0266
Benzo[a]pyrene (B[a]P)	0.000000755	0.0000012
1,3-Butadiene	0.0154	0.0289
Formaldehyde	0.259	0.438
Diesel particulate matter (DPM)	0.602	1.02

Exposure to Off-road Sources

Much less is known about changes in quantity and composition of emissions from off-road engines over time. Data from the U.S. suggest that while on-road diesel exhaust has decreased over the years, off-road DE may have increased. Off-road sources contribute about half of the total ambient particulate matter concentrations (US EPA, 2000). There are insufficient data to estimate the contribution of off-road sources to total DE exhaust in Toronto, therefore only on-road emissions data were used in the estimation of health risk due to DE.

Non-cancer Effects of Diesel Exhaust

Short-term exposure to high levels of DE has been associated with eye, nose, and throat irritation as well as with nausea, cough and phlegm. There is some evidence that DE may affect the immune system and exacerbate allergies. Studies in experimental animals have shown lung damage after long-term exposure to DE, but most studies in humans have not found significant non-cancer effects related to long-term DE exposure. Epidemiological studies have shown an association between air pollution and day-to-day changes in mortality, hospital emergency visits for lung and heart disease and changes in lung function. The contribution of DE to these effects is however not known (IPCS, 1996; US EPA, 2000).

Various agencies have developed maximum recommended exposure levels for the non-cancer effects of DE (Table 2). The World Health Organisation (WHO) Regional Office for Europe derived an air guideline for the non-cancer effects of DE using two methods (WHO, 2000). The values obtained were 2.3 and 5.6 $\mu\text{g}/\text{m}^3$ (1-year average). This is similar to the California Reference Exposure Level (REL) of 5 $\mu\text{g}/\text{m}^3$ (CalEPA, 1998). In its 2000 assessment of DE, the U.S. EPA revised its inhalation reference concentration (RfC) of 14 $\mu\text{g}/\text{m}^3$ (24-hr average). It notes that this is almost the same as the long-term $\text{PM}_{2.5}$ National Ambient Air Quality Criteria of 15 $\mu\text{g}/\text{m}^3$ (US EAP, 2000). Estimated ambient levels of DE in Toronto are less than these values.

Table 2: Recommended Ambient Air Quality Levels for Diesel Exhaust

Non-cancer Effects	World Health Organisation	2.3 - 5.6 $\mu\text{g}/\text{m}^3$ (1-year average)
	California	5 $\mu\text{g}/\text{m}^3$ (24-hour average)
	U.S. Environmental Protection Agency	14 $\mu\text{g}/\text{m}^3$ (24-hour average)
Cancer (1 x 10 ⁻⁶ lifetime risk)	World Health Organisation	0.014 – 0.0625 $\mu\text{g}/\text{m}^3$
	California	0.0033 $\mu\text{g}/\text{m}^3$
Note: Diesel exhaust is normally measured as diesel particulate matter or DPM		

Cancer Effects of Diesel Exhaust

There is general consensus among various agencies across the world that DE is a probable human carcinogen by inhalation. There is insufficient data to evaluate the potential of DE to cause cancer through oral or dermal routes of exposure. Studies have found an increase in risk of developing cancer among railway workers, truck drivers, heavy equipment operators and professional drivers. Several studies in rats have also resulted in an increase in lung tumours, but the evidence in other animals is weak. Lung cancer is the cancer most often associated with DE exposure. Evidence that DE could also cause bladder cancer is weak (US EPA, 2000).

The way in which DE may cause cancer and the component of DE that may be most responsible for cancer effects is not known. Many of the organic compounds present in DE are known to have mutagenic and carcinogenic properties. At high exposure levels DPM appears to be most responsible for cancer effects. Whether this is also the case at lower levels generally found in the environment is unknown (US EPA, 2000). Several attempts have been made to estimate the cancer potency of DE. However, these have severe limitations and no consensus exists on an acceptable approach.

WHO has used data in rats to estimate unit risk values for cancer (WHO, 2000). Using four different studies WHO calculated a range of 1.6×10^{-5} to 7.1×10^{-5} per $\mu\text{g}/\text{m}^3$. This would suggest that a lifetime exposure to DE between 0.014 and $0.0625 \mu\text{g}/\text{m}^3$ could result in a one in one million excess risk of cancer.

The California Environmental Protection Agency has proposed a unit lifetime cancer risk of 3.0×10^{-4} per $\mu\text{g}/\text{m}^3$ diesel particulate matter (CalEPA, 1998). This would be equivalent to one in one million excess cancer risk for lifetime exposures to $0.0033 \mu\text{g}/\text{m}^3$ DPM. This was derived from data on exposed workers and based on evidence that suggested unit risks between 1.5×10^{-4} and 15×10^{-4} per $\mu\text{g}/\text{m}^3$. This estimate has been widely criticised as overestimating the risk.

In 2000, the U.S. EPA released a revised draft health assessment for DE (US EPA, 2000). It concluded that because the mechanism by which DE causes cancer in experimental animals is not relevant to humans, animal studies are not suitable to quantify the risk for DE. As well, uncertainties in the human data due to factors such as possible confounding with smoking and change in DE composition over the years are too high to accurately estimate the risk. However, it concluded that data available suggest that current exposure levels in the U.S. may pose a lifetime cancer risk above the one in one million benchmark. Some segments of the population, such as people with a high exposure to off-road sources, and children who may be more sensitive to early-life exposures, could be at higher risk. Overall DE exposure in the U.S. may pose a lifetime cancer risk of between 1 in 100,000 and 1 in 1,000. This should not be seen as an accurate measure of risk but rather as an indicator of the potential significance of the lung cancer hazard posed by exposure to DE.

The Multiple Air Toxics Exposure Study

The Multiple Air Toxics Exposure Study (MATES-II) identified DE as the most significant contributor to the cancer potential of air pollution in Los Angeles and the surrounding area. This

was derived using data from extensive monitoring of toxic air contaminants and the California cancer potency estimates. Since no similar survey has been carried out in Toronto and the cancer potency used overestimates the risk of DE, it is not possible to use the results of MATES II to draw conclusions on the relative contribution of DE to cancer from air pollution in Toronto.

Estimating Health Risk from On-road Diesel Exhaust in Toronto

Risk is composed of two elements – the level of exposure to a substance and the degree of hazard the substance poses. The main source of exposure to DE is through inhalation, and thus only this route of exposure was considered when estimating the risk of cancer due to DE in Toronto.

The estimated outdoor levels of the various components of DE as shown in Table 1. To take into consideration the fact that some people could be exposed to more than the average DE concentration the risk was also estimated using the reasonable maximum air concentration. The time spent outdoors also affects exposure. People who work outdoors will have higher exposures to DE than those who work indoors since levels of DE are higher outdoors than indoors and because outdoor workers are likely to be involved in greater physical activity. The risk was calculated for both a typical indoor and outdoor worker assuming that levels of DE indoors were half those outdoors. Finally the various length of residence in Toronto (average - 9 years, reasonable maximum - 30 years, and lifetime) was also factored in.

Diesel exhaust is a complex mixture. There are two basic approaches to estimating the risk caused by complex mixtures: adding the risk of individual components of the mixture (Method 1) and assessing the mixture as a whole based on the level of an indicator substance (Method 2). Each approach has its advantages and disadvantages. More details on the derivation of these risk estimates are available in the report “Estimated Human Health Risk from Exposure to Diesel Exhaust in Toronto” prepared by ToxProbe Inc. for Toronto Public Health (TPH, 2002a).

Method 1

To estimate the cancer risk of DE the risks due to the following components were added: acetaldehyde, acrolein, benzene, 1,3-butadiene, formaldehyde, and PAHs (Table 3). This resulted in an estimated risk for an indoor worker of between 6.02×10^{-8} and 9.3×10^{-7} and for an outdoor worker between 7.22×10^{-8} and 1.12×10^{-6} (Table 4).

One way to estimate if exposure levels are of concern is to calculate an exposure ratio. This compares concentration in the environment against the health benchmark. Exposure ratios greater than one suggest that exposures are above the benchmark. The exposure ratio for non-cancer effects ranged between 0.17 and 0.38. For cancer effects, comparing these to a one in a million benchmark, the exposure ratio was 0.06 – 1.12. Using this method, only outdoor workers exposed to DE at reasonable maximum air concentrations were exposed to levels that were estimated to cause more than one in one million lifetime excess cancer risk (Table 5).

Table 3: Potency of Diesel Exhaust Components

Substance	Unit risk per $\mu\text{g}/\text{m}^3$ *	RfC ($\mu\text{g}/\text{m}^3$)
1,3 -Butadiene	6.3×10^{-6}	0.33
Acetaldehyde	2.2×10^{-6}	9
Acrolein	-	0.02
B[a]P (as surrogate for PAH)	2.3×10^{-2} **	-
Benzene	4.1×10^{-6}	-
Formaldehyde	8.0×10^{-7}	-
Diesel PM	3×10^{-4} †	14

* US EPA values unless otherwise noted
 ** Ontario Ministry of the Environment (OMOE, 1997)
 † California Environmental Protection Agency (CalEPA, 1998)

Table 4: Lifetime Exposure Estimates Using Method 1

	9-year exposure at average air levels ($\mu\text{g}/\text{m}^3$)	30-year exposure at average air levels ($\mu\text{g}/\text{m}^3$)	Lifetime exposure at maximum air levels ($\mu\text{g}/\text{m}^3$)
Indoor workers			
1,3 -Butadiene	1.03×10^{-3}	3.44×10^{-3}	1.61×10^{-2}
Acetaldehyde	1.96×10^{-2}	6.52×10^{-2}	3.11×10^{-1}
Acrolein	2.44×10^{-4}	8.13×10^{-4}	3.97×10^{-3}
B[a]P (as surrogate for PAH)	5.06×10^{-8}	1.69×10^{-7}	6.73×10^{-7}
Benzene	1.11×10^{-3}	3.71×10^{-3}	1.49×10^{-2}
Formaldehyde	1.74×10^{-2}	5.79×10^{-2}	2.45×10^{-1}
Outdoor workers			
1,3 -Butadiene	1.24×10^{-3}	4.13×10^{-3}	1.94×10^{-2}
Acetaldehyde	2.35×10^{-2}	7.84×10^{-2}	3.73×10^{-1}
Acrolein	2.93×10^{-4}	9.77×10^{-4}	4.77×10^{-3}
B[a]P (as surrogate for PAH)	6.08×10^{-8}	2.03×10^{-7}	8.08×10^{-7}
Benzene	1.34×10^{-3}	4.46×10^{-3}	1.78×10^{-2}
Formaldehyde	2.09×10^{-2}	6.95×10^{-2}	2.94×10^{-1}

Table 5: Exposure Ratios Using Method 1

	9-year exposure at average air levels	30-year exposure at average air levels	Lifetime exposure at maximum air levels
Indoor workers			
Cancer	0.06	0.2	0.93
Non-cancer	0.166	0.166	0.29
Outdoor workers			
Cancer	0.07	0.24	1.12
Non-cancer	0.2	0.2	0.4

Table 6: Lifetime Exposure Estimates Using Method 2

	9-year exposure at average air levels ($\mu\text{g}/\text{m}^3$ DPM)	30-year exposure at average air levels ($\mu\text{g}/\text{m}^3$ DPM)	Lifetime exposure at maximum air levels ($\mu\text{g}/\text{m}^3$ DPM)
Indoor workers	2×10^{-2}	6.75×10^{-2}	2.85×10^{-1}
Outdoor workers	2.4×10^{-2}	8.1×10^{-2}	3.42×10^{-1}

Table 7: Exposure Ratios Using Method 2

	9-year exposure at average air levels	30-year exposure at average air levels	Lifetime exposure at maximum air levels
Indoor workers			
Cancer	12	41	171
Non-cancer	0.03	0.03	0.05
Outdoor workers			
Cancer	15	49	205
Non-cancer	0.03	0.03	0.055

The chief disadvantage of Method 1 is that while the mixture is made up of hundreds of components, data are available only on a few of these. This method also assumes that the risk of exposure is additive and does not accurately reflect possible interactions. This method therefore will likely underestimate the risk. Since this calculation does not consider exposure to off-road diesel sources and it includes the risk of only a small number of the many contaminants in DE, this estimate can be considered a lower bound to the potential risk of cancer from DE in Toronto.

Method 2

In the second approach, exposure to DPM was used as a surrogate for all diesel components in conjunction with the California cancer potency estimate of 3×10^{-4} per $\mu\text{g}/\text{m}^3$. This gave an estimated cancer risk of between 1.2×10^{-5} and 1.71×10^{-4} for an indoor worker and 1.46×10^{-5} and 2.05×10^{-4} for an outdoor worker (Table 6). With this method, exposure ratios for non-cancer effects were smaller ranging between 0.03 and 0.055. However for cancer effects exposure ratios were much higher and ranged from 12 for the least exposed to 205 for the most exposed (Table 7). This is equivalent to about one in 100,000 and 2 in 10,000 excess cancer risk

There are about 1000 deaths from lung cancer in Toronto every year (TPH, 2000). Exposure to cigarette smoke is the major cause of cancer and although particulate pollution has been associated with an increase in cancer, DE is only one of many sources of particles in Toronto's air. Given these factors Method 2 clearly overestimates the potential risk of DE. While it is conceptually preferable to estimate the risk based on the mixture as a whole, it assumes that the indicator chemical correctly reflects the composition of the mixture, which does not appear to be the case.

Given this, it is likely that the risk of cancer from DE in Toronto is between the one in one million and one in 100,000 lifetime cancer risk. This is lower than the U.S. EPA estimate, which may be a reflection that, when it comes to air pollution, Toronto is in "the middle of the pack". However, there could be some situations where the risk is higher, but there is insufficient data at this time to assess these in Toronto. The California EPA has compared the potential risk from different activities using diesel-fuelled engines. Of the activities considered, idling school buses and the maintenance of emergency vehicles had the lower risk associated with them. Of intermediate ranking were truck stops and low-volume expressways. Distribution centres and stationary (off-road) engines were estimated to pose a higher risk. Areas close to high volume expressways (20,000 heavy-duty trucks per day) were identified as posing the most risk (CalEPA, 2000).

Initiatives to Reduce Emissions from Diesel Engines

Diesel Emission Control in the U.S.

Until the 1990s regulation of mobile sources focussed on light-duty motor vehicles. The passage of the U.S. Clean Air Act amendments of 1990 spurred regulations of emissions from other types of vehicles. Since then, a series of regulations was adopted that addresses different types of sources, both on-road and off-road, including locomotives and marine engines (US EPA, 2001c). These regulations are expected to reduce emissions from these diesel engines, including

carcinogens, by 90-95 percent (US EPA, 2001a, 2001b). As well, programmes are in place to encourage the retrofitting of existing engines (US EPA, 2001c).

There are several technologies, such as catalytic converters and diesel hybrid engines, that reduce emissions from diesel engines (CalEPA, 2000). The use of these technologies in conjunction with ultra low-sulphur diesel (15-ppm sulphur) can result in emissions from diesel engines similar to those from new gasoline or compressed natural gas engines. To enable on-road vehicles to meet the U.S. 2007 emission standards, sulphur content for on-road diesel in the U.S will be reduced to 15-ppm between 2006 and 2009 (US EPA, 2001a).

At present the sulphur level in off-road diesel is not regulated in the U.S. However, many users of off-road equipment use on-road fuel because it is more readily available and results in less engine wear and tear (US EPA, 2001d). The lower levels of sulphur in the fuel reduces emissions of sulphur dioxide, sulphate and PM emissions from diesel engines (US. EPA, 2001a). The U.S. EPA is still evaluating the need to control off-road DE sources beyond the regulations adopted in 1998. If this is found to be necessary, the U.S. EPA is committed to introducing new rules by 2004 (US EPA, 2001d). Meanwhile, California has already adopted rules that set the maximum allowable sulphur content in both on-road and off-road diesel sold in California to 15-ppm by 2006 (Cal EPA, 2000).

Diesel Emission Control in Canada

As part of its commitment to implement the Ozone Annex to the 1991 Canada-United States Air Quality Agreement, Environment Canada has announced several initiatives on diesel fuel and diesel engines. This includes the development and implementation of regulations for vehicles, off-road equipment and fuel, the expansion of inspection and maintenance programmes for in-use heavy-duty vehicles. New regulations for heavy-duty vehicles and engines have been proposed with some provisions taking effect in 2004 and others in 2006 (Environment Canada, 2002). In addition, Environment Canada has indicated that control of DE from diesel engines used in construction and in agriculture will be proposed for 2004 (Environment Canada, 2001). These will ensure that Canadian standards are as stringent as are those of the U.S. EPA. Environment Canada has also implemented voluntary agreements with manufacturers of off-road diesel engines to supply cleaner engines to the Canadian market before new regulations are in place (Environment Canada, 2001).

Transport Canada is the authority that regulates the locomotives and marine engines (greater than 50 horsepower) in Canada. An agreement between the Canadian government and the Railway Association of Canada provides for new locomotives to meet the U.S. emission standards. Canada is signatory to the International Convention for the Prevention of Pollution from Ships (MARPOL). Annex VI of this convention includes provisions to control emissions from ships. Canada plans to ratify the protocol and incorporate Annex VI into the Canada Shipping Act early in 2003.

City of Toronto Initiatives

The Corporate Green Fleets Strategy and route optimization initiatives by Solid Waste Management Services of Works and Emergency Services that have been adopted to help address pollutants linked to smog and climate change will also have an impact on DE emissions from the City's fleet. As well, in 1999, the City implemented a low-sulphur fuel purchasing practice. Although this practice was the emissions of pollutants linked to smog, it is also expected to contribute to a reduction in DE from the City's fleet.

Recommendations

At its meeting of June 2001, the Board of Health adopted a report "Air Quality and Federal Standards for Sulphur in On-Road Diesel". This report supported the proposal by the Federal Minister of the Environment to establish a 15-ppm sulphur standard for on-road diesel by June 2006. It also urged the Minister to introduce incentives to promote the early introduction of low-sulphur diesel and to harmonize sulphur standards for off-road diesel with those for on-road fuels. Given the important role of fuel quality in reducing emissions from diesel engines, the Board of Health should re-iterate its position and request that Environment Canada and Transport Canada negotiate with the industry the early introduction of ultra-low sulphur fuels low-emission technology on the Canadian market.

The reduction of exposure to DE has focussed on regulating emissions from diesel engines. Strategies that aim at reducing vehicle use, increased fuel efficiency and alternative fuels can equally play a role in reducing DE. The City has already taken initiatives that have helped reduce vehicle emissions, including diesel emissions, and as part of the implementation of the Environmental Plan the City is developing an inter-departmental Air Quality Strategy. This strategy should consider measures to further reduce emissions of, and exposure to, DE.

Conclusions

Diesel exhaust contributes to the overall level of air pollution in Toronto. Air pollution is associated with an increase in premature mortality and hospitalisation for heart and lung disease. Recent studies have linked air pollution with an increase in lung cancer and DE has been classified as a probable carcinogen. Therefore, although the data is insufficient to accurately estimate the risk, it is likely that DE contributes to the burden of cancer in Toronto.

There have been many advances in diesel engine technology that can result in significant reductions in emissions. The U.S. has already introduced legislation that will reduce emissions from both on-road and off-road diesel engines. Many of these need low sulphur diesel to ensure their optimal performance. The Federal government has proposed regulation to harmonise vehicle emission standards and sulphur in on-road diesel standards to those of the U.S. The Board of Health should re-iterate its support for the adoption of a 15-ppm limit for sulphur in diesel and encourage the Federal government should negotiate with the industry to make ultra low-sulphur diesel and low-emission technology available as soon as possible.

In the development of its Air Quality Strategy, the City can consider opportunities to reduce emission of, and exposure to, DE.

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