RESEARCH REPORT

Workplace Exposure Standard for Diesel Particulate Matter

Prepared for:

Safe Work Australia
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EXECUTIVE SUMMARY

Background and Objectives

Workplace exposure standards (WES) set out the airborne concentration of a substance or mixture in air that must not be exceeded. While a WES represents a concentration of an airborne contaminant that is not expected to cause ill health effects in exposed workers, they are not a dividing line between a healthy or unhealthy working environment. WES can be presented in a number of parameters, with each of the parameter values generally based on the ill health effect that occurs at the lowest airborne concentration of a chemical, i.e. the 'critical effect'.

As part of the review of the WES for airborne contaminants, WSP Australia Pty Ltd (WSP) and an independent peer reviewer concluded with respect to diesel engine exhaust (DEE) that the data available from the agreed sources in the pre-defined methodology for the review were inadequate to determine a WES value and that a review of additional sources be conducted (SWA 2019a).

DEE is made up of a complex mixture of hundreds of chemicals consisting of gaseous, adsorbed organics and particulate components found in the exhaust emissions from diesel engines; diesel particulate matter (DPM) refers to the particulate fraction of DEE. The composition of this mixture varies depending on engine type, operating conditions, fuel, lubricating oil, and whether an emission control system is present.

Due to the increasingly stringent pollution emission standards over the last two decades, engine technology has evolved from older 'traditional diesel engines' (TDE) to 'new technology diesel engines' (NTDE). With the application of sophisticated emission control devices in NTDE, emissions of particulates and other constituents are >90% lower than in TDE exhaust. TDE engines are generally considered to be any engines manufactured before 2007 and not equipped with after-treatment devices.

Safe Work Australia (SWA) engaged SLR Consulting Australia Pty Ltd (SLR) to:

- identify relevant independent, peer reviewed research and literature on the health effects of exposure to DEE and the impact engine types and year of manufacture have on the composition of DEE;
- if indicated in the research, recommend an encompassing health-based WES with supporting advisory notations for all DEE compositions present in Australia, based on the adverse effect that occurs at the lowest airborne concentration; and
- if not feasible, to make recommendations for individual WES values and notations that would be protective of exposure to the different compositions of DEE present in Australia.

A wide range of scientific literature, including human and animal data, was investigated. The research was focused on finding information that could inform the exposure-response relationship and whether NTDE exhaust toxicity differs markedly from TDE exhaust.

Results

The literature review revealed that the critical health effects associated with exposure to DEE include lung irritation, which upon long-term exposure, can progress to an inflammatory response and lung cancer. DEE has also been shown to have an effect on cardiovascular parameters in human controlled exposure studies as well as being associated with cardiopulmonary disease in large-scale human investigations. DEE can also increase the response to other allergens but does not appear to be an allergen itself.

EXECUTIVE SUMMARY

As DEE is a complex mixture, most human and animal studies do not measure the majority of its constituents. However, there are several indicators of DEE in the particulate and/or gaseous fraction of DEE that appear to be associated with the health effects of concern. These include DPM (expressed as elemental carbon, or EC), nitrogen dioxide (NO_2), aldehydes (e.g. formaldehyde, acetaldehyde), and polycyclic aromatic hydrocarbons (PAHs). Although the literature indicates mass-based concentrations may not be the best indicator to describe the toxicity of the particulate fraction of DEE, unfortunately there is insufficient information to justify derivation of a non-mass-based exposure limit for DPM at this time.

Experimental animal studies indicate that the chronic pulmonary toxicity and carcinogenicity of the particulate fraction of DEE is likely similar to other poorly soluble particulate substances where lung cancer in rats is due to a non-specific particle overload effect. However, occupational human exposure studies have shown associations for an increased risk of lung cancer at exposures that do not appear to be sufficient to cause lung overload. This suggests that a number of different mechanisms may be operable in the toxicity of DEE. Mutagenicity data indicate both the particles themselves and adsorbed chemicals like PAHs may contribute to a mutagenic mechanism for lung cancer. In addition, gaseous components of DEE (particularly NO_2 and aldehydes) may be direct irritants and/or carcinogens themselves, this means the effects are independent of particulate exposures, which is also evident with NTDE exhaust. It is uncertain whether a threshold or non-threshold concentration-response is operable for DEE and lung cancer.

There is insufficient information to recommend a WES for DEE as a whole because DEE composition varies considerably under different conditions (e.g. type of engine, emission control device, load, operation, etc.) which can result in varied toxicological responses. Instead, a WES has been derived using DPM as an indicator compound in DEE, keeping in mind that exposures to other constituents of potential concern in DEE (i.e. NO_2, PAHs and aldehydes) should be managed as well. For DPM, there is sufficient information from experimental animal studies and controlled human exposure studies to enable recommendation of a WES as respirable EC (REC). The epidemiological information, although considered insufficient on its own to be used for quantitative WES derivation, is used as supporting information to put the recommended WES for REC into context.

A number of approaches for establishing a health-based 8-hour time-weighted average (TWA) for DPM have been explored. They included derivation using data from controlled human exposure studies and derivation using data from experimental studies in rats. The resulting candidate WES values are for REC in the range of 7.5-25 µg REC/m^3. Consideration of potential PAH content adsorbed to DPM indicates the candidate WES values would likely be protective of lung cancer from PAH exposure. In addition, the candidate WES values are well below the REC concentrations in epidemiological studies that have been associated with oxidative DNA damage or pulmonary effects (114 and 56 µg REC/m^3, respectively) and at the low end of the range of approximate epidemiological exposure estimates that have been associated with an increased risk of lung cancer (8-67 µg REC/m^3).

Recommendations

The literature supports the view that exposure to DPM should be low. This is important to minimise the development of ill effects.

The following recommendations are made to protect workers from the risks of developing DEE-related lung disease.

EXECUTIVE SUMMARY

- An 8-hour TWA of 15 µg REC/m^3 for DPM as respirable elemental carbon is recommended to be applied. This is applicable to DPM from both NTDE and TDE. This is the approximate midpoint of the derivations discussed in the results section. The candidate DPM WES value derived herein is an estimate of the concentration of DPM to which workers may be exposed for a lifetime without the likelihood of appreciable harm from non-cancer or cancer effects.
- A 'Carcinogenicity Category 1A' notation is recommended for DPM based on the weight of evidence from both human and animal studies indicating DEE is a lung carcinogen.
- In addition, it is recommended the candidate WES for DPM be applied in conjunction with appropriate management measures to control and/or minimise exposures to other indicators of potential concern within DEE including NO_2, PAHs, and aldehydes to ensure the risk of health effects from the mixture as a whole is adequately controlled.

CONTENTS

CONTENTS

DOCUMENT REFERENCES

TABLES

FIGURES

CONTENTS

APPENDICES

List of Commonly Used Abbreviations and Definitions

Abbreviation/Term	Definition
ALARP	As low as reasonably practicable.
BAL(F)	Bronchoalveolar Lavage (Fluid). This is a diagnostic method of the lower respiratory system, in which a bronchoscope is passed through the mouth or nose into an airway in the lungs, with a measured amount of fluid introduced and then collected for examination.
BaP	Benzo(a)pyrene.
$BMCL_{10}$	Lower Benchmark Concentration for a 10% Response. The BMC is the concentration of a substance inhaled that is associated with a specified measure or change of a biological effect, in this case 10%. The BMCL is the lower one-sided confidence limit on the BMC.
DEE	Diesel Engine Exhaust.
DEP	Diesel Exhaust Particulates. Synonymous with DPM (Diesel Particulate Matter).
Diesel soot	Diesel soot appears to be a term used in some of the literature consulted to refer to DPM.
DPF	Diesel Particulate Filters. A DPF is a device designed to remove diesel particulate matter (DPM) or soot from the exhaust gas of a diesel engine.
DPM	Diesel Particulate Matter. Particulate matter, as defined by most emission standards, is filterable material sampled from diluted and cooled exhaust gases. This definition includes both solids, as well as liquid material that condenses during the dilution process. The basic fractions of DPM are carbonaceous solids and heavy hydrocarbons derived from the fuel and lubricating oil. In cases where the fuel contains significant sulfur, hydrated sulfuric acid can also be a major component. DPM contains a large portion of the polycyclic aromatic hydrocarbons (PAHs) found in engine exhaust. DPM includes small solid primary soot particles of diameters below 40 nm and their agglomerates of diameters up to 1 µm as well a nucleation mode of particles consisting almost entirely of condensed liquid.
DOC	Diesel Oxidation Catalysts. DOCs are catalytic converters designed specifically for diesel engines and equipment to reduce carbon monoxide (CO), hydrocarbons (HCs) and particulate matter (PM) emissions. DOCs are simple, inexpensive, maintenance-free and suitable for all types and applications of diesel engines.

Abbreviation/Term	Definition
EC	Elemental Carbon. Carbon that is present in atmospheric particulate matter can be classified into three basic forms: carbonate carbon, organic compounds, and EC. Carbonate carbon comprises the salts of the carbonate ion (CO_2^{2-}) and the bicarbonate ion (HCO_3^-), whereas organic carbon comprises hundreds to thousands of organic compounds. EC is pure carbon, of which there are two types (graphite and diamond). EC is frequently used as a surrogate for measuring concentrations of diesel particulate matter. While both organic carbon and EC are present in diesel engine exhaust, there are many other sources of organic carbon that atmospheric concentrations of it cannot be stated, with any reliability, as being solely from diesel engine exhaust. However, respirable EC generally has only one source in the workplace: exhaust. The terms EC and black carbon (BC) have been widely interchanged in the literature in the past. Both BC and EC consist of carbonaceous airborne particle species formed from incomplete combustion of carbonaceous fuel, have light-absorbing characteristics, and are linked with possible human health impacts. However, they are not measures of the same carbonaceous particle properties and should not be viewed as interchangeable. EC is operationally defined based on thermal properties rather than light absorbing properties; however, because EC measurements capture a major fraction of BC, EC has been used as a surrogate measure of BC. The two measurements are generally well correlated when measuring emissions from the same source, despite some differences in total mass measured (Briggs and Long 2016).
EGR	Exhaust Gas Recirculation. Exhaust gas recirculation (EGR) is an effective strategy to control NO_x emissions from diesel engines. The EGR reduces NO_x through lowering the oxygen concentration in the combustion chamber, as well as through heat absorption. Several configurations have been proposed, including high- and low-pressure loop EGR, as well as hybrid systems. EGR is also used in gasoline engines, primarily in order to reduce pumping work and increase engine efficiency.
fDEP	Filtered Diesel Exhaust Particulates. Synonymous with fDPM.
fDPM	Filtered Diesel Particulate Matter.
HEC	Human Equivalent Concentration. The human concentration (for inhalation exposure) of an agent that is believed to induce the same magnitude of toxic effect as the experimental animal species concentration.
HR	Hazard Ratio. Hazard ratios are measures of association widely used in prospective studies. It is the result of comparing the hazard function among exposed to the hazard function among non-exposed. The HR has also been defined as, the ratio of (risk of outcome in one group)/(risk of outcome in another group), occurring at a given interval of time
Ig	Immunoglobulin. Immunoglobulins are also known as antibodies, a Y-shaped protein used by the immune system to identify and neutralise foreign materials including some bacteria and viruses. There are different types of Igs including IgG, IgM, IgA, IgD, and IgE.
LOEC	Lowest Observed Effect Concentration. This is the lowest exposure level at which there are observable effects when comparing the exposed population and its appropriate control group. The effect, in this case, is not adverse.
LOAEC	Lowest Observed Adverse Effect Concentration. This is the lowest exposure level at which there are statistically and/or biologically significant changes in frequency or severity of adverse effects between the exposed population and its appropriate control group. The effect, in this case, is considered adverse.

Abbreviation/Term	Definition
NOAEC	No Observed Adverse Effect Concentration. The greatest concentration or amount of a substance at which there are no statistically and/or biologically significant changes in the frequency or severity of adverse effects between the exposed population and its appropriate control. Some effects may be produced at this level, but they are not considered as adverse, nor immediate precursors to specific adverse effects. In an experiment with several NOAECs, the assessment focus is primarily on the highest one for a given critical effect, leading to the common usage of the term NOAEC as the highest exposure without adverse effect.
NOEC	No Observed Effect Concentration The greatest concentration or amount of a substance at which there are no observable effects when comparing the exposed population and its appropriate control group.
NOx	Nitrogen Oxides. NO_x is a generic term for the nitrogen oxides that are most relevant for air pollution, namely nitric oxide (NO) and nitrogen dioxide (NO_2).
NO_2	Nitrogen Dioxide.
NSR	NO_x Storage-Reduction Catalyst
NT	No Exhaust After-Treatment.
NTDE	New Technology Diesel Engines. These engines were developed in the mid-2000s and involve use of inbuilt emission control measures (e.g. DPFs, DOCs) to minimise emissions to air (particularly DPM and NO_x) from the diesel combustion process. NTDE exhaust is defined as diesel exhaust from post-2006 and older retrofit diesel engines that incorporate a variety of these technological advancements, including electronic controls, ultra-low-sulfur diesel fuel, oxidation catalysts, and wall-flow DPFs.
OEL	Occupational Exposure Limit. The OEL typically represents a regulatory limit for airborne contaminants that must not be exceeded. It is synonymous with the Workplace Exposure Standard (WES) used in Australia, but the terminology 'OEL' has been used more broadly in this report when referring to international versions of these values.
OR	Odds Ratio. This is a measure of association between an exposure and an outcome in epidemiological studies. The OR represents the odds that an outcome will occur given a particular exposure, compared to the odds of the outcome occurring in the absence of that exposure. ORs are most commonly used in case-control studies, however they can also be used in cross-sectional and cohort study designs as well.
PAH	Polycyclic Aromatic Hydrocarbons. PAHs are a class of chemicals that occur in coal, crude oil, and gasoline. They are also produced when coal, oil, gas, wood, garbage, and tobacco are burned, as well as in combustion of diesel. Several PAHs are carcinogenic.
Peak Limitation	A maximum or peak airborne concentration of a substance determined over the shortest analytically practicable period of time that does not exceed 15 minutes. Peak limitations are set for some substances where exposure can induce acute effects after relatively brief periods to high concentrations. Excursions above the peak limitation exposure standard are not permitted at any time under the model WHS laws.

Abbreviation/Term	Definition
PM	Particulate Matter. This is the term used to describe a mixture of solid particles and liquid droplets found in air. PM includes $PM_{10,}$ inhalable particles with diameters generally less than or equal to 10 µm, and $PM_{2.5}$, fine inhalable particles with diameters generally less than or equal to 2.5 µm.
POD	Point of Departure. The concentration-response point that marks the starting point for low-concentration extrapolation. The POD may be a NOAEC/LOAEC, but ideally is established from BMD modelling of the experimental data. The POD generally corresponds to a selected estimated low-level of response (e.g. 1 to 10% incidence for a quantal effect). Depending on the mode of action and available data, some form of extrapolation below the POD may be employed for low-concentration risk assessment or the POD may be divided by a series of uncertainty factors to arrive at a guideline value protective of human exposures.
REC	Respirable Elemental Carbon. This is EC measured by a size selective device conforming to a sampling efficiency curve which has an approximate 50% cut-point of 4 µm, as defined by ISO 7708. This is also the mass fraction of inhaled EC that penetrates to the unciliated airways (i.e. the gas-exchange region of the lung).
RfC	Reference Concentration. An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a NOAEC, LOAEC, or BMC, with uncertainty factors generally applied to reflect limitations of the data used. This terminology is used by the United States Environmental Protection Agency and is equivalent to an ambient air guideline value.
ROS	Reactive Oxygen Species. A type of unstable molecule that contains oxygen and that easily reacts with other molecules in a cell. A build-up of ROS in cells may cause damage to DNA, RNA, and proteins, and may cause cell death. Reactive oxygen species are free radicals. Also called oxygen radical.
SCR	Selective Catalytic Reduction. This is an advanced active emissions control technology system that injects a liquid-reductant agent through a special catalyst into the exhaust stream of a diesel engine. The reductant source is usually automotive-grade urea, otherwise known as diesel exhaust fluid. The diesel exhaust fluid sets off a chemical reaction that converts NO_x into nitrogen, water and tiny amounts of carbon dioxide (CO_2), natural components of the air we breathe, which is then expelled through the vehicle tailpipe.
SEM	Scanning Electron Microscopy. This is a type of microscopy technique that produces images of a sample by scanning the surface with a focused beam of electrons.
SO_2	Sulfur Dioxide.
STEL	Short-Term Exposure Limit. This is the time-weighted average maximum airborne concentration of a substance calculated over a 15-minute period. Under the model WHS laws, the STEL must not be exceeded at any time during an 8-hour working day, even if the exposure during the full day is less than the TWA. Exposures at the STEL must not be longer than 15 minutes and must not be repeated more than 4 times per day. There must be at least 60 minutes between successive exposures at the STEL.

Abbreviation/Term	Definition
SVOC	Semi-Volatile Organic Compounds. SVOCs are not as volatile as VOCs. The lower the boiling point for a compound to evaporate into the air, the more volatile it is. Thus, a SVOC, being less volatile, has a higher boiling point at which it evaporates into the air. SVOCs typically have boiling point ranges of 240-260°C to 380-400°C (US EPA 2022).
TDE	Traditional Diesel Engines. These are engines that are not considered NTDE. Increasingly stringent emissions standards (1988–2010) for particulate matter (PM) and nitrogen oxides (NO_x) in diesel exhaust have helped stimulate major technological advances in diesel engine technology and diesel fuel/lubricant composition, resulting in the emergence of NTDE. Traditional Diesel Engines are any engines that are not NTDE.
TEF	Toxicity Equivalency Factor. In this report, this term is used to describe the toxicity potency of a PAH relative to that of one of the most potent PAHs, benzo(a)pyrene (BaP).
TEQ	Toxic Equivalence Quotient. A TEQ expresses an aggregate measure of toxicity based on a number of contributing compounds. Contributing compounds are assigned a weighted factor (i.e. a TEF) relative to the most toxic compound contributing to the aggregate. The sum of PAHs in analytical measurements is often reported as BaP TEQ.
TLV	Threshold Limit Value (American Conference of Governmental Industrial Hygienist terminology). Threshold Limit Values (TLVs®) refer to airborne concentrations of chemical substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed, day after day, over a working lifetime, without adverse health effects.
TWA	Eight-hour Time-Weighted Average. This is the maximum average airborne concentration of a substance when calculated over a specific timeframe. Under the model WHS laws, this is an 8-hour working day, for a 5-day working week. With respect to a TWA, during periods of daily exposure to an airborne contaminant, exposure above this value is permitted for short periods, if they are compensated for by equivalent exposures below the exposure standard during the working day.
UF	Uncertainty Factor. This is a number (equal to or greater than 1) used to divide the POD to estimate the NOAEC for the whole human population. UFs may be applied for extrapolation from animals to humans, to account for human variability, for database uncertainties, for the use of subchronic studies when deriving a guideline value to protect against chronic exposures, for use of a LOAEC instead of a NOAEC, etc.
$\mu g/m^3.yr$	Micrograms Per Cubic Metre Years. This is a cumulative measure of an exposure concentration over a specific duration, in this case a number of years. The number of years over which the exposure is accumulated must be stated.
ULSD	Ultra-Low Sulfur Diesel. ULSD is a cleaner-burning diesel fuel that contains 97% less sulfur than low-sulfur diesel. ULSD was developed to allow the use of improved pollution control devices that reduce diesel emissions more effectively but can be damaged by sulfur. The sulfur content in ULSD is typically a maximum of 15 mg/kg.

Abbreviation/Term	Definition
VOC	Volatile Organic Compound. Volatile organic compounds (VOCs) are any materials made up of carbon, hydrogen, and potentially oxygen that can easily evaporate into a gas under very low boiling points. VOCs are organic chemicals that have a high vapour pressure at room temperature. High vapor pressure correlates with a low boiling point, which relates to the number of the chemical's molecules in the surrounding air, a trait known as volatility. VOCs typically have boiling points at 50-100°C to 240-260°C (US EPA 2022).
WES	Workplace Exposure Standard. In Australia, under the model WHS laws, a WES for a particular chemical sets out the airborne concentration limit of that substance or mixture that must not be exceeded. WES are not intended to represent acceptable exposure levels for workers, and do not identify a dividing line between a healthy or unhealthy working environment.

1 Introduction and objectives

As part of the review of the workplace exposure standards (WES) for airborne contaminants, WSP Australia Pty Ltd (WSP) and an independent peer reviewer concluded with respect to diesel engine exhaust (DEE) that the data available from the agreed sources in the pre-defined methodology for the review were inadequate to determine a WES value and that a review of additional sources be conducted (SWA 2019a).

Composition of DEE (and proportions of constituents) may vary depending on various factors such as engine type, fuel type and operating conditions (SWA 2019a). Thus, for potential derivation of a WES it is imperative to delineate in a methodical manner which constituents are of most critical toxicological relevance in DEE (if possible) and whether this varies based on engine age and type, and what exposure concentrations of these constituents are (or are not) associated with adverse health effects.

Safe Work Australia (SWA) engaged SLR Consulting Australia Pty Ltd (SLR) to produce a research report to inform a WES and supporting advisory notations for DEE. The objectives of the Project were to:

- identify relevant independent, peer reviewed research and literature on the health effects of exposure to DEE and the impact engine types and year of manufacture have on the composition of DEE;
- if indicated in the research, recommend an encompassing health-based WES with supporting advisory notations for all DEE compositions present in Australia based on the adverse effect that occurs at the lowest airborne concentration; and
- if not feasible, to make recommendations for individual WES values and notations that would be protective of exposure to the different compositions of DEE present in Australia.

Outside of the scope of the Project was for the WES derivation to take into consideration feasibility of measurement and attainability in current workplaces.

1.1 What is a WES?

A WES refers to an exposure standard listed in the *Workplace Exposure Standards for Airborne Contaminants* (SWA 2019b) and represents the airborne concentration of a particular substance or mixture that must not be exceeded. There are three types of WES:

- 8-hour time-weighted average (TWA);
- short term exposure limit (STEL); and a
- peak limitation.

An 8-hour TWA is the average airborne concentration of a particular substance permitted over an 8-hour working day and a 5-day working week. These are the most common types of WES. It is preferable to keep exposure limits continually below the 8-hour TWA exposure standard. However, during periods of continuous daily exposure to an airborne contaminant, the 8-hour TWA exposure standard allows short term excursions above the exposure standard provided they are compensated for by extended periods of exposure below the standard during the working day (SWA 2013).

STELs are expressed as airborne concentrations of substances, averaged over a period of 15 minutes. STELs must not be exceeded at any time during an 8-hour working day, even if the TWA is not exceeded (SWA 2013, 2019c). Workers should not be exposed at the STEL concentration continuously for longer than 15 minutes, or for more than 4 such periods per 8 hour working day. There must be a minimum of 60 minutes between successive exposures at the STEL concentration (NOHSC 1995, SWA 2019b). Effectively, this means that exposure at the STEL could occur collectively for a total period of 1 hour during an 8-hour workday.

Generally, STELs are established to minimise the risk of acute adverse effects in nearly all workers including (NOHSC 1995, SWA 2013):

- intolerable irritation;
- irreversible tissue change; and
- narcosis to an extent that could precipitate workplace incidents.

STELs are recommended when there is evidence from human and/or animal studies that adverse health effects can be caused by high, short-term exposures (NOHSC 1995, SWA 2013).

Peak limitations are WES parameters representing a maximum airborne concentration of a particular substance determined over the shortest analytically practicable period of time, which does not exceed 15 minutes. A peak limitation may be applicable for a substance that can induce immediate effects upon relatively brief exposures to high concentrations. A peak limitation must not be exceeded at any time (SWA 2013).

2 Methodology

2.1 Literature search

SLR undertook a methodical literature search to identify relevant national and international agency documents and reviews, supplemented by a detailed search of the most recent peer-reviewed scientific literature (Figure 2-1). The study objectives (Section 1) and several targeted research questions helped focus the literature search (Section 2.2).

The search for relevant agency reviews used the search terms “diesel exhaust” OR “diesel emissions” and included all agencies listed in Appendix A.

Principal scientific databases, namely Science Direct, PubMed (including ToxLine), Embase and MedLine were searched to identify papers without date limitations addressing the adverse health effects and composition of diesel exhaust. Critical relevant articles were identified from titles and abstracts and full text sourced wherever possible. The search terms were combinations of “diesel exhaust” OR “diesel emissions” AND “health” OR “toxicity” OR “composition” OR “constituents”. A supplemental Google® search was also carried out and the first 15 pages of results were scanned. The bibliographies of agency reviews were also scanned to identify and source relevant papers not already sourced in the literature search.

The literature was interrogated to identify critical adverse health effects from exposure to DEE, concentration-response relationships for identified critical adverse effects, comparisons of toxicity and composition of traditional diesel engine (TDE) exhaust and new technology diesel engine (NTDE) exhaust.

The sourced literature was refined, using the inclusion criteria outlined in Appendix A, to enable critical review of those most relevant to the research questions. The epidemiological studies were assessed for relevant health effect data and tabulated by extracting qualitative and quantitative information on the study population, exposure level and duration, health effect and findings (Appendix B). Data extraction for epidemiological studies focused on those with potential exposure-response information and those that had not been previously reviewed in detail in the various agency reviews that were sourced. Environmental exposure studies were not included in the review. Epidemiological information is described in Section 6.2.

Relevant experimental animal toxicological information was also summarised in tabular form (Appendix C). Summarised studies were limited to those providing adequate concentration-response information for the inhalation route of exposure, and the focus was on studies published after the various agency reviews (i.e. ~2007 or later). Less importance was placed on non-inhalational studies or studies that only examined a single exposure. Those studies identified as providing crucial exposure-response information for comparison of TDE exhaust with NTDE exhaust were described in more detail (Section 6.3).

The relevant health effects of DEE, along with mechanistic considerations and the exposure concentrations associated with the critical effect(s), were summarised in subsequent sections. This information was then consolidated to make and support the recommendations detailed in this report in Section 7.

A list of all references cited in this report is provided in Section 9.

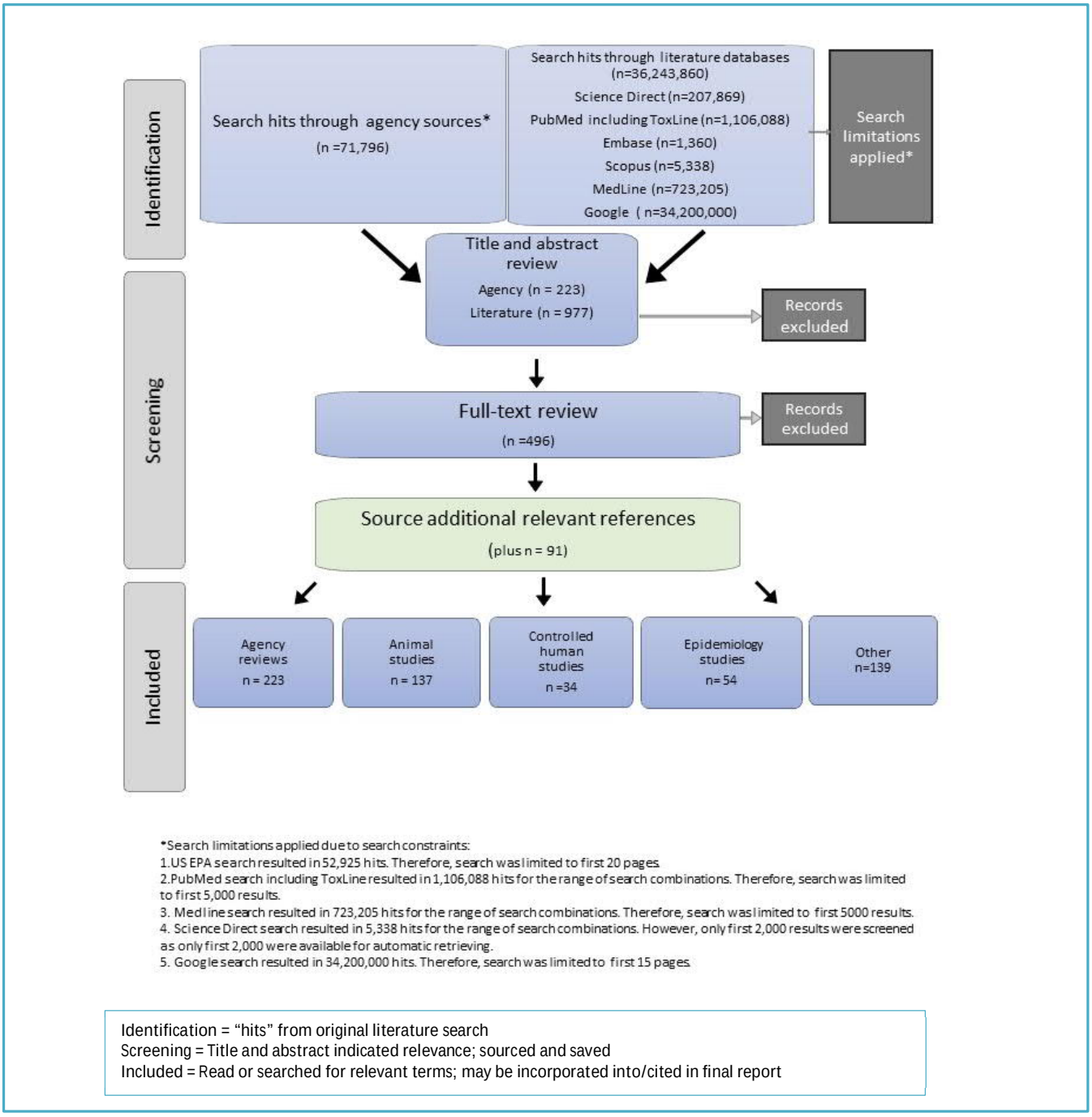


Figure 2-1 Summary of methodical literature search process

2.2 Research questions

The following research questions, devised to be in line with the objectives of this project, guided the literature review.

1. What is the composition of DEE?
2. How does the composition vary by engine type?

3. How does the composition vary by year of manufacture?
4. What are the health effects associated with exposure to DEE?
5. Does the weight of evidence in humans and/or animals suggest any advisory notations should be recommended for DEE?
6. If so, what advisory notations are recommended?
7. Are there specific critical constituents in DEE most commonly associated with these health effects?
8. Does exposure to DEE cause health effects over and above those of particulates *per se*?
9. Is there a threshold for the health effects associated with DEE exposure?
10. Is there sufficient (epidemiological and/or experimental animal) information to enable recommendation of a WES for DEE as a whole?
11. If not, is there sufficient (epidemiological and/or experimental animal) information to enable recommendation of a WES for critical components of DEE?
12. If so, what WES is recommended?

3 What is DEE?

3.1 Background

DEE is a complex mixture containing hundreds of different chemical species. Concentrations of these different species can change markedly depending on engine type, speed, load, whether accelerating or decelerating, starting temperature and the use of exhaust after-treatment devices (Landwehr et al. 2019, SCOEL 2017, Taxell and Santonen 2016, WHO 1996).

Diesel engine technology has steadily increased in complexity partly due to the increasingly stringent pollution emission standards over the last two decades. Engine technology has evolved from older, very basic mechanical fuel injection systems to the modern very high-pressure common rail electronic fuel injection systems, which allows both more finely atomised fuel to be injected and fuel injections to be electronically timed with potential for multiple injections per combustion event in order to cause the least exhaust emissions possible (Landwehr et al. 2019). Diesel particulate filters (DPF) and other similar exhaust after-treatment devices such as diesel oxidation catalysts (DOC), exhaust gas recirculation (EGR) and nitrogen oxide (NO_x) traps and selective catalytic reduction (SCR) for NO_x control were introduced to further limit pollution caused by diesel engines. In order for the exhaust after-treatment devices to be used to full capacity, sulfur concentration in diesel fuels also had to decrease because high sulfur levels degrade the after-treatment devices (Landwehr et al. 2019). This led to legislation changes starting in the mid 2000s that introduced ultra-low sulfur diesel (ULSD) into circulation across much of the world, decreasing sulfur levels in diesel fuel from above 500 ppm to below 15 ppm (Landwehr et al. 2019).

The EURO, US EPA and the US TIER classification systems have been developed as emission standards for light-heavy vehicles on road, heavy duty vehicles on road and off-road engine emissions respectively (Landwehr et al. 2019). Most engines classified as EURO IV, US EPA 2007 or US TIER 4, and above, require exhaust after-treatment devices, such as a DPF and DOC, for compliance, and engines classified as EURO IV and above generally require the latest high pressure common rail electronic fuel injection systems. In a mining setting in Australia, all trucks and cars that can be driven ‘on road’ are required to meet EURO classifications as adopted in Australia (see Figure 3-1i) (Landwehr et al. 2019). All other diesel equipment uses the US TIER ‘off road’ classifications (see Figure 3-1ii). New emission standards, not shown in Figure 3-1, EURO VII and US TIER 5, are currently under development and are expected to be implemented in 2025-2028 (CARB 2022, T&E 2022). According to Landwehr et al. (2019) and AIOH (2017), the majority of diesel engines currently used in underground mining in Australia are pre-2007 older technology transitional engines- TIERs 1-3, and thus do not contain exhaust after-treatment devices such as DPFs.

i)

Emission Standard	Year of Introduction (Europe)	Year of Introduction (Australia)[a]	Emission				
			CO (g/kWh)	HC (g/kWh)	NO_x (g/kWh)	PM (g/kWh)	Particle Number (1/kWh)
EURO I	1992	1994/1995	4.5	1.1	8.0	0.36	
EURO II	1996	2002/2003	4.0	1.1	7.0	0.25	
EURO II	1998	2002/2003	4.0	1.1	7.0	0.15	
EURO III	2000	2002/2003	2.1	0.66	5.0	0.10	
EURO IV	2005	2007/2008	1.5	0.46	3.5	0.02	
EURO V	2008	2010/2011	1.5	0.46	2.0	0.02	
EURO VI	2013	NA[b]	1.5	0.13	0.40	0.01	$8.0x10^{11}$

a= variable phase-in periods for new vehicle models vs existing models.

b= not applicable as Euro VI has not been introduced for heavy vehicles in Australia.

ii)

Emission Standard	Year of Introduction (US)[a]	Emission (g/kWh)				
		CO	HC	$HC+NO_x$	NO_x	PM
TIER 1	1996	11.4	1.3		9.2	0.54
TIER 2	2001	3.5		6.4		0.2
TIER 3	2006	3.5		4		0.2
TIER 4i	2011	3.5	0.19		2	0.02
TIER 4f	2014	3.5	0.19		0.4	0.02

a= The authors could not find any federally mandated emission limits for "off road" diesel engines in Australia.

Figure 3-1 i) EURO standards for 'on road' heavy duty diesel engines and ii) examples of US TIER standards for 'off road' heavy duty engines (reproduced from Landwehr et al. 2019)

A vast amount of primary and secondary literature, for example scientific and agency reviews (AIOH 2017, BauA 2017, Bugarski et al. 2011, Carex Canada 2020, DFG 2014, Health Canada 2016a, 2017; HCOTN 2019, IARC 2014, NRCWE 2018, NTP 2021, SCOEL 2017, Steiner et al. 2016, Taxell and Santonen 2016, TEMA 2012, US EPA 2002a) have been published on the composition and toxicity of DEE. Many of these have focused on DEE from the older TDE technology with only a few of the more recent reviews concentrating in part on NTDE exhaust (Greim 2019, Hallberg et al. 2017, HEI 2015a, Landwehr et al. 2019, Taxell and Santonen 2016, Weitekamp et al. 2020). Since the composition of DEE from TDE and NTDE can vary markedly from a quantitative perspective and potentially also from a qualitative perspective (Sections 3.2 and 3.4), this has led to speculation whether the toxicity and potential health effects from exposure to DEE from the two engine technology types are still comparable. Such considerations are important when considering deriving a DEE WES for occupational exposure. Many workplaces may still contain a combination of vehicles powered by TDE and NTDE (and/or transitional engines)[1], as the lead time to complete replacement with NTDE can be long.

[1] This is when TDE may be retrofitted with DPFs or other treatment devices with the aim of reducing emissions.

3.2 Composition of DEE

DEE consists of a complex mixture of gaseous, adsorbed organics and particulate components; diesel particulate matter (DPM) refers to the particulate fraction of DEE (AIOH 2017, Budroe et al. 2012, Bugarski and Timko 2007). The composition of this mixture varies depending on engine type, operating conditions, fuel[2], lubricating oil[3], and whether an emission control system is present (Budroe et al. 2012, Cantrell and Watts 1997, Health Canada 2016a, Wang et al. 2019).

The components of DEE are detailed below.

- The gaseous phase of DEE is a complex mixture consisting principally of the products of complete combustion, small amounts of the oxidation products of sulfur and nitrogen, and compounds derived from the fuel and lubricant. It consists largely of the same gases found in air, such as nitrogen, oxygen, carbon dioxide and water vapour, as well as carbon monoxide (AIOH 2017, Carex Canada 2020, HCOTN 2019, WHO 1996). Volatile hydrocarbons in the gaseous phase of DEE may include aldehydes (e.g. formaldehyde, acetaldehyde), alkanes, alkenes, aromatic compounds [including benzene, toluene, and polycyclic aromatic hydrocarbons (PAHs)] (Budroe et al. 2012). The gaseous phase makes up approximately 99% of the mass of whole DEE (Taxell and Santonen 2016). Principal components of the gaseous phase of DEE include the following.
    - Nitrogen oxides. At high temperatures, molecular nitrogen (N_2) from the intake air will react with oxygen (O_2) and hydrocarbons (HC) to form gaseous NO_x emissions, or oxides of nitrogen (NO and NO_2).
    - Carbon monoxide (CO). Compared to the CO emissions of a gasoline engine, the CO concentration in DEE is lower because diesel engines have a higher amount of available oxygen, or overall lean mixtures (Bugarski et al. 2011, Health Canada 2017). This makes it possible to run this type of engine in enclosed worksites where gasoline engines should not be used (Carex Canada 2020).
    - Gas-phase hydrocarbons consisting of volatile and semi-volatile organic compounds (VOCs and SVOCs). Various compounds including formaldehyde, acetaldehyde, isobutyl aldehyde, crotonaldehyde, propionaldehyde, benzaldehyde, propiolactone, aliphatic ketones, lactones, esters and alcohols, aromatic esters, ketones, anhydrides, phenols and dihydric phenols have been previously identified in DEE from TDE (Merchant 1982). PAHs are also known to be present in the gaseous phase of DEE (Geldenhuys et al. 2022)[4].

[2] A large number of publications found in the literature searches undertaken for this report dealt with changes in DEE emissions due to the use of alternatives to petroleum-based fuels, with a large focus on biodiesel (a generic term for various types of diesel fuels produced from biomass such as vegetable oils, animal fats, etc.). These publications have not been reviewed in detail as they are considered outside of the scope of this project, which focuses on petroleum-based fuel. Bugarski et al. (2011) found the effects of biodiesel blends on DEE emissions are not consistent over the range of possible applications, and they are also somewhat unpredictable. Some engine technologies are more responsive to biodiesel blends than others. In addition, engine operating conditions play a major role in defining the characteristics of the emissions when using fatty acid methyl ester fuels.

[3] Some products resulting from the combustion of lubricating oil are increased organic emissions of DPM, gaseous sulfuric emissions, and non-combustible DPM (or ash). The sulfuric and ash content of newer fuel and lube oil blends have been reduced considerably in recent years (Bugarski et al. 2011).

[4] Note PAHs are found in the gaseous phase of DEE and can also be found adsorbed to the particulate fraction of DEE.

- Sulfur dioxide (SO_2), which forms when sulfur in the fuel and lubrication oil oxidises during the combustion process.

- The particulate fraction of DEE (i.e. DPM, see Figure 3-2) consists of a solid carbon core and ultra-fine droplets of a complex mix of SVOCs as well as inorganic substances, which largely include four by-products of diesel combustion [i.e. elemental carbon (EC), organic carbon (OC), ash, and sulfuric compounds] (Bugarski et al. 2011, Cantrell and Watts 1997, HCOTN 2019, WHO 1996).
    - The organic fraction of DPM contains adsorbed compounds such as aldehydes, alkanes, alkenes, aliphatic hydrocarbons, PAHs and PAH-derivatives (Budroe et al. 2012). These are the non-gas-phase organics that have mass and, therefore, contribute to total DPM mass (Bugarski et al. 2011). Note as discussed above many of these components are found in the gaseous phase of DEE but can also be adsorbed to DPM.
    - The solid particulate fraction consists mainly of very small particles (typically 15-30 nm diameter) that rapidly agglomerate together to form clumps of particles, which are themselves typically <1 µm aerodynamic size. High resolution scanning electron microscopy (SEM) has demonstrated that the basic diesel particle consists of an irregular stacked graphitic structure, nominally called EC[5] (AIOH 2017, Bugarski and Timko 2007, Rogers and Davies 2005) (Vedula 2011).

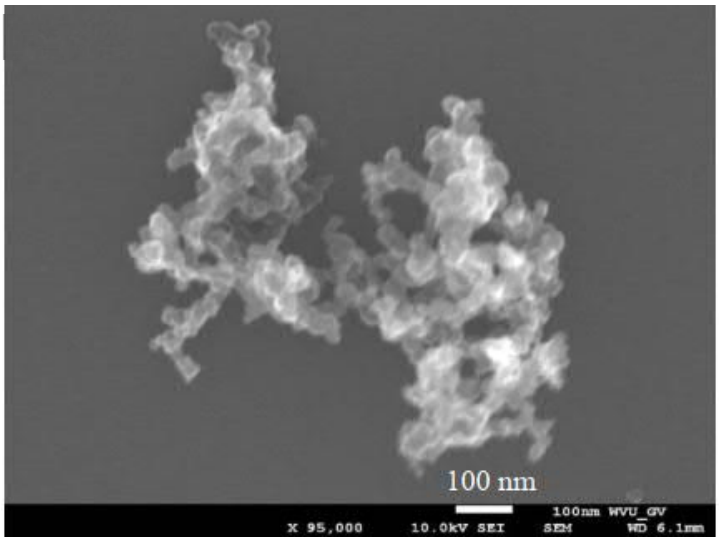
      The graphitic nature and high surface area of these very fine carbon particles means they have the ability to adsorb hydrocarbons (the semi-volatile organic carbon droplets and vapours) originating from the unburnt fuel, lubricating oils and the compounds formed in the complex chemical reaction during the combustion cycle. Due to their small size, DPM aerosols behave similarly to the surrounding gases. They have much longer residence times in the atmosphere than larger mechanically generated particles, which are removed quite quickly by gravitational settling. In addition, a large portion of DPM is deposited in the human respiratory tract and can penetrate to the alveoli of the lungs where gas exchange occurs (AIOH 2017, Bugarski et al. 2011, NTP 2021, US EPA 2002a). Several jurisdictions have concluded that the particulate fraction of DEE is most likely responsible for any carcinogenic potential (see also Section 4.2) (AIOH 2017, Health Canada 2016a, IARC 2014, Neumann et al. 2019, SCOEL 2016)[6].

[5] Diesel engines function by allowing a mixture of fuel (a hydrocarbon, C_XH_Y) and intake air, which includes oxygen (O_2), nitrogen (N_2), and carbon dioxide (CO_2), to ignite under high temperatures and pressures formed by compression. This form of combustion allows overall cylinder conditions to be 'lean' (fuel poor or oxygen rich), which promotes good efficiency and, as a result, a high conversion of fuel (C_XH_Y) into carbon dioxide (CO_2) and water vapour (H_2O). However, fuel injection (typically used in diesel engines) also creates 'rich' regions, or localised areas within the fuel injection plume that lack the amount of O_2 necessary for proper combustion of fuel. If temperatures are hot enough, fuel will burn without the presence of O_2 within these regions, creating charred remains, or solid carbon soot, also referred to as EC. Once the EC is formed, most of it will combine with O_2 and burn during later stages of the combustion process. However, the remainder will be emitted from the engine exhaust as solid PM, forming the core of a typical diesel-particle agglomerate. The formation of EC during combustion and expulsion is therefore driven by three primary factors: temperature, residence time, and availability of oxidants (Bugarski et al. 2011).

[6] IARC (2014) noted there is sufficient evidence in experimental animals for the carcinogenicity of whole DEE and DEE particulate matter and extracts of DEE particles, but inadequate evidence for gas-phase DEE.

- DPM also contains transitional metals in ash (Bugarski and Timko 2007). Fuel and lubricating oil often contain a number of additives (detergents, dispersants, etc.), which are composed of metallic elements. When these fluids are consumed during combustion, these metallic elements can form inorganic solids. Normal wear of metallic engine components is another, though less substantial, source of ash generation (Bugarski et al. 2011). The contribution of ash to DPM mass is often lower in comparison with other forms of PM emissions.
- Sulfuric compounds are another contributor to DPM emissions. The sulfur in the fuel converts to SO_2. During the emissions process, SO_2 can react with other compounds in the exhaust and form solid sulfates, which contribute to overall DPM emissions (Bugarski et al. 2011).

50% load



Full load

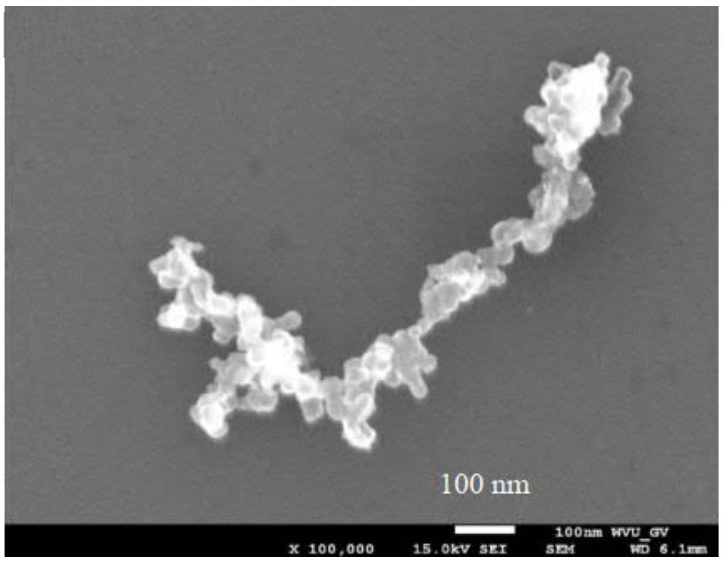


Figure 3-2 SEM images of DPM at different loads of a heavy-duty diesel engine (from Vedula 2011)

Some components of DEE have been analytically well characterised. A complete mass balance for all DEE, however, is not available since source fuel varies (DFG 1990, 2014).

3.3 Emission controls

The three most commonly applied emission control systems installed in diesel engines are the DOC, DPF and NSR/SCR (see Figure 3-3).

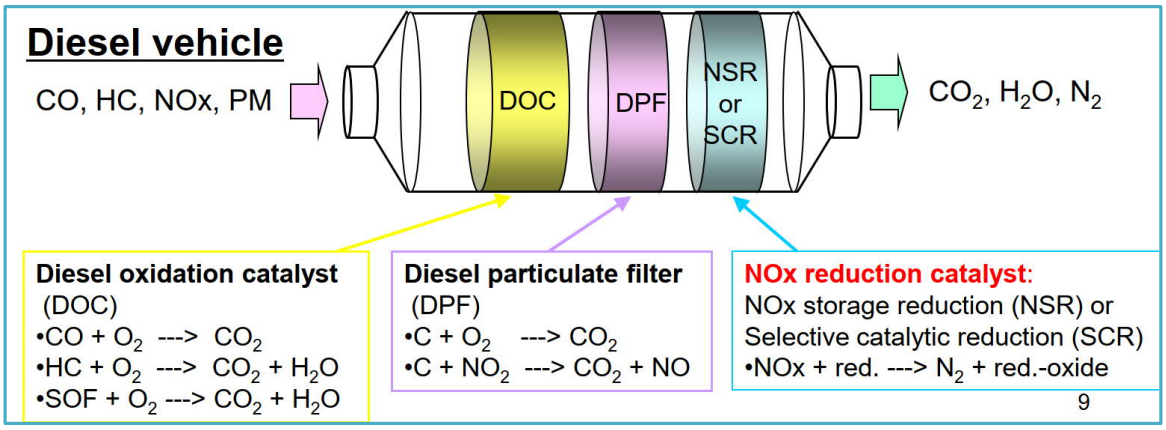


Figure 3-3 Diagram of main emission control systems in diesel engines (reproduced from HAS 2012)

NO_x emissions are typically controlled using SCR technology. Almost any attempt at lowering NO_x emissions results in an increase in DPM and vice versa; this is because NO_x formation increases at higher combustion temperatures and 'lean' conditions, while DPM mass formation increases at lower combustion temperatures and 'rich' conditions (Bugarski et al. 2011, Cauda et al. 2010). Hence, NTDE require sophisticated combinations of various in-cylinder and after-treatment technologies. The effectiveness of DPFs in removing aerosols has been found to be strongly influenced by engine operating mode. The concentration of nucleation mode aerosols tends to be higher at high-load modes than at low-load modes (Bugarski et al. 2008, 2009). With the use of SCR technology, expected reductions in NO_x emissions from heavy-duty engines range from 55-90%, depending on the application and test method used (Bugarski et al. 2011).

With respect to SO_2 and sulfuric compounds in DPM, the transition toward ultralow sulfur diesel (ULSD) and low-sulfur content lubricants has promoted control over these emissions (Bugarski et al. 2011, Taxell and Santonen 2016).

DOCs are often used within NTDE exhaust systems as a secondary control of organic carbon emissions, but DPFs may also play a role in reducing these emissions. EC formation is reduced at the source by increasing the surface area contact of fuel and air during combustion so that the conversion rate of fuel into CO_2 and H_2O is high. This includes promoting lower local fuel/air ratios in contemporary engines through a number of in-cylinder controls and/or capturing these particles within the exhaust system using DPFs (Bugarski et al. 2011).

3.4 TDE vs. NTDE

Using a DPF, DOC and other such exhaust after-treatment devices such as is employed in NTDEs, the concentrations of components of DEE change dramatically. A DPF is capable of removing approximately 90% by mass of PM (Bugarski et al. 2007, 2011; Health Canada 2016a, US EPA 2010a, c). EC is preferentially removed and ratios of EC to organic carbon reduce from approximately 3 to 0.512 (Bugarski et al. 2004, HCOTN 2019, Landwehr et al. 2019). In exhaust without a DPF, EC makes up approximately 75% of PM by weight, which reduces to approximately 13% after the use of a DPF (HCOTN 2019, NRCWE 2018, SCOEL 2017, Taxell and Santonen 2017, US EPA 2002a). Average accumulation mode[7] particle sizes also decrease from >40 nm to approximately 25 nm with the use of a DPF and in the ultrafine particle range, larger sized particles closer to 100 nm in size are removed from the exhaust more successfully than smaller sizes (Bugarski et al. 2007, 2020; Landwehr et al. 2019). Figure 3-4 provides an overview of the typical composition of DPM in TDE and NTDE exhaust.

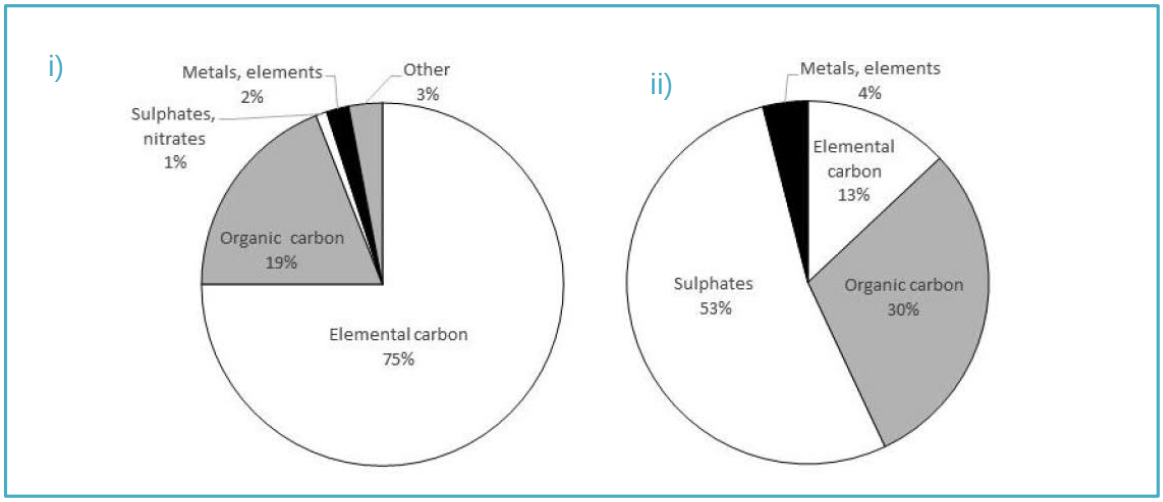


Figure 3-4 Typical composition of DPM emitted by i) 1990-2000 diesel engine (TDE) and ii) post-2006 diesel engine (NTDE). Reproduced from Taxell and Santonen (2016)

NTDE also emit much less sulfur (as sulfates) due to the transition to ULSD and as a consequence there are more detectable numbers of ultrafine droplets of SVOCs (as these are less readily adsorbed onto the surface of the reduced quantities of carbon particulates) (AIOH 2017). A four-fold increase in percentage of NO_2 in total NO_x may be seen when engines are fitted with a DOC and engine is operated at high-load modes (Bugarski et al. 2007, HCOTN 2019).

[7] From a size distribution perspective, DPM consists of two distinctive modes: an accumulation mode with a median around 100 nm and a nucleation mode characterised by a peak below 30 nm. While the accumulation mode is the predominant mode present for exhaust without aftertreatment technologies, high number concentrations related to nucleation mode have been recorded on treated exhausts (Cauda et al. 2012, Taxell and Santonen 2016).

As part of the Advanced Collaborative Emissions Study (ACES), exhaust emissions from four different 2007 model year US Environmental Protection Agency (EPA)-compliant heavy-duty highway diesel engines were measured. The engines were equipped with exhaust high-efficiency catalysed DPFs. Compared with other work performed on 1994- to 2004-technology engines, average emission reductions in the range of 71 to 99% were observed for metals, other elements, EC, inorganic ions, and gas- and particle-phase VOCs and SVOCs. On average, particle number emissions with the 2007 engines were 90% lower than the particle number emitted from a 2004-technology engine. EC represented only 13% of the PM mass emitted from the 2007 technology engines (Khalek et al. 2011).

In a subsequent study, Khalek et al. (2015) found that heavy-duty 2010-compliant diesel engines (Tier 4) equipped with a high-efficiency catalysed DPF and powered by ULSD fuel had emissions of PAHs, nitro-PAHs, hopanes and steranes, alcohols and organic acids, alkanes, carbonyls, dioxins and furans, inorganic ions, metals and elements, EC, and particle number that were substantially (90 to >99%) lower than pre-2007-technology engine emissions. Lower concentrations of PAHs were also found by Braun et al. (2010) in Euro 3 compliant engine exhaust compared to TDE exhaust.

Hesterberg et al. (2008) investigated the emission concentrations of PM, several volatiles and PAHs in TDE exhaust compared with NTDE equipped with a DOC or catalysed particulate filters (traps). From this information, it is not clear that the composition of NTDE exhaust is fundamentally different from TDE exhaust. Clear reductions in emissions were evident for PM (i.e. 95-99%) whereas reductions of some other compounds such as PAHs, benzene and 1,3- butadiene in NTDE exhaust may be lower (i.e. 80-90%) (Hesterberg et al. 2008). Although most constituents exhibit a clear reduction in total emissions in NTDE exhaust, the proportions of individual constituents relative to that of PM may change (Budroe et al. 2012). Liu et al. (2010) made similar findings with reductions in alkanes, aliphatic aldehydes, hopanes and steranes achieved by more than 95%. Large reductions were also observed for three of four biphenyl aromatics between 47.2 and 69.9% (noting that 2-methylbiphenyl concentrations increased), most of the PAHs between 51.6 to 98.4% (with an increase in methyl fluorenes), in nitro-PAHs by more than 76.2% and also in oxygenated PAHs (52.9 to 99.2%).

In a subsequent study, Hesterberg et al. (2011) has shown NTDE exhaust to have greater resemblance to particulate emissions from compressed natural gas or petrol engines than to that of TDE. Large reductions in total emissions of PM, sulfate/nitrate, hydrocarbons, EC and ash were observed for NTDE exhaust. Sulfate was one of the few DEE species with some consistent findings of increased emissions in NTDE as compared to TDE exhaust, but this species is generally regarded to be of low toxicity (Hesterberg et al. 2011).

Overall, the information reviewed indicates NTDEs, depending on the controls put in place, have achieved reductions in emissions of various constituents of DEE ranging from 47-99% when compared to TDEs. Although reductions in the amounts of certain emissions are observed, the identity of DEE constituents does not appear to differ.

4 Health effects of DEE exposure

Inhalation is the most important route of occupational exposure to DEE. Although the adverse health effects of the gaseous fraction of DEE have been known for some time, research in the last two decades has focussed on the particulate component of DEE since it has been found to be able to induce various health effects. In addition, DEE are known to be associated with non-health aspects such as malodour, and visual and nuisance pollution (AIOH 2017), which are not the subject of this report.

DEE has been demonstrated to cause several adverse effects in animals and humans. These effects include respiratory, cardiovascular and immune system toxicity (Budroe et al. 2012). DEE has also been shown to induce genotoxicity and cancer in both animals and humans (IARC 2014, NTP 2021).

4.1 Pulmonary toxicity

Exposure to DEE can cause inflammation in the lungs, which may aggravate chronic respiratory symptoms and asthma. In epidemiological studies, chronic exposures have been associated with cough, increased sputum production, and lung function changes (Carex Canada 2020, CDC 2011, US EPA 2002a), and there is suggestive evidence that there may be an increased risk of chronic obstructive pulmonary disease with increased exposure to DEE (Hart et al. 2012).

Studies in experimental animals indicate that short-term DEE exposure can cause an increase in airway resistance and reactivity, and that short- and long-term DEE exposures can result in respiratory inflammation, pulmonary hyper- and metaplastic changes and fibroses (DFG 2014, Health Canada 2016a, US EPA 2002a) [8]. In addition, many studies have demonstrated that DEE and DPM exposures can lead to cytotoxic effects in cell culture (Health Canada 2016a).

Prolonged exposure to high concentrations has been associated with accumulation of particles in macrophages, changes in lung cell populations, fibrotic effects and squamous metaplasia, which appeared to be associated with impaired pulmonary clearance (DFG 2014, IARC 2014, US EPA 2002a).

The evidence, reviewed in detail in Section 6, is considered to be sufficient for the relationship between DEE exposure and adverse respiratory health outcomes to be considered causal, based on clear evidence of adverse respiratory symptoms, decrements in lung function and inflammatory responses from multiple controlled human exposure studies (for references, see Section 6.1) and supporting evidence of enhanced airway responsiveness and respiratory inflammation from toxicological studies (for references, see Section 6.3). This conclusion is in line with recent agency reviews (ATSDR 2018, IARC 2014, Health Canada 2016a). The concentration-response for these effects and suitability for derivation of a WES is discussed in Section 6.

[8] Exposure of rats, guinea-pigs, and cats to DEE from TDE with a particle content of 6,000 µg/m^3 for about four weeks altered lung function, including a 35% increase in pulmonary flow resistance in guinea-pigs and a 10% decrease in vital capacity (expiratory flow) in cats. Histopathologically, focal thickening of alveolar walls, a significantly increased type-II cell labelling index, and accumulations of particle laden macrophages were found. The accumulations were located near the terminal bronchioles and became larger, due to macrophage attachment (sequestration), during a subsequent recovery period (WHO 1996).

4.2 Carcinogenicity

Based on animal and epidemiological studies, it is evident that exposure to DPM from TDE is associated with an increased risk of lung cancer, albeit the increase is not always consistent, and many studies suffer from limitations (DFG 2014, IARC 2014, NTP 2021, SCOEL 2017). Common limitations of many occupational epidemiological studies with DEE include small study sizes, no adjustment for common potential confounders such as smoking and exposure to other occupational carcinogens, and limited exposure information rendering exposure misclassification or imprecise exposure estimates a potential problem (Gamble et al. 2012, HCOTN 2019, IARC 2014, SCOEL 2017, TEMA 2012, US EPA 2002a)[9]. Recent studies have also shown sporadic significant associations with other cancers (e.g. rectal, bladder, oestrogen receptor negative breast cancer, and childhood brain tumours associated with parental occupational exposure) (Kachuri et al. 2016, Koutros et al. 2019, Talibov et al. 2019, Pedersen et al. 2021, Peters et al. 2013), but numbers of studies investigating these endpoints are too few to draw any firm conclusions (see also Figure 4-1). HEI (2015a) and IARC (2014) concluded that the evidence for an association of DEE exposure with cancers other than lung and bladder cancers is inadequate.

The International Agency for Research on Cancer (IARC) first determined that DEE was likely to be carcinogenic to humans in 1989, classifying it as a Group 2A carcinogen (probably carcinogenic to humans). In 1998, an advisory group to the IARC Monographs Program suggested that DEE be re-evaluated based on a study on underground miners in the United States [referred to as the Diesel Exhaust in Miners Study or DEMS)[10] (see also Section 6.2). Following the publication of the study's results in March 2012, IARC re-evaluated its classification of DEE. On June 12, 2012, IARC reclassified DEE as a Group 1 carcinogen (carcinogenic to humans). This determination was based on 'sufficient evidence' for lung cancer and 'limited evidence' for bladder cancer in humans (IARC 2014). The most informative studies on DEE exposure and lung cancer were based on occupational cohorts of miners, railroad workers and workers in the transport industry with better characterised exposure relative to earlier studies; these studies supported a positive association between exposure to DEE and risk of lung cancer, but the exposure-response relationship for the effect has been subject to much debate. This debate is discussed in Section 6.2.

[9] IARC (2014) noted the major limitation in many studies was the paucity of data relating job titles to exposure to DEE, and the inclusion of studies in meta-analyses with varying quality of exposure information. Moreover, most of the individual studies evaluated risks for occupations and not for exposure to DEE. A further limitation of the earlier studies was their inclusion of subjects who worked before or at the beginning of the diesel era or workers with a latency period that was inadequate to attribute any increases in cancer to exposure to DEE (IARC 2014).

[10] The original DEMS study included 12,315 workers exposed to DEE at eight US non-metal mining facilities. The study was originally initiated in part due to the many other studies that had been conducted at that time that had found an association between DEE and lung cancer but had made no quantitative exposure estimates. Different aspects of the study, along with numerous reanalyses of the data from the study, have been published in several scientific articles (Attfield et al. 2012, Coble et al. 2010, Costello et al. 2016, 2018; Crump and van Landingham 2012, Crump 2014, Crump et al. 2015, 2016; Ferguson et al. 2020, Morfeld and Spallek 2015, Neophytou et al. 2016, Silverman et al. 2012, Stewart et al. 2010, 2012; Vermeulen et al. 2010a, 2010b, 2014, 2020). The results of these various publications are discussed in more detail in Section 6.2 and summarised in Appendix B.

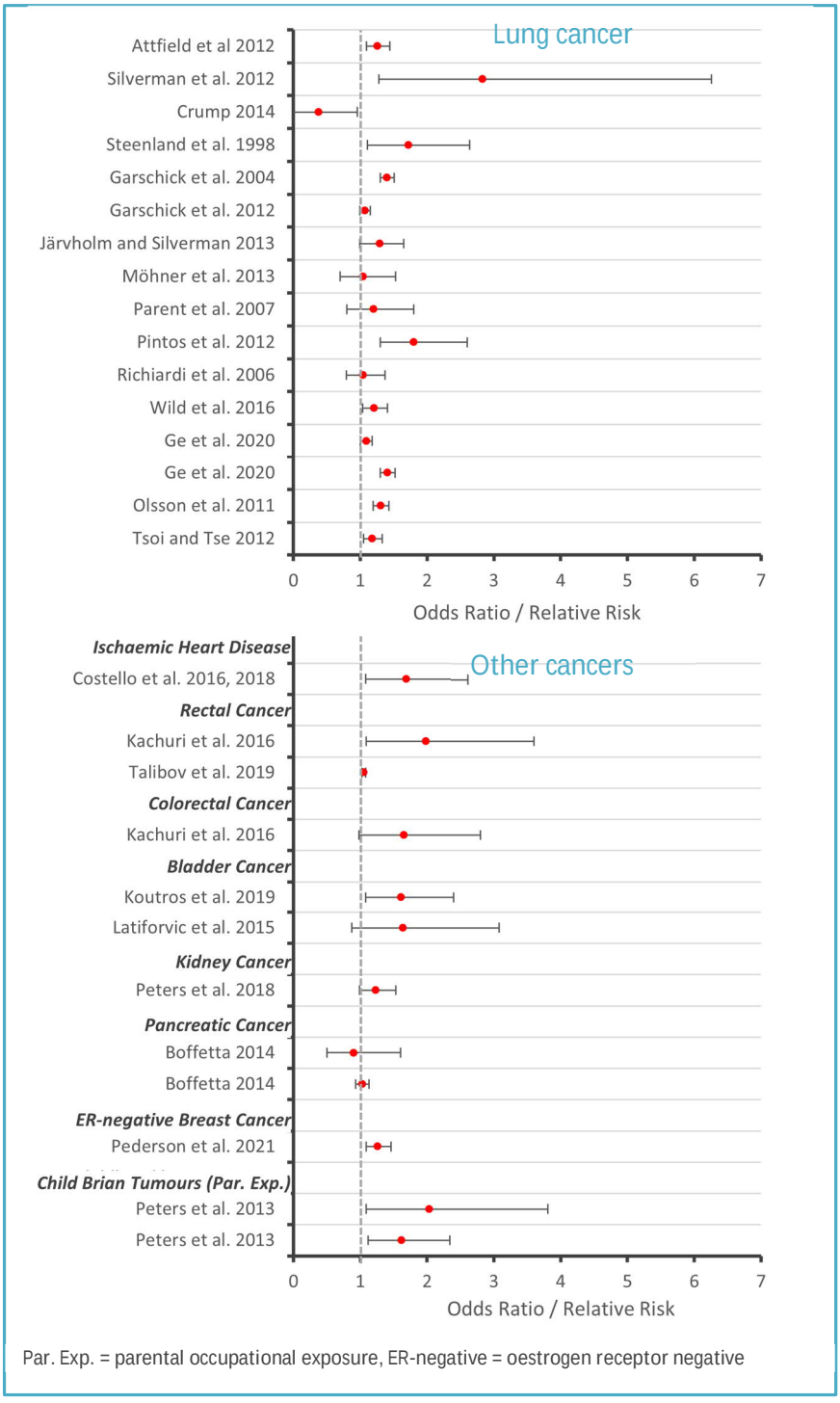


Figure 4-1 Summary of select odds ratios / risk ratios for lung cancer and other cancers from recent literature (compiled from data in Appendix B)

In animal carcinogenicity studies, a statistically significant increase in lung tumour frequency (primarily adenomas, squamous cell tumours and adenocarcinomas) was seen in all studies in which rats were exposed to TDE concentrations of DPM above 2,000 µg/m^3 for more than two years (DFG 2014, HCOTN 2019, IARC 2014, Taxell and Santonen 2016, WHO 1996). However, the majority of studies in other experimental animals have found no increased incidence in lung tumours at TDE DPM exposure concentrations ranging from 2,000 to 12,000 µg/m^3 (see Table 4-1). The gaseous phase of DEE, free of particles, and filtered DEE induced no tumours in rats (DFG 2014, IARC 2014, Ishihara and Kagawa 2002, 2003; NRCWE 2018, NTP 2021, Taxell and Santonen 2016).

In a recent experimental inhalation study in which rats were exposed to whole DEE from NTDE [3-12 µg DPM/m^3 (≈ 1-3 µg respirable EC or REC/m^3), 0.2-8 mg NO_2/m^3], no tumours were found (McDonald et al. 2015). This indicates the reduction in particulate concentration from the emission controls in NTDE likely attenuates the carcinogenicity of DPM; it is unknown whether this is due to the reduction in particulates *per se* or the reduction in concentrations of adsorbed genotoxic compounds, e.g. PAHs[11].

Table 4-1 Summary of experimental animal carcinogenicity study outcomes for TDE

DPM concentrations (µg/m^3)	Animal studied	Study finding in relation to lung carcinogenicity	References
≤ 2,000	Rats	No carcinogenic effects	Studies summarised in DFG 1990, 2014; IARC 2014, NRCWE 2018, TEMA 2012
≥ 2,000	Rats	↑ tumour frequency	Several studies summarised in DFG 2014, HCOTN 2019, IARC 2014, Taxell and Santonen 2016, WHO 1996
2,000	Monkeys	No ↑ tumour incidence (1)	Lewis et al. 1989
2,000-4,000	Mice (ICR and C57B1/N)	No ↑ tumour incidence	Takemoto et al. 1986
4,000	Mice (NMRI)	Significant ↑ in tumour incidence	Heinrich et al. 1986
4,000	Mice (NMRI, C57B1/6N)	No ↑ tumour incidence	Heinrich et al. 1995
4,000	Hamsters	No ↑ tumour incidence	Heinrich et al. 1986
6,000 for 12 mo, then 12,000 for 12 mo	Mice (Sencar)	Slightly ↑ tumour incidence (F only)	Pepelko and Peirano 1983
6,000 for 12 mo, then 12,000 for 12 mo	Cats	No ↑ tumour incidence (1)	Pepelko and Peirano 1983

[11] The critical potential adverse health effects of concern associated with PAH exposure are lung and skin cancer, and in relation to DEE exposure, there is a possibility that PAHs adsorbed to DEE particulates may be responsible for cancer induction (see Section 5.1). Benzo(a)pyrene (BaP) is typically used as an indicator compound for assessing the carcinogenic hazard and risk of a PAH mixture. Based on a comparison of potencies of individual PAHs, their concentrations are generally translated using toxicity equivalency factors (TEFs) to a total PAH concentration expressed as BaP toxicity equivalency quotients (TEQ). Numerous PAHs have been identified in DEE (Khalek et al. 2011, Liu et al. 2010). Naphthalene is the predominant PAH typically found in DEE, followed by other 'standard' PAHs with very small amounts (i.e. 0.04-0.2%) of nitro-PAHs (see Appendix E). Based on their TEFs, the majority of PAHs have lower potency compared with the indicator compound BaP. The exception is 6-nitrochrysene which, according to Kelly et al. (2021) is 11x more potent than BaP. However, due to the very low amounts of this PAH in DEE (Appendix E) it is unlikely to have a marked influence on overall BaP TEQ.

DPM concentrations (µg/m^3)	Animal studied	Study finding in relation to lung carcinogenicity	References
7,100	Mice (CD-1)	No ↑ tumour incidence	Mauderly et al. 1996
6,600	Hamsters	No ↑ tumour incidence	Brightwell et al. 1989

↑ = increased. DPM = Diesel particulate matter. mo = months. F = Females.

1. It should be noted monkeys and cats were only exposed for two years, which is inadequate for evaluation of carcinogenicity in these species (IARC 2014).

Various international organisations have assessed in detail lung cancer risk after exposure to DEE. Based on their interpretation of the toxicological and epidemiological data, regulatory authorities in USA, Europe and Canada have concluded that sufficient evidence exists to indicate that DPM from TDE presents an increased risk of lung cancer, although the absolute quantification of potency remains unclear (AIOH 2017, DFG 2014, Health Canada 2016a, US EPA 2002a, IARC 2014, BauA 2017)[12]. Potency and the exposure-response relationship for DPM exposure and lung cancer in humans has been subject to much debate which is discussed further in Section 6.2.

This has meant, for example, that the US EPA (2002a) has not developed a quantitative estimate of cancer risk, because of "*inadequate exposure-response data from human studies"* and a determination that "*doses at which toxicity was observed in rats were much higher than expected environmental exposure levels"* (US EPA 2002a). Many international agencies and scientists who have derived WES for DPM have not used quantitative estimates of cancer risk from epidemiological studies for the derivation (see Section 7.1).

Nevertheless, two international agencies have considered the occupational epidemiological information to be of sufficient quality to be used for quantitative derivation of a WES (HCOTN 2019, NRCWE 2018) (see Section 7.1). The use of the epidemiological information in quantitative assessments has been the subject of much debate in the scientific community (further discussed in Section 6.2).

[12] For example, US EPA (2002a) considered DEE (from TDE) to be likely carcinogenic to humans by inhalation based on: (1) strong, but less than sufficient, evidence for a causal association between DEE exposure and increased lung cancer risk in workers from varied occupations with DEE exposure; (2) supporting positive results in genotoxicity tests with DEE and organic constituents; (3) knowledge that a number of components of DEE have produced positive results in genotoxicity and carcinogenicity tests; and (4) positive results in cancer bioassays with rodents exposed to high intratracheal instillation doses of whole DEE, in skin painting studies using extracts of organic whole DEE, and in many chronic inhalation rat studies showing a positive lung cancer response at high exposures. A quantitative estimate of cancer risk, however, was not developed, because of inadequate exposure-response data from human studies and a determination that doses at which toxicity was observed in rats were much higher than expected environmental exposure levels (US EPA 2002a).

4.3 Other adverse effects

Irritation and neuro-physiological effects

Short-term exposure to DEE in humans has been shown to cause irritation of the eyes, throat, and bronchi, as well as neuro-physiological symptoms such as light-headedness, nausea, and respiratory symptoms (Carex Canada 2020, CDC 2011, SWA 2020a, b; UK HSE 2012a, WHO 1996). There is considerable variability in the reported DEE detection threshold, at or above which some of these effects may occur (US EPA 2002a). Controlled human exposure studies have reported that short-term DEE exposure was associated with subjective symptoms (e.g. eye and nose irritation, smell, headache, "throat and chest symptoms") (Giles et al. 2018, Mudway et al. 2004, Rudell et al. 1996b, Wierzbicka et al. 2014), altered lung function, respiratory inflammation and respiratory oxidative responses (see Appendix D). DEE exposure was associated with increased measures of airway resistance, indicative of bronchoconstriction, in healthy and asthmatic individuals (Koch et al. 2021, Mudway et al. 2004). In addition, both DEE and DPM can induce a range of inflammatory responses in human airways (Health Canada 2016a). AIOH (2017) noted that the level of eye and upper respiratory tract irritation is significantly reduced at DPM exposure concentrations of 200 µg/m^3, or approximately 100 µg/m^3 REC (see also Section 6.1).

Cardiovascular effects

In environmental epidemiological studies, increased mortality rates have been reported in association with exposure to DEE, particularly in the elderly and those with cardiopulmonary conditions (Carex Canada 2020). Short-term controlled exposure studies in humans have also observed effects on cardiovascular measures (see Appendix D). Health Canada (2016a) concluded that the evidence is suggestive of a causal relationship between chronic DEE exposure and adverse cardiovascular health outcomes, based on epidemiological studies and supporting long-term animal toxicological studies.

Immunological effects

DEE exposure has also been shown to increase immunological response to other allergens. Environmental epidemiological studies on asthma and DEE indicate that particularly children living near busy roads have an increased risk of developing asthma and asthma-like symptoms (DFG 2014). In short-term human studies, asthmatics showed increased bronchial hyperreactivity and an increase in sputum levels of Interleukin 6 (IL-6, a TH2-type lymphocyte and inflammatory cytokine), after inhalation of DEE (Nordenhäll et al. 2001); no allergic mechanism is assumed here. A large number of studies are available regarding the modes of action of DEE on the respiratory immune system. DEE can stimulate the formation of other TH2 lymphocytes (IL-4, IL-5, IL-6 and IL-10) and immunoglobulin E (IgE) production, have an effect stimulating red blood cells and increase the expression of signalling molecules (referred to as chemokines) and the formation of oxidants by DEE. In addition, DEE has been demonstrated to act as an adjuvant[13] in sensitisation studies in various animal models and humans (Alexis and Carlsten 2014, DFG 2014, Finkelman 2014, Health Canada 2016a, Inoue and Takano 2011, Jung et al. 2021, Kim et al. 2011, Muñoz et al. 2019). However, DEE does not appear to be a sensitiser itself.

[13] An adjuvant is a substance that enhances the immune system's response to the presence of an antigen or foreign substance.

Reproductive effects

Multiple experimental animal studies have shown DEE exposure may result in histopathological changes in male and female reproductive systems, altered sperm morphology parameters, delayed sexual maturation and developmental neurotoxicity (Health Canada 2016a, Taxell and Santonen 2016, Ema et al. 2013), however the data are somewhat inconsistent.

5 Mechanisms of toxicity

IARC (2014) concluded that the mechanisms by which DEE induces cancer in humans are complex and that no single mechanism appears to predominate. Organic solvent and physiological fluid extracts of DEE particles and several of their individual components are genotoxic, and some are carcinogenic, generally through a mechanism that involves DNA mutation. These modifications include the formation of bulky DNA adducts and oxidised DNA bases. Both the organic and particulate components of DEE can generate oxidative stress through the formation of reactive oxygen species (ROS), which can be generated from washed particles, fresh particles, arene quinones formed by photochemical or enzymatic processes, metals and the phagocytosis process, and as a result of the inflammatory process (IARC 2014). ROS can lead directly to the formation of oxidatively modified DNA and DNA adducts from the by-products of lipid peroxidation. They can also cause lipid peroxidation, which generates cytotoxic aldehydes, and initiate a signaling cascade that leads to inflammation, resulting in further induction of oxidative stress, which can then cause cell proliferation and cancer.

5.1 Mutagenicity

It has been hypothesised that the inorganic and organic substances (nitro-PAH and PAH adducts) attached to the soot core of DPM are mainly responsible for the development of DEE-associated lung tumours in rodents (Cohen and Nikula 1999, DFG 2014). Nevertheless, the gaseous phase of DEE is also mutagenic to bacteria and contains a series of carcinogens including acetaldehyde, acrolein, benzene, 1,3-butadiene, formaldehyde, ethylene oxide, propylene oxide and naphthalene (IARC 2014, Taxell and Santonen 2016). Both the gaseous and particulate phase of DEE may contain carcinogenic PAHs and nitro-PAHs, whereas metals are likely only present in the particulate phase (IARC 2014).

In DPM, the fraction of PAHs only accounts for a small percentage of the total DPM mass. It is assumed that the metabolism of the substances attached to the DPM core and released in the organism leads to mutagenic metabolites (DFG 2014, Taxell and Santonen 2016). PAHs in DPM adhere strongly to the surface of particles. About 50% of the PAHs adsorbed onto diesel particles is cleared from the lung within one day, but the retention half-times for the remaining PAHs can be 18-36 days. Studies with 3 H-benzo[α]pyrene (BaP) and 14C-nitropyrene show that when PAHs are associated with particulate matter, their clearance from the lungs is significantly delayed in comparison with the clearance of inhaled gaseous PAHs not associated with particulate matter (Taxell and Santonen 2016, WHO 1996). However, it has also been suggested that since biological fluids are far less efficient at extracting potentially mutagenic organic compounds from DPM than organic solvents (which are typically used for mutagenicity assays), mutagenic chemicals are likely tightly bound to DPM and may not be bioactive *in vivo* (TEMA 2012).

Numerous experiments investigating the mutagenicity of DEE (and individual components) from both TDE and NTDE have been published. Earlier studies focused on whether various types of exhaust after-treatment technologies would attenuate the genotoxicity of TDE DEE. For example, Nylund et al. (2004) investigated the mutagenicity in the Ames assay of DEE from engines with no exhaust after-treatment (NT), an OC and a continuously regenerating particulate filter (CRT), the latter similar to the technology used in NTDE. Nylund et al. (2004) found mutagenicity to be similar for both NT and OC engines, but markedly lower in engines with a CRT. Hallberg et al. (2017) found that emissions from 2007-compliant diesel engines (NTDE) resulted in a lack of measurable DNA damage as measured in lung tissue, serum, and the hippocampus of rats from a chronic toxicology study.

In a human controlled exposure study (Rudell et al. 1999b) in which TDE exhaust air used in the chamber (with and without filtration) was tested for mutagenicity in the Ames assay: all DEE, with and without filter combinations ($PM_{0.8}$ = 110-290 µg/m^3, NO_2 = 0.3-0.7ppm), and with and without metabolic activation, had a significantly higher mutagenic effect than control air exposures ($PM_{0.8}$ <30 µg/m^3, NO_2 <0.02 ppm). The unfiltered DEE (at $PM_{0.8}$ = 290 µg/m^3, NO_2 = 0.7ppm) gave the highest mutagenic effect and all DEE samples gave a higher mutagenic effect in the presence of metabolic activation than in its absence (Rudell et al. 1999b). This does suggest lowering the DPM content of TDE exhaust lowers the mutagenic activity of the mixture, and in this study metabolic activation increased the mutagenicity of the mixture which may suggest PAHs to be a contributing factor to the observed effect.

DPM and their organic extracts from TDE have been shown to cause point mutations, chromosome aberrations, DNA damage and sister chromatid exchange *in vitro* in bacteria and mammalian cells (HCOTN 2019, IARC 2014, NRCWE 2018). Numerous nitroarenes, derivatives of PAHs, which are present in the condensate, have proved to be particularly genotoxic (DFG 1990, 2014; IARC 2014)[14]. Components of DEE are also genotoxic *in vivo* so that application of the particles from the exhaust fumes or of their organic extracts to various species leads to micronucleus formation, increased sister chromatid exchange and somatic mutations (NRCWE 2018). *In vivo* metabolites and DNA adducts, which are formed from DEE and the nitroarenes that they contain, are similar to those formed in bacteria and mammalian cells *in vitro*. Interestingly, positive responses were also found in the absence of metabolic activation, i.e. in some studies, mutagenicity of DEE appeared to be PAH-independent and potentially due to the gaseous fraction of DEE (US EPA 1981c, WHO 1996).

While the exact mechanism of DEE-induced carcinogenesis is not known, according to Budroe et al. (2012) it is entirely plausible that genotoxic carcinogens such as PAHs, benzene and 1,3-butadiene found in DEE contribute to that carcinogenic process. Thus, the available data do not indicate that NTDE exhaust should be considered to be fundamentally different in terms of the constituents that drive the potential risk of harm associated with exposure compared with that from older TDE (Budroe et al. 2012). Essentially, this means that the risk profile of the exhaust (regardless of whether it comes from NTDE or TDE) depends entirely on the exposure concentrations of the constituents of concern; from the information reviewed, it is considered implausible that NTDE exhaust contains novel constituents that somehow change the hazard profile compared to TDE exhaust. Most DEE aerosol mutagenicity appears associated with the particulate phase (to which mutagenic and carcinogenic PAHs are generally adsorbed), and lung tumour induction in laboratory rats is primarily associated with the particulate phase (Cantrell and Watts 1997).

Hence it follows that if one decreases the concentration of particulates in DEE then one would expect lower mutagenic activity. Indeed, recent chronic inhalation studies with DEE from a heavy-duty NTDE did not show local or systemic genotoxicity or oxidative DNA damage in rodents (HEI 2015a). This suggests that NTDE and after-treatment technologies may decrease the genotoxic potency of DEE, likely attributable to the significant reduction of particulate matter (and therefore adsorbed PAHs) in the exhaust (Taxell and Santonen 2016).

[14] Carcinogenicity may result from metabolism to the ultimate carcinogenic aryl nitrenium ion that binds covalently to DNA to form adducts (IARC 2014).

5.2 Inflammation

The most plausible mechanism of action for adverse respiratory effects is likely irritation of the respiratory tract, leading to an inflammatory response (Health Canada 2016a)[15]. Thus, if the inflammatory response from DEE exposure is prevented, further adverse pulmonary changes should also be prevented.

One hypothesised mechanism of action for the development of lung tumours in rodents after exposure to DEE is that the mechanism mainly consists of inflammatory proliferative effects of DPM on lung cells (DFG 2014, NTP 2021, Taxell and Santonen 2016). The soot core of DPM is a poorly soluble particle of low toxicity. The pulmonary toxicity typical of poorly soluble particles is characterised by an inflammatory proliferative and subsequently fibrotic effect. This process is caused by macrophages that are activated, after the particles have been absorbed, and secrete various cytokines and chemokines to an increased extent. These signalling proteins lead to the migration of inflammatory cells, which in turn produce oxygen and nitrogen radicals. The radicals cause damage and proliferation of the pulmonary epithelial tissue and pulmonary parenchymal tissue (DFG 2014, IARC 2014, Taxell and Santonen 2016). Lower particle loads do not produce lung cancer because they do not trigger compensatory inflammatory responses in the lung, i.e. the inflammatory response has an exposure threshold below which the effects are not initiated (IARC 2014).

It is now widely accepted that the tumourigenic effect of DEE in rats is likely an unspecific overload reaction to the large mass of particles deposited in the lungs, to which the rat is particularly predisposed (DFG 1990, 2014; IARC 2014, NTP 2021, TEMA 2012, US EPA 2002a). This is supported (DFG 1990, 2014) by the fact that:

i) no respiratory tract tumours were found in hamsters in three inhalation experiments with exposure conditions like those that had produced tumours in the rat; and

ii) it was not expected that a tumourigenic effect would be demonstrable for DEE because of the relatively low PAH content of the particulate fraction where the BaP concentration in the inhalation experiment was less than 50 ng/m^3. [16]

Additional support comes from the experiment by Heinrich et al. (1995) where female rats were exposed to carbon black, TiO_2, four concentrations of DPM, and particle-free DEE. The rats were exposed by whole body inhalation for up to 24 months, 18 h/day, 5 d/week. After the exposure period, the rats were then kept in clean air conditions for an additional 6 months. The exposure concentrations of DPM were 7,000 µg/m^3, 2,500 µg/m^3, and 800 µg/m^3, which coincide with 61.7 g/m^3 x h, 21.8 g/m^3 x h, and 7.4 g/m^3 x h (Heinrich et al. 1995). The concentration of carbon black and TiO_2 was 102.2 g/m^3 x h and 88.1 g/m^3 x h, respectively. At 20 months of exposure, the incidences of bronchioloalveolar hyperplasia were 98/100 for the high exposure concentration of

[15] The inflammatory effects of DEE are mediated by redox-sensitive mitogen-activated protein (MAP) kinase and nuclear factor kappa B (NF-κB) cascades, which are responsible for the production of inflammatory cytokines, chemokines and adhesion molecules (Taxell and Santonen 2016). Cytokines and chemokines, including tumour necrosis factor alpha (TNF-α) and interleukins (ILs), together with the adhesion molecules are involved in the recruitment and activation of inflammatory cells in the lungs. Activated leukocytes produce large quantities of ROS causing further oxidative damage to the surrounding cells. The inflammatory cascade also includes activation of phospholipase A2, leading to an increase in local vasodilation and vasopermeability to enhance the accumulation of inflammatory cells. Besides the inflammatory effects, oxidative stress inside a cell may also lead to cytotoxicity through mitochondrial release of pro-apoptotic factors (Taxell and Santonen 2016).

[16] In another inhalation experiment the emissions from pyrolysed pitch, which are rich in PAH but poor in particles, induced tumours in only 18% of the rats; the emissions contained about 90 µg BaP/m^3; the animals were exposed to this high concentration in the second year of life (DFG 1990, 2014).

DPM, 96/100 for carbon black, and 96/100 for TiO_2. The intensity and frequency of interstitial fibrosis increased with particle exposure concentration. No differences could be detected among the high DEE exposure, the carbon black, and the TiO_2 groups. Particle-laden macrophages and particles in the alveolar region were observed in the lungs of all exposed rats. The comparison showed that, regardless of the particle type used in this study, the lung tumor rate increased with increasing particle exposure concentration (NTP 1998).

This pulmonary overload mechanism is not considered to be relevant to general population exposures to DEE (Health Canada 2016a) but may be relevant for certain high occupational exposures where DEE is not well-controlled.

6 Concentration-response information to assist in WES derivation

6.1 Controlled human exposure studies

A summary of the controlled human exposure studies reviewed as part of this study is provided in Appendix D. The majority of controlled human exposure studies were conducted over a short time period of 1-2 hours exposure to DEE generated from TDE. Most of the DEE exposure concentrations were around 300 µg/m^3, which was either a no observed effect concentration (NOEC) or a lowest observed effect concentration (LOEC) in the various studies (Figure 6-1). At this level of DEE exposure, there are a few reports of irritation to the eyes, nose and throat or 'subjective symptoms' (Rudell et al. 1999b, Mudway et al. 2004, Wierzbicka et al. 2014), although it is unclear whether this irritation can be attributed to the particulate fraction of DEE (HCOTN 2019). For example, the irritation effects observed at 280 µg/m^3 in the study by Wierzbicka et al. (2014) are likely attributable to the aldehydes and/or NO_2 in the exhaust[17]. It is noted the aldehyde content of DEE in the other controlled exposure studies is not known. The NO_2 content, where reported, varied markedly between studies (0.02-1.6ppm). No clear relationship between NO_2 concentration in the DEE used in the acute controlled exposure studies and effects observed can be discerned from the available information.

The majority of studied effects involved investigating pulmonary inflammatory markers or effects on the cardiovascular system, likely due to the need for less invasive techniques than would be required for other systems (Landwehr et al. 2019). Some studies have shown that an initial airway inflammation response may occur as early as after 2 hours of exposure to DEE at 100-200 µg/m^3 (DFG 2014, Nightingale et al. 2000).

Three studies found indicative LOECs shown in Figure 6-1 that are lower than these concentrations. They are:

- Andersen et al. (2019) monitored individuals travelling inside diesel trains for 6 hours/day over 3 days and compared lung function and cardiovascular function parameters with people travelling on electric trains. They found small decreased lung function and increased DNA strand breaks in peripheral blood mononuclear cells (PBMC) at 10.3 µg/m^3 (as BC) compared to those travelling on electric trains. However, it is noted this study was the only controlled exposure study to report concentrations as BC and thus is not readily directly comparable to the other studies. In addition, the exposure in this study could not be blinded. The authors indicate the time-integrated exposure to PM in their study (216 µg*h/m^3 per day or 648 µg*h/m^3 per 3-day exposure period) is similar to other controlled exposure studies (300-900 µg*h/m^3).
- Glück et al. (2003) is a chronic exposure study rather than an acute controlled exposure study, where 194 non-smoking customs officers who regularly travel inside diesel fuelled trucks (8.4 hours/day, 42 hours/week, 5 years) showed signs of chronic inflammation of the nasal mucous membrane with no evidence of progression of these changes. Their DEE (as "diesel soot")[18] exposure concentrations ranged between 31 and 60 µg/m^3. It is noted chronic non-allergic rhinitis is normally considered adverse, because it can be associated with symptoms such as stuffy/runny nose, sneezing, mucus in the throat and cough, which can be debilitating if persistent. However, it is noted Glück et al. (2003) make no comment regarding whether the workers monitored in the study experienced any symptoms. The concentrations identified therefore are considered to represent a minimal LOAEC (i.e. a LOAEC of minimal severity) for this effect in this report.

[17] Concentrations of formaldehyde and acetaldehyde in this study were 400 and 200 µg/m^3, respectively (Wierzbicka et al. 2014) which are above their respective irritation thresholds (SWA 2019c, d).
[18] "Diesel soot" was not defined by Glück et al. (2003). Soot appears to be a term used in some of the literature consulted to refer to DPM.

- Nightingale et al. (2000) exposed 10 non-smoking healthy volunteers for 2 hours at rest to DPM at 200 µg/m^3 (measured as PM_{10}) or air in a double-blind, randomised crossover study. Study authors measured a number of lung and cardiovascular function parameters for up to 4 hours after exposure with repeat measurements at 24 hours. There were no changes in cardiovascular parameters or lung function; the only effect observed was an increase in sputum neutrophils and myeloperoxidase at 4 hours compared with controls, but no change in concentrations of inflammatory markers was observed. The effects observed were biochemical changes which are not adverse.

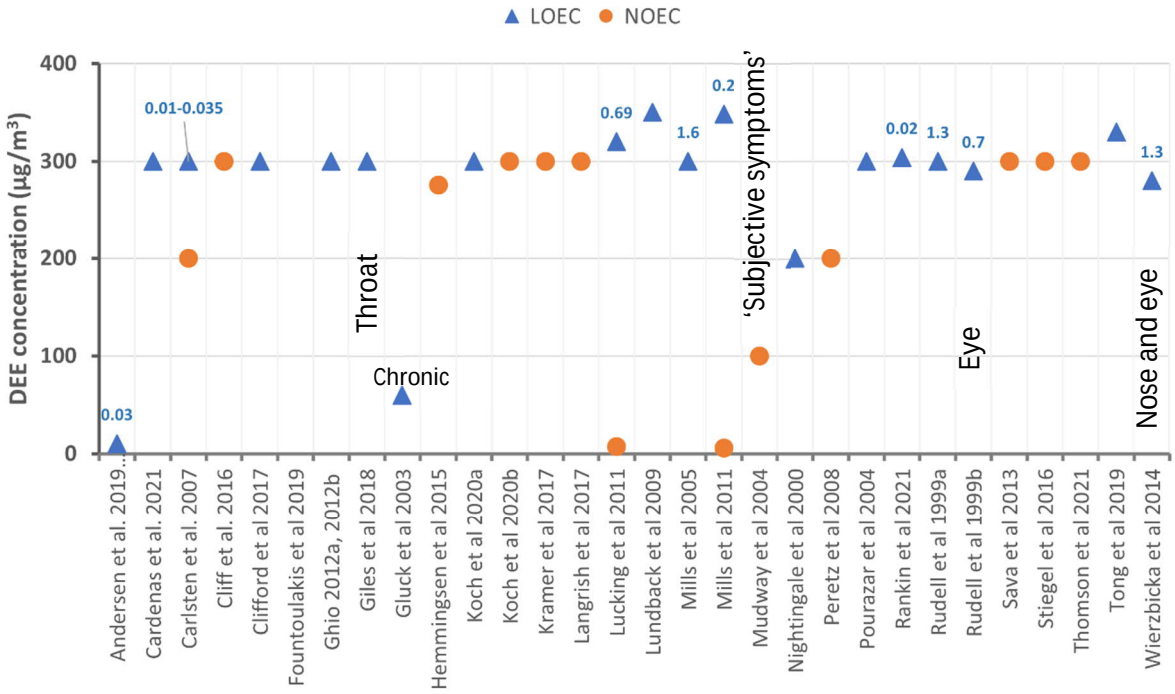
NOECs identified in the controlled exposure studies ranged from 6 to 300 µg/m^3; the NOECs at the lower end of the range are influenced by exposure concentration spacing. For example, Lucking et al. (2011) found that compared with filtered DEE (DPM = 7.2 µg/m^3), unfiltered DEE inhalation (DPM = 320 µg/m^3) was associated with decreased vasodilatation and increased *ex vivo* thrombus formation. The NOEC for these effects may in fact be higher than shown in Figure 6-1, but this is unable to be determined due to the exposure concentration spacing used.

After reviewing a large number of acute controlled exposure studies in humans, Ghio et al. (2012a, b) concluded that there appears to be a threshold concentration of DPM approximating 300 µg/m^3 (usually measured as $PM_{2.5}$) for pulmonary inflammation. Health Canada also reviewed available studies and based on the consistency of results, considered a LOEC of 100 µg/m^3 DPM was defensible as a point of departure (POD) for derivation of a short-term exposure guidance value (Health Canada 2016a).

Mills et al. (2011) is the only controlled human exposure study found conducted with NTDE (EU Stage V, EPA Tier 4). They generated unfiltered ($PM_{2.5}$ = 348 µg/m^3, NO_2 = 0.2 ppm) and filtered ($PM_{2.5}$ = 6 µg/m^3, NO_2 = 0.2 ppm) exhaust and exposed healthy non-smokers to the DEE for 2 hours and monitored cardiovascular parameters. Inhalation of unfiltered and filtered DEE increased systolic blood pressure, but only unfiltered DEE attenuated vasodilation to three vasodilators. This indicates that particulate filtration in NTDE may attenuate adverse cardiovascular effects, and at least some of these effects appear to be due to the particulate component of DEE since NO_2 concentrations in both treatments were the same.

The available human controlled exposure studies indicate the following PODs could tentatively be used together with other relevant information for derivation of a candidate WES:

- an acute NOEC of 100 µg/m^3 DEE (measured as $PM_{2.5}$) for airway inflammation (Mudway et al. 2004); and
- a chronic minimal LOAEC of 31-60 µg/m^3 as diesel soot (presumed to be REC, no further details provided) for chronic rhinitis (Glück et al. 2003).



NO_2 concentrations provided in bold blue text. LOEC = lowest observed effect concentration, NOEC = no observed effect concentration

Figure 6-1 Summary of DEE NOECs and LOECs with corresponding NO_2 concentrations where they were available from (mostly) acute controlled human exposure studies with DEE (see Appendix D for study details)

6.2 Epidemiological studies

6.2.1 Studies with exposure-response information

Appendix B provides a summary of epidemiological information found as part of the literature review undertaken for this report. As indicated previously, not every epidemiological study is included in Appendix B. The focus was on identifying those published subsequent to most of the major agency reviews. Of the 54 studies scanned, most (if not all) of the occupational epidemiology studies evaluated exposure to TDE exhaust. Many occupational exposure studies focus on historical data obtained before 2000, when sulfur levels in fuel were high (>500 ppm and in some cases >5000 ppm) and diesel engines were not equipped with exhaust after-treatment devices (see Appendix B). Only four studies (Garschick et al. 2012, Möhner et al. 2013, Silverman et al. 2012, Steenland et al. 1998)[19] included exposure measurements and/or complex retrospective estimates of exposure[20]. In a further three studies that found significant associations of DEE exposure with lung cancer risk, there was no clear exposure-response relationship (e.g. Ahlberg et al. 1981, Hansen 1993, Menck and Henderson 1976). A summary of the principal lung cancer mortality findings[21] from each of these studies is provided in Table 6-1.

[19] Garschick et al. (2012) and Steenland et al. (1998): trucking industry; Silverman et al. (2012): non-metal mining industry; Möhner et al. (2013): potash/salt mining industry.

Table 6-1 Summary of lung cancer outcomes from epidemiological studies with DEE that have included exposure measurements and/or complex retrospective estimates of exposure

Study	Group	REC exposure concentration (µg/m³ or µg/m³.yr) (95% CI) [1]		Lung cancer mortality (95% CI)	
Attfield et al. 2012 - miners [2]	Whole cohort	87.0 (85.2-88.8) µg/m³		SMR = 1.26 (1.09-1.44)	
	Ever-underground workers (15 yr lagged excluding <5 tenure)	0-<20 µg/m³.yr (ref)	0-<2 µg/m³	HR = 1.00 (ref)	1.00 (ref)
		20-<40 µg/m³.yr	2-<4 µg/m³	HR = 1.39 (0.36-5.39)	0.93 (0.19-4.49)
		40-<80 µg/m³.yr	4-<8 µg/m³	HR = 0.82 (0.17-4.03)	1.00 (0.31-3.18)
		80-<160 µg/m³.yr	8-<16 µg/m³	HR = 2.69 (0.99-7.37)	1.79 (0.65-4.92)
		160-<320 µg/m³.yr	16-<32 µg/m³	HR = 2.67 (0.98-7.27)	2.01 (0.74-5.50)
		320-<640 µg/m³.yr	32-<64 µg/m³	HR = 2.21 (0.82-5.97)	3.20 (1.36-7.51)
		640-<1280 µg/m³.yr	64-<128 µg/m³	HR = 5.01 (1.97-12.76)	2.11 (0.81-5.48)
		≥1280 µg/m³.yr	≥128 µg/m³	HR = 2.39 (0.82-6.94)	3.01 (1.20-7.71)
Silverman et al. 2012 - miners [2]	Whole cohort, lagged 15 yrs	0-<3 µg/m³.yr	0-<1 µg/m³	OR = 1.0 (ref)	1.0 (ref)
		3-<72 µg/m³.yr	1-<6 µg/m³	OR = 0.74 (0.4-1.38)	1.11 (0.57-2.07)
		72-<536 µg/m³.yr	6-<57 µg/m³	OR = 1.54 (0.74-3.2)	1.90 (0.9-3.99)
		≥536 µg/m³.yr	≥57 µg/m³	2.82 (1.28-6.26)	2.28 (1.07-4.87)
Steenland et al. 1998 - truckers	No lag	0-174 µg/m³.yr (as EC)		OR = 1.2 (0.79-1.81)	
		174-268 µg/m³.yr (as EC)		OR = 1.16 (0.77-1.75)	
		268-360 µg/m³.yr (as EC)		OR = 1.39 (0.91-2.11)	
		>360 µg/m³.yr (as EC)		OR = 1.72 (1.11-2.64)	
	5-year lag	0-169 µg/m³.yr (as EC)		OR = 1.08 (0.72-1.62)	
		169-257 µg/m³.yr (as EC)		OR = 1.10 (0.74-1.65)	
		257-331 µg/m³.yr (as EC)		OR = 1.36 (0.90-2.04)	
		>331 µg/m³.yr (as EC)		OR = 1.64 (1.09-2.49)	
Garschick et al. 2012 - truckers		<371 µg/m³.mth (EC)	<3.6 µg/m³	HR = 1.00 (ref)	1.00 (ref)
		371-<860 µg/m³.mth (as EC)	3.6-<5.4 µg/m³	HR = 1.18 (0.92-1.51)	1.15 (0.93-1.43)

[20] Occupational cohort and case-control studies for workers mainly exposed to DEE included studies with non-metal/potash miners, trucking industry workers, truck and bus drivers, transport maintenance workers, railroad workers, dock workers, and heavy equipment workers. Most of these studies estimated exposure in a qualitative way by job title and years of work. In a few studies semi-quantitative and quantitative exposure levels in the past were estimated using job exposure matrices, current exposure levels (elemental or total carbon), or available emission data. Potential confounding in part of the studies included smoking habits, and exposure (in the past or present) to various other potential carcinogenic substances than the substances known to be present in DEE (HCOTN 2019).

[21] The findings for lung cancer mortality (or incidence) were considered to be the most relevant for consideration of exposure-response relationships since the epidemiological evidence for this effect is strongest (see also Section 4.2).

Study	Group	REC exposure concentration (µg/m^3 or µg/m^3.yr) (95% CI) [1]		Lung cancer mortality (95% CI)	
	Whole cohort, 5-year lag (unadjusted for duration of work)	860-<1803 µg/m^3.mth (as EC)	5.4-<7.9 µg/m^3	HR = 1.16 (0.88-1.53)	1.11 (0.89-1.39)
		≥1803 µg/m^3.mth (EC)	≥7.9 µg/m^3	HR = 1.12 (0.83-1.52)	1.06 (0.84-1.34)
Möhner et al. 2013 – potash miners	Whole cohort, assuming log-linear exposure-response	Per 1,000 µg/m^3.yr		OR = 1.04 (0.70-1.53)	
	Whole cohort, conditional logistic regression	<983 µg/m^3.yr [mean = 624 µg/m^3.yr]		OR = 1.00 (ref)	
		983-1550 µg/m^3.yr [mean = 1279 µg/m^3.yr]		OR = 1.77 (0.85-3.69)	
		>1550 µg/m^3.yr [mean = 2375 µg/m^3.yr]		OR = 1.04 (0.47-2.27)	

CI = confidence interval. SMR = standardised mortality ratio. yr = year. HR = hazard ratio. ref = referent group. OR = odds ratio. REC = respirable elemental carbon. EC = elemental carbon. Bolded values indicate findings that are statistically significantly different from the referent group.

1. This is reported as an average exposure concentration or as a cumulative exposure concentration for a particular worker subgroup.
2. Used data from DEMS study.

One of the largest studies, which has also been subject to numerous critiques and reanalysis, is the Diesel Exhaust in Miners Study (DEMS) study[22]. The original study included 12,315 workers exposed to DEE at eight US non-metal mining facilities. The DEMS study was originally initiated in part due to the many other studies that had been conducted at that time that had found an association between DEE and lung cancer but had made no quantitative exposure estimates. Retrospective exposure estimates of the DEMS data were derived for all surface and underground jobs, by year and facility, from year of introduction of diesel-powered equipment in the facility (1947-1967) to December 31, 1997 (Attfield et al. 2012, Silverman et al. 2012). Thus, data gathered as part of the original DEMS study concluded at the end of 1997, with the best association between lung cancer and exposure found for exposures that lagged 15 years. This means that the most recent engine that the DEMS study would have potentially included was from 1983 and therefore can be classed as TDE. No measurements of REC were available during the time of the study. Therefore, to obtain retrospective REC exposure estimates REC measurements from personal samplers collected during the 1998-2001 DEMS surveys, at seven of the eight study facilities originally used (the eighth facility had closed in 1993) were retrospectively extrapolated. This was done using the relationship between EC and carbon monoxide (CO)[23] in contemporary measurements, estimates of diesel emission changes over time, and job exposure matrices. This allowed the construction of retrospective EC exposure concentrations (Vermeulen et al. 2010a, b).

[22] Different aspects of the study, along with numerous reanalyses of the data from the study, have been published in several scientific articles (Attfield et al. 2012, Coble et al. 2010, Costello et al. 2016, 2018; Crump and van Landingham 2012, Crump 2014, Crump et al. 2015, 2016; Ferguson et al. 2020, Morfeld and Spallek 2015, Neophytou et al. 2016, Silverman et al. 2012, Stewart et al. 2010, 2012; Vermeulen et al. 2010a, 2010b, 2014, 2020). The results of these various publications are summarised in this Section and also in Appendix B.

[23] TEMA (2012) indicated the core assumptions for the exposure assessment methodology that serve as the foundation for the DEMS study are that: (i) CO and PM emissions from different diesel engines correlate sufficiently well; (ii) historical CO emissions correlate sufficiently well with and can be estimated based upon aggregate engine horsepower; and (iii) the overall correlation of CO and PM emissions from different diesel engines is sufficiently proportional and linear to allow for

As shown in Table 6-1, a significant increase in the standardised mortality ratio (SMR) for lung cancer in the whole cohort was obtained: 1.26 (95% CI, 1.09-1.44) (Attfield et al. 2012). When considering hazard ratios (HRs) for lung cancer mortality in 'ever-underground' workers (who had higher DEE exposures), HRs increased with increasing cumulative REC exposure to a maximum HR of 5.01 (95% CI = 1.97-12.76) in the 640 to <1280 μg/m^3.yr category compared with the reference category (0 to <20 μg/m^3.yr). Interestingly as shown in Table 6-1, the HR declined at higher cumulative exposures. Average REC intensity HRs rose to a plateau around 32 μg/m^3, where it became statistically significant (p=0.008), but then declined in the 64-<128 μg/m^3 group and rose again in the ≥128 μg/m^3 group (Attfield et al. 2012; see also Table 6-1).

This apparent non-monotonic exposure-response relationship is unusual for the presumed mechanisms of toxicity of DEE (see Section 5). Silverman et al. (2012) describes a nested case-control study of the DEMS population. The study authors found statistically significant increasing trends in lung cancer risk with increasing cumulative REC and average REC intensity. Cumulative REC, lagged 15 years, yielded a statistically significant positive gradient in lung cancer risk overall (P_{trend} = .001). The odds ratio (OR) for lung cancer with workers in the top quartile (i.e. ≥536 μg/m^3.yrs) was 2.83 (1.28-6.26) compared with the reference category of workers in the lowest quartile (0-<3 μg/m^3.yrs) (see Table 6-1). There was an interaction observed between smoking and 15-year lagged cumulative REC ($P_{interaction}$ = 0.086) such that the effect of each of these exposures was attenuated in the presence of high levels of the other. Estimates of DEE exposure in this study undoubtedly had some imprecision despite effort to minimise misclassification. It is noted the exposure-response in this study was not entirely clear. An increase in risk was shown at low-to-moderate exposure levels followed by a plateau / potential decline at high levels.

Steenland et al. (1998) conducted exposure-response analyses among workers in the trucking industry (original study published by Steenland et al. 1990 was case-control) (994 lung cancer cases, 1085 controls who died in 1982-1983), and adjusted for smoking. Past exposures were estimated as a function of the number of heavy-duty trucks on road, particulate emissions of diesel engines over time, and leaks from trucks' exhaust systems. Regardless of the assumptions about past exposure, all analyses resulted in significant positive trends in lung cancer risk with increasing cumulative exposure. As shown in Table 6-1, the ORs for the highest exposure quartile (cumulative REC >331 or >360 μg/m^3.yrs) were significantly different from others. The study authors noted their results depend on very broad assumptions on exposure estimates with extrapolations to earlier time periods and that it is unknown whether drivers were also exposed to leaky emissions from their own trucks. IOM (2015) considered the exposure estimates from Steenland et al. (1998) to be much less reliable than for another trucker study (Garschick et al. 2012, described below), and very likely to be strongly positively biased. IOM (2015) did not consider the Steenland et al. (1998) study suitable to define an exposure-response relationship. TEMA (2012) pointed out that similar elevations in lung cancer incidence in truck drivers have been found prior to dieselisation and that this suggests that potentially lifestyle factors or an unidentified occupational agent other than DEE might be responsible for the low elevations in relative risk reported in the trucking industry studies.

Garschick et al. (2012) conducted a retrospective cohort study to assess lung cancer mortality risk among 31,135 male US trucking industry workers. A statistical model was used by the study authors to estimate historical work-related exposures to EC, which resulted in a wide range of cumulative EC exposures (50th percentile was 1,061 μg/m^3.mths for entire cohort). Duration of employment was found to be inversely associated with lung cancer risk (consistent with a healthy worker survivor effect). The authors found a suggestive linear-response relationship for each 1000μg/m^3.mths cumulative EC based on a 5-yr lag, the HR was 1.07 (0.99-1.15). Average EC exposure was not associated with a significant relative risk (see Table 6-1). As the 95% confidence interval overlaps 1, this association can be considered suggestive in this study, but was not statistically significant.

1:1 scaling over the years of the study. They present arguments in their report that none of these assumptions is necessarily correct (TEMA 2012).

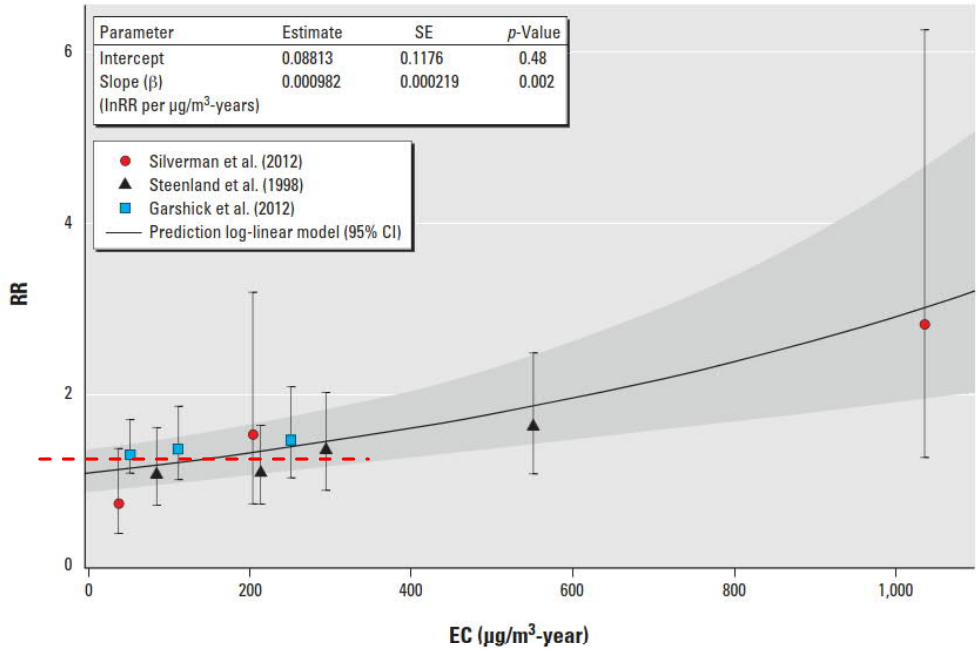
Möhner et al. (2013) reanalysed lung cancer risk associated with occupational exposure to DEE in 5,819 potash miners (employed for at least one year after 1969), while controlling for potential confounders (e.g. smoking, previous occupational history). TC and EC measurements from 1991 were used for designing a job-exposure matrix. Estimated cumulative mean REC exposure differed for each decade ranging from 672 µg/m^3.yrs (in <1920) to 1,957 µg/m^3.yrs (in 1940-1949). The authors' analysis did not show any notable association between cumulative REC exposure and lung cancer risk. Introducing cumulative REC exposure as a continuous variable into the conditional logistic regression model yielded an OR of 1.04 [95% CI: 0.70–1.53], adjusted for smoking and previous employment. The authors concluded the study results give no evidence for an association between REC exposure and lung cancer risk. The study authors noted the exposure transformation of 1991 measured data may have increased the chance of exposure misclassification. Lung cancer cases in this study were low (n=61), which resulted in wide confidence intervals.

6.2.2 Meta-analyses and reanalyses of exposure-response information

Vermeulen et al. (2014) derived a meta exposure-response curve for DEE and lung cancer mortality using data from the US DEMS study and the US occupational cohorts from Steenland et al. (1998) and Garschick et al. (2012). The authors excluded the study by Möhner et al. (2013) from the analysis because mean cumulative EC exposure in the reference exposure category (624 µg/m^3.yrs) was higher than almost all non-reference exposures of the other studies and there was a lack of detail on the derivation of the EC exposure metric. The authors used as relative risk (RR) the ORs for cumulative EC exposure categories with a 5-year lag from Steenland et al. (1998), the HRs for cumulative EC exposure categories with a 5-year lag (excluding the mechanics) in the Garshick et al. (2012) study, and the ORs for cumulative EC exposure with a 15-year lag from Silverman et al. (2012).

From the three studies, 10 study-specific categorical RR estimates for lung cancer mortality were extracted, covering a cumulative exposure range from 37 to 1,036 µg REC/m^3.yrs. Figure 6-2 shows the predicted exposure-response curve from Vermeulen et al. (2014) based on a log-linear model. The dark grey shaded area represents the 95% confidence interval estimates. Combining the three studies, a pooled slope (β) factor of the exposure-response curve was estimated by Vermeulen et al. (2014) of 0.00098 (natural logarithm of the RR (lnRR) for a 1 µg/m^3.year increase in REC; 95% CI, 0.00055 – 0.00141). Background exposure was taken into account for the lowest exposure groups, i.e. the intercept approached the background levels. It is noted that formal tests of heterogeneity were not undertaken as the authors indicated them to be of limited value.

The approach of extrapolating risk from a slope to exposures lower than those observed in the occupational studies is not without uncertainty, especially bearing in mind the considerable uncertainties in any retrospective exposure assessment. It is also noted the estimates included in the model differed regarding exposure lag (5 or 15 yr). Visually, from Figure 6-2 the confidence intervals are wide and if the top two data points are excluded, the regression appears to be almost a horizontal straight line (see red dotted line in figure drawn in by SLR). In addition, the 95% confidence intervals from almost all estimates in the figure encompass a RR of 1, indicating no significant association with cumulative EC.



SE = Standard Error; 95% CI = 95% confidence interval; RR = Relative Risk
Red dotted line was drawn in by SLR to visually demonstrate the apparent lack of a marked exposure-response relationship in the lower cumulative REC data points included in the meta-analysis.

Figure 6-2 Predicted 'pooled' log-linear exposure-response curve of cumulative REC and relative risk of lung cancer mortality, adapted from Vermeulen et al. (2014)

Following the publication of the DEMS study and Vermeulen et al. (2014) study, a number of reanalyses, critiques and responses to critiques ensued. These are summarised in Appendix F, and principal findings from each summarised in Table 6-2 below.

Table 6-2 Summary of reanalyses, critiques and reviews on quantitative DEE exposure and lung cancer risk published since Vermeulen et al. (2014) paper

Study reference	Group description	REC exposure concentration (µg/m³ or µg/m³.yr) (95% CI) or comment if not provided [1]	Lung cancer mortality risk outcome (95% CI)
Vermeulen et al. 2014 [3]	Meta-analysis, log-linear model: Steenland et al. 1998 (5-yr lag), Garschick et al. 2012 (5-yr lag), Silverman et al. 2012 (15-yr lag)	Pooled slope (ß) = 0.00098 per 1 µg/m³.yr REC (0.00055-0.00141)	
Crump 2014 [1]	Reanalysis of Vermeulen et al. 2014 using all 5-yr lags, log-regression model	RR = 0.38 (-0.03-0.96)	
Vermeulen et al. 2014b	Response to Crump 2014: sensitivity analysis including different lags	Pooled slope (ß) = 0.00065 per 1 µg/m³.yr REC (0.00028-0.001)	
Crump et al. 2015		DEMS_REC1: 383.5-<902.7 µg/m³.yrs	OR = 2.73 (1.08-6.88)
		DEMS_REC1: ≥902.7 µg/m³.yrs	OR = 5.04 (1.77-14.30)

Study reference	Group description	REC exposure concentration (µg/m^3 or µg/m^3.yr) (95% CI) or comment if not provided [1]	Lung cancer mortality risk outcome (95% CI)
	Reanalysis of DEMS data using conditional logistic regression adjusted for smoking and radon exposure. The paper provides a large number of ORs. Only statistically significant ORs with an apparent exposure-response are presented in this table for 'ever underground' workers with cumulative REC lagged 15 yrs	DEMS_REC2: 318.2-<782 µg/m^3.yrs	OR = 4.21 (1.52-11.71)
		DEMS_REC2: ≥782 µg/m^3.yrs	OR = 4.19 (1.43-12.26)
		DEMS_REC3: 157.1-<521.7 µg/m^3.yrs	OR = 2.72 (1.16-6.37)
		DEMS_REC3: 521.7-<957.4 µg/m^3.yrs	OR = 3.21 (1.15-8.98)
		DEMS_REC3: ≥957.4 µg/m^3.yrs	OR = 4.67 (1.61-13.57)
		REC1: 649.2-<1287.8 µg/m^3.yrs	OR = 3.96 (1.45-10.80)
		REC1: ≥1287.8 µg/m^3.yrs	OR = 2.58 (0.81-8.25)
		REC2: 556.5-<1101.4 µg/m^3.yrs	OR = 4.13 (1.43-11.93)
		REC2: ≥1101.4 µg/m^3.yrs	OR = 3.07 (1.07-8.84)
		REC3: ≥693.9 µg/m^3.yrs	OR = 4.26 (1.3-13.95)
		REC4: 329.8-<964.2 µg/m^3.yrs	OR = 3.64 (1.3-10.18)
		REC4: ≥964.2 µg/m^3.yrs	OR = 3.92 (1.22-12.56)
		REC5: 231.4-<711.9 µg/m^3.yrs	OR = 2.73 (1.14-6.58)
		REC5: 711.9-<1241.4 µg/m^3.yrs	OR = 2.97 (1.09-8.12)
		REC5: ≥1241.4 µg/m^3.yrs	OR = 3.55 (1.24-10.14)
		REC6: 204.9-<717 µg/m^3.yrs	OR = 3.22 (1.21-8.54)
		REC6: ≥717 µg/m^3.yrs	OR = 4.50 (1.42-14.24)
Crump et al. 2016	Further reanalysis of DEMS data (Silverman et al. 2012), using newly developed estimates of REC exposures revising diesel use & ventilation rates. Applied conditional logistic regression. Results presented for HP-CFM REC estimates "with radon" controls.	0-<6.6 µg/m^3.yrs (ref)	OR = 1.0 (ref)
		6.6-<129 µg/m^3.yrs	OR = 1.02 (0.55-1.90)
		129-<891 µg/m^3.yrs	OR = 1.20 (0.56-2.56)
		≥891 µg/m^3.yrs	OR = 1.37 (0.5-3.77)
Möhner 2016	Reanalysis of DEMS data (Silverman et al. 2012), unconditional logistic regression, result for 'ever underground' miners, adjusted for smoking.	OR = 0.92 (0.66-1.30)	
Morfeld and Spallek 2015	Reanalysis of data used by Vermeulen et al. 2014 using different modelling approaches and explored impact of various variables.	Of all meta-analyses, Vermeulen et al. (2014) gave highest risk estimates. Authors express uncertainty with using these estimates in quantitative risk assessment. [2]	

Study reference	Group description	REC exposure concentration (µg/m^3 or µg/m^3.yr) (95% CI) or comment if not provided (1)	Lung cancer mortality risk outcome (95% CI)
Vermeulen et al. 2020	Reanalysis of Vermeulen et al. 2014 to address two critiques (use of historical CO measurements to calibrate exposure model & potential confounding by radon).	Effect estimates for lung cancer using these alternative REC exposures or adjusting for radon typically changed by <10% when compared with the original estimates.	
Möhner and Wendt 2017	Review of available epidemiological information to provide exposure-response relationship.	Concluded an upper bound for cumulative REC exposure of 2.5 mg/m^3.yrs seems sufficient to prevent detectable lung cancer risk. This value corresponds to an average cumulative exposure value of 50 µg/m^3 for a 45-yr working lifetime.	
Sun et al. 2014	Critical evaluation of 42 cohort & 32 case-control studies (1970-2013).	Concluded that epidemiological studies published to date do not allow a valid quantification of the association between DEE and lung cancer, although such an association cannot be ruled out	

CI = confidence interval. REC = respirable elemental carbon. RR = relative risk. OR = odds ratio. Bolded values indicate findings that are statistically significantly different from the referent group.

1. It is noted the lower confidence interval for the RR is negative, which seems unusual. According to Crump (2014), "*There are other limitations of the analysis by Vermeulen et al. (2014): Garshick et al. (2012) employed a second measure of diesel exposure (exposure duration), which Vermeulen et al. did not account for in the analysis; and Vermeulen et al. used very crude exposure summaries (e.g., midpoints of exposure intervals)".*
2. Morfeld and Spallek (2015) concluded "*....the present re-analysis also revealed that the results of the meta-regression study by Vermeulen et al. [1] should not be used in any quantitative lung cancer risk evaluation without reservations, as the results vary significantly depending on the input data selected and the statistical methods used. This is particularly true for the low exposure region."*
3. Vermeulen et al. (2014) paper also included in this table for completeness.

It is clear from Table 6-2 and the discussion presented in Appendix F that there is considerable debate in the scientific community as to the strengths and utility of the exposure-response relationships for lung cancer risk from occupational epidemiological studies with DEE. Qualitatively, the studies do support a statistically significant association between DEE exposure and lung cancer risk. However, from a quantitative assessment perspective, the extrapolation to low exposures (lower than those in the reference groups) and apparent non-monotonicity of the relationship (which is not supported by the hypothesised mechanisms for DEE-induced lung cancer)[24] arguably limits the utility of the information for deriving a WES. It is also somewhat concerning that various reanalyses of the same dataset have resulted in vastly different quantitative estimates, highlighting the uncertainties inherent in any retrospective exposure assessment and application of statistical models of best fit. In addition, when using the exposure metric of cumulative REC to inform an average WES, it is obviously dependent on the exposure duration of each of the individual workers in the various cohorts. For example, a cumulative REC exposure of 500 µg/m^3.yrs could be represented by 40 years of exposure at an average REC concentration of 12.5 µg/m^3 but could just as well be represented by 10 years of exposure at a concentration of 50 µg/m^3.

It is therefore concluded that the occupational epidemiological information on its own is not considered to be sufficient for quantitative use in derivation of a WES. It has nevertheless been considered by putting the candidate WES values into context through comparison with cumulative REC exposures that were found to be associated with a significantly increased risk of lung cancer (see Section 7.3.4).

[24] Alternatively there may be other (unknown) confounders which could be influencing the risk estimates.

6.3 Experimental animal studies

6.3.1 Studies best suited for DPM WES derivation

Naturally, a large number of animal experiments have been identified in the reviews and literature consulted (see also Appendix C). Many only applied single exposure concentrations and monitored for subtle biochemical changes which may provide insight into mechanistic considerations but are not applicable for use in derivation of a WES. Exposure concentrations of the DEE applied in experimental animal studies ranged widely from tens (when filtered) to thousands µg DPM/m^3 (when unfiltered) and typically 0.1 to approximately 5 ppm NO_2. Very few studies monitored for other constituents such as aldehydes.

The primary target organ for the non-carcinogenic effects of DEE is the lungs. The most sensitive endpoint of an adverse effect after chronic inhalation of DEE is considered to be the inflammatory proliferative response in the rat both with increased number of inflammatory cells and specific enzymes and cytokines in the bronchioalveolar lavage as well as with hyperplastic changes in the alveolar epithelium (BauA 2017). Since rats were found to be the most sensitive experimental animal species[25] to DEE inflammatory, proliferative and consequent neoplastic lung changes, the inhalation studies in which rats were exposed to various concentrations of DPM over a long period of time (i.e. 2 years), are considered the most informative for derivation of a WES.

The most relevant chronic no observed adverse effect concentrations (NOAECs) from experimental animal studies with TDE exhaust include those summarised in Table 6-3. Based on the similarity of the exposure timeframe in the Mauderly et al. (1987) and Henderson et al. (1988) study to that potentially experienced in an occupational exposure scenario, this study is likely better suited to deriving a WES than the study by Ishihara and Kagawa (2003) and Ishinishi et al. (1988, cited in US EPA IRIS 2003).

Table 6-3 Chronic inhalation experimental animal studies most suited for DPM WES derivation

Study	Diesel engine / exhaust type	Exposure duration / test animal	Exposure concentrations (µg/m^3) [NO_2 in ppm]	Effects	NOAEC (LOAEC)
Ishihara and Kagawa 2003	TDE	2-yr (16h/d, 6d/week), Wistar rats	10, 200, 1100, 2800 [1.1, 0.2, 1.03, 2.96ppm]	Variable ↑ in macrophages, leukocytes, lymphocytes & phospholipids.	200 µg/m^3 (1100 µg/m^3) (1)
Mauderly et al. 1987, Henderson et al. 1988	TDE	2-yr (7h/d, 5d/week), F344 rats	350, 3500, 7000 [0.1, 0.3, 0.7ppm]	Chronic inflammatory changes in the lungs	350 µg/m^3 (3500 µg/m^3) (2)

[25] Mice, rats, hamsters, cats, and monkeys generally did not exhibit drastic decreases in body weight or reduced survival times after long-term inhalation of DPM up to 4,000 µg/m^3. Dose-related toxic effects seen in all species after long-term inhalation of DEE were: increases up to 400% in lung weight; pulmonary inflammation measurable by biochemical (cytoplasmic marker enzymes, collagen) and cytological (increase in polymorphonuclear neutrophils) parameters; impairment of lung mechanics; increasing numbers of particle-laden macrophages with focal accumulations (sequestration) under overload conditions; and subsequent proliferative alterations of epithelial cells and onset of fibrosis (WHO 1996).

Study	Diesel engine / exhaust type	Exposure duration / test animal	Exposure concentrations (µg/m^3) [NO_2 in ppm]	Effects	NOAEC (LOAEC)
Ishinishi et al. 1988, as cited in US EPA IRIS 2003 (3)	TDE	2.5-yr (16h/d, 6d/week)	110, 410, 1180, 2320 (LD) OR 460, 960, 1840, 3720 (HD)	Minor bw changes (4), equivocal alterations in liver & kidney function. Respiratory morphology changes at >460µg/m^3	410 (LD) 460 (HD)

bw = body weight. LD = light-duty. HD = heavy-duty. Blue shading in table indicates NOAEC selected for derivation of candidate WES for DPM using experimental animal studies (see Section 7.3.2).

1. Kato et al. (2000) describe the histopathological findings from the study by Ishihara and Kagawa (2003). In the experimental group exposed to the lowest concentration of DPM, histological changes were focal and found scattered, essentially classified as slight. They included bronchiolisations in the transition area (alveolar duct) of the airways to the alveolus and proliferation of type II cells in the alveoli, in which increased macrophages had accumulated and infiltration of the interstitium in the alveolar area with macrophages, mast cells, lymphocytes. The authors also assessed the morphological changes in the highest dose group as mild. BauA (2017) considered the morphological changes of the lowest dose group can still be regarded on the border for adversity and the DPM concentration of 200 µg/m³ diesel soot can be considered as a NOAEC.
2. Only at the lowest particle concentration of 350 µg/m³ were there no biochemical, cytological and morphological signs of an inflammatory response, except for elevation of beta-glucuronidase in bronchioalveolar lavage (BAL) after 18 months but not after 24 months. Alveolar particle clearance was not impaired compared to the control animals after 24 months. The longer daily exposure timeframe in the Ishihara and Kagawa (2003) study likely explains the differences in the NOAECs observed between that study and the Mauderly et al. (1987) and Henderson et al. (1988) studies. In addition, the NO_2 concentration in the Mauderly et al. (1987) study (0.1 ppm) was half that in Ishihara and Kato (2003). The NOAEC of 350 µg/m³ from the Mauderly et al. (1987) and Henderson et al. (1988) studies was selected for derivation of a WES for DPM using experimental animal studies (see Section 7.3.2).
3. The reference cited in US EPA IRIS (2003) is as follows: Ishinishi, N., Kuwabara, N, Takaki, Y, et al. (1988) Long-term inhalation experiments on diesel exhaust. In: Diesel exhaust and health risks: Results of the HERP studies. Ibaraki, Japan: Japan Automobile Research Institute, Inc., Research Committee for HERP Studies; pp. 11-84. Attempts were made to source the original reference, but it could not be located online.
4. The body weight of rats exposed to 2320 µg DPM/m³ was 15-20% less than controls throughout the study. While no histopathological changes were observed in the lungs of rats exposed to 460 µg/m³ DPM or less, at higher concentrations, severe morphological changes were observed, including shortened and absent cilia in the tracheal and bronchial epithelium, marked hyperplasia of the bronchiolar epithelium, and swelling of the Type II cellular epithelium. There was no difference in the degree of changes in pulmonary pathology at similar exposure concentrations between the LD and the HD series. No NO_2 concentrations were reported by US EPA IRIS (2003).

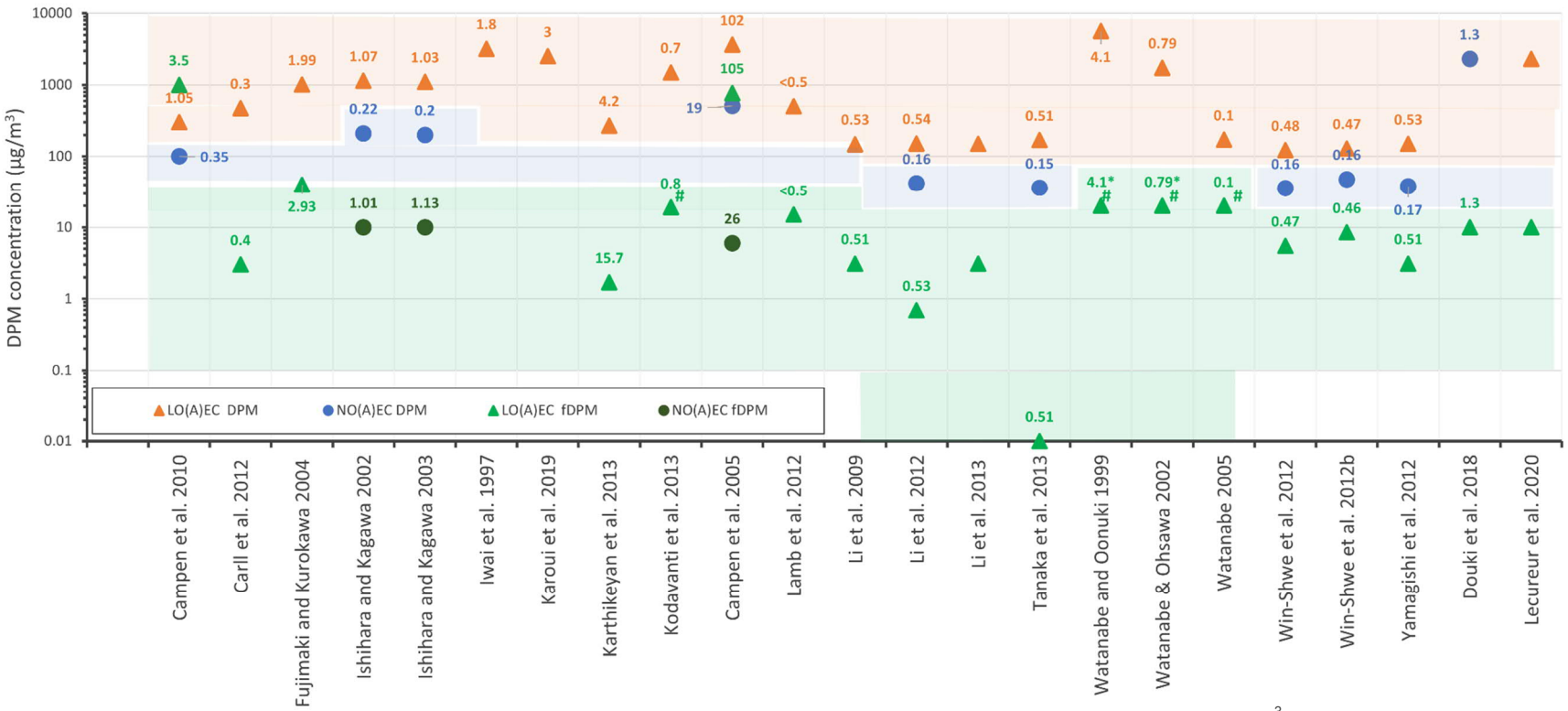
6.3.2 Studies useful for comparing TDE and NTDE exhaust toxicity

In a long-term (130 weeks) inhalation study in rats applying exhaust from a NTDE (particulates were in ultrafine range 20-40nm in diameter), mild alveolar and bronchial epithelial hyperplasia, mild fibrotic lesions, and a mild progressive decrease in pulmonary function mainly in the smallest airways consistent with the morphological changes were observed at 4.2 ppm NO_2 (12 µg DPM/m^3, ~ 3 µg EC/m^3), determined to be the LOAEC of this study (McDonald et al. 2015). Corresponding but slightly milder effects were reported in the same study for rats exposed at 3.6 ppm NO_2 (13 µg DPM/m^3) for 13 weeks. The findings were largely associated with NO_2. No histopathological changes were detected after a 130-week exposure at ≤ 0.9 ppm NO_2 (5 µg DPM/m^3, ~ 1 µg EC/m^3) or a 13-week exposure at ≤ 1.0 ppm NO_2 (≤ 4 µg DPM/m^3) leading to a NOAEC of 0.9 ppm NO_2 (HCOTN 2019, HEI 2015a, NRCWE 2018, Taxell and Santonen 2016). No genotoxic effects were found with this DPM (HEI 2012). The study did not directly compare TDE and NTDE exhaust but is one of only a few that have investigated NTDE exhaust toxicity.

McDonald et al. (2004a) compared lung inflammation in mice exposed by inhalation 6 h/day for 7 days to exhaust generated from a diesel engine using '2003' fuel and no exhaust after-treatment (234 µg DPM/m^3, ~0.21 ppm NO_2) or generated from the same engine under the same operating conditions but using low-sulfur-fuel and a particle trap (7 µg DPM/m^3, ~0.19 ppm NO_2)[26]. Mice exposed to unfiltered DEE had increased levels of lung inflammatory markers. No significant effects of inflammatory markers were observed in mice exposed to filtered DEE (McDonald et al. 2004a). Since the NO_2 concentrations were the same in both filtered and unfiltered DEE, it is likely that the higher DPM concentration in unfiltered exhaust contributed to the observed effects.

In order to further consider whether there is likely to be a difference in the toxicological potency of DPM from TDE and NTDE exhaust, animal studies that included concurrent results for unfiltered DPM and filtered DPM (fDPM) were collated (see blue shaded studies in Appendix C). Concentrations of particulates from these select studies are shown below in Figure 6-3. For the purposes of this exercise, both LO(A)ECs and NO(A)ECs are considered. The figure has been shaded to represent three different regions; i) LO(A)ECs identified for unfiltered DPM in animal studies (orange shaded area), ii) NO(A)ECs identified for unfiltered DPM in animal studies (blue shaded area), and iii) LO(A)ECs or NO(A)ECs identified for fDPM in animal studies (green shaded area). In some instances, the fDPM concentration was not reported however it was assumed to be low, nominally 20µg/m^3, for the purposes of this exercise. The values shown above data points on the figure refer to the NO_2 concentrations (ppm) reported from the relevant study. An asterisk next to these values (for fDPM) denotes that the NO_2 concentration in the study was not reported. In these instances, the NO_2 concentration for fDPM was assumed to be the same as that reported for the corresponding unfiltered DPM result. The NO_2 concentrations at each DPM exposure concentration at which no effects or some effects (adverse or non-adverse) were observed are also presented as a scatter plot in Figure 6-4. Note that NO_2 was often measured concurrently to DPM in these studies; it is therefore sometimes not easy to tease out the cause of the effects observed: whether they be due to DPM or NO_2 exposure.

[26] NO_2 was not directly measured in this study due to analyser failure (McDonald 2004a). In a corresponding study, McDonald (2004b) determined NO_2 to represent 10% of total nitrogen oxides (NO_x). Therefore, NO_2 concentrations are estimated in this report to be 0.21 ppm with no treatment and 0.19 ppm with treatment based on reported NO_x concentrations of 2.1ppm and 1.9ppm respectively (see Table 4, McDonald 2004a).



Values shown above shapes are nitrogen dioxide (NO_2) concentrations in ppm, # = fDPM concentration assumed to be low (nominally <20µg/m^3)

* = NO_2 concentration for fDPM assumed to be the same as DPM value, DPM = diesel exhaust particulate matter, fDPM = filtered DPM

Figure 6-3 Experimental animal NO(A)ECs/LO(A)ECs from studies concurrently measuring effects of unfiltered DPM and filtered DPM (fDPM).

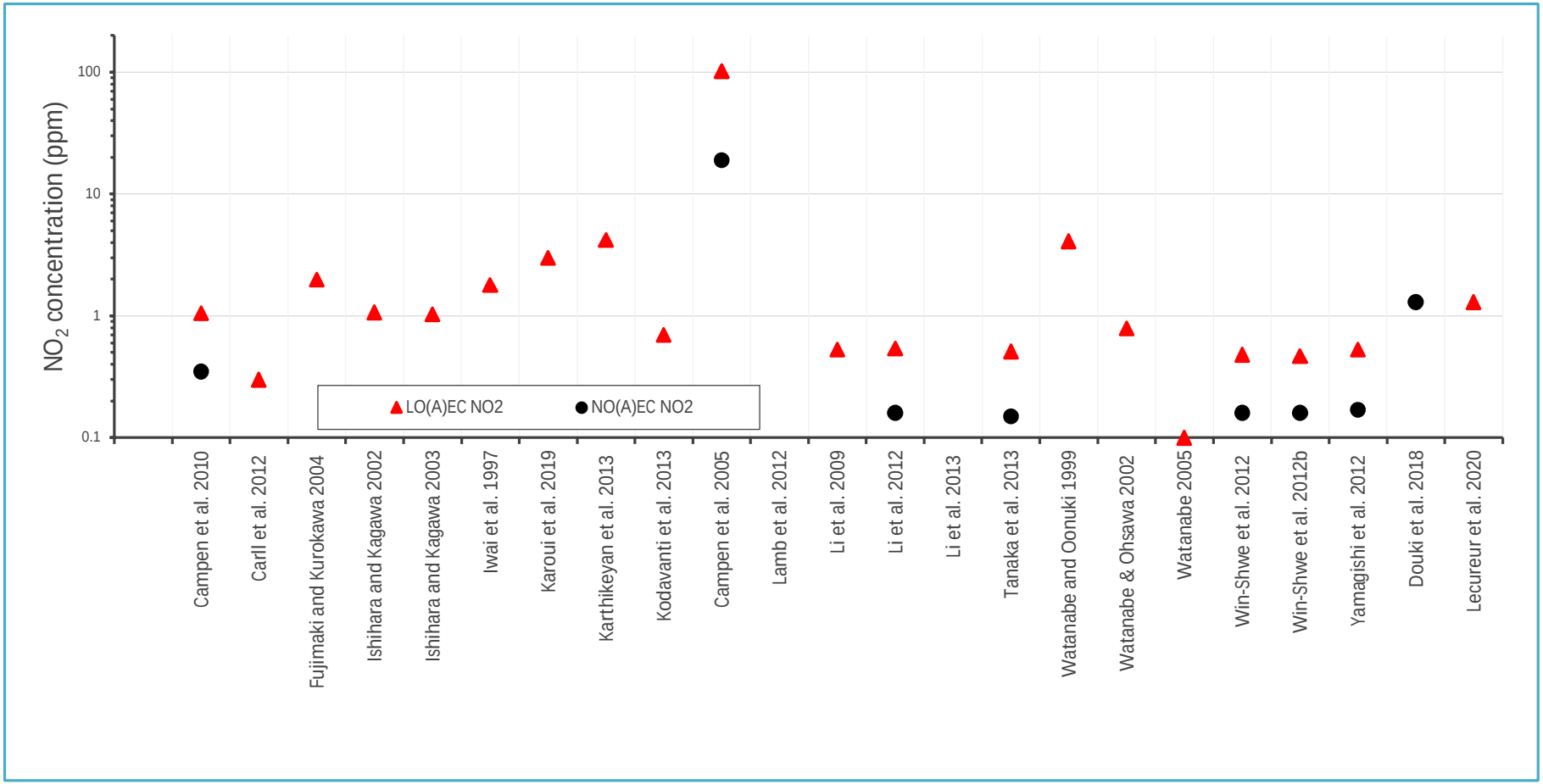


Figure 6-4 Scatter plot of NO_2 NO(A)ECs and LO(A)ECs from studies shown in Figure 6-3.

From Figure 6-3 and Figure 6-4 it is evident that:

- The LO(A)ECs for unfiltered DPM in these studies range from 100µg/m^3 to above 1,000µg/m^3 (see orange shaded area in Figure 6-3).
- The LO(A)ECs and NO(A)ECs for fDPM are considerably lower (<40µg/m^3, see green shaded area in Figure 6-3), with Campen et al. (2010) being an exception (with a fDPM LO(A)EC of 1,000 µg/m^3) but this study did not obtain a NO(A)EC for fDPM.
- The NO(A)ECs for unfiltered DPM (ranging between 30µg/m^3 and 200µg/m^3, see blue shaded area in Figure 6-3) tend to lie in the region between the LO(A)ECs for unfiltered DPM and fDPM.
- These ranges in LO(A)ECs/NO(A)ECs indicate that it is unlikely that the particulate fraction of DEE is responsible for the effects observed. Rather, the effects are likely attributable to the gaseous portion (rather than the particulate portion) of DEE. Various authors have attributed the changes observed to NO_2 (Fujimaki and Kurokawa 2004, Watanabe 2005, Li et al. 2009, 2012 and 2013; Campen et al. 2010, Gordon et al. 2012, Lamb et al. 2012, Karthikeyan et al. 2013, Tanaka et al. 2013, Dhouki et al. 2018, Karoui et al. 2019, Lecureur et al. 2020, Weitekamp et al. 2020, Greim 2019) and in at least one instance have also hypothesised that aldehydes may be contributing to the observed changes (Lecureur et al. 2020)[27].
- Most of the studies appeared to observe effects when NO_2 concentrations were greater than approximately 0.3 ppm (see Figure 6-4).

It is noted that the data are somewhat limited in that generally only one exposure group for fDPM was included in the study; this means although it appears some of the effects observed occur at lower concentrations in the fDPM treatments compared to the DPM treatments this may simply be a function of the limited concentrations tested. It seems to be that the NO_2 concentrations show a more consistent differential between where no effects are observed (~0.1-0.2ppm) and where effects are observed (~>0.3ppm). Nevertheless, there are also a few exceptions to this (Campen et al. 2005, Watanabe 2005).

These data provide tentative support for the conclusion that DPM from TDE is not likely to be in principle of different toxicity to DPM from NTDE. The lower concentration of DPM in NTDE appears to reduce the toxicity of DPM, however the concentrations of gaseous compounds such as NO_2 and aldehydes may increase compared with TDE (see Section 3.4)[28] and therefore need to be monitored in conjunction with DPM in management of DEE exposures in the workplace.

[27] Lecureur et al. (2020) exposed rats to unfiltered or filtered DEE for 3 hours/day, 5 days/week for 3 weeks and through genomic analysis in rat lungs, found a modest regulation of gene expression level (lower than 2-fold) and a higher number of genes regulated downstream of the filter (i.e. for filtered DEE). The concentration of aldehydes was higher downstream of the filter (i.e. 200 µg/m^3 vs. 40 µg/m^3) whereas BTEX, alkane and PAH concentrations were lower indicating aldehydes in the gaseous phase of DEE may have a higher influence on observed animal responses of NTDE than the particulate fraction.

[28] This is also evident from data for Campen et al. (2010), Carll et al. (2012), Fujimaki and Kurokawa (2002), Karthikeyan et al. (2013) in Figure 6-3.

7 WES derivation

7.1 Other WES in the literature

A number of international agencies, professional organisations, and/or scientists have derived occupational exposure limits (OELs) for DEE. These are equivalent to a WES in Australia, but in some instances the health basis is unclear and they are not always necessarily legally enforceable. In this report the terminology WES has been used although it is recognised the values may have different names depending on the organisation or author that derived them. These are summarised in descending order in Table 7-1 and span five orders of magnitude from 0.005 to 160 µg/m^3, with the vast majority expressed as REC.

The lowest WES have been derived by Denmark and the Netherlands, which both used the exposure-response relationship from the Vermeulen et al. (2014) meta-analysis of three epidemiological studies.

The AIOH (2017) supports the maintenance of DPM levels (measured as submicron EC[29]) as low as reasonably practicable (ALARP) below an 8-hour TWA guidance exposure value of 100 µg/m^3, with the provision of applying a TWA value of 50 µg/m^3 as an action level, which triggers investigation of the sources of exposure and implementation of suitable control strategies. The AIOH (2017) considers this to be a balance between the factors of minimising irritation and minimising the potential risk of lung cancer to a level that is not detectable in a practical sense in the workforce.

US EPA (2002a) considered the human exposure-response data available at the time to be too uncertain to derive a confident quantitative estimate of cancer unit risk, and with the chronic rat inhalation studies not being predictive for environmental levels of exposure, US EPA chose not to develop a quantitative estimate of cancer unit risk. Nevertheless US EPA (2002a) did derive an ambient reference concentration (RfC) of 5 µg/m^3 for DPM[30]. This was based on the highest human equivalent NOAEC (i.e. NOAEC$_{HEC}$) associated with no apparent effects in experimental animal studies of 144 µg/m^3 (the experimental NOAEC was 460 µg/m^3) from a 30-month rat inhalation study by Ishinishi et al. (1988). To obtain the RfC, this POD was divided by two types of uncertainty factors (UFs): a factor of 3 for interspecies extrapolation uncertainties, and a factor of 10 for interindividual human variation in sensitivity. An evaluation of the interspecies extrapolation issues for dosimetric and pharmacodynamic equivalence between rats and humans showed that although some adjustments could be accounted for, there remained a residual uncertainty, and thus an uncertainty factor of 3 out of a possible factor of 10 was used. In the absence of mechanistic or specific data, a default value of 10 was considered appropriate by US EPA (2002a) to account for possible human variability in sensitivity, particularly for children and people with pre-existing respiratory conditions. For a worker population, which principally consists of healthy adults, it could be argued that an uncertainty factor of 3 instead of the default of 10 is likely appropriate. Adjusting then from a 24-hour to an 8-hour exposure timeframe would result in a WES of 48 µg/m^3 [NOAEC$_{HEC}$ 144 µg/m^3 ÷ UF of 9 x 24/8 hours]. This WES (adapted from the US EPA 2002a RfC to a worker population) is also included in Table 7-1.

[29] AIOH (2017) indicate that REC is similar to submicron EC. It is noted the toxicological and epidemiological literature on which a WES can be based typically involved measurement of REC, hence why the recommended indicator for a candidate WES in this report is REC.

[30] DPM is defined by US EPA (2002a) by the measurement procedures summarised in the Code of Federal Regulations (Title 40 CFR, Part 86, Subpart N). These procedures define DPM as the mass of material collected on a filter at a temperature of 52°C or less after dilution of the exhaust with air. The particle filter sizing was not provided by US EPA (2002a) and could not be found in the CFR document quoted.

Landwehr et al. (2019) recently reviewed research findings relevant to setting appropriate WES, focusing specifically on newer engine and after-treatment technologies[31]. Their review found that exposure to DEE from both older and new technology classifications was found to cause negative health impacts on the lungs, heart and brain including increased risk of cancer, increased blood pressure, increased risk of thrombosis, neuroinflammation and increased DNA damage. Subjects with asthma, allergy or respiratory disease were more at risk of negative effects caused by DEE exposure than healthy subjects. According to the study authors, health impacts were found to occur even in studies using exhaust concentrations below the recommended AIOH (2017) WES TWA of 100 µg/m^3 of submicron EC (Landwehr et al. 2019). Based on the results of their review, Landwehr et al. (2019) recommended that an 8-hour TWA DPM concentration below 50 µg/m^3, around 35 µg/m^3 of REC, is more appropriate in order to limit health effects. The reasoning provided is that this concentration is below the acute exposure concentrations where effects were still found in asthmatics and below the exhaust concentrations that found the highest lung cancer risks. In addition, according to Landwehr et al. (2019) this concentration is supported by *in vivo* exposure studies, where exposure concentrations at 50 µg/m^3 only resulted in mild health effects.

The American Conference of Industrial Hygienists (ACGIH) is generally one of the organisations that other organisations look to for guidance on WES. The ACGIH has placed DEE on its list of agents under study, but does not currently recommend a Threshold Limit Value (TLV, i.e. WES) for DEE. The ACGIH first proposed a TLV (as an 8-hour TWA) of 150 µg/m^3 for DEE (on the basis of DPM) in its 1995-1996 Notice of Intended Changes (NIC). At that time, it assigned a designation of A2 (suspected human carcinogen) to DEE. That proposed limit was later lowered to 50 µg/m^3 (as total DPM) and was replaced in 2002 with a proposed TLV of 20 µg/m^3, expressed as EC. The proposed carcinogenicity classification remained as A2. The ACGIH withdrew DEE from the NIC in its 2003 edition of *Threshold Limit Values for Chemical Substances and Physical Agents*. DEE was placed on the "Under Study" list in 2016 and remains there as of January 1, 2022 (ACGIH 2022).

Based on evidence of increased lung cancer risk at very low levels, Carex Canada (2020)[32] recently recommended that Canadian jurisdictions move towards a WES for DEE based on EC of 20 µg/m^3 for the mining industry and 5 µg/m^3 for other workplaces to protect worker health. The higher WES recommended for the mining industry takes into account the feasibility of implementation in this industry and is meant as an interim target in a staged approach to eventually have one harmonised WES for all workers (Carex Canada 2020).

Table 7-1 Recommended WES for DEE in the literature

Source	WES (arranged in descending order)	Basis
US Mine Safety and Health Administration (MSHA), as summarised in Barn et al. (2021)	TWA of 160 µg/m^3 respirable total carbon (TC)	• Basis not described. Applies since 2006 to underground metal and non-metal mines.

[31] The authors searched the PubMed database for studies published since 2005 focussing on the health effects of whole DEE exposure. Studies were separated into the methodology used (whether they exposed human, animal or tissue) and type of engine used to generate the exhaust. Engines that used exhaust after-treatment devices including both a DOC and a DPF were classified as NTDEs. All other studies were classified as using older technology engines.

[32] Carex Canada (CARcinogen EXposure) is a multi-institution team of researchers and specialists with expertise in epidemiology, risk assessment, toxicology, geographic systems and knowledge mobilisation. The purpose of CAREX Canada is to provide a body of knowledge about Canadians' exposures to known and suspected carcinogens.

Source	WES (arranged in descending order)	Basis
Australian Institute for Occupational Hygienists (AIOH 2017)	TWA of 100 µg/m^3 (as submicron EC) (action level: 50 µg/m^3)	• Experience has shown that irritant effects decrease markedly when workplace exposures are controlled below this WES. • Möhner and Wendt (2017) note that an upper bound for the cumulative exposure of 2.5 mg/m^3.years REC seems to be sufficient to prevent a detectable increase of lung cancer. This value they put as corresponding to an average annual exposure value of 50 µg/m^3 REC assuming an 'exposed' working life of 45 years.
WorkSafe NZ (2020)	TWA of 100 µg/m^3 REC	Basis for value not found in literature consulted. Notation "Diesel exhaust is a confirmed carcinogen". Note same value also used in Switzerland (as summarised by Barn et al. 2021).
Hebisch and Wolf 2018, Hebisch et al. 2017, TRGS-552	TWA of 50 µg/m^3 REC STEL of 400 µg/m^3 REC	Basis not described in this publication. TWA likely based on BauA (2017), see below. Same TWA value also used by EU (Barn et al. 2021).
German Federal Institute for Occupational Safety and Health (BauA 2017) It is noted the European Parliament has adopted the same WES in December 2018 (Carex Canada 2020); Luxembourg also lists the same WES (JOGDL 2021).	TWA of 50 µg/m^3 REC	• Experimental NOAEC of 350 µg/m^3 for inflammatory changes in the lung in 2-year rat study (Mauderly et al. 1987, Henderson et al. 1988). Alveolar particle clearance was not impaired at this dose. This was converted to a NOAEC$_{HEC}$ using two different approaches. (1) • The resulting NOAEC$_{HEC}$ values from the two different approaches were 72 and 34 µg EC/m^3. • A WES of 50 µg/m^3 REC was recommended, which lies in between the two WES derived above.
Derived in this report based on US EPA (2002a) identified highest NOAEC$_{HEC}$ (2)	48 µg/m^3	No apparent effects in 30-month rat study by Ishinishi et al. (1988). NOAEC$_{HEC}$ calculated by US EPA to be 144 µg/m^3. This was used by US EPA (2002a) to derive an RfC in ambient air for non-cancer effects. Taking into account exposure adjustment (24 vs. 8 hours) and potential lower sensitivity of occupational populations, the resulting WES was derived by SLR as follows: [NOAEC$_{HEC}$ 144 µg/m^3 ÷ UF of 9 x 24/8 hours]. See text for description. (2)
Landwehr et al. (2019)	TWA of 35 µg/m^3 REC	• Based on review of available toxicological and epidemiological studies of DEE from older and new technology engines published since 2005, which indicated that health effects may occur below 100 µg/m^3 and the use of after-treatment devices had little to no impact on resulting health effects of DEE exposure.

Source	WES (arranged in descending order)	Basis
BHP Billiton (IOM 2015)	TWA of 30 µg/m^3 REC	• Concluded there is a marked elevation in lung cancer risk from DEE even at relatively low levels of exposure. • Acknowledged it may be impractical to set a meaningful health based WES for workplace exposure but concluded that evidence was strong enough to support recommending controlling exposures to DPM to lowest level that is technically achievable.
California Department of Public Health USA, as noted in Barn et al. (2021)	TWA of 20 µg/m^3 as diesel particulates	Equates to an excess lung cancer risk of one in 1,000 over a working lifetime, which is considered an acceptable workplace risk in California (CDC 2011).
Finnish Institute of Occupational Health, as reported in Barn et al. (2021)	TWA of 5 µg/m^3 REC (general workplaces) TWA of 20 µg/m^3 REC (mines, underground construction)	Basis not provided in Barn et al. (2021) or Carex Canada (2020), which cite these WES. A search on the Finnish Institute of Occupational Health website did not provide the background documentation for these values.
Health Council of the Netherlands (HCOTN 2019)	TWA of 1.03 µg/m^3 REC (prohibition risk level) TWA of 0.011 µg/m^3 REC (target risk level)	• Based on predicted exposure-response curve for lung cancer calculated in meta-analysis of epidemiological studies by Vermeulen et al. (2014), i.e. a pooled slope (β) factor of 0.00098 (lnRR for a 1 µg/m^3.year increase in REC). • Based on 'prohibition' level of 4 extra cases of lung cancer deaths per 1,000 (4 x 10^{-3}) for 40 years of occupational exposure. The concentration at the 'target' risk level of 4 extra cases per 100,000 (4 x 10^{-5}) was 0.011 µg REC/m^3.
Danish National Research Centre for the Working Environment (NRCWE 2018)	Provided WES for different lung cancer risk levels: 0.45 µg/m^3 at 1 in 1,000 0.05 µg/m^3 at 1 in 10,000 0.005 µg/m^3 at 1 in 100,000	• Based on predicted exposure-response curve calculated in meta-analysis of epidemiological studies by Vermeulen et al. (2014), i.e. a pooled slope (β) factor of 0.000982 (lnRR for a 1 µg/m^3.year increase in REC). (3)

WES = Workplace exposure standard (used as terminology to describe OELs in this report); EC = Elemental carbon; R = Respirable; NOAEC = No observed adverse effect concentration; HEC = Human equivalent concentration; RR = Relative risk; TC = Total carbon.

1. In the first approach: the HEC for poorly soluble particle agglomerates based on the macrophage volume from Krombach et al. (1997) and Pauluhn (2011) is as follows: HEC/CT = 0.008 x 1110 x 0.15 x (DF_T/DF_H) = 1.33 x (DF_T/DF_H), where DF is the deposition fraction (percent/100), T is the animal (rat), H is human. From this HEC a candidate WES was derived by accounting for the proportion of the HEC that is EC [12% is the organic extractable proportion from the diesel soot particles according to Mauderly et al. (1987), 27% constitutes sulfates and ash according to Hesterberg et al. 2011, leaving 61% being EC]. Since the observed inflammation from different inhalation studies on different species (rat, hamster, mouse) (e.g. Edler et al. (2005)) indicates the rat to be the most sensitive, the default variability factor was changed from 5 to 3 (calculation: HEC = 1.33 x 0.094/0.101 (DF_T/DF_H) x 350 µg/m³ (NOAEC) x 0.5 (approximate EC portion) /3 (variability) = 72 µg EC/m³.

   In the second approach: the same NOAEC was used but adjusted for the higher human respiratory volume in the workplace due to physical activity and the change in exposure timeframes. This resulted in a WES of 68 µg DPM/m³ (34 µg EC/m³) [calculation: 350 µg/m³ x 0.5 (approximate proportion EC) /3 (variability) x7/8 (exposure time adjustment) x6.7/10 (inhalation rate adjustment)= 34 µg/m³].
2. It is recognised this value has not been published in the literature but is a simple adjustment from an RfC for public exposure derived by US EPA (2002a) to workplace exposures. It is noted the resulting value is similar to the mid-range of other values in this table.
3. Based on Denmark lifetime risk of developing lung cancer being 4.9% for men and 4.5% for women, RRs were calculated for excess lung cancer risks of 1 in 1000 (i.e. RR = (49+1)/49 = 1.02), 1 in 10,000 (RR 1.002), and 1 in 100,000 (RR 1.0002). These RRs were used to solve for exposure in the equation: Exposure (EC in µg/m^3.years) = Ln(RR) ÷ β and subsequently divided by 45 years of workplace exposure.

7.2 Candidate indicators for WES

The complexity of DEE, and the fact that DEE mass balance changes depending on fuel type, makes it difficult and resource intensive to analyse for all individual exhaust constituents to determine exposures.

EC has been widely used a surrogate for DPM, especially in epidemiological studies, since it has been shown to provide the best proxy[33] of diesel particulate emissions (especially from TDE), is relatively free of interferences and is chemically stable, unlike the adsorbed OC fraction (AIOH 2017, Carex Canada 2020, SCOEL 2016, 2017). However, EC has been shown to vary depending on engine load (Bugarski et al. 2011, Cauda et al. 2012). In addition, mass-based exposure assessments are not always predictive of risk of adverse effects: in some cases, respirable particle surface area and detailed surface compositional or morphological properties better correlate with toxicity or offer an explanation of seeming anomalies in epidemiological findings of disease risk (Bugarski and Timko 2007, Cauda et al. 2012, Landwehr et al. 2019).

Landwehr et al. (2019) comment that exposure limits based on both the mass of EC and the mass of total PM are limited in their long-term applicability. This is because DPFs remove particles from the exhaust, however they preferentially select for elemental carbon above other particle types, skewing the exhaust output and potentially eliminating EC as a predictive measure for overall exhaust exposure, making a WES based on EC potentially unreliable for NTDE exhaust (Landwehr et al. 2019, Taxell and Santonen 2016). For NTDE, NO_x may be a more reliable indicator of DEE exposure (Landwehr et al. 2019).

Nevertheless, the selection of an appropriate indicator for derivation of a WES is reliant on the measurements available in toxicological and human exposure data. Thus, for DPM, the indicators with the most information for delineating an exposure-response relationship are REC and DEE $PM_{2.5}$ or PM_1 (the latter measures often used in controlled exposure studies, summarised in Section 6.1 and Appendix D). Most of the latest WES for DPM found in the literature are based on a measurement of REC (see Section 7.1). For the gaseous fraction of DEE, NO_2 appears to be an important indicator for adverse health effects, especially for NTDE (see Section 6.3). In addition, as indicated in Section 6.1, the irritation effects observed in controlled human exposure studies with DEE may be attributable to the aldehydes and/or NO_x in gaseous DEE.

There is also potential that the PAHs in the soluble organic fraction of DPM may be responsible for some of the effects observed in genotoxicity studies with DEE (see Section 5.1). Therefore, PAHs may be another potential useful indicator group of compounds when used in concert with EC, NO_x, and aldehydes to inform on the overall potential toxicity of DEE.

7.3 Approaches to deriving a WES for DEE

Two approaches have been investigated for the derivation of a candidate WES for DEE, along with two additional complementary considerations. The methods are considered to be complementary so as to cater for the uncertainties in relation to the mechanisms of action for the critical health effects in humans and the necessity of using multiple indicators to define the toxicity of a complex mixture like DEE. The approaches involve the following.

1. Derivation of a WES for DPM using an acute NOEC and chronic minimal LOAEC from controlled human exposure studies that would prevent irritation and also subsequent pulmonary inflammation (which is considered the most likely mechanism for acute and chronic adverse pulmonary effects).

[33] Although total carbon (TC) has also been used as a surrogate for DPM because it accounts for over 80% of DPM, TC can be prone to interferences such as cigarette smoke and oil mist, which are organic carbon (OC) aerosols that generally belong to the same size category as diesel aerosols (Bugarski et al. 2011).

2. Derivation of a WES for DPM using a NOAEC from chronic experimental animal studies. If the mechanism for lung cancer from DEE exposure in humans were due to an inflammatory response, the exposure-response relationship would have a threshold, and the WES derived with this method would be protective of lung cancer in humans.
3. Consideration whether the candidate WES values for DPM derived using approach 1 and 2 above would be protective of lung cancer if the responsible constituents in DEE (from both TDE and NTDE) were carcinogenic PAHs and the mechanism of action for lung cancer in humans were due to genotoxicity (resulting in a non-threshold exposure-response).
4. Although the occupational epidemiological information on its own is not considered to be sufficient for quantitative use in derivation of a WES (see discussion in Section 6.2), the candidate WES values derived using approach 1 and 2 above are put into context by comparing them with the cumulative REC exposures that were found to be associated with a significantly increased risk of lung cancer.

7.3.1 Derivation using controlled human exposure studies

As discussed in Section 6.1, the available human controlled exposure studies indicate the following PODs could tentatively be used together with other relevant information for derivation of a candidate WES:

- an acute NOEC of 100 µg/m^3 DEE (measured as $PM_{2.5}$) for airway inflammation (Mudway et al. 2004);
- a chronic minimal LOAEC[34] of 31-60 µg/m^3 as diesel soot (presumed to be REC, no further details provided) for chronic rhinitis (Glück et al. 2003).

Typically, uncertainty or safety factors are applied to a POD when deriving health-based guideline values. These cater for various aspects of ambiguity associated with toxicokinetic and toxicodynamic variability between humans, interspecies differences in toxicokinetics and toxicodynamics, use of a LOAEC instead of a NOAEC, and when needed other uncertainties in the derivation of the guideline (enHealth 2012a).

Given that the acute exposure studies used to select the POD included evaluation of potentially sensitive subgroups, i.e. subjects with asthma (Sava et al. 2013, Thomson et al. 2021), no UF is considered to be required to account for further susceptibility in a healthy worker population. Typically, an UF of 10 would be applied to a NOAEC from a short-term exposure study, however since the POD is a NOEC (rather than a NOAEC), and prevention of airway inflammation should prevent chronic adverse effects if the mechanism of toxicity is operable via a threshold exposure-response curve, an UF of 3 is considered sufficient. Application of an UF of 3 for use of a NOEC from a short-term exposure study to derive a chronic WES results in a candidate WES of 33 µg/m^3 DEE (i.e. 100 µg/m^3 ÷ 3 = 33 µg/m^3), which, when adjusted for EC which was potentially present in TDE exhaust at the time (i.e. ~75%, HCOTN 2019), results in a candidate WES of 25 µg/m^3 (as REC).

The exposure midpoint in the chronic study by Glück et al. (2003) was approximately 45 µg/m^3 (presumably as REC). Since the study population was a worker population and extrapolation is being done to another hypothetical worker population (i.e. no children or elderly who may potentially be more sensitive to the effects of DEE exposure), use of an additional UF to account for potential human variability is not considered to be required. An UF of 3 is applied for use of a minimal LOAEC, since there was no evidence of progression for the chronic rhinitis observed. This results in a candidate WES of 15 µg/m^3 REC.

[34] Chronic non-allergic rhinitis is considered adverse because it can be associated with symptoms such as stuffy/runny nose, sneezing, mucus in the throat and cough, which can be debilitating if persistent. However, it is noted Glück et al. (2003) make no comment regarding whether the workers monitored in the study experienced any symptoms. The POD has been considered to be a minimal LOAEC in this report.

7.3.2 Derivation using chronic experimental animal studies

For chronic exposure to DEE, respiratory effects are the critical health effect in experimental animals. Rats are the most sensitive species (Health Canada 2016a). The chronic rat NOAEC of 350 µg DPM/m^3 for exposure for 7 hours/day, 5 days/week for 24 months was considered the most appropriate animal NOAEC to use for derivation of a candidate WES.

Adjusting this NOAEC to an 8-hour exposure gives an adjusted NOAEC of:

$NOAEC_{adj}$ = 350 µg DPM/m^3 x 7/8 hours = 306 µg DPM/m^3

The following UF are considered appropriate:

- a standard default UF of 10x for interspecies variability in toxicokinetics and toxicodynamics (enHealth 2012a); and
- the standard default parameter for potential differences between humans is 10x (enHealth 2012a) and comprises toxicokinetic and toxicodynamic considerations. These are primarily based on the presence in the general population of very young, old and infirmed persons that are potentially more vulnerable to exposure to chemicals. There is much less variability between workers who are presumably healthy at the start of employment. An uncertainty factor of 3x is therefore considered appropriate.

Application of a composite UF of 30x to the $NOAEC_{adj}$ 306 µg DPM/m^3 results in a concentration of 10 µg DPM/m^3, which when adjusted for EC content potentially present in TDE exhaust (i.e. ~75%, HCOTN 2019), results in a candidate WES of 7.5 µg/m^3 (as REC).

7.3.3 Consideration of PAHs

One possible mechanism for the carcinogenicity of DEE is genotoxicity due to carcinogenic PAHs adsorbed to the DPM (see Section 5.1). If it is assumed this mechanism is at play, it is important to consider whether the candidate WES values derived in Sections 7.3.1 and 7.3.2 would be protective of a carcinogenic effect due to PAHs in DEE.

As discussed in Footnote 11 in Section 4.2, it is likely highly conservative to assume that the total PAH content of DEE has the same potency as BaP. In animal experiments with NTDE (Euro IV engine) total PAHs made up only approximately 0.04% of total particulate concentrations in raw exhaust[35] (Karoui et al. 2019, Lecreure et al. 2020). A similar percentage (0.05%) was found in a Euro III engine exhaust (Oravisjärvi et al. 2014). PAHs may consist of up to approximately 1% of DPM mass in TDE (SCOEL 2016, Taxell and Santonen 2016, US EPA 2002a).

[35] Total PAH content in raw exhaust (containing 23,000 µg/m^3 DPM) was approximately 10 µg/m^3 (i.e. 0.04%).

Thus at candidate WES of 7.5-25 µg REC/m^3 derived in Sections 7.3.1 and 7.3.2 (i.e. ~10-33 µg DPM/m^3, HCOTN 2019), maximum PAH concentrations could be expected to be 0.1-0.33 µg/m^3 in TDE exhaust and 0.004-0.013 µg/m^3 in NTDE. Making the highly conservative assumption that all PAHs in DEE are as potent as BaP, and using the US EPA (2017) inhalation unit risk factor[36] of 6 x 10^{-4} (µg/m^3)$^{-1}$, estimated cancer risks for occupational exposure timeframes would be in the range of 8.0 x 10^{-7} (i.e. 8 in 10,000,000) to 6.6 x 10^{-5} (i.e. 6.6 in 100,000)[37]. These estimated cancer risks are generally in the range of or slightly above the 'target' acceptable risk levels (of 1 in 100,000 or 1 in a million) used by Australian regulatory authorities to assess the risk of chemical exposures to the general public (enHealth 2012a). Considering the high-level conservatism embedded into this calculation, PAH concentrations estimated at the candidate WES values for DPM are unlikely to present a concern, especially considering most modern vehicles will be equipped with emission control devices.

7.3.4 Consideration of occupational epidemiological information

Although the occupational epidemiological information is not considered sufficient on its own to form the primary basis for derivation of a WES (see Section 6.2), it is useful to consider some of the quantitative estimates for lung cancer risk and pulmonary toxicity to place into context the candidate WES derived using the other approaches (Sections 7.3.1-7.3.3). Some of the quantitative estimates available from the various studies are as follows.

- Du et al. (2020) found a small change in lung function in underground miners in an Australian gold mine at an average EC personal exposure concentration of 56 µg/m^3. [38] The change observed was small and within the normal variation of repeated tests, rendering the clinical significance of the change uncertain.
- Duan et al. (2016) found significantly increased concentrations of a marker for oxidative DNA damage in diesel engine testing workers (employed for 1-37 years) exposed to an average of 114 ± 66.7 µg REC/m^3 compared with matched controls exposed to 11.8 ± 0.61 µg REC/m^3 (by static air sampling). As the PAH concentrations[39] that these workers were exposed to were relatively high, it is uncertain whether the effect observed was due to particulate exposure, PAH exposure, or both. This effect could potentially be indicative of an early molecular change in the progression to lung cancer, but, on its own, is not regarded as sufficient to use for derivation of a WES.

[36] An inhalation unit risk factor is an expression of the incremental risk associated with increase in exposure by a single unit of exposure measure. It has also been defined as the plausible upper-bound estimate of the probability of a response (in this case cancer) from a chemical over a lifetime. It is derived from the slope of the linearised concentration-response relationship and usually expressed in units of incremental risk per µg/m^3 in air (enHealth 2012a).

[37] Calculated as follows: Unit risk factor [6x10^{-4} (µg/m^3)$^{-1}$] x Concentration of PAHs in air (0.004-0.33 µg/m^3) x 8 hr/24 hr

[38] It is noted this study is limited by small sample size, measures of DEE may contain inherent bias, and the study authors were unable to adjust for diurnal variation influence on lung function.

[39] It is noted total PAH concentrations were 4.94 ± 1.3 in the exposure group compared with 0.035 ± 0.001 µg/m^3 in the control (reference) group (Duan et al. 2016). Using the US EPA (2017) inhalation unit risk factor of 6 x 10^{-4} (µg/m^3)$^{-1}$ for benzo(a)pyrene, and assuming all PAHs measured by Duan et al. (2016) have the same potency as benzo(a)pyrene, this would equate to an estimated cancer risk of 9.9 x 10^{-4} (i.e. 9.9 in 10,000 people).

- Silverman et al. (2012) found the highest cumulative REC exposure quartile (15-year lag) cumulative REC exposure to be statistically significant for an increased lung cancer risk. The cumulative REC was ≥536 µg/m^3.yrs. The period of time for the cumulative exposure experienced by miners in this group was not stated in the Silverman et al. (2012) paper. Attfield et al. (2012) provide information on the overall cohort of the DEMS study in which the mean underground tenure is stated to be 8 years. It is, however, uncertain whether this underground tenure timeframe can be related directly back to the cumulative EC exposure reported by Silverman et al. (2012). Although a crude estimate since some workers may have been exposed at higher concentrations for a shorter timeframe, if it is assumed this cumulative REC came from an evenly spread exposure over a 40-year occupational lifetime, this would equate to an average assumed exposure concentration of approximately 13 µg REC/m^3. If it is assumed to have occurred over an 8-year timeframe the average exposure concentration would have been approximately 67 µg REC/m^3.
- The highest exposure quartile in the Steenland et al. (1998) trucker lung cancer mortality study was >331 µg EC/m^3.yrs, but the mean exposure timeframe associated with this cumulative exposure estimate was not provided in the paper. Assuming evenly 40-year working lifetime exposure, this equates to an average assumed exposure concentration of approximately 8 µg EC/m^3.
- Möhner and Wendt (2017) noted that an upper bound for the cumulative REC exposure of 2.5 mg/m^3.yrs seems to be sufficient to prevent a detectable increase of lung cancer risk based on their review of available studies. This value corresponds to an average annual exposure value of 63 µg/m^3 REC assuming exposure is evenly spread over a working lifetime of 40 years, noting that mean exposure timeframes associated with cumulative exposure estimates in the various lung cancer epidemiology studies are not typically provided.

The candidate WES values of 7.5-25 µg REC/m^3 (derived in Section 7.3.1 and 7.3.2 using controlled human exposure studies and experimental animal studies) are well below the concentrations in epidemiological studies (described in the dot points above) that have been associated with oxidative DNA damage or non-adverse pulmonary effects (114 and 56 µg REC/m^3, respectively) and at the low end of the approximate exposure estimates that have been associated with an increased risk of lung cancer mortality (8-67 µg REC/m^3), assuming cumulative REC can be translated evenly to a 40-year working lifetime (which is unlikely to be the case) (see Figure 7-1).

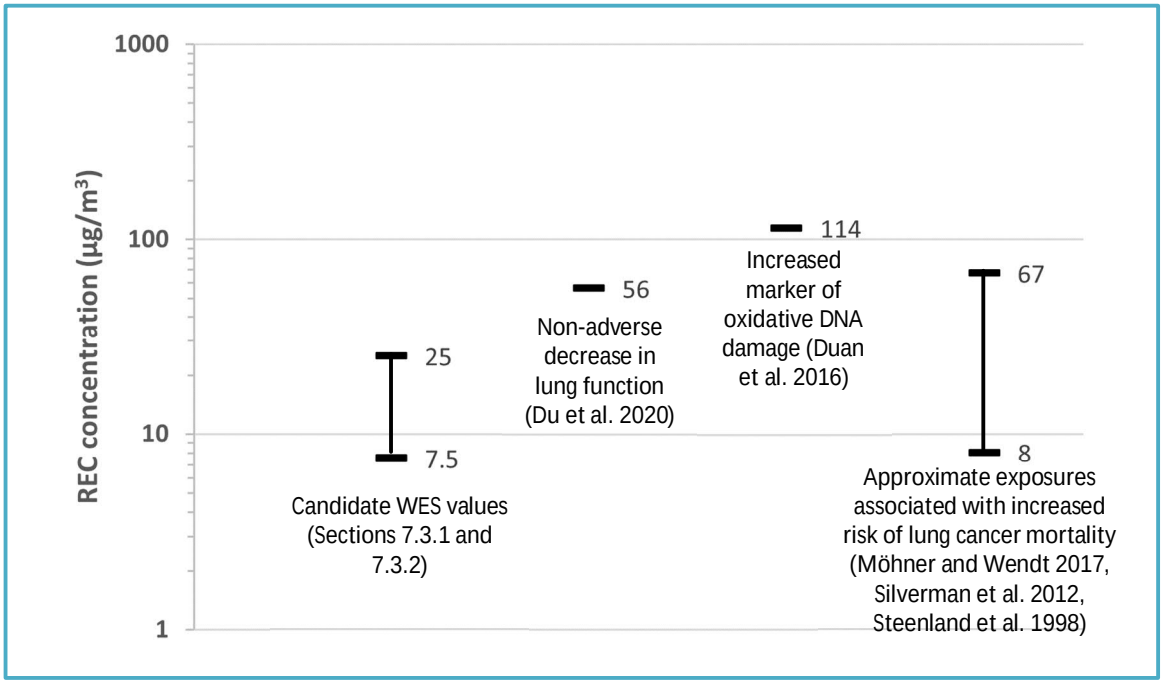


Figure 7-1 Candidate WES values in relation to epidemiological study findings

7.4 Summary of candidate WES and recommendations

A number of approaches for establishing a health-based 8-hour TWA for DPM have been explored. They included derivation using an acute or chronic POD from controlled human exposure studies (see Section 7.3.1) and derivation using a chronic NOAEC from experimental studies in rats (see Section 7.3.2). The resulting candidate WES values are in the range of 7.5-25 µg REC/m^3. Consideration of potential PAH content adsorbed to DPM indicates the candidate WES values would likely be protective of lung cancer from adsorbed PAH exposure (see Section 7.3.3). In addition, the candidate WES values are well below the concentrations in epidemiological studies that have been associated with oxidative DNA damage or non-adverse pulmonary effects (114 and 56 µg REC/m^3, respectively) and at the low end of the approximate epidemiological exposure estimates that have been associated with an increased risk of lung cancer (8-67 µg REC/m^3) (see Section 7.3.4).

The approximate midpoint of the three derivations in Section 7.3.1 and 7.3.2 is a concentration of 15 µg REC/m^3; this value is recommended. The recommended WES value derived herein is an estimate of the concentration of REC to which workers may be exposed for a lifetime without the likelihood of appreciable harm from non-cancer or cancer effects. The recommended WES for REC can be applied to DEE from both TDE and NTDE, although it is recognised compliance with this WES may prove difficult in certain workplaces where TDEs are still prominently used. The weight of evidence is considered sufficient to assign a 'Carcinogenicity Category 1A' notation to DEE.

Since DEE is a complex mixture, and since data have shown the toxicity of DEE (especially from NTDE) may be associated with gaseous components of DEE, it is recommended the candidate WES for REC be applied in conjunction with appropriate management measures to control and/or minimise exposures to other indicators of potential concern within DEE including NO_2, PAHs, and aldehydes[40] to ensure the risk of health effects from the mixture as a whole is adequately controlled.

[40] The presence of aldehydes could not be explicitly considered in the derivation of a WES for DPM since very few studies have actually measured aldehydes (see Section 6.3). At least one study (Lecureur et al. 2020) hypothesised that aldehydes

8 Conclusions

In this section, succinct responses are provided to each research question that guided the literature review.

Research Q1: What is the composition of DEE?

DEE consists of a complex mixture of hundreds of chemicals consisting of gaseous, adsorbed organics and particulate components found in the exhaust emissions from diesel engines; DPM refers to the particulate fraction of DEE. The composition of this mixture varies depending on engine type, operating conditions, fuel, lubricating oil, and whether an emission control system is present.

Research Q2/Q3: How does the composition vary by engine type/year of manufacture?

Due to the increasingly stringent pollution emission standards over the last two decades, engine technology has evolved from older 'traditional diesel engines' (TDE) to 'new technology diesel engines' (NTDE). With the application of sophisticated emission control devices in NTDE, emissions of particulates (especially elemental carbon) and other constituents (e.g. PAHs, nitro-PAHs, hopanes and steranes, alcohols and organic acids, alkanes, carbonyls, dioxins and furans, inorganic ions, metals and elements) are >90% lower than in TDE exhaust. TDE engines are generally considered to be any engines manufactured before 2007 and not equipped with after-treatment devices.

Research Q4: What are the health effects associated with exposure to DEE?

The critical health effects associated with exposure to DEE include pulmonary irritation which, upon long-term exposure, can progress to an inflammatory response and lung cancer. DEE has also been shown to have an effect on cardiovascular parameters in human controlled exposure studies as well being associated with cardiopulmonary disease in epidemiological investigations. DEE can also increase the response to other allergens but does not appear to be an allergen itself.

Research Q5/Q6: Does the weight of evidence in humans and/or animals suggest any advisory notations should be recommended for DEE? If so, what advisory notations are recommended?

Yes, a 'Carcinogenicity Category 1A' notation is recommended based on the weight of evidence from both epidemiological and experimental animal studies indicating DEE is a lung carcinogen.

may be contributing to the observed lung transcriptional changes in rats after exposure to DPM and filtered DPM from NTDE exhaust. Since there is some indication that the concentration of aldehydes may increase in NTDE exhaust compared with TDE, it makes sense to monitor for the presence of aldehydes in order to prevent irritation (and any potential progression to neoplastic changes) in workers.

Research Q7: Are there specific critical constituents in DEE most commonly associated with these health effects?

As DEE is a complex mixture, most human and animal toxicological studies do not measure the majority of the constituents. However, there are several indicators of DEE that appear to be associated with the health effects of concern. These include DPM (expressed as REC), NO_2, aldehydes (e.g. formaldehyde, acetaldehyde), and PAHs. Although the literature indicates mass-based concentrations may not be the best indicator to describe the toxicity of the particulate fraction of DEE, suggesting instead that complementary indicators such as surface area and physical properties of DPM be also used, unfortunately there is insufficient information in the literature reviewed to justify derivation of a non-mass-based exposure limit for DPM at this time.

Research Q8: Does exposure to DEE cause health effects over and above those of particulates *per se*?

Experimental animal studies indicate that the chronic pulmonary toxicity and carcinogenicity of the particulate fraction of DEE is likely similar to other poorly soluble particulate substances where rat carcinogenicity is associated with a non-specific particle overload effect. However, occupational epidemiological studies have shown associations for a significantly increased risk of lung cancer at exposures which do not appear to be sufficient to cause pulmonary overload. This suggests that a number of different mechanisms may be operable in the toxicity of DEE. Mutagenicity data indicate both the particles themselves and adsorbed chemicals like PAHs may contribute to a genotoxic mechanism for carcinogenicity. In addition, gaseous components of DEE (particularly NO_2 and aldehydes) may be direct irritants and/or carcinogens themselves, which means the effects are independent of particulate exposures such as is tentatively evident with NTDE exhaust.

Research Q9: Is there a threshold for the health effects associated with DEE exposure?

There are thresholds associated with most health effects of DEE exposure. With respect to lung cancer, although the rat toxicological data indicate there is a threshold, the epidemiological data suggest DEE may increase lung cancer risk at very low exposure concentrations. It is uncertain whether a threshold or non-threshold concentration-response is operable for DEE and lung cancer.

Research Q10: Is there sufficient (epidemiological and/or experimental animal) information to enable recommendation of a WES for DEE as a whole?

No, because DEE composition varies considerably under different conditions (e.g. type of engine, emission control device, load, operation, etc.), which can result in varied toxicological responses. Instead, it is suggested a WES be derived and/or applied for a number of indicator compounds in DEE.

Research Q11: If not, is there sufficient (epidemiological and/or experimental animal) information to enable recommendation of a WES for critical components of DEE?

For DPM, there is sufficient information from experimental animal studies and controlled human exposure studies to enable recommendation of a WES. The epidemiological information, although considered insufficient on its own to be used for quantitative WES derivation, is used as supporting information to put the recommended WES for DPM into context. In addition, it is recommended the candidate WES for DPM be applied in conjunction with appropriate management measures to control and/or minimise exposures to other indicators of potential concern within DEE including NO_2, PAHs, and aldehydes to ensure the risk of health effects from the mixture as a whole is adequately controlled.

Research Q12: If so, what WES is recommended?

A number of approaches for establishing a health-based 8-hour TWA for DPM have been explored. They included derivation using an acute or chronic POD from controlled human exposure studies and derivation using a chronic NOAEC from experimental studies in rats. The resulting candidate WES values are in the range of 7.5-25 µg REC/m^3. Consideration of potential PAH content adsorbed to DPM indicates the candidate WES values would likely be protective of lung cancer from PAH exposure. In addition, the candidate WES values are well below the concentrations in epidemiological studies that have been associated with oxidative DNA damage or pulmonary effects (114 and 56 µg REC/m^3, respectively) and at the low end of the range of approximate epidemiological exposure estimates that have been associated with an increased risk of lung cancer (8-67 µg REC/m^3).

The approximate midpoint of the three derivations is at a concentration of 15 µg REC/m^3; this value is recommended. The candidate WES value derived herein is an estimate of the concentration of DPM (measured as REC) to which workers may be exposed for a lifetime without the likelihood of appreciable harm from non-cancer or cancer effects. The recommended WES for REC can be applied to DEE from both TDE and NTDE.

9 References

This bibliographical list contains references that were cited in the body of the report. Appendix G contains a bibliographical list of literature screened as being potentially relevant in the literature search conducted, but these references have not been cited in the body of the report.

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APPENDIX A

Literature review strategy

Search strategy

A methodical literature search was undertaken to identify relevant national and international agency documents and reviews, supplemented by a detailed search of a wide range of peer-reviewed scientific literature without date limitation. The project objectives helped focus the literature search:

1. identify relevant independent, peer reviewed research and literature on the health effects of exposure to DEE and the impact engine types and year of manufacture have on the composition of DEE;
2. if indicated in the research, recommend an encompassing health-based WES with supporting advisory notations for all DEE compositions present in Australia based on the adverse effect that occurs at the lowest airborne concentration; and
3. if not feasible, to make recommendations for individual WES values and notations that would be protective of exposure to the different compositions of DEE present in Australia.

The national and international agencies that were searched for publicly available reports and reviews using the query "diesel exhaust OR diesel emissions" are listed below in alphabetical order.

ACGIH – American Conference of Governmental and Industrial Hygienists
AICIS – Australian Industrial Chemicals Introduction Scheme (formerly NICNAS)
AIOH – Australian Institute of Occupational Hygienists
ATSDR – Agency for Toxic Substances and Disease Registry
Australian State Health Departments and Environmental Protection Authorities
BaUA – Bundesanstalt für Arbeitsschutz und Arbeitsmedizin (German Federal Institute of Occupational Safety and Health
CDC – Centers for Disease Control
DFG MAK – Deutsche Forschungsgemeinschaft (German Research Foundation
DWEA – Arbejdstilsynet (Danish Working Environment Authority)
European AfsHW – European Agency for Safety and Health at Work
ECHA C FIOH – Finnish Institute of Occupational Health
HAS – Health and Safety Authority, Health Canada
IARC – International Agency for Research on Cancer
INRS – Institut National de Recherche et de Securite (National Institute of Research and Security)
INSHT – Instituto Nacional de Seguridad e Higiene en el Trabajo (National Institute of Occupational Safety and Hygiene)
International Programme on Chemical Safety (IPCS INCHEM)
JOGDL – Journal Officiel du Grand-Duche de Luxembourg (Official Journal of the Grand Duke of Luxembourg)
JSOH – Japan Society for Occupational Health
NH&MRC – National Health and Medical Research Council
NTP – US National Toxicology Program
NZ DoL – New Zealand Department of Labour, OECD = Organisation for Economic Co-operation and Development
OEHHA – Office of Environmental Health and Hazard Assessment (California)
Ontario MoL – Ontario Ministry of Labour
RIVM – Dutch National Institute of Health (Netherlands)
SWA – Safe Work Australia
SCOEL – Scientific Committee on Occupational Exposure Limits
SUVA – Swiss Accident Insurance Fund
SWEA – Swedish Work Environment Authority
TCEQ – Texas Commission on Environmental Quality
UK HSE – United Kingdom Health and Safety Executive UK, UK Government
UNEP – United Nations Environment Programme
US EPA – United States Environmental Protection Agency
WorkSafe BC – WorkSafe British Columbia
Gov of Alberta – Government of Alberta
Quebec CfOHS – Quebec Commission for Occupational Health and Safety

WHO – World Health Organization
Work Safe NZ – Work Safe New Zealand

Principal scientific databases, namely Science Direct, PubMed (incl. ToxLine), Embase and MedLine, were searched without date limitations using the terms below. In addition, a general Google® search was undertaken and limited to the first 15 pages of results. For the CDC search, results were limited to the first 200 results as the relevance decreased with subsequent results and large numbers of non-relevant documents appeared. Search locations and terms queried were:

Science Direct ["diesel" and "health"]
["diesel" and "toxicity"]
["diesel" and "exhaust"]
["diesel" and "composition"]

PubMed including ToxLine ["diesel" and "health"]
["diesel" and "toxicity"]
["diesel exhaust" and "emissions"]
["diesel exhaust" and "composition"]
["diesel exhaust" and "constituents"]

Embase ["diesel" and "health"]
["diesel" and "toxicity"]
["diesel exhaust" and "composition"]

MedLine ["diesel" and "health"]
["diesel" and "toxicity"]
["diesel exhaust" and "emissions"]
["diesel exhaust" and "composition"]
["diesel exhaust" and "constituents"]

Google ["diesel" and "health"]
["diesel" and "toxicity"]
["diesel exhaust" and "emissions"]
["diesel exhaust" and "composition"]
["diesel exhaust" and "constituents"]

Study selection criteria

Potentially relevant articles were identified from titles and abstracts. Full-text articles were sourced wherever possible for those studies that clearly met the inclusion criteria or where eligibility to meet the inclusion criteria was unclear. The sourced literature was refined, as per the inclusion criteria outlined below, to enable critical review of those most relevant to the research questions.

Inclusion criteria	Exclusion criteria
Complete study available for review	No abstract or full text available
Study in English, German, Spanish or French	Study in language other than English, German, Spanish or French
Study contains original data	Unpublished report/unable to retrieve

Inclusion criteria	Exclusion criteria
Study examines effects related to chemical exposure or provides relevant information on DEE composition	Study does not examine health effects or relevant exposure levels or provides information on DEE composition
Route of exposure relevant to Workplace Exposure Standard (WES) development (e.g. inhalation, intratracheal instillation).	Study examining dermal/intravenous/subcutaneous or intraperitoneal exposure
Study focused on the mixture of concern (DEE)	Endpoint studied not relevant to human health
Relevant animal model and endpoints examined	Multiple routes possible/unknown route of exposure
Study examines effects related to chemical exposure or levels of chemical exposure	Epidemiological studies that examine environmental population exposures to DEE where concentration-response is not clearly defined.

Screening method

Results have been screened as follows.

Preliminary title screen

Total number of search results for both the agency search and individual scientific databases have been recorded into a spreadsheet. Spreadsheets with search results and screening outcomes (i.e. reasons for exclusion at content screen) were kept for project records.

Numbers of overall results with duplicates eliminated (numbers of "hits" per search, and numbers excluded) are presented in Figure 2-1.

During the search the titles of each document were scanned and only those titles were recorded in the spreadsheets which were considered potentially relevant (for consideration in the content screen, see below) as per the exclusion criteria. Where the relevance of a particular search document seemed uncertain, the researcher discussed the matter with a subject expert prior to making a decision OR the result was considered potentially relevant and included.

Content screen

The abstracts and/or full text content of reports/reviews selected for inclusion from the preliminary title screen were reviewed by a subject expert to determine which reports/reviews to include in the research report. Only reports/reviews which provided information relevant to answering the research questions have been taken through the content screen.

Data extraction

There was a need to evaluate and integrate data from diverse evidence streams, as information was collected from controlled human exposure studies, epidemiological studies, workplace exposure studies, animal toxicological and mechanistic studies. Due to the sheer volume of results obtained (i.e. over 1,500 papers/reports passed the content screen) and the large number of agency reviews, resources were focused on the following:

The epidemiological studies were assessed for relevant health effect and exposure data, and for those where multiple publications with overlapping data for the same study (e.g. publications reporting subgroups, additional outcomes or exposures, or longer follow-up) were identified, the primary study was that with the longest follow-up period, the most recent publication date, and/or the most relevant health and exposure assessment.

Studies deemed useful following this step were reviewed in more detail and their bibliographies were checked for relevant papers. The relevant epidemiological studies were tabulated by extracting qualitative and quantitative information on the study population, exposure level and duration, health effect and findings (Appendix B). Data extraction for epidemiological studies focused on those with potential exposure-response information and those which had not been previously reviewed in detail in the various agency reviews that were sourced. Environmental exposure studies were not included in the review.

Relevant experimental animal toxicological information was summarised in tabular form (Appendix C). Summarised studies were limited to those providing adequate concentration-response information for relevant exposure routes, and the focus was on studies published after the various agency reviews (i.e. ~2007 or later). Less importance was placed on non-inhalational studies or studies that only examined a single exposure concentration. Those studies identified as providing crucial information for comparison of Traditional Diesel Engine (TDE) Exhaust with New Technology Diesel Engine (NTDE) Exhaust were described in more detail.

Similarly, relevant controlled human exposure studies with DEE were summarised in Appendix D. Again, the focus was on those studies which had not been previously reviewed by the various agency reports.

Although a large number of *in vitro* studies were found in the literature search, these were considered of limited value for concentration-response determination and thus were not evaluated in detail unless they directly compared TDE exhaust with NTDE exhaust. Many of these *in vitro* investigations used concentrations that are very high, e.g. 50-200 µg/ml, which over 24 hours would equate to an exposure concentration of ~1,717 to 79,000 µg/m^3 according to proportionality noted by Savary et al. (2018)[41].

References for Appendix A

Savary C. C., Bellamri N., Morzadec C., Langouët S., Lecureur V. and Vernhet L. (2018). *Long term exposure to environmental concentrations of diesel exhaust particles does not impact the phenotype of human bronchial epithelial cells.* Toxicology In Vitro 52: 154-160.

[41] Savary et al. (2018) noted for an exposure concentration of DEE of 79 µg/m^3 over 24 hours, a mass deposition of 0.05 to 2.3 µg/cm^2 would be expected in the tracheobronchial and alveolar regions of the lung. 2 µg/cm^2 is approximately equal to 10 µg/ml in an *in vitro* experiment.

APPENDIX B

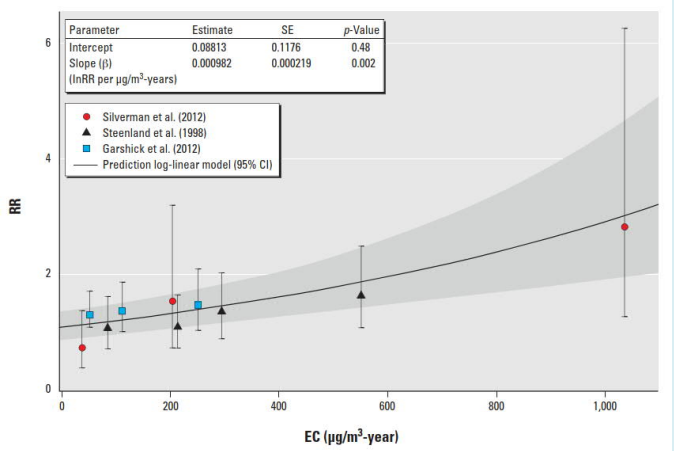
Summary of findings in epidemiological studies.

N=54 papers (Studies using data from DEMS study highlighted in light blue)

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Attfield et al. 2012, Coble et al. 2010, Stewart et al. 2010, 2012; Vermeulen et al. 2010a, 2010b (DEMS study)	Cohort mortality study	12,315 workers exposed to DE at 8 US non-metal mining facilities	Mean (95% CI) exposures in the 8 mines ranged from 78.1 (72-84.2) µg/m3 REC in limestone mine to 216.1 (207-225.2) in potash mine B for ever-underground workers. Surface-only workers range: 0.9 (0.8-0.9) in potash mine D to 3.2 (3.1-3.3) µg/m3 REC in salt mine E. Cumulative REC exposure and average REC intensity modelled at specific cut-points Average NO_2 levels underground ranged from 0.1-0.6ppm (~10x higher than surface).	Mean year of first exposure to DE in 8 mines ranged from 1967 to 1976. Mean underground tenure ranged from 6.6 to 9.1 years.	Mortality	SMRs (95% CI): • Lung cancer: 1.26 (1.09-1.44) • Oesophageal cancer: 1.83 (1.16-2.75). • Pneumoconiosis: 12.2 (6.82-20.12). All-cause, bladder cancer, heart disease, COPD not elevated. Hazard ratios for lung cancer mortality increased with increasing 15-yr lagged cumulative REC exposure for ever-underground workers with ≥5 yrs of tenure to a max in 640-<1280 µg/m^3.yr category cf. reference (0-<20 µg/m^3.yr) but declined at higher exposures (i.e. no concentration-response).	Elevated hazard ratios were also seen for surface workers. Surrogate data were used for extrapolation to past exposures. Information on smoking was not available. Historical measurements & surrogate exposure data, with industrial hygiene measurements, used to derive retrospective quantitative estimates of REC. No clear statistically significant concentration response for cumulative REC exposure or average REC intensity.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Silverman et al. 2012 (DEMS study)	Nested case-control	As above (198 lung cancer deaths, 562 incidence density-sampled controls).	As above	As above	Lung cancer	• Statistically significant increasing trends in lung cancer risk with increasing cumulative REC and average REC intensity. • Cumulative REC, lagged 15 years, yielded statistically significant positive gradient in lung cancer risk overall (P trend = .001). • OR for workers in top quartile (i.e. ≥536 µg/m^3.yrs) was 2.83 (1.28-6.26) cf with lowest quartile (0-<3 µg/m^3.yrs). • Among heavily exposed workers (ie, above the median of the top quartile [REC ≥ 1005 µg/m 3-y]), risk was approximately three times greater (OR = 3.20, 95% CI = 1.33 to 7.69) than that among workers in the lowest quartile of exposure. • Among never smokers, ORs were 1.0, 1.47 (0.29, 7.50), and 7.30 (1.46, 36.57) for workers with 15-year lagged cumulative REC tertiles of <8, 8 to <304, and ≥304 µg/m^3.yr, respectively. • Observed interaction between smoking and 15-year lagged cumulative REC (P interaction = .086) such that the effect of each of these exposures was attenuated in the presence of high levels of the other.	Adjusted for cigarette smoking and other confounders (e.g. history of employment & respiratory disease). However, data on smoking & other confounders were derived from next-of-kin interviews. Estimates of DEE exposure undoubtedly had some imprecision despite effort to minimise misclassification. Exposure-response was not clear. Increase in risk was shown at low-to-moderate exposure levels followed by plateau / decline at high levels.

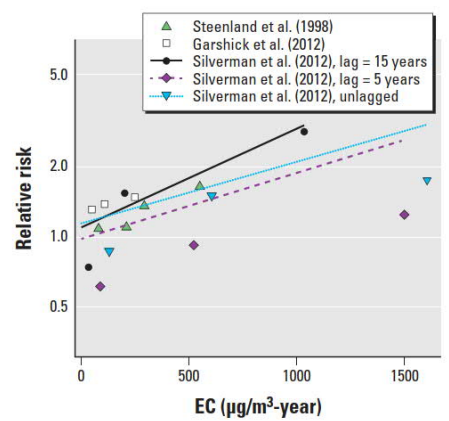
Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Vermeulen et al. 2014 (Meta exposure response including DEMS study and 2 others)	Meta-exposure-response study	Three US occupational cohort studies (Garschick et al. 2012, Silverman et al. 2012, Steenland et al. 1998). Excluded study by Möhner et al. (2013) because mean cumulative EC exposure in reference exposure category (624 µg/m^3.yrs) was higher than almost all non-reference exposures of the other studies.	See individual studies.	See individual studies.	Lung cancer mortality	• Estimated a lnRR of 0.00098 (95% CI: 0.00055, 0.0014) for lung cancer mortality with each 1-µg/m^3.year increase in cumulative EC based on a linear meta-regression model. • Corresponding lnRRs for the individual studies ranged from 0.00061 to 0.0012. • Estimated numbers of excess lung cancer deaths through 80 years of age for lifetime occupational exposures of 1, 10, and 25 µg/m3 EC were 17, 200, and 689 per 10,000, respectively. For lifetime environmental exposure to 0.8 µg/m3 EC, they estimated 21 excess lung cancer deaths per 10,000.	Formal tests of heterogeneity not undertake (of limited value). Results extrapolated to exposures lower than those observed in the occupational studies. Estimates differed regarding exposure lag (5 or 15 yr). Considerable uncertainty in retrospective exposure assessment.



Visually, from figure confidence intervals are wide and if top two data points are excluded, the regression appears to be almost a straight line.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Costello et al. 2016, 2018 (DEMS study)	Cohort mortality study	7,122 male underground workers from DEMS study (n=191 IHD mortality cases).	Various cut-points selected for cumulative REC exposure (0-9, 10-19, 20-39, 40-159, 160-639, 640-1279, 1280-2559, ≥2560 µg/m^3.yrs) and cumulative respirable dust (RD).	Not provided (see Attfield et al. 2012)	Mortality from ischaemic heart disease (IHD)	None of the HR for cumulative REC were significant. Only average RD, but not average REC, showed significant associations with the ≥ 1.5 mg/m3 exposure, but patterns were not monotonic. Trends significant for cumulative & average RD exposure (p<0.01). Using category containing change point as referent, HR for top category were 1.69 (1.08-2.62) for cumulative REC and 1.54 (0.92-2.57) for average REC.	Noteworthy is no clearly significant concentration-response. Not adjusted for smoking. Only the nested case-control study of lung cancer included smoking information, and those data were insufficient to adjust for smoking in the cohort analysis of IHD.
Ferguson et al. 2020 (DEMS study)	Cohort mortality study	Sub-cohort of 11,667 males in DEMS study (n=3732 surface only, n=7935 ever underground) (n=140 cases)	Ever-underground vs. surface workers. • Respirable dust: 1.7 vs. 0.61 mg/m3 • Cumulative resp dust: 14 vs 7.18 mg/m3 • REC: 92.1 vs 0.94 µg/m3 • Cumulative REC: 631.9 vs 13.9 µg/m3	Mean duration of employment was 10 yrs for ever-underground workers, 12.82 yrs for surface workers.	Chronic Obstructive Pulmonary Disease (COPD) mortality	• Not significant for all workers or ever-underground workers. • For surface workers, only significant for middle exposure group (>9-22 µg/m3 cumulative REC) for 0 or 10-yr lag but not high one.	Potential confounders were measured among a subset of people but not entire cohort (e.g. medical history, prior employment, socioeconomic status, etc). Lack of control for smoking, an important risk factor for COPD.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Neophytou et al. 2016 (DEMS study)	Previous studies have not accounted for the healthy worker survivor bias using methods that address time-varying confounding affected by previous exposure. Healthy worker survivor bias may occur if individuals who leave work, and thus are no longer exposed, are at greater risk of the adverse health outcome. With work status as a time-varying confounder, standard statistical methods will not be adequate to estimate an unbiased effect of the exposure on the outcome when work status is also affected by prior exposure. Thus, if termination of employment is affected by exposure, the effect of diesel exhaust on lung cancer may be even higher than previously reported. Authors applied an accelerated failure time model to assess the effect of exposures to REC on time to termination of employment among non-metal miners who ever worked underground (n=8,307). Then applied parametric g-formula to assess how possible interventions setting REC limits would have changed lifetime risk of lung cancer.				Median time to termination was 36% shorter (95% confidence interval = 33%, 39%), per interquartile range width increase in REC exposure. lung cancer risk decreased with more stringent interventions, with a risk ratio of 0.8 (95% confidence interval = 0.5, 1.1) comparing a limit of ≤25 µg/m3 REC to no intervention. The fraction of cases attributable to diesel exposure was 27% in this population.		
Crump and van Landingham 2012 (Reanalysis of DEMS study)	Authors re-estimated yearly REC exposures i) using data from mines to estimate CO-REC relationship rather than assuming a linear relationship, ii) using different method for assigning values to non-detect CO, and iii) taking account of statistical uncertainty to estimate bounds for REC exposures.				Analysis resulted in significantly different exposure estimates than original study. Authors did not conduct reanalysis of health effect associations in this publication.		
Crump 2014 (Critique of Vermeulen et al. 2014 DEMS study analysis)	Reanalysis of data in Vermeulen et al. (2014) using all 5-year lags. Critique: *There are other limitations of the analysis by Vermeulen et al. (2014): Garshick et al. (2012) employed a second measure of diesel exposure (exposure duration), which Vermeulen et al. did not account for in the analysis; and Vermeulen et al. used very crude exposure summaries (e.g., midpoints of exposure intervals).*				Revised RR (95% CI): • All 5-yr lags: 0.38 (-0.03-0.96). Similar results obtained using 0-year lag.		Re-analysis suggests no significant association.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Vermeulen et al. 2014 (response to Crump 2014 critique)	*Results for an analysis using a 5-year lag in DEMS were not published, although the 5-year lag had been included in analyses to examine changes in model fit as a function of lag. From those analyses (Silverman et al. 2012, Supplementary Figure 1), it is apparent that a 5-year lag showed the worst model fit of all lags (0-25 years); thus, it does not make sense to use this particular analysis as the primary exposure-response relation simply because the label "5-year lag" coincides with that of the other two studies.* *We acknowledge that the interpretation of the risk function may be affected by differences in exposure between lag times. For this reason, we performed sensitivity analyses that included different lags from each study; overall results changed only slightly. We extended our earlier sensitivity analyses by including the unpublished 5-year lagged data from Silverman et al. (Silverman DT, personal communication). We note again, however, that these 5-year lagged data from the DEMS do not fit nearly as well as the 15-year lagged data. We stress our belief that they should not be the primary data for use in any risk assessment/meta-regression.* *Figure 1 includes the individual risk estimates for all three studies, with three alternative lag times for DEMS. It is clear from Figure 1 that the 5-year lagged risk estimates for DEMS selected by Crump for his meta-analysis are considerably lower than those of the two trucking studies and the alternative lag times for DEMS. We also included in the figure three different regression lines based on the two trucking studies and one of three different sets of results for DEMS, obtained using the exposure data lagged 0, 5, and 15 years. All models are fitted using the full estimated covariance matrix to appropriately account for the correlation between categorical point estimates from the same study, a correction ignored by Crump. The lowest meta-regression slope, using the 5-year lagged exposure results from DEMS, a) is higher than that reported by Crump using the variance estimates only; b) is statistically significant (0.00065; 95% CI: 0.00028, 0.0010); and c) falls within our previous sensitivity analyses (Vermeulen et al. 2014). It is also clear that the exposure-response function derived using the 15-year lagged exposure data from DEMS is a much better fit overall.*  **Figure 1.** Predicted exposure–response curve of cumulative elemental carbon (EC) and lung cancer risk using different lag-times based on a log-linear regression model using relative risk estimates from the three cohort studies.						

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Crump et al. 2015 (Reanalysis of DEMS study)	Reanalysis of DEMS case-control data to evaluate suitability for quantitative risk assessment. Re-analysis used conditional logistic regression and adjusted for cigarette smoking in a manner similar to the original DEMS analysis. However, also includes were additional estimates of DEE exposure & adjustment for radon exposure.					Without adjusting for radon, results were similar to those in the original DEMS analysis: all but one of the nine DEE exposure estimates showed evidence of an association between DEE exposure and lung cancer mortality, with trend slopes differing only by about a factor of two. When exposure to radon was adjusted, the evidence for a DEE effect was greatly diminished, but was still present in some analyses that utilized the three original DEMS DEE exposure estimates. A DEE effect was not observed when the six alternative DEE exposure estimates were utilized, and radon was adjusted. No consistent evidence of a DEE effect was found among miners who worked only underground. OR (95%CI) for ever underground workers with cumulative exposure lagged 15 years were significant for some exposure cutoffs when not adjusted for radon exposure. A statistically significant OR with an apparent concentration response appears to be observed for the following estimates: • DEMS_REC1: At exposures ≥ 383.5 µg/m^3.yrs • DEMS_REC2: At exposures ≥ 318.2 µg/m^3.yrs • DEMS_REC3: At exposures ≥ 157.1 µg/m^3.yrs • REC1: At exposures ≥ 649.2 µg/m^3.yrs • REC2: At exposures ≥ 556.5 µg/m^3.yrs • REC3: At exposures ≥ 693.9 µg/m^3.yrs • REC4: At exposures ≥ 329.8 µg/m^3.yrs • REC5: At exposures ≥ 231.4 µg/m^3.yrs • REC6: At exposures ≥ 204.9 µg/m^3.yrs ORs were not significant when analysis was limited to workers who only worked underground (presumed to have higher DE exposure).	It is noted when examining the exposure cut-points (for cumulative or average REC) many of the ORs are not statistically significant (their 95% CI encompass 1). Where they are significant, they do not show a clear exposure-response except for ever underground workers with cumulative exposure lagged 15 yrs (see previous column).

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Crump et al. 2016 (reanalysis of DEMS study)	Developed new estimates of REC exposures using historical data on use of diesel equipment, diesel engine horsepower (HP), mine ventilation rates, and the documented reduction in particulate matter emissions per HP in diesel engines from 1975 through 1995. These new REC estimates were applied in a conditional logistic regression of the DEMS nested case-control data very similar to the one applied in the original DEMS analyses.			None of the trend slopes calculated using the new REC estimates were statistically significant ($p > 0.05$). The trend slopes were smaller by roughly factors of five without control for radon exposure and factors of 12 with control for radon exposure compared to those estimated in the original DEMS analyses. Also, the 95% confidence intervals for these trend slopes had only minimal overlap with those for the slopes in the original DEMS analyses.			*These results underscore the uncertainty in estimates of the potency of diesel exhaust in causing lung cancer based on analysis of the DEMS data due to uncertainty in estimates of exposures to diesel exhaust.*
Morfeld and Spallek 2015 (reanalysis of DEMS study)	Performed extended re-analysis using different modelling approaches (fixed and random effects regression analyses, Greenland/Longnecker method) and explored the impact of varying input data (modified coefficients of Garshick et al. 2012, results from Crump et al. 2015 replacing Silverman et al. 2012, modified analysis of Moehner et al. 2013).			*We reproduced the individual and main meta-analytical results of Vermeulen et al. 2014. Of all the meta-analyses we performed, the evaluation of the data as used by Vermeulen et al. resulted in the highest risk estimates. However, our analysis demonstrated a heterogeneity of the baseline relative risk levels between the three studies (Vermeulen did not report on this). Heterogeneity refers to a systematic difference in the baseline risk of the three studies (i.e., setting exposure to zero). Other authors in other situations reject combinations of studies even with considerably less pronounced heterogeneity. This uncertainty in the baseline level renders the use of the analysis by Vermeulen et al. a problem for risk estimates in the lower exposure region.* *The meta-coefficient was estimated to be about 10–20 % of the main effect estimate in Vermeulen et al. 2014 in this analysis.* *….the present re-analysis also revealed that the results of the meta-regression study by Vermeulen et al. [1] should not be used in any quantitative lung cancer risk evaluation without reservations, as the results vary significantly depending on the input data selected and the statistical methods used. This is particularly true for the low exposure region.*			
Vermeulen et al. 2020 (reanalysis of DEMS study)	To address two issues that have been raised in critique of DEMS study analysis (use of historical CO measurements to calibrate exposure model and potential confounding by radon), authors developed alternative REC estimates using models that did not rely on CO but rather on estimated use of diesel equipment, mine ventilation rates and changes in DEE emission rates over time.			Validation of the new REC exposure estimates indicated that they overestimated historical REC by 200–400% (absolute differences >170–400 µg/m3 REC and relative differences >100%), compared with only 10% for the original estimates. Exposure levels from these models were >1000 µg/m3 for many jobs. REC exposure levels >1000 µg/m3 have not been reported in the literature and seem unrealistically high. Effect estimates for lung cancer using these alternative REC exposures or adjusting for radon typically changed by <10% when compared with the original estimates.			

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Steenland et al 1998	Original study was case-control, this is an exposure-response study based on the original study.	994 lung cancer cases, 1085 controls (died in 1982-1983). Analysis was restricted to long-haul drivers (n=1237), short-haul drivers (n=297), dockworkers (n=164), mechanics (n=88) and those outside the trucking industry (n=120).	Exposures estimated based on a 1990 industrial hygiene survey. Past exposures estimated as a function of # of heavy duty trucks on road, particulate emissions of diesel engines over time, and leaks from trucks' exhaust systems. Median EC (across all groups): 372.9 (range 0.45-2439.9) $\mu g/m^3$.yrs. Without background and assuming 4.5gm/mile emission.	Not reported	Lung cancer	• Regardless of assumptions about past exposure, all analyses resulted in significant positive trends in lung cancer risk with increasing cumulative exposure. • OR (95% CI) for highest quartile were significantly different from others. No lag >360 $\mu g/m^3$.yrs 1.72 (1.11-2.64); 5-yr lag >331 $\mu g/m^3$.yrs 1.64 (1.09-2.49).	Adjusted for age, race, smoking, diet and reported asbestos exposure Results depend on very broad assumptions on exposure estimates with extrapolations to earlier time periods. Unknown whether drivers also exposed to leaky emissions from their own trucks.
Bassig et al. 2017	Cross-sectional molecular epidemiology study	54 males occupationally exposed to DEE in a diesel engine manufacturing facility, 55 unexposed male controls from representative workplaces in China.	EC (background-adjusted): 48.5 ± 22.1 µg/m3 (EC was not correlated with PM2.5).	Mean employment duration for exposed workers: 20 ± 7 years	Plasma levels of 64 immune / inflammatory markers. Compared to findings from nested case-control study of these markers & lung cancer risk, conducted among never-smoking women in Shanghai.	Levels of 9 markers that were associated with lung cancer risk in Shanghai study were altered in DEE-exposed workers in the same direction as the lung cancer associations. Two of these (CRP and CCL15/MIP-1D) were observed in workers with increasing EC levels (p trend <0.05).	Small sample size, prevented sufficient analysis with smoking status. All male participants which may not directly compare with female markers.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Dai et al. 2018 (same group as in Bassig et al. 2017)	Cross-sectional molecular epidemiology study	41 diesel engine testing workers, 46 unexposed controls	$PM_{2.5}$ (mg/m3) (unadjusted for background): 0.37 ± 0.07, 0.15 ± 0.07 (adjusted) EC (µg/m3): 58.1 ± 24 (unadjusted), 47± 24 (adjusted) Organic carbon (µg/m3): 138.1 ± 27.2 (unadjusted), 69.4 ± 27.2 (adjusted)	Mean employment duration for exposed workers: 20.8 ± 6 years	Serum levels of 6 inflammatory biomarkers.	• MIP-1β significantly ↓ by 37% ($p<0.001$) with decreasing trend in $PM_{2.5}$ in all subjects & exposed subjects only. • IL-8 and MIP-1β significantly lower in workers with highest exposure tertile to PM2.5 (>397 µg/m3).	*Given that IL-8, MIP-1b, and MCP-1 are chemokines that play important roles in recruitment of immunocompetent cells for immune defence and tumour cell clearance, the observed lower levels of these markers with increasing PM2.5 exposure may provide insight into the mechanism by which DEE promotes lung cancer.*
Du et al. 2019	Statistical association (point-in-time observational)	80 underground miners and 20 surface miners at Australian gold mine	Underground workers (median exposures): EC: 56.47 µg/m3 VOCs: 108.6 µg/m3 NO2: 0.33 µg/m3 Particle number: 52,740 Particle size: 57.8nm Surface workers: EC: 0.97 µg/m3 VOCs: 48.03 µg/m3 NO2: 0.02 µg/m3 Particle number: 8763 Particle size: 50.4nm	12 hours (end of shift measurements)	Lung function (by spirometry)	Cross-shift reduction in Z-score value of FEV1/FVC in underground miners was statistically significantly greater than those of surface miners. The cross-shift change in Z-score value of FEV1/FVC was associated with exposure to higher concentration of EC and particle number, but not with VOCs, NO2 and particle size.	Small sample size, measures of DEE may contain inherent bias, unable to adjust for diurnal variation influence on lung function.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Duan et al. 2016	Statistical association (point-in-time observational)	101 male diesel engine testing workers and 106 matched controls	Exposed vs. controls: • PM2.5: 288.3 ± 107.9 vs. 91.9 ± 3.36 µg/m3 • EC: 114 ± 66.7 vs 11.8 ± 0.61 µg/m3 • NO2: 0.299 ± 0.15 vs. 0.04 ± 0.0005ppm • PAHs: 4,937 ± 1305 vs. 34.85 ± 1.43 ng/m3	8.31 years (range 1-37 years)	Post-shift urine samples analysed for 1-hydroxypyrene (an internal exposure marker for DEE). Levels of DNA strand breaks and oxidised purines measured. Urinary 8-OHdG determined.	• Significantly higher urinary 1-OHP (p<0.001). • Significantly higher parameters for oxidative DNA damage in normal comet and FPG-comet assay, dose- and time-dependent. • No significant differences in urinary 8-OHdG levels.	Authors indicate this study shows DEE induces DNA damage which could be used as an early biomarker for DEE risk assessment.
Gamrad-Streubel et al. 2021 (Abstract only)	Epidemiological health study	Underground salt and potash miners (n=801) and maintenance workers (n=202) cf. above ground workers (n=243).	Personal exposure measurements among underground workers exceed OELs 19% for NO2 (0.5 ppm), 33% for NO (2 ppm) and 56% for EC-DPM (50µg/m3).	Not stated in abstract.	Biomarkers of acute and chronic health effects (specific biomarkers not listed in abstract).	• Majority of biomarkers in reference ranges. • 80-100% were similar among all exposure groups. • Acute effects biomarkers not statistically significantly different between post- and pre-shift samples in any group. • Graphical evaluations and ANCOVA results did not identify consistent associations between exposure quintiles and biomarkers of health effects for underground personnel	*Study results suggest that underground exposures don't have a negative effect on the health of underground salt and potash miners.*

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Garschick et al. 2004	Cohort mortality study	54,973 US railroad workers (n=4,351 lung cancer deaths)	No exposure concentrations available. Exposures grouped by job code and information from hygiene survey.	Study duration: 38 yrs (1959-1996)	Lung cancer mortality	• RR for workers operating trains with 5 yr exposure lag: 1.4 (1.3-1.51). • No increase with increasing years of work. • Adjustment for smoking (using broad adjustment factors) attenuated RR to between 1.17 and 1.27.	Adjusted for healthy worker survivor effect & age. *Although a contribution from exposure to coal combustion products before 1959 cannot be excluded, these results suggest exposure to DE contributed to lung cancer mortality.*
Garschick et al. 2012	Retrospective cohort mortality study	31,135 male workers employed in the unionised US trucking industry in 1985.	No exposure measurements. Statistical model used to estimate historical work-related exposures to EC. Wide range of cumulative EC exposure (50th percentiles were 1,061 µg/m^3.mths for entire cohort).	Mean cumulative yrs of work in the study cohort was 21.6 years	Lung cancer mortality	• Duration of employment inversely associated with lung cancer risk (consistent with healthy worker survivor effect). • Suggestive linear-response relationship for each 1000µg/m3 months cumulative EC based on 5-yr lag, HR was 1.07 (0.99-1.15). • Average exposure not associated with relative risk.	Suggestive association, not statistically significant.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Järvholm and Silverman 2003	Cohort mortality study	6,364 male truck drivers and 14,364 drivers of heavy construction vehicles (n=119,984 controls) in Sweden	No exposure measurements.	Not specified.	Lung cancer mortality	• Heavy construction vehicle drivers had no increased risk of lung cancer compared to controls. Significant inverse trend risk with increasing use of cabins. • Truck drivers had increased risk of lung cancer (SIR = 1.29, 0.99-1.65).	SIR was not significant as 95% CI did not encompass 1. Unable to quantify intensity & duration of DEE exposure.
Kachuri et al. 2016	Case-control study	N=931 colon and n=840 rectal cancer cases and n=1360 controls from 7 Canadian provinces in 1994-1997.	No exposure measurements. Occupational hygienists, blinded to case–control status, assigned exposures to each job for 3 dimensions: concentration, frequency, and reliability.	Ranged from >0 to >31 years.	Colon and rectal cancer	• OR for colorectal cancer: 1.65 (0.98-2.8). • OR for rectal cancer: 1.98 (1.09-3.6). (for highest attained exposure concentration) • Risks were not elevated for colon cancer. • Prolonged (>10 yrs) exposure at high concentrations was associated with high risks of rectal cancer (OR = 2.33, 0.94-5.78, p-trend = 0.02).	Adjusted for age, province, use of proxy, respondents, smoking, BMI, physical activity, alcohol intake, processed meats, and exposure to asbestos & aromatic amines. Findings suggestive of modest association between high concentrations of DEE rectal cancer.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Koutros et al. 2020	Case-control study	N=1944 cases, n=2135 controls pooled from two case-control studies in US and Spain.	Lifetime occupational histories combined with exposure-oriented questions were used to estimate cumulative exposure to respirable elemental carbon (REC) Cumulative µg/m3 REC-yrs: Medians 15-39 Interquartile ranges: 3-116	Duration of diesel-exposed jobs (medians) ranged from 23-29 yrs.	Urothelial cell carcinoma of the bladder	• Workers with cumulative REC>396 µg/m^3.yrs had OR of 1.61 (1.08-2.4). • When US and Spain studies were separated, the OR was not longer significant. • Increased risk was found for all lag intervals evaluated (5-40 yrs).	Non-differential exposure misclassification may have occurred. No info on exposure intensity.
Latifovic et al. 2015	Case-control study	658 bladder cancer cases, 1360 controls from Canadian National Enhanced Cancer Surveillance System.	Job-exposure matrix constructed from information on lifetime occupational histories and supplemented by expert review to assign values for each job. (no exposure concentrations provided)	Cutoff groups for duration of exposure ranged from <7 to ≥26 yrs.	Bladder cancer	• Men ever exposed to DEE at increased risk (OR = 1.64, 0.87-3.08) but result not significant. • Those with >10 yrs exposure at high concentrations had higher OR (2.45, 1.04-5.74).	Adjusted for proxy respondent, province, aget, cigarette pack-yrs, cumulative asbestos & silica exposure. Used self-reported occupational histories, could not account for exposure variability.
Lee et al. 2010	Statistical association (point-in-time observational)	9 males and 2 female diesel engine exhaust inspectors from central Taiwan	Environmental and personal concentrations of $DEP_{2.5}$ were 107.25±39.76 (mean±SD) and 155.96±75.70 µg/m3	Information not provided.	Urinary 8-OHdG (biomarker for oxidative stress)	• The level of urinary 8-OHdG in the exposed group was higher than that of the control group, by 7.47 µg/g creatinine (P = 0.028). Smoking and cooking at home were not found to be a confounder in this study.	PAH levels not measured in study.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
León-Mejía et al. 2019	Statistical association (point-in-time observational)	120 DEE exposed mechanics and 100 non-exposed controls in Columbia.	PM2.5: 250 µg/m3 Alkylnaphthalenes made up ~1.2% of the dust collected on filter.	Mean duration of service 12 ± 10 yrs (3-35 yrs)	Biomarkers for cytotoxicity and genotoxicity in peripheral blood lymphocytes.	• Significant differences found for various biomarkers between exposed and non-exposed group.	Significant correlations were also found between some of the biomarkers, meat and vitamin consumption, alcohol intake & family cancer history. It is unclear whether results were adjusted for these factors.
León-Mejía et al. 2020	Statistical association (point-in-time observational)	123 DEE exposed mechanics in Columbia. (as above)	Not specified in this paper. Presumably same cohort as above		Polymorphism effect on genotoxicity profiles.	• Found an association between various polymorphisms and the genotoxicity findings.	Indicates genetic polymorphisms play a role in potential susceptibility to adverse effects of DEE.
Möhner et al. 2013 (reanalysis of potash miner study)	Cohort study	5,819 potash miners (employed for at least 1 year after 1969).	TC and EC measurements from 1991 used for designing job-exposure matrix. Cumulative mean REC exposure (µg/m^3.yrs) differed for each decade ranging from 672 (in <1920) to 1,957 (in 1940-1949).	Not clearly stated, but many miners recruited into study had already worked 13 yrs in potash mine before diesel technology was introduced. Mean underground tenure was 20 yrs.	Lung cancer mortality	• Analysis did not show any notable association between cumulative REC exposure and lung cancer risk. • Introducing cumulative REC exposure as a continuous variable into the conditional logistic regression model yielded an odds ratio of OR = 1.04 [0.70–1.53]95 % adjusted for smoking and previous employment. • The study results give no evidence for an association between REC exposure and lung cancer risk.	Exposure transformation of 1991 measured data may have increased chance of exposure misclassification. Lung cancer cases low (n=61) resulting in wide confidence intervals.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Lotz and Kersten 2012 (Additional analysis of potash mining cohort in Germany) (Abstract only)	Reanalysis of cohort study	568 miners (mean age 45 yrs) re-examined after a 5 yr period.	Mean individual lifetime exposure concentrations were 10.1 mg/m3 of inhalable and 1.46 mg/m3 of respirable salt dust, 0.12 mg/m3 of diesel exhaust (EC), 0.78ppm NO2 and 1.59ppm NO.	Information not provided in abstract		• Analysis indicated clear concentration response relationships in both models and confirmed results of previous study. • With linear regression, loss of FEV1 of 11.5 mL/yr was predicted in relation to average exposure in 5-yr period, with a stronger effect for respirable dust by itself. • Impossible to determine effect of a single exposure component separately because of high correlation of individual concentrations (>0.8).	
Parent et al. 2007	Population based case-control	857 male lung cancer patients, 533 controls, 1349 patients with other cancer types in Montreal Canada (1979-1985)	Industrial hygienists translated each job description into indices of exposure to several agents, including engine emissions. No EC measurements.	Mean duration of exposure was 20 years.	Lung cancer	• When cancer controls were considered, there was no excess risk to DEE exposure. • When population controls were studied, the OR after adjustments for potential confounders, were 1.2 (95% confidence interval: 0.8, 1.8) for any exposure and 1.6 (95% confidence interval: 0.9, 2.8) for substantial exposure.	OR obtained were not statistically significant (overlapping CIs).

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Pedersen et al. 2021	Case-control	38,375 women <70 yrs with incident breast cancer and 5 breast cancer-free controls per case in Denmark.	Full employment history was obtained for all study subjects from a nationwide pension fund, and exposure to diesel exhaust and PAH was assessed using a job exposure matrix. No EC concentrations reported.	By summing years of employment in all exposed calendar periods. Ranged from 1 to >20 years.	Breast cancer	• No noteworthy associations were observed for overall breast cancer in women exposed to DEE. • DEE modestly elevated the risk of estrogen receptor negative breast tumors before age 50 [OR 1.26, 95% confidence interval (CI) 1.09–1.46]. • No notable risk patterns were generally observed for PAH exposure.	Not possible to account for potential confounding of certain lifestyle factors (e.g. obesity, alcohol consumption, physical inactivity, use of oral contraceptives, etc). May still be explained by confounding.
Peters et al. 2013	Case-control	306 cases of childhood brain tumours and 950 controls in Australia	No exposure concentrations. Parental occupational exposure assessed via questionnaire based on job characteristics obtained from detailed occupational histories.	Not stated.	Childhood brain tumours from parental occupational exposure	• Increased risks were observed for maternal exposure to DEE any time before the child's birth (OR 2.03, 95% CI 1.09–3.81) and paternal exposure around the time of the child's conception (OR 1.62, 95% CI 1.12–2.34). • No clear associations with other engine exhausts were found.	Parents of cases may consider past exposures more than control parents, so recall bias may result in overestimated relative risk estimates. However, information on jobs and tasks instead of self-reported exposures was used, where the latter is more prone to recall bias

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Peters et al. 2018	Case-control	712 kidney cancer cases, 2457 controls from National Enhanced Cancer Surveillance System (NECSS) in Canada (1994-1997)	No exposure concentrations. Self-reported questionnaires were used to obtain information on lifetime occupational history and cancer risk factors. Two hygienists, blinded to case status, coded occupational histories for diesel and gasoline exhaust exposures using concentration, frequency, duration, and reliability.	Not stated.	Kidney cancer in men	• Workers who had ever been exposed to engine exhausts were more likely to have kidney cancer than those who were never exposed (OR diesel = 1.23, 95% CI = 0.99–1.53; OR gasoline = 1.51, 95% CI = 1.23–1.86). • Those men with high cumulative exposure to both gasoline and diesel exhaust had a 76% increased odds of kidney cancer (95% CI = 1.27–2.43).	OR for diesel exhaust was not significant.
Pintos et al. 2012	Population based case-control	Study I (1979-1986) comprised 857 cases and 533 population controls; study II (1996-2001) comprised 736 cases and 894 population controls.	A detailed job history was obtained, from which lifetime occupational exposure to 294 agents was inferred, including diesel engine emissions. No exposure concentrations provided.	Not stated per se, but categories split ranged from 1 to >20 yrs.	Lung cancer	• Increased risks of lung cancer were found in both studies. The pooled analysis showed an OR of lung cancer associated with substantial exposure to DEE of 1.80 (95% CI 1.3 to 2.6). • The risk associated with substantial exposure was higher for squamous cell carcinomas (OR 2.09; 95% CI 1.3 to 3.2) than other histological types. • Joint effects between DEE and tobacco smoking are compatible with a multiplicative synergistic effect.	Adjusted for socio-economic factors, smoking history and selected occupational carcinogens. Potential for exposure misclassification, lack of quantitative exposure data.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Richiardi et al. 2006	Population based case-control	595 lung cancer cases (diagnosed in 1991-1992) and 845 controls in Turin, Italy.	Occupational histories used to evaluate probability, intensity and frequency of exposure. No exposure concentrations provided.	Not stated	Lung cancer	• OR for ever exposure to DEE was 1.04 (95% confidence interval 0.79–1.37). • No association was found with intensity, probability and duration of exposure.	Adjusted for age, sex, smoking and having worked in occupations entailing exposure to known lung carcinogens.
Rumchev et al. 2020	Statistical association (point-in-time observational)	N=2598 miners in Western Australia for which exposure measurements were available.	Secondary data collected from 2006 to 2012 Measured EC concentrations ranged between 0.01 mg/m3 and 1.00 mg/m3 and tended to significantly decrease over the study period ($p < 0.001$).	Study period 2006-2012	Prevalence of respiratory symptoms	• Underground mine workers were exposed to significantly higher ($p < 0.01$) median EC concentrations of 0.069 mg/m3 (IQR 0.076) when compared to surface workers' 0.038 mg/m3 (IQR 0.04). • Overall, 29% of the miners reported at least one respiratory symptom, with the highest frequency recorded for cough (16%). Respiratory symptoms have also declined with time in line with reduced EC.	Reliance on self-reported respiratory symptoms without medical diagnoses, reliance on secondary sourced exposure data.
Rynning et al. 2019	Statistical association (point-in-time observational)	N=69 tunnel finishing workers (TFW) and 69 referents in Norway	No exposure concentration measurements.	Not stated.	Peripheral blood mononuclear cell (PBMC) bulky DNA adducts & expression of microRNAs, circulating free arachidonic acid in plasma	• PBMC from TFW showed significantly higher levels of DNA adducts compared with referents. • Levels of DNA adducts were also related to smoking habits.	

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Talibov et al. 2019	Case-control study	181,709 colon cancer and 109,227 rectal cancer cases diagnosed between 1961 and 2005 in Finland, Iceland, Norway and Sweden.	Diesel exhaust and gasoline exposure values were assigned by country-specific job-exposure matrices. No exposure concentrations provided.	Assumed to be 45 years, unless occupations changed throughout census.	Colorectal cancer	• Diesel exhaust exposure was associated with a small increase in the risk of rectal cancer (odds ratio = 1.05, 95% confidence interval 1.02-1.08). • Gasoline exposure was not associated with colorectal cancer risk.	Adjusted for physical strain at work and occupational exposure to benzene, formaldehyde, ionizing radiation, chlorinated hydrocarbons, chromium, and wood dust.
Wang et al. 2018	Statistical association (point-in-time observational)	Cohort of 137 diesel engine testers and 127 referents.	REC not reported.	8.5 years median exposure (Q1-Q3: 5.6-9.6 yrs)	Urinary metabolites, FeNO, serum markers & spirometry to assess local & systemic inflammation.	• A 19% reduction in CC16 and a 94% increase in CRP were identified in DETs compared with non-DETs (all p values $<10^{-4}$), which were further corroborated by showing a dose–response relationship with internal dose for DE exposure (all p values <.04) and a time–course relationship with DE exposure history (all p values <.005). • Mediation analysis showed that 43% of the difference in FEV1 between DETs and non-DETs can be explained by circulating CC16 and CRP (permuted p <.001). • An inverse dose-dependent relationship between FeNO and internal dose for cigarette smoke was identified (p = 0.0003).	Cross-sectional design does not allow for assessing effect of local and systemic inflammatory markers measured at baseline and their change over time on subsequent FEV1 decline.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Wild et al. 2016	2-phase case-control	246 cases, 531 controls in an area with high lung cancer rates in Northeast France	Cumulative expert-based exposure scores obtained from subset of 215 cases and 269 controls stratified on smoking. Exposure concentrations not reported.	Not stated.	Lung cancer	• Based on this 2PCC design, statistically significant dose–response relationships were obtained for asbestos, crystalline silica, PAH, and diesel motor exhaust. • OR for DEE exposure & lung cancer was 1.205 (1.035-1.402), when split into tertiles only highest tertile was statistically significant (2.13, 1.07-4.22).	The 2PCC design may be the design of choice when resources allow only a limited number of subjects with a full expert-based exposure assessment.
Zhang et al. 2015 (same group as Zhang et al. 2016)	Statistical association (point-in-time observational)	117 DEE-exposed workers and 112 non-DEE exposed workers	Exposed vs, non-exposed. PM2.5: 267.45 vs 91.88 µg/m3 EC: 113.69 vs 11.81 µg/m3 Total PAHs: 4.76 vs 0.03 µg/m3	8.09 ± 5.73 years	Urinary levels of OH-PAHs as well as indicators of genetic damage in peripheral blood lymphocytes (PBLs), i.e. micronucleus (MN), nucleoplasmic bridge (NPB), and nuclear bud (NBUD) frequencies.	• The DEE-exposed workers exhibited significantly higher MN, NPB, and NBUD frequencies than the non-DEE-exposed workers (P <0.05). Among all study subjects, increasing levels of all 4 urinary OH-PAHs, on both quartile and continuous scales, were associated with increased MN, NPB, and NBUD frequencies (all P <0.05).	Results show that exposure to DEE can induce increases in MN, NPB, and NBUD frequencies in PBLs and suggest that DEE exposure level is associated with MN frequencies.
Zhang et al. 2016	Statistical association (point-in-time observational)	117 DEE-exposed workers and 112 non-DEE exposed workers	Urinary PAH concentrations. See above for exposure measurements.	Exposure duration ranged from 0 to >8 years.	Methylation levels of three DNA damage response-related genes.	• DEE-exposed workers showed significantly lower mean promoter levels. • Increasing quartiles of urinary summed OH-PAHs was associated with hypomethylation of a number of genes.	Provides potential suggestive mechanism for mediation of cancer risk. Did not measure nutrition factors, known to impact DNA methylation.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Meta-analyses or review papers or papers of interest							
Boffetta (2014)	Meta-analysis	26 studies investigating risk of pancreatic cancer and occupational exposure to DE (n=5 routine statistics, n=11 case-control, n=10 cohort)	Not provided	Few studies reported results according to duration of exposure.	Pancreatic cancer	No consistent pattern emerged. Meta-RR (95% CI): • Case-control: 0.9 (0.5-1.6). • Cohort: 1.03 (0.93-1.13).	*Overall evidence leads to conclusion of absence of an association.* *The strongest (albeit still quite weak in absolute terms) evidence in favour of an association comes from the studies based on routinely collected data, which have the weakest design, while the results of cohort studies, those with the strongest design, do not point toward an association.*

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Boffetta et al. 2004	Meta-analysis (no formal pooling possible)	9 case-control studies, 9 based on routine statistics (no cohort studies found).	Not specified	Not specified	Leukaemia (in particular acute myeloid leukaemia).	The available studies do not consistently suggest an increased risk of leukaemia, and specifically AML, among workers exposed to DE. For none of the occupational groups potentially exposed to DE, the results suggest an association, and sporadic positive results are counterbalanced by negative associations and might result from reporting bias. DE exposure does not appear to be associated with increased risk of leukaemia.	*The available evidence does not support the hypothesis of an association between DE exposure and risk of leukemia, and of AML in particular. Although studies might suffer from lack of power, misclassification of exposure and outcome, it seems unlikely that these factors obscure an existing association.*

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Ge et al. 2020	Pooled case-control analysis	16,901 lung cancer cases and 20,965 controls.	EC job-exposure matrix used for exposure assessments constructed based on 4,417 EC measurements from other sources.	Exposure duration categories ranged from 1->29 years.	Lung cancer mortality (excess lifetime risks or ELRs).	• In men, ORs were 1.09 (1-1.18) and 1.41 (1.3-1.52) for the lowest and highest cumulative exposure groups, respectively. • Associations were strongest for squamous and small cell carcinomas and weaker for adenocarcinoma. • Association was observed regardless of smoking history. • ELRs for 45 yrs of exposure at 50, 20, and 1 µg/m3 estimated to be 3%, 0.99% and 0.04%, respectively. • Increased risk among lowest exposure group was seen at median EC of 3.3 µg/m^3.yrs.	Controlled for smoking and exposure to other occupational carcinogens. Did not account for changes in exposure at different time periods, which could have underestimated exposure. EC data was collected from 1985-2016, whereas subjects were exposed from 1923-2020.
Olsson et al. 2011	Pooled case-control analysis	13,304 lung cancer cases and 16,282 controls from 11 case-control studies conducted in Europe and Canada.	A general population job exposure matrix based on ISCO-68 occupational codes assigned no, low, or high exposure to DEE.	Duration of exposure for cases ranged from 1 to >30 years.	Lung cancer	• Cumulative DEE exposure was associated with ↑ lung cancer risk in highest quartile vs unexposed (OR 1.31, 1.19-1.43) with significant exposure-response relationship (p<0.01).	Adjusted for smoking and other occupational exposures.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Sun et al. 2014	Systematic review	42 cohort studies & 32 case-control studies	To facilitate comparison between studies, a job-exposure matrix (JEM) of DEE exposures was created based on 4,000 historical industrial measurements.	Varies.	Lung cancer	• Neither cohort nor case-control studies indicate a clear exposure-response relationship between DEE exposure and lung cancer. • Epidemiological studies published to date do not allow a valid quantification of the association between DE and lung cancer.	Only two studies contain industrial hygiene measurement data (Silverman et al. 2012, Möhner et al. 2013). In all remaining studies, the exposure assessments are based on expert judgements. Differences in expert opinion can have a strong influence on estimated exposure-response relationships.

Study reference	Type of study	Population studied	Exposure concentrations	Exposure duration	Health effects assessed	Findings	Comments
Tsoi and Tse 2012	Systematic review / meta-analysis	19 studies investigating professional driver risk of lung cancer (1996-2011).	No exposure concentrations provided.	6 to >30 years, depending on the study.	Lung cancer	• Significantly increased risk of lung cancer (pooled smoking-adjusted RR 1.18, 95% CI 1.05 to 1.33) among professional drivers after combining four cohort studies and nine case-control studies. • A higher pooled RR was observed among smoking-adjusted studies reporting 10 yrs or more of employment (RR 1.19, 95% CI 1.06 to 1.34) as compared with the study having a shorter duration of employment (6 years) (RR 1.00, 95% CI 0.92 to 1.09).	Significant heterogeneity across studies. Potential confounding effects in some studies by smoking, and potential confounding by other occupational carcinogens. None of the studies distinguished between short & long-distance drivers.

References for Appendix B

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APPENDIX C

Animal studies potentially informative for concentration response

Note: Blue shaded rows identify those animal studies with information for DEP and fDEP considered in the report when evaluating whether the effects (adverse or biochemical) observed are related to the gaseous or particle component of diesel exhaust.

Bolded text in rows identify those long-term animal studies with three or more exposure concentrations considered for identification of a departure point (e.g. NOAEC based on adverse effects).

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Bai et al., 2011a, b	TDE (2002 model year turbocharged direct-injection 5.9-L Cummins, using Number 2 Diesel Certification Fuel with 454ppm sulfur) (Gould et al. 2008)	Inhalation exposure study in ApoE knockout mice	DEP (NO_2 also measured)	200 µg/m^3 ($PM_{2.5}$) (6h/d, for 5d/wk, for 7 wk) (as provided in Gould et al. 2008)(1)	↑plaque foam cell formation (concomitant with ↑oxidative stress markers and ↑expression of iNOS and CD36 in aortic root in the lungs)	Not identified	200 µg/m^3 (35µg/m^3 averaged over 24 hours) (NO_2 = 0.035ppm)
Bemis 2015	NTDE Detroit Diesel 2007 model Series 60 14L heavy-duty diesel engine	Long-term inhalation study (micronucleus formation) in Wistar Han rats	Diesel exhaust (NO_2)	NO_2 = 0.1, 0.8 or 4.2ppm (16h/d, 5d/wk, up to 24M) (NB: DEP = 23.3, 20 or 27.3µg/m^3)	No sex-based differences in micronuclei frequency were observed in the rats.	NO_2 = 4.2ppm (DEP <30µg/m^3)	-
Bernal-Meléndez et al 2016	Not determined (Engine type, fuel used and sulfur content not stated)	Short term reproduction inhalation exposure study in pregnant rabbits	DEP	1,000 µg/m^3 (2h/day, 5d/wk, GD 3 to 28)	Neuro-olfactory development in pups (nanosize particles in in olfactory sensory neurons and glomerular layer of the olfactory bulb as well as ↑ serotonin levels and ↓ dopamine levels and metabolites in olfactory bulb).	Not identified	1,000 µg/m^3 (NO_2 concentrations not reported)
Bernal-Meléndez et al. 2019	TDE (presumed as no catalytic converter fitted and no mention of diesel fuel type used) 25KVA Loxam engine, with a 500 nm particle filter (fuel used and sulfur content not stated)	Short-term repeated inhalation study (gestational, developing brain) in New Zealand white pregnant female rabbit	DEP	1,000µg/m^3 DEP exhaust (2h/d, 5d/wk, from GD3 to GD27) (NO_2 concentration not reported)	Nano-sized particles were observed in cilia and cytoplasm of the olfactory sensory neurons in the olfactory mucosa and in the cytoplasm of periglomerular cells in the olfactory bulbs of exposed foetuses. Moreover, cellular and axonal hypertrophies were observed throughout olfactory tissues. Concomitantly, foetal serotoninergic and dopaminergic systems were affected in the olfactory bulbs. Moreover, the neuromodulatory homeostasis was disturbed in a sex-dependent manner in olfactory tissues. At birth, the olfactory sensitivity to 2MB2 was reduced in exposed PND2 pups	Not identified	1,000µg/m^3 (NO_2 = 0.7ppm, Valentino et al. 2016, Table S2)
Burchiel et al. 2004	TDE (2000 model Cummins ISB Turbo Diesel 5.9-l engine using Number 2 Diesel Certification Fuel, sulfur content not stated)	Immunotoxicity whole body exposure inhalation study in AJ mice	DEP	30, 100, 300, and 1000 µg/m^3 (6hr/d, 7 d/week for 6Ms)	Suppressed proliferative response of splenic T cell mitogenesis (an indicator of systemic immunity in females AJ mice)	Not identified	30 µg/m^3 (NO_2 concentrations not reported)
Campen et al. 2003	TDE (2000 Cummins 5.9l ISB turbo engine using Number 2 Diesel Certification Fuel, 340ppm sulfur)	Short-term inhalation study (cardiovascular effects) in spontaneously hypersensitive mice	DEP	0, 30, 100, 300, & 1000µg/m^3 DEP exhaust (6h/d for 7d)	↑heart rate from 30µg/m^3 with prolongation of the PQ interval from 100µg/m^3	Not identified (heart rate) 30µg/m^3 (PQ Interval)	30µg/m^3 (heart rate) 100µg/m^3 (PQ Interval)
Campen et al. 2005	TDE (Presumed) (Single-cylinder Yanmar diesel generator engine (YDG5500), using grade number 2 diesel fuel)	Acute exposure inhalation study (cardiovascular effects) in ApoE deficient mice and C57BL/ 6J mice	DEP & fDEP (NO_2 also measured)	fDEP: 6 or 770µg/m^3 DEP: 512 or 3,634µg/m^3 (6h/d, for 3d) (Exposures conducted at same exhaust dilutions)	↓ heart rate (ApoE-/-) and T-wave area (high exposure) in both fDEP (high exposure) and DEP (high exposure) exposed groups	fDEP: 6µg/m^3 (NOx = 26ppm) DEP: 512µg/m^3 (NOx =19 ppm)	fDEP: 770µg/m^3 (NOx = 105ppm) DEP: 3,634µg/m^3 (NOx = 102ppm)
Campen et al. 2010	TDE (2000 Cummins 5.9l ISB turbo engine using Number 2 Diesel Certification Fuel, 340ppm sulfur)	ApoE knockout Mice inhalation exposure study	DEP & fDEP (NO_2 also measured)	unfiltered: 100, 300, and 1000 µg/m^3 filtered; 1000 µg/m^3 (6h/d, 7d/wk, for 50 ds) (Exposures conducted at same exhaust dilutions)	Exacerbation of atherosclerosis (changes in plaque composition, ↑transcription of several markers of vascular remodelling including MMP-9 and ET-1 and ↑ staining of macrophages). Filtering did not affect the result.	DEP: 100 µg/m^3 (NO_2 = 0.35ppm presumed) (2)	DEP: 300 µg/m^3 (NO_2 = 1.05ppm presumed) (2) fDEP: 1,000 µg/m^3 (NO_2 = 3.5ppm presumed) (2)
Carll et al. 2012	NTDE (presumed based on web search indicating it complies with Euro V) Single-cylinder, air-cooled, direct injection, 320cm3 Yanmar L70 V diesel engine with diesel fuel (32 ppm sulfur)	Acute exposure inhalation study (cardiovascular effects) in SHHF rats	DEP & fDEP (NO_2 also measured)	fDEP: 3µg/m^3 DEP: 472µg/m^3 (4h) (Exposures occurred to same air volume)	↓ heart rate, prolonged PR interval and ↓ blood pressure in the fDEP and DEP exposed groups. ↑ blood pressure, HRV triangular index, ↓decreased T-wave amplitude, ↑ atrioventricular (AV) block Mobitz II arrhythmias and ↑QTc and in the fDEP exposed group only.	Not identified	fDEP: 3µg/m^3 (NO_2 = 0.4ppm) DEP: 472µg/m^3 (NO_2 = 0.3ppm)

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Carll et al. 2013	NTDE (presumed based on web search indicating it complies with Euro V) 4.8 kW (6.4 hp) Yanmar L70 V engine using low sulfur diesel (32ppm).	Acute (single exposure) inhalation study (cardiovascular effects) in spontaneously hypertensive heart failure rats	DEP (NO_2 also measured)	0 & 515µg/m^3 DEP exhaust (6h)	↑ cardiac output, left ventricular volume (end diastolic and systolic), stroke volume, HRV, and atrioventricular block arrhythmias. ↑ HRV correlated with postexposure changes in bradyarrhythmia frequency, repolarization, and echocardiographic parameters ↑electrocardiographic measures of ventricular repolarization	Not identified	500µg/m^3 (NO_2 = 0.66ppm)
Cassee et al. 2012	TDE presumably (Ingersoll Rand (4IRD5AE) engine using standard diesel fuel, sulfur content not stated)	Subacute inhalation exposure study in ApoE knockout mice	DEP (NO_2 also measured)	1,700 µg/m^3 (with and without 9ppm caesium oxide) (20 min, 1hr or 3hr/d, 5d/wk, for 4 wks).	↑atherosclerotic plaque size in animals exposed to DEP Note; Addition of caesium oxide decreased the number and surface area of DEP and increased gaseous co pollutants and may reduce atherosclerosis however differences observed in caesium exposed and unexposed groups were small.	Not identified	1,700 µg/m^3 (NO_2 = 2ppm)
CDC 1984, Gowdy et al. 2008	TDE (engine not stated)	Chronic exposure inhalation study in rats	DEP	2,000 µg/m^3 (7hrs/d, 5d/wk, for 2yrs).	Depressed alveolar macrophages.	Not identified	2,000 µg/m^3 (NO_2 concentrations not reported)
Chang et al. 2018	TDE (Presumed) (Single-cylinder Yanmar diesel generator engine (YDG5500), fuelled with standard highway-grade number 2 diesel fuel)	Prenatal exposure inhalation study (neurodevelopmental) in male and female C57Bl/6J mice	DEP	0 & 250 - 300µg/m^3 DEP exhaust (6h/d, 5d/wk, Embryonic Day 0 to PND21)	↓ social interaction, ↑repetitive behaviour, and ↓or altered communication.	Not identified	250 - 300µg/m^3 (NO_2 = 1.8ppm, based on past experiments with this experimental setup)
Chen et al. 2021	TDE (presumed) (Nissan QD32, fuel type and sulfur content not stated)	Acute inhalation exposure study in male SD-rats	DEP	200 and 1,000 µg/m^3 (24hrs/d, 7d/wk, for 14 days).	Ultrastructural changes in NTS, Glial activation in the brain, ↑numbers of microglia and astrocytes in the NTS, as well as NGF expression in the NTS. ↑Inflammatory cytokines (IL-1β, IL-6, and TNF-α).	Not identified	200 µg/m^3 (NO_2 concentrations not reported)
Cole et al. 2016	TDE (presumed) (Yanmar YDG5500 Diesel generator using Number 2 Diesel Certification Fuel with ultra-low sulfur fuel)	Single acute exposure inhalation study in C57BL/6 mice	DEP	250 - 300µg/m^3 (6 hours).	Neuroinflammation and oxidative stress in brain [↑ lipid peroxidation and ↑ pro-inflammatory cytokines (IL-1a, IL-1b, IL-3,IL-6, TNF-a) in various brain regions]	Not identified	250 - 300µg/m^3 (NO_2 concentrations not reported)
Cole et al. 2020	TDE (presumed) (Yanmar YDG5500 Diesel generator using Number 2 Diesel Certification Fuel, sulfur content not stated)	Developmental inhalation exposure study in C57BL/6 mice	DEP	250µg/m^3 (6 hours, GD0 to PND21).	Biochemical/molecular and behavioural alterations M: ↑ Pax6, Tbr1, Tbr2, Sp1, and Creb1 on PND3 M & F: ↓Tbr2+ intermediate progenitor cells in the PND60 hippocampal dentate gyrus	Not identified	250µg/m^3 (NO_2 concentrations not reported)
Conklin and Kong 2015	NTDE Detroit Diesel 2007 model Series 60 14L heavy-duty diesel engine	Long-term inhalation study (cardiovascular) in Wistar Han rats	Diesel exhaust (NO_2)	NO_2 = 0.1, 0.8 or 4.2ppm (16h/d, 5d/wk, up to 24M) (NB: DEP = 23.3, 20 or 27.3µg/m_3)	↑in soluble intercellular adhesion molecule 1 (sICAM-1) and interleukin-6 (IL-6) levels and ↓ in total and non-high-density-lipoprotein cholesterol (non-HDL) levels in plasma in female rats after 24M (medium or high exposure groups). These effects were not observed in male rats, and no changes in cardiac fibrosis or aorta morphometry resulting from DE exposure were observed in either sex.	NO_2 = 0.1µg/m^3	NO_2 = 0.8µg/m^3
de Brito et al. 2018	TDE (presumed) (DE electrical generator, BD-2500 CFE using metropolitan diesel fuel (50ppm sulfur content) with a blend of 5% BD)	Single exposure acute inhalation study in adult male Balb/C mice	DEP (NO_2 also measured)	600 and 1,200 µg/m^3 (2 hours).	Haemodynamic effects, alterations in blood parameters (↑ erythrocytes, ↑ haemoglobin at 600 and 1200, haematocrit and reticulocytes at 1200), bone marrow cellularity (↑ in eosinophils at 600, ↑ in myelocytes at 1,200, ↓ in metamyelocytes, band neutrophils and plasmocytes at 600), bronchoalveolar lavage fluid (↑ in macrophages and total cells at 600) and lung histology (↑ neutrophils density). Increased expression of receptor of endothelin-B and vascular cell adhesion molecule 1 in bronchial epithelium were also observed.	Not identified	600µg/m^3 (NO_2 = 31µg/m^3, i.e. 0.02 ppm)

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Douki et al. 2018	NTDE (Euro4-compliant supercharged common rail direct injection diesel engine using diesel fuel with <3ppm sulfur)	Repeat exposure (lung oxidative stress and genotoxicity) inhalation study in male Wistar rats	DEP & fDEP (NO_2 also measured)	fDEP: <10µg/m^3 DEP <10 and 2,300µg/m^3 (3h/d, 5h/wk, for 3 wks) (unclear whether same volume of exhaust was used for both exposures, but likely to be the case)	Statistically significant increase in the anti- gH2AX antibody in lungs of rats in fDEP exposed group but not the DEP exposed group (although there was an increase in this measured in the DEP exposed group compared to the control although not statistically significant). Most other changes observed were considered to not be statistically significant (e.g. small induction of g-H2AX and acrolein adducts was observed to a greater extent in filtered compared to unfiltered exhaust). No changes identified in bulky adducts and 8-oxodGuo were noted.	DEP: 2,300 µg/m^3 (NO_2 = 1.3ppm)	fDEP <10 µg/m^3 (NO_2 = 1.3ppm)
Durga 2015	Not specified (on-road vehicles)	Sub-chronic neurotoxicity (nose only) inhalation exposure study in male Wistar rats	DEP	330,000, 500,000 & 1,000,000µg/m^3 (3) (4h/d, 5d/wk, for 90d) *Note concentrations appear to be unusually high(3)	Increased inflammation, DNA damage and oxidative stress at the highest concentration (↑ pro-inflammatory cytokines, amyloid beta 42 (Aββ42), reactive oxygen species (ROS), hydrogen peroxide (H2O2), nitrate (NO3−), nitrite (NO2−) and apurinic/apyrimidinic sites (AP) at varying degrees at different sections of rat brain). TNF was increased in the midbrain at all concentrations.	Not identified	330,000µg/m^3 (NO_2 concentrations not reported)
Ehsanifar et al. 2019a	Not stated (Light-duty 2776cc, 4-cylinder, diesel engine, Khodro Diesel Co. using standard diesel fuel, sulfur content not stated)	Sub-chronic neurotoxicity inhalation exposure study in NRMI male mice	DEP	350-400µg/m^3 (3h, 6h and 8h/d, 5h/wk, for 12 wks)	Inflammation and oxidative stress changes in olfactory bulb and hippocampus for 3h, 6h and 8h exposure groups, markedly coinciding with the results of behavioural alterations (rapid induction of MDA and nitrite oxide in brain regions and neuronal nitric oxide synthase nNOS mRNA followed by IL6, IL1α, and TNFα in olfactory bulb and hippocampus).	Not identified	350-400 µg/m^3 (NO_2 concentrations not reported)
Ehsanifar et al. 2019b	Not stated (Light-duty 2776cc, 4-cylinder, diesel engine, Khodro Diesel Co.)	Prenatal inhalation exposure study in adult female NRMI mice offspring	DEP	350-400µg/m^3 (2h, 4h and 6h/d, 21 days during gestation)	Anxiety and impairment of spatial learning and memory in adult male mice offspring (↑ time spent in open arms (OAT) in all exposed groups, ↑IL6 HI and entries in open arms (OAE) in the 4h and 6h group, ↑IL1b HI and TNFa HI in the 6h group as well as ↓ Morris water maze probability, N-methyl-D-aspartate receptor subunit 2A (NR2A), and 3B (NR3B), and Nissl staining in the hippocampus in the 4h and 6h groups). (NB: Study authors state the effects were likely caused by gaseous compounds, but do not report on concentrations of gaseous compounds in exhaust).	Not identified	350-400 µg/m^3 (NO_2 concentrations not reported)
Ehsanifar et al. 2021	Not stated (Light-duty 2776cc, 4-cylinder, diesel engine using standard diesel fuel, sulfur content not stated)	Sub-chronic neurotoxicity inhalation exposure study in adult male and female NRMI mice offspring	DEP	350-400µg/m^3 (6h/d, 5d/wk for 14 weeks)	Neuroinflammation and oxidative stress observed in female and male mice with male mice more susceptible to changes observed. ↑ in inflammatory cytokines (IL1a, IL1b, TNFa, and IL6), heme oxygenase-1 (HO-1) N-methyl-D-aspartate receptor subunit 2A (NR2A), and 2B (NR2B), and neuronal Nitric Oxide Synthase (nNOS). (NB: Authors state the effects were likely caused by gaseous compounds, but do not report on concentrations of gaseous compounds in exhaust).	Not identified	350-400 µg/m^3 (NO_2 concentrations not reported)
Fujimaki and Kurokawa 2004	Not stated	Sub-chronic exposure inhalation study (immune system) in BABL/c mice injected intraperitoneally with sugi basic protein (SBP)	DEP & fDEP (NO_2 also measured)	fDEP: 40µg/m^3 DEP: 1,010µg/m^3 (12h/d, 7d/wk, for 5wks) (exhaust pre-diluted prior to exposure indicating exposure was to same volume of air)	↑ chemokine production in both fDEP and DEP exposed groups.	Not identified	fDEP: 40µg/m^3 (NO_2 = 2.93ppm) DEP: 1,010µg/m^3 (NO_2 = 1.99ppm)

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Gerlofs-Nijland et al. 2010	Not stated 35 KVA diesel generator (Bredenoord, Apeldoorn, The Netherlands)	Pulmonary and cardiovascular short-term exposure inhalation study in male Fisher F344 rats	DEP (NO_2 also measured)	150µg/m^3 (6h/d, 5d/wk for 4 weeks)	↓numbers of white blood cells, ↓ von Willebrand factor protein in the circulation, ↓lung tissue factor activity in conjunction with ↓ generation in lung tissue thrombin.	Not identified	150µg/m^3 (NO_2 = 918µg/m3, i.e. 0.49 ppm)
Goodson et al. 2017	TDE (Presumed as standard diesel fuel used) (Single-cylinder Yanmar diesel generator engine (YDG5500), fuelled with standard highway-grade number 2 diesel fuel)	Prenatal exposure inhalation study (cardiomyocyte transcription, DNA methylation and metabolic perturbation) in male and female C57Bl/6J mice	DEP (NO_2 also measured)	0 & 300µg/m^3 DEP exhaust (6h/d, 5d/wk, Embryonic Day 0.5 to Embryonic Day 17.5)	Cardiomyocytes exhibit ↓metabolic, ↓ methylation of DNA in neonatal cardiomyocytes	Not identified	300µg/m^3 (NO_2 = 0.06ppm)
Gordon et al. 2012	NTDE (presumed based on web search indicating it complies with Euro V) Single-cylinder, air-cooled, direct injection, 320cm3 Yanmar L70 V diesel engine with diesel fuel (32 ppm sulfur)	Acute exposure inhalation study (respiratory system) in Wistar-Kyoto rats	DEP & fDEP (NO_2 also measured)	fDEP: not stated DEP: 1,900µg/m^3 (5h/d for 2d or 5h/d, 5d/wk for 4wks) (same volume of exhaust was used for both exposures)	↑ BALF neutrophils, blood pressure, BALF GGT activity and BALF GGT activity in both fDEP and DEP exposed groups. ↓ heart rate in both fDEP and cardiac contractility in the DEP exposed groups ↓ QA interval (4 weeks) in the fDEP exposed group.	Not identified	fDEP: Not stated DEP: 1,900µg/m^3 (NO_2 = 10-30ppm in filtered & unfiltered treatments)
Greve et al. 2020	Not stated (Single-cylinder, air-cooled, direct injection, 320cm3 Yanmar L70 diesel engine)	Neurotoxicity inhalation study in WYK rats	DEP	0, 50, 150, 500µg/m^3 (4 weeks)	Neuroinflammation (impaired TREM2 expression, ↑ Lyz2, and ↑ Cx3crl at 150µg/m^3 and ↓in microglial-vessel association at 500µg/m^3).	50µg/m^3 (NO_2 concentrations not reported)	150µg/m^3 (NO_2 concentrations not reported)
Hallberg et al. 2015	NTDE Detroit Diesel 2007 model Series 60 14L heavy-duty diesel engine	Long-term inhalation study (genotoxicity) in Wistar Han rats	Diesel exhaust (NO_2)	NO_2 = 0.1, 0.8 or 4.2ppm (16h/d, 5d/wk, up to 24M) (NB: DEP = 23.3, 20 or 27.3µg/m^3)	Levels of 8-OHdG in serum at 12M and 24M at the mid and high exposure levels. Some other scattered changes were noted however, these differences did not follow an exposure-dependent pattern. Hippocampal concentrations of TBARs also showed some small and scattered changes at different exposures and time points however these changes were not considered to be treatment-related. Exposure to diesel exhaust in these rats did not produce any significant increase in oxidative damage to lipids or damage to DNA in the form of strand breaks.	NO_2 = 4.2ppm	Not relevant
Hashimoto et al. 2001	Not stated	Sub-chronic (nose only) exposure inhalation asthma study in female Hartley guinea pigs	DEP	3,000µg/m^3 (12h/d, 7d/wk, for 8 wks with and without sensitisation to ovalbumin)	Non-sensitised animals: Nil Sensitised animals: ↓ volume density, ↑ concentration of sialic acid, eosinophils and albumin and ↑ number of eosinophils.	3,000µg/m^3 (non-sensitised animals)	3,000µg/m^3 (sensitised animals) (NO_2 concentration not reported)
Hazari et al. 2017	Not determined Yanmar diesel generator using low sulfur diesel fuel (32 ppm)	Acute (single exposure) inhalation study (cardiovascular effects) in Wistar–Kyoto (WKY) and spontaneously hypertensive (SH) rats	DEP	0 & 150µg/m^3 DEP exhaust (4h) challenged with dobutamine 24h later	↓ reserve of refractoriness in spontaneously hypertensive rats and WKY rats (NB: the change in spontaneously hypertensive rats was 8x greater than in WKY rats)	Not identified	150µg/m3 in dobutamine challenged mice (NO_2 concentration not reported)
Hemmingsen et al. 2009	Not stated. (diesel exhaust particle standard reference material 2975)	Prenatal inhalation study in pregnant mice	DEP	20,000µg/m^3 (1h/d, day 7 – 19 during gestation)	↓ daily sperm production in adulthood (male offspring). Note: Lack of effects on body and testes weight, AGD, and plasma levels of testosterone and oestradiol	Not identified	20,000µg/m^3
Henderson et al. 1988	TDE 1980, 5.7L Oldsmobile engine using a standardized certification fuel (D-2 Diesel Control Fuel) (sulfur content not stated)	Long-term inhalation study (pulmonary changes) in F344/Crl rats and CD-1 mice	DEP (NO_2 also measured)	DEP: 350, 3,500, or 7,000µg/m^3 (7 h/d, 5 d/wk, for 24M).	A NOAEC of 350 µg/m3 was identified for rats and mice for chronic inflammatory changes in the lungs at higher doses (3,500 and 7,000 µg/m^3 for 7 hours/day, 5 days/week, for 24 months).	350 µg/m^3 (NO_2 = 0.1ppm)	3,500µg/m^3 (NO_2 = 0.3ppm)
Hue-Beauvais et al. 2019	Not determined 25KVA Loxam engine, with a 500 nm particle filter (fuel used and sulfur content not stated)	Prenatal inhalation study (mammary gland and milk composition) in New Zealand White rabbit	DEP (NO_2 also measured)	0 & 1,000µg/m^3 DEP exhaust (2h/d, 5d/wk, GD3 to GD27, nose only)	Mammary alveolar lumina contained numerous fat globules, ↑ stearoyl CoA reductase expression in mammary epithelia. Changes in milk composition was also noted.	Not identified	1,000µg/m^3 (NO_2 = 0.7ppm, Valentino et al. 2016, Table S2)

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Hullmann et al. 2017	Not determined Common-rail motor, 100 kVA, 35 KW load (fuel type and sulfur content not stated)	Long-term exposure inhalation study (neurodevelopmental) in female 5X Familial AD (5XFAD) mice and their wild-type female littermates	DEP (NO_2 also measured)	0 & 950µg/m^3 DEP exhaust (6h/d, 5d/wk, for 3 or 13 weeks)	Plaque formation accelerated and greater motor function impairment was observed ↓ grip strength and motor coordination, ↑ cortical Aβ plaque load and ↑ whole brain homogenate Aβ42 levels	Not identified	950µg/m^3 (NO_2 = 1.56ppm)
Ishihara and Kagawa 2002	TDE (presumed based on year of study) (7.41-liter diesel engines using standard oil No. 2, 3,900ppm sulfur)	Chronic inhalation (cardiac and pulmonary) study in Harley male guinea pigs	DEP & fDEP (NO_2 also measured)	DEP: 210µg/m^3, 1,140µg/m^3 or 2,940µg/m^3 fDEP: 10µg/m^3 (16h/d, 6d/wk, for 6, 12, 18, or 24M). (exhaust pre-diluted prior to exposure indicating exposure was to same volume of air)	DEP induced continuous inflammation, overproduction of mucus, and phospholipids in the lung as evident by biochemical changes for C4 in plasma, ↑ eosinophil counts, ↑ total protein, ↑ LDH, ↑ sialic acid and ↑ phospholipids in the medium and high exposed groups from as early as 12 months exposure. Animals exposed to the medium level of diesel exhaust without particulate matter showed significantly less increase of these biomarkers as compared with animals exposed to the same level of diesel exhaust with particulate matter.	fDEP: 10µg/m^3 (NO_2 = 1.13ppm) DEP: 210µg/m^3 (NO_2 = 0.22ppm)	DEP: 1,140µg/m^3 (NO_2 = 1.07ppm)
Ishihara and Kagawa 2003	TDE (presumed based on year of study) (7.41-liter diesel engines using standard oil No. 2, 3,900ppm sulfur)	Long-term inhalation study (pulmonary effects) in male Wistar rats and CD-1 mice	DEP & fDEP (NO_2 also measured)	fDEP: 10µg/m^3 DEP: 200, 1100, or 2800µg/m^3 (16 h/d, 6 d/wk, for up to 24M). (exhaust pre-diluted prior to exposure indicating exposure was to same volume of air)	The NOAEC in the rat lung was determined to be between 200 µg/m^3 and 1,000 µg/m^3 for DEP. In this study, male Wistar rats were exposed to DEP at a DPM level of 200, 1100, or 2800 µg/m^3 for 16 hours/day, 6 days/week, for up to 24 months. Another animal group in this study was exposed to particle-free gaseous phase of DEE (mean particle concentration of 10µg/m^3; NO_2 concentration was 1.1 ppm at 6M, 1.13ppm at 12M, 1.12 ppm at 18M, 1.12 ppm at 24M). There were no treatment-related adverse effects in the 200 µg/m^3 group or the fDEP exposed group, whereas in the 1,100 µg/m^3 group there were variable increases in number of macrophages, leukocytes, lymphocytes and phospholipids. In the animals that inhaled particle-free DEE, only a slight increase in the number of leukocytes was observed after 24 months.	fDEP: 10µg/m^3 (NO_2 = 1.13ppm) DEP: 200 µg/m^3 (NO_2 = 0.2ppm)	DEP: 1,100µg/m3 (NO_2 = 1.03ppm)
Iwai et al. 1997	TDE Light duty 2369 ml diesel engine (for commercial vehicles)	Chronic inhalation (and intratracheal) study (lung tumour) in male and/or female Fischer F344 Rats.	DEP & fDEP (NO_2 also measured)	DEP = 9,400µg/m^3, fDEP concentration not stated, NO_2 = 1.8ppm (8h/d, 7d/wk for 6, 12 or 24M) ii) DEP = 3,200µg/m^3, fDEP = not stated, NO_2 = 1.8ppm (8h/d, 6d/wk for 48M) iii) DEP = 5,100µg/m^3, fDEP = not stated, NO_2 = 1.8ppm (18/d, 3d/wk for 48M). (exhaust pre-diluted prior to exposure indicating exposure was to same volume of air)	Soot particles deposited in the alveolar macrophages and in the interstitial tissues with chronic inflammatory cell infiltration and tumours in the lung (Adenoma and adenocarcinoma [originating from Type II alveolar cells]) were observed. Effects were associated with carbon core. The filtered exhaust group demonstrated the same lung tumour incidence of 4 % as the clean air control group, indicating no enhancing effect of tumour formation in the gaseous component of the exhaust.	Not observed	3,200µg/m^3 (NO_2 = 1.8ppm)
Karoui et al. 2019	NTDE (Euro4-compliant supercharged common rail direct injection diesel engine using commercial low sulfur diesel, 3ppm).	Acute and repeat exposure inhalation study in male Wistar rats (mitochondrial and cardiac dysfunctions)	DEP & fDEP (NO_2 also measured)	DEP = 2,500µg/m^3 fDEP = tested but concentration not provided. (3h/d, 5d/wk for 3wks) (exhaust pre-diluted prior to exposure indicating exposure was to same volume of air)	Disturbance in echocardiographic parameters, which persisted and worsened after repeated exposures and other biochemical changes (↑ in LV end-diastolic and end-systolic diameters parameters and a ↓ in oxygen consumption, Complex 1 activity, and ATP production). The presence of the fDEP did not modify the cardiovascular dysfunction revealing an important implication of the gas phase in this response. There were sustained cardiovascular and mitochondrial effects attributable to the gas phase, possibly aldehydes and/or NO_2.	Not identified	fDEP = not stated (NO_2 not stated) DEP = 2,500µg/m3 (NO_2 = 3ppm)

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Karthikeyan et al. 2013	2004 Cummins ISM 280, 10.8 l, inline 6-cylinder heavy-duty diesel engine using commercial ultralow sulfur diesel	Acute exposure inhalation study (respiratory system) in F344 rats	DEP & fDEP (NO_2 also measured)	fDEP: 1.7 µg/m^3 DEP: 269µg/m^3 (4h/d for 1d or 4h/d for 3 days) (exhaust pre-diluted prior to exposure indicating exposure was to same volume of air)	↑ BALF protein (single), BALF neutrophils (single), ALF macrophages (repeated) and cytokine production (single) and ↓ BALF neutrophils (repeated) in both fDEP and DEP exposed groups. fDEP resulted in heightened injury and inflammation, consistent with the 4-fold increase in NO_2 concentration	Not identified	fDEP: 1.7 µg/m^3 (NO_2 = 15.7ppm) DEP: 269µg/m^3 (NO_2 = 4.2ppm)
Kim et al. 2016	TDE (presumed based on year of engine manufacture, 1990's) Light-duty (2,740-cc), 4-cylinder diesel engine (4JB1 type, Isuzu Automatic Co. using standard diesel fuel)	Sub-chronic inhalation study (airway inflammation) in BALB/c mice	DEP	100µg/m^3 & 3,000µg/m^3 (1h/d, 5d/wk for3M)	DEP exposure increased AHR, inflammation, lung fibrosis, and goblet cell hyperplasia in a mouse model. Biochemical changes (↑ AHR, inflammatory cytokine [IL-5, IL-13, and interferon-γ], and levels of vascular endothelial growth factor in the low and high exposure groups and ↑neutrophils, IL-10 and lymphocyte in the high exposure group) were observed with ↑ collagen content and ↑ lung fibrosis in the high exposure group	100 µg/m^3 (lung fibrosis) (NO_2 concentration not stated)	100µg/m^3 (biochemical changes) 3,000 µg/m^3 (lung fibrosis) (NO_2 concentration not stated)
Kobayashi 2000	TDE presumed based on year of engine manufacture Light-duty (2,740-cc), 4-cylinder diesel engine (4JB1 type, Isuzu Automatic Co. using standard diesel fuel).	Sub-chronic inhalation study (allergenic) in male Hartley guinea pigs	DEP	1,000µg/m^3 & 3,000µg/m^3 (5wks)	Exposure to DEP enhanced the number of sneezes and the amount of nasal secretions induced by OVA. Titres of specific anti-OVA-IgG and anti-OVA-IgE also significantly increased in DEP-exposed animals. Exposure to DEP also augmented the number of eosinophils that infiltrated both the nasal epithelium and the sub-epithelium induced by OVA.	Not identified	1,000µg/m^3
Kodavanti et al. 2013	NTDE (presumed based on web search indicating it complies with Euro V) 4.8 kW (6.4 hp) Yanmar L70 V engine using low sulfur diesel fuel (32 ppm)	Acute inhalation study (pulmonary and cardiovascular impairment) in male Wistar Kyoto (WKY) rats	DEP & fDEP (NO_2 also measured)	fDEP: 19µg/m^3 (4wks) DEP: 1,494µg/m^3 (4wks) (5h/d for 2d or 5h/d, 5d/wk for 4wks) (unclear whether same volume of exhaust was used for both exposures, but likely to be the case)	↑ BALF neutrophils (WKY rats) and BALF GGT activity (2 days, 4 weeks) in both fDEP and DEP exposed groups. Acute and 4-week gas and DEP and fDEP exposures increased neutrophils and γ-glutamyl transferase (γ-GT) activity in lavage fluid of WKY and SH rats. DE (4 weeks) caused pulmonary albumin leakage and inflammation in SH rats. Marked increases occurred in aortic mRNA after 4-week DEP or fDEP in SH (eNOS, TF, tPA, TNF-α, MMP-2, RAGE, and HMGB-1).	Not identified	fDEP: 19µg/m^3 (NO_2 = 0.8ppm) DEP: 1,494µg/m^3 (NO_2 = 0.7ppm)
Lamb et al. 2012	NTDE (presumed based on web search indicating it complies with Euro V) Single-cylinder, air-cooled, direct injection, 320cm3 Yanmar L70 V diesel engine with diesel fuel (32 ppm sulfur)	Acute exposure inhalation study (respiratory system) in Wistar-Kyoto and SH rats	DEP & fDEP (NO_2 also measured)	fDEP: 15 or 21 DEP: 168 or 425µg/m^3 (4h) (Exposures occurred to same air volume)	Immediate electrocardiographic alterations in cardiac repolarization (ST depression) and atrioventricular conduction block (PR prolongation) as well as bradycardia in SH rats in the fDEP exposed group. Ostexposure ST depression, ↑sensitivity to the pulmonary C fiber agonist capsaicin in SH rats and ↓ heart rate in WKY rats in the DEP exposed group (low and / or high). ↑ Susceptibility to apnea after capsaicin in DEP group only	Not identified	fDEP: 15 or 21µg/m^3 (NO_2 = <0.5ppm) DEP: 168µg/m^3 (NO_2 = <0.5ppm)
Lecureur et al. 2020	NTDE Light duty 4 cylinder direct-injection engine (with oxidation catalyst and with or without a filter) (Euro IV) using low sulfur diesel fuel	Short-term exposure inhalation study (lung transcriptional signatures) in rats	fDEP & DEP (NO_2 also measured)	fDEP: <10µg/m^3 DEP <10 and 2,300µg/m^3 (described in Douki et al. 2018) (3h/d, 5h/wk, for 3 wks) (unclear whether same volume of exhaust was used for both exposures, but likely to be the case)	A modest regulation of gene expression level (lower than 2-fold) and a higher number of genes regulated downstream of the filter. Aldehydes were higher downstream of the filter whereas BTEX, alkanes and PAHs were lower indicating aldehydes and the gaseous phase may have a higher impact than particles.	Not determined	fDEP <10 µg/m^3 (NO_2 = 1.3ppm) DEP: 2,300 µg/m^3 (NO_2 = 1.3ppm)

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Li et al. 2008	TDE (presumed based on use of standard fuel) 2369-cc diesel engine (Isuzu Motor) using commercial light oil (sulfur content not stated)	Sub-chronic exposure inhalation study (lung transcriptional signatures) in Nrf2 deficient C57BL/6 mice and wild type mice	DEP	<100µg/m^3 (7h/d, 5d/wk, for 8 wks)	DEP-induced oxidative stress and host antioxidant responses play some role in the development of DEP-induced airway inflammation. Nrf2 knockout mice: ↑ airway hyperresponsiveness and of ↑ lymphocytes counts, ↑ eosinophils, ↑ inflammatory cytokines (IL-12 and IL-13), and ↑ activation-regulated chemokine (TARC). Wild type mice: ↑ expression of antioxidant enzyme genes	Not identified	<100µg/m^3
Li et al. 2009	NTDE 8-L diesel engine J08C, Hino Motors using low sulfur diesel fuel	Prenatal exposure inhalation study (testicular function) in immature male rats	DEP & fDEP (NO_2 also measured)	fDEP: 3.1µg/m^3 DEP: 148.9µg/m^3 (5h/d, from GD1 to GD19) (exhaust pre-diluted prior to exposure indicating exposure was to same volume of air)	Results suggest that prenatal exposure to nano-rich and filtered DEP leads to endocrine disruption after birth and suppresses testicular function in male rats. DEP & fDEP: ↓ relative weights of the seminal vesicle and prostate to body weight, ↓ concentrations of testosterone, progesterone, corticosterone, and follicle stimulating hormone in serum and steroidogenic acute regulatory protein and17β-hydroxysteroid dehydrogenase mRNA and ↑ immunoreactive inhibin in serum. DEP only: ↑follicle stimulating hormone receptor mRNA	Not identified	DEP: 148.86µg/m^3 (NO_2 = 0.53ppm) fDEP: 3.1µg/m^3 (NO_2 = 0.51ppm)
Li et al. 2012	NTDE 8-L diesel engine J08C, Hino Motors using low sulfur diesel fuel	Sub-chronic exposure inhalation study (adrenocortical function) in Nrf2 deficient C57BL/6 mice and wild type mice	DEP & fDEP (NO_2 also measured)	fDEP: 0.69µg/m^3 DEP: 41.73µg/m^3 or 152µg/m^3 (5h/d, 5d/wk, for 8 wks) (exhaust pre-diluted prior to exposure indicating exposure was to same volume of air)	DEP or fDEP may disrupt adrenocortical function in adult male mice (↓ progesterone and corticosterone in high exposure DEP and fDEP groups)	DEP: 41.73µg/m^3 (NO_2 = 0.16ppm) fDEP: Not identified	DEP: 152µg/m^3 (NO_2 = 0.54 ppm) fDEP: 0.69µg/m^3 (NO_2 = 0.53ppm)
Li et al. 2013	TDE 8L diesel engine J08C, Hino Motors using low-sulfur diesel fuel (JIS No. 2 light oil)	In-utero exposure inhalation study (reproductive developmental effects) in pregnant Fischer rats	DEP & fDEP	fDEP: 3.1µg/m^3 DEP: 148.9µg/m^3 (5h/d, from GD1 to GD19) (exhaust pre-diluted prior to exposure indicating exposure was to same volume of air)	↓ maternal progesterone and ↓ maternal relative spleen and liver weight in both fDEP and DEP exposed groups. ↑ maternal luteinizing hormone and corticosterone in both fDEP and DEP exposed groups. ↑ oestradiol in fDEP exposed group only. Altered induced corpus luteum gene expression and foetal body weight and length in both fDEP and DEP exposed groups.	Not identified	fDEP: 3.1µg/m^3 (NO_2 concentration not stated) DEP: 148.9µg/m^3 (NO_2 concentration not stated)
Liu et al. 2018	Not determined (0.2-L diesel engine, TP168F, Tuopu Motors using low-sulfur NO.0 diesel fuel (sulfur content not stated.	Short-term inhalation study (pulmonary effects) in male CD-1 mice	DEP	0 & 350µg/m^3 DEP exhaust (5h/d, for 7 days)	DEP induced i) pulmonary arterial hypertension-phenotype accompanied with increased right ventricular systolic pressure (RVSP), right ventricle hypertrophy and pulmonary arterial thickening, ii) proliferation of vascular smooth muscle cells (VSMCs) and apoptosis of endothelial cells in pulmonary artery and ii) accumulation of CD45þ lymphocytes and CD68þ macrophages surrounding and infiltrating pulmonary arteriole. ↑↑Inflammatory cytokines (TNF-a, IL6 & IL13 and ↑ T helper 17 (Th17) and Th2 cells.	Not identified	350µg/m^3 (NO_2 = 500µg/m^3, i.e. 0.27ppm)
Mauderly et al. 2014	TDE (2000 Model Cummins ISB 5.9L turbocharged engine burning No. 2 fuel with 371ppm sulfur)	Repeat exposure inhalation study in F344 rats and A/J mice	DEP	996µg/m^3 DEP exhaust (6h/d, 7d/wk, for 3 days to 6 months)	↓clearance of bacteria from the lung, ↑ lung cytotoxicity, and ↑ oxidant stress and pro-atherosclerotic responses in aorta	Not identified	996µg/m^3 (NO_2 = 4.4ppm)

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
McDonald et al. 2004b	TDE and NTDE (Single-cylinder Yanmar diesel generator engine (YDG5500), fuelled with either i) number 2 diesel fuel with 371ppm sulfur or ii) ECD1 low sulfur fuel with 14ppm sulfur and catalyst system)	Short-term exposure inhalation study in C57B1/6 mice using DEP or DEP generated with emission reduction technologies (DEP ER)	DEP & DEP ER (NOx also measured)	236µg/m^3 DEP exhaust or 7.5µg/m^3 DEP exhaust from engine with low sulphur fuel and catalyst trap (6h/d for 7d)	DEP: ↑ lung viral burden and lung histopathology, ↑ inflammatory cytokines (TNFα, IL6 & IFN-gamma) and haeme oxygenase DEP ER: - (NB: NO_2 was not directly measured in this study due to analyser failure (McDonald 2004). In a corresponding study, McDonald (2004b) determined NO_2 to represent 10% of total nitrogen oxides (NO_x). Therefore, NO_2 concentrations are estimated in this report to be 0.21 ppm with no treatment and 0.19 ppm with treatment based on reported NO_x concentrations of 2.1ppm and 1.9ppm respectively (see Table 4, McDonald 2004a).	DEP: Not identified DEP ER: 7.5µg/m^3 (NOx = 1.9ppm, 0.19 ppm NO_2 estimated from NO_x)	DEP: 236µg/m^3 (NOx = 2.1ppm, 0.19 ppm NO_2 estimated from NO_x) DEP ER: Not identified (but expected to be >7.5µg/m^3)
McDonald et al. 2015	NTDE Detroit Diesel 2007 model Series 60 14L heavy-duty diesel engine	Long-term inhalation study (carcinogenicity, pulmonary and systemic effects) in Wistar Han rats	Diesel exhaust (NO_2)	NO_2 = 0.1, 0.8 or 4.2ppm (16h/d, 5d/wk, up to 24M) (NB: DEP = 23.3, 20 or 27.3µg/m^3)	Absence of pre-neoplastic lung lesions, primary lung neoplasia, or neoplasia of any type attributable to exposure. Lung lesions (minimal to mild epithelial hyperplasia with minimal increase in fibrous stroma) in the highest exposure level. Subtle accumulation of pulmonary alveolar macrophages. These findings progressed from 3months to 12 months but did not progress further. Biochemical changes in the lung tissue and lavage fluid that indicated mild inflammation and oxidative stress in the highest exposure group. A mild progressive decrease in pulmonary function, which was more consistent in females than males was also observed. NO_2 may have been the primary driver of the biologic responses to NTDE in the present study.	NO_2 = 0.8ppm	NO_2 = 4.2ppm
Miyabara et al. 1998	TDE (Light-duty (2,740-cc), 4-cylinder diesel engine (4JB1 type, Isuzu Automatic Co.) with standard diesel fuel	Sub-chronic inhalation study (allergic airway inflammation) in ICR mice challenged with ovalbumin	DEP (NO_2 also measured)	3,000µg/m^3 DEP soot (12h/d for 5 to 6wks)	Diesel exhaust enhanced allergic airway inflammation, hyperplasia of goblet cells, and airway hyperresponsiveness caused by ovalbumin sensitization ↑ infiltration of eosinophils, neutrophils and inflammatory cytokines (IL5) in lung tissue and ↑ IgG1 production.	Not identified	3,000µg/m^3 (NO_2 = 4.08ppm)
Moreira et al. 2020	TDE (stationary diesel electrical generator (BD-2500 CFE; Branco used with metropolitan diesel containing 10 ppm sulphur)	Inhalation study (pulmonary) in C57BL/6 mice (with saline or porcine pancreatic elastase (PPE) instillation)	DEP (NO_2 also measured)	600µg/m^3 DEP (1h/d for 5 to 6wks)	DEP and PPE instillation: ↓ Htis values and in Gtis , ↑ number of neutrophils and ↑ numbers of total cells in BAL, macrophages, epithelial cells, and lymphocytes in DEP and PPE instillation group, ↑ LM values, ↑ Caspase-3 expression in the lung parenchyma, ↑ collagen fibres,↑ inflammatory cytokines (IL4, IL10 & IL13), ↑ IRF-4 and Arg-1 expression DEP and Saline instillation: ↑ Caspase-3 expression in the lung parenchyma, ↑ collagen fibres, ↑ cytokines (IL4, IL10 & IL13)	Not identified	600µg/m^3 (NO_2 = 4.08ppm)
Heidari Nejad et al. 2015	Not stated Light duty indirect injection diesel engine using Australian Diesel (sulfur content not stated)	Acute exposure inhalation study (neurovascular inflammation) in male and female BALB/c mice	DEP (NO_2 also measured)	20,000µg/m^3, 30,000µg/m^3 DEP exhaust (4d/wk, for 2 wks)	↑ abundance of parenchymal IgG and parenchymal glial fibrillar acidic protein	Not identified	20,000 µg/m^3 (NO_2 = 5.18ppm)
Nikula et al. 1995	TDE 1998 LH6 6.2L V8 engines (General Motors) using D-2 control fuel. (sulfur content not stated)	Long term exposure inhalation study (pulmonary and carcinogenicity) in F344 rats	DEP (NO_2 also measured)	0, 2,500 or 6,500µg/m^3 DEP (16h/d, 5d/wk, for 24M)	DEP accumulated progressively in lungs of exposed rats and resulted in nonneoplastic legions as well as malignant and benign neoplasms in lungs (adenoma and adenocarcinoma) of female and male rats. It was suggested in this study that the organic fraction in DE does not play an important role in carcinogenicity of DEP in rats	Not identified	2,500µg/m^3 (NO_2 = 0.73ppm)

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Nway et al. 2017	TDE (presumed) 8L diesel engine J08C, Hino Motors (fuel type and sulfur content not stated)	Prenatal inhalation study (developmental) in pregnant C3H/HeN (TLR4-intact) and C3H/HeJ (TLR4-mutated) mice	DEP (NO_2 also measured)	0 or 85µg/m^3 DEP (5h/d from GD14 to PND10) NB: EC/TC ratio = 0.40	↓ performance in the test phase of spatial learning which was more prominent in C3H/HeJ neonatal mice. ↑ mRNA expression levels of the NMDA receptor subunits (NR1, NR2B), proinflammatory cytokines, tumour necrosis factor-a and cyclooxygenase-2, oxidative stress marker, haeme oxygenase-1, and microglial marker, Iba1, in the hippocampus in the neonatal C3H/HeN male mice but not females	Not identified	85µg/m^3 (NO_2 = 1ppm)
Ogliari et al. 2013	TDE (Stationary diesel electrical generator, BD-2500 CFE; Branco, using metropolitan diesel and 3% biodiesel with 500ppm sulfur)	Intrauterine and postnatal exposure inhalation study (developmental) in male and female Swiss mice (males were removed after mating)	DEP	0 or 600µg/m^3 DEP (1h/d during pregnancy and a further 60d)	↓in the proportion of primordial follicles in intrauterine-exposed animals, postnatally exposed animals, and animals exposed during both phases. ↓ primary follicle proportion in animals exposed during pregnancy.	Not identified	600µg/m^3 DEP (NO_2 concentration not reported)
Olivo et al. 2022	TDE (Stationary diesel electrical generator, BD-2500 CFE China, using metropolitan diesel with 10 ppm sulfur and 5% soybean biodiesel)	Sub-chronic inhalation study (pulmonary and cardiovascular function) in BALB/C male mice which were or were not submitted to a 10-week exercise training program	DEP	0 or 577µg/m^3 DEP (1h/d during pregnancy and a further 60d)	Untrained animals: ↓ heart rate variability, ↓ elastance of the respiratory system, ↑ inflammatory cytokines (IL23 & IL 12p40) and expression of inducible nitric oxide synthase, and ↑ bronchoalveolar lavage fluid, macrophages, neutrophils and lymphocytes, the density of polymorphonuclear cells and the proportion of collagen fibres in the lung parenchyma Trained animals: ↑amount of collagen fibres and expression of inducible nitric oxide synthase	Not identified	577µg/m^3 DEP (NO_2 concentration not reported)
Reed et al. 2004	TDE (2000 Cummins 5.9L ISB turbo engine with diesel fuel No. 2 with 371ppm sulfur)	Long-term and subchronic exposure inhalation study in F344 rats and A/J mice	DEP (NO_2 also measured)	0, 30, 100, 300, or 1000µg/m^3 DEP exhaust (6h/d, 7d/wk, for 7d or 6M)	↓ serum cholesterol in the high exposure group after 7d although had lessened by 6 months. ↓clotting Factor VII in the high exposure group after 6 months. ↑ serum gamma-glutamyl transferase in the mid-high and high exposure groups. Carcinogenic potential as determined by micronucleated reticulocyte counts and proliferation of adenomas in A/J mice were unaffected by 6M of exposure.	100µg/m^3 DEP, 6h/d (NO_2 = 0.4ppm)	300µg/m^3 DEP, 6h/d (NO_2 = 0.8ppm)
Ribeiro et al. 2019	TDE (Stationary diesel electrical generator, BD-2500 CFE China) (NB: Diesel fuel type and sulfur content not stated)	Short-term exposure inhalation study (pulmonary) in C57BL/6 male mice	DEP	0 or 1,200µg/m3 DEP exhaust (1h/d, 7d/wk, for 30d)	Older mice presented decreased pulmonary resistance and elastance, increased macrophage infiltration and decreased tumor necrosis factor (TNF) and interleukin 10 (IL-10) levels in the BALF, reduced activities of the antioxidant enzymes glutathione peroxidase (GPx) and glutathione reductase (GR), and increased activity glutathione S-transferase (GST); increased lung volumes with decreased elastic fiber and increased airway collagen content. SIRT1 gene expression was decreased in older animals, but protein levels were increased. DE exposure increased macrophage infiltration and oxidative stress in the lungs of animals of both ages. SIRT6 gene expression was decreased by DE exposure, with increased protein levels. In older animals, DE affected lung structure and collagen content.	Not identified	1,200µg/m^3 DEP, 1h/d (NO_2 concentration not stated in main report, graph in supplementary information shows it fluctuated between 0-300ppm)
Ritz et al. (2011)	TDE (Presumed) (Engine and fuel type not stated)	*In utero* exposure inhalation study (multigenerational, ESTR mutation analysis) in pregnant C57Bl/6 mice	DEP	0 or 19,000µg/m^3 DEP exhaust (1h/d, GD7 to GD19)	↑mutation frequency of male mice exposed *in utero* to DEP. No evidence for increased expanded simple tandem repeat (ESTR) loci mutation rates in females exposed *in utero* to DEP relative to control females	Not identified	19,000µg/m^3 (NO_2 concentrations not stated)

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Rousseau-Ralliard et al. 2021	Not determined 25KVA Loxam engine, with a 500 nm particle filter (fuel used and sulfur content not stated)	Prenatal exposure inhalation (nose only) study (cardiometabolism) in New Zealand white female rabbit	DEP (NO_2 also measured)	0 or 1,000µg/m^3 DEP exhaust (2h/d, 5d/wk from 3d to 27d post conception)	Male offspring: Age-related ↑in blood pressure, glycaemia, and perirenal fat mass and ↓ HDL-cholesterol and fat-to-body weight ratio was observed in males exposed during pregnancy. Female offspring: ↑ triglycerides and ↓bone density. Metabolic challenges triggered or amplified some biological responses, especially in females.	Not identified	1,000µg/m^3 (NO_2 = 0.7ppm, Valentino et al. 2016, Table S2)
Rousseau-Ralliard et al. 2019	Not determined 25KVA Loxam engine, with a 500 nm particle filter (fuel used and sulfur content not stated)	*In-utero* exposure inhalation (multi-generational) study in New Zealand white female rabbit	DEP	0 or 1,000µg/m^3 DEP exhaust (2h/d, 5d/wk from 3d to 27d post conception)	F1: ↑hepatic neutral lipid contents without change in the blood biochemistry. F2: ↑placental lipid contents, with ↑ monounsaturated fatty acids and ↓pro-inflammatory arachidonic acid (AA), without placental structural changes. ↑ proportion of anti-inflammatory n-3 polyunsaturated fatty acids in F2 plasma and ↓ AA. Proteasome complex and ubiquitin pathways genes were over-represented and ion channel function and inflammation pathways genes were under-represented in exposed animals.	Not identified	1,000µg/m^3 (NO_2 = 0.7ppm, Valentino et al. 2016, Table S2)
Russell et al. 1981	TDE 6-cylinder Nissan engine (fuel type and sulfur content not stated)	Sub-chronic exposure inhalation study (heritable effects) in mice	DEP	6,000µg/m^3 DEP exhaust (8h/d, 7d/wk, for 10 wks)	The results of all genetic tests in both sexes were negative. ↓ in the number of ovulations and ↑ in interval between mating opportunity and mating	Not identified	6,000µg/m^3
Sato et al. 2001	TDE (presumed) (engine and fuel type not stated)	Short-term inhalation study in F344 rats	DEP	0, 300 or 3,000µg/m^3 DEP exhaust (12h/d, 7d/wk, for 4 weeks)	↑ contents of chondroitin sulphate and hyaluronan (both components of extracellular matrix) in the surroundings of the bronchi at 300 & 3000µg/m^3 ↑ 8-OHdG positive cells and proliferating cell nuclear antigen-positive cells in areas of the glycosaminoglycans at 3000µg/m^3	Not identified	300µg/m^3 (NO_2 concentration not stated)
Shaheen et al. 2016	TDE (four-cylinder 2179-cc diesel engine, AA-4LE2; Isuzu Motors with diesel fuel. NB: sulfur content not stated)	Short-term inhalation study (pulmonary effects) in male C57BL/6J mice	DEP	0 or 100µg/m^3 DEP exhaust (8h/d, for 1 or 7days)	DEP had no effect on the expression of Nos2, a biomarker of oxidative stress Ceramide production in the bronchial epithelial cells and Sphk1 mRNA expression was induced after DEP exposure	Not identified	100µg/m^3 (NO_2 concentration not stated)
Sharkhuu et al. 2010	NTDE (presumed based on web search indicating it complies with Euro V) 4.8 kW (6.4 hp) Yanmar L70 V engine using low sulfur diesel (32ppm).	*In utero* exposure inhalation study (immunity, pulmonary effects) in pregnant BALB/c mice	DEP (NO_2 also measured)	0, 800 or 3,100µg/m^3 DEP exhaust (8h/d , for GD9 to GD18)	Inhaled DEP affected pregnancy outcome but did not alter bodyweight of the offspring. Lung TLR4 mRNA expression, the number of neutrophils in the bronchoalveolar lavage fluid (BALF) and splenic T cells expressing CD45+CD3+CD4+ and CD4+CD25+ were differentially affected (increased or decreased) at one or both exposure concentrations.	Not identified	800µg/m^3 (NO_2 = 0.4ppm)
Singh and Arora 2017 (Abstract only)	TDE (Euro II) and NTDE (Euro IV) (engine type, fuel used and sulfur content not stated)	Inhalation study in Balb/c mice	DEP	No concentrations stated (5d/wk, for 3wks)	↑ in AHR and elastance and inflammation in the lungs. These changes were more evident in mice exposed to Euro II than Euro IV.	Not determined	Not determined
Singh and Arora 2021	NTDE (L100V diesel engine, Euro tier IV) (NB: Fuel type and sulfur content not stated)	Intermediate exposure inhalation study (pulmonary function) in C57BL/6 mice	DEP	No concentrations stated (30min/d, 5d/wk, for 8wks)	↑lung resistance and tissue elastance with ↓in inspiratory capacity ↑ macrophages, neutrophils and monocytes in BALF ↑ inflammation and alveolar wall thickening in lungs ↑ mucus producing goblet cells plus black soot in lungs ↑ α-SMA and fibronectin in lung	Not determined	Not determined

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Sunil et al. 2009	TDE (presumed) (Yanmar YDG5500 Diesel generator using premium low sulfur, <500 ppm, and 40-weight motor oil (Proline HD40)	Inhalation study (pulmonary effects) in aged mice (2M and 18M)	DEP (NO_2 also measured)	0, 300, or 1000µg/m^3 DEP exhaust (3h once or 3h/d, for 3d)	Structural alterations in the lungs of older but not younger mice, including patchy thickening of the alveolar septa and inflammatory cell localization in alveolar spaces (more pronounced at highest concentration). ↑in BAL nitrogen oxides and expression of lipocalin 24p3 in older mice but not in younger mice. Tumour Necrosis Factor alpha (TNFα) expression was upregulated in lungs and cytokines (IL6 and IL8) were elevated in younger and older mice. ↓ constitutive expression of manganese superoxide dismutase (MnSOD) in younger mice and not detectable in older mice	Not identified	300µg/m^3 (NO_2 = 4.3ppm)
Tanaka et al. 2013	TDE (presumed) 8L diesel engine J08C, Hino Motors (fuel type and sulfur content not stated)	Sub-chronic exposure inhalation study (immune system) in ICR mice	DEP & fDEP (NO_2 also measured)	fDEP: 0µg/m^3 DEP: 36 or 169µg/m^3 (5h/d, 5d/wk, for 8wks) (exhaust pre-diluted prior to exposure indicating exposure was to same volume of air)	↑ ovalbumin-induced eosinophilic airway inflammation, cytokine and chemokine production and production/release of myeloperoxidase into alveolar space in both fDEP and DEP (high concentration) exposed groups.	DEP: 36µg/m^3 (NO_2 = 0.15ppm)	fDEP: 0µg/m^3 (NO_2 = 0.51ppm) DEP: 169µg/m^3 (NO_2 = 0.51ppm)
Thompson et al 2019	NTDE (presumed based on web search indicating it complies with Euro V) 4.8 kW (6.4 hp) Yanmar L70 V engine using low sulfur diesel (16ppm).	Acute exposure inhalation study (cardiovascular dysfunction) in Spontaneously Hypertensive (SH) Rats	DEP (NO_2 also measured)	0, 150 or 500µg/m^3 DEP exhaust (for 4h)	Serum from DE-exposed rats had significant changes in multiple serum proteins and caused ↓ rat aortic endothelial cells (RAECs)NOS activity at 150 & 500µg/m^3 and ↑ REAC VCAM-1 expression at 500µg/m^3. While rats exposed to DEP demonstrated increased heart rate at the start of LVp assessments, heart rate, systolic pressure, and double product fell below baseline in DEP-exposed rats compared to filtered air during recovery from dobutamine, indicating dysregulation of post-exertional cardiovascular function.	Not identified	150µg/m^3 (NO_2 = 0.1ppm)
Tong et al. 2019	Not stated (36-kW diesel generator, Model 3W991A, Dayton Electric MFG. CO. using ultra-low sulfur diesel (sulfur content not stated)	Acute exposure inhalation study (cardiovascular effects) in female C57Bl/6 mice	DEP (NO_2 also measured)	0, 330 (photochemically aged), or 460 (fresh) µg/m^3 DEP exhaust (for 4h)	Fresh DEP: ↓ left-ventricular-developed pressure and min and max contractility (dP/dt) Aged DEP: ↓ left-ventricular-developed pressure and max contractility (dP/dt) Greater reductions observed with fresh DEP.	Not identified	Fresh DEP: 460µg/m^3 (NO_2 = 0.29ppm) Aged DEP: 330µg/m^3 (NO_2 = 0.23ppm)
Umezawa et al. 2018	TDE (four-cylinder 2179-cc diesel engine, AA-4LE2; Isuzu Motors with diesel fuel. NB: sulfur content not stated)	Inhalation study (liver toxicity) in C57Bl/6 mice (normal or n-3 PUFA–deficient).	DEP (NO_2 also measured)	100µg/m^3 DEP exhaust (8h/d, for 4wks)	In n3-PUFA deficient mice (compared to normal mice): ↑ hepatic lipid droplets accumulation and expression of genes promoting fatty acid synthesis (Acaca, Acacb, and Scd1), ↑ plasma leptin and the expression of fatty acid synthesis-related genes (Acacb, Fasn, and Scd1) and potentially enhanced hepatic fatty acid synthesis and subsequently accumulation of lipid droplets. DEP exposure and intake of n-3 PUFA–deficient diet may be an additional risk factor for the incidence of non-alcoholic fatty liver disease.	Not identified	100µg/m^3 (NO_2 = 0.15ppm)
Valberg and Crouch 1999	Not relevant (meta-analysis)	Meta-analysis of rat lung tumours from lifetime inhalation of diesel exhaust	DEP	Not relevant	No tumorigenic effect of DEP in rats exposed to <600µg/m^3. Upper-bound human UR of 9.3 x 10^6 per µg/m^3 estimated. Meta analysis of the low-exposure data in rats does not support a lung cancer risk for DEP exposure at non overload conditions. Average ambient concentrations of DEP (0-3µg/m^3) are < 1% of the concentration associated here with a threshold of tumour response in the rat bioassay.	Threshold response between 200 and 600µg/m^3	Upper-bound human UR of 9.3 x 10^6 per µg/m^3

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Valentino et al. 2016a	Not determined 25KVA Loxam engine, with a 500 nm particle filter (fuel used and sulfur content not stated)	Maternal exposure (nose only) inhalation (developmental) study in New Zealand white rabbits	DEP (NO_2 also measured)	1,000µg/m^3 DEP exhaust (2h/d, 5d/wk for 20d during gestation)	Mid gestation: growth retardation with ↓ head length and umbilical pulse. Near term: ↓ foetal head length, plasma insulin and IGF1 concentrations. ↓ placental efficiency, ↓ placental blood flow and ↓ foetal vessel volume. Non-aggregated and "fingerprint" nanoparticles were observed at various locations, in maternal blood space, in trophoblastic cells and in the foetal blood, demonstrating transplacental transfer. Adult female offspring were bred with control males. Although foetoplacental biometry was not affected near term, second generation foetal metabolism was modified by grand-dam exposure with ↓plasma cholesterol (p= 0.008) and ↑ triglyceride concentrations.	Not identified	1,000µg/m^3 (NO_2 = 0.7ppm)
Valentino et al. 2016b (abstract only)	Not determined 25KVA Loxam engine, with a 500 nm particle filter (fuel used and sulfur content not stated)	Prenatal inhalation (reproductive) study in rabbits	DEP (NO_2 also measured)	1,000µg/m^3 DEP exhaust (2h/d, 5d/wk for 20d during gestation)	↓ foetal head length and umbilical pulse at GD14. ↓ foetal head length, foetal plasma insulin and IGF1, foetal/placental weight ratio, placental blood flow and foetal vessel volume at GD28 Nanoparticles observed in maternal blood space, placenta and foetal blood. ↑ fat globules number/volume in mammary alveolar and milk fatty acid content. ↑blood pressure, glycemia, and perirenal fat mass and ↓ HDL-cholesterol in F1 adult males and increased and ↑ plasma triglycerides in females ↑ epididymal sperm DNA fragmentation. F2 generation (Female offspring were bred to control males): ↓foetal plasma cholesterol and ↑ triglyceride concentrations.	Not identified	1,000µg/m^3 (NO_2 = 0.7ppm)
Watanabe and Oonuki 1999	TDE (presumed based on study year) (309-cc NFAD50 Yanmar diesel engine, fuel type and sulfur content not stated)	Sub-chronic inhalation study in F344 rats	DEP & fDEP (NO_2 also measured)	fDEP: Not stated DEP: 5,630µg/m^3 (6h/d, 5d/wk for 3M) (exposure was to same volume of air)	fDEP and DEP: ↑ serum levels of testosterone and oestradiol and ↓ follicle-stimulating hormone, sperm production, activity of cular hyaluronidase and step 18 and 19 spermatids in stage VI, VII, and VIII tubules in the testes. fDEP only: ↓ luteinizing hormone	Not identified	fDEP: Not stated (NO_2 not stated) DEP: 5,630µg/m^3 (NO_2 = 4.1ppm)
Watanabe & Ohsawa 2002	TDE (presumed based on study year) (309-cc NFAD50 Yanmar diesel engine, fuel type and sulfur content not stated)	*In-utero* exposure inhalation study (reproductive developmental effects) in male offspring of pregnant Fischer rats	DEP & fDEP (NO_2 also measured)	fDEP: not stated DEP: 1,730µg/m^3 (6h/d, from GD7 until birth) (exposure was to same volume of air)	↑ serum testosterone and ↓ relative spleen and thymus weight in both fDEP and DEP exposed groups.	Not identified	fDEP: not stated (NO_2 not stated) DEP: 1,730µg/m^3 (NO_2 = 0.79ppm)
Watanabe 2005	TDE (presumed based on study year) (309-cc NFAD50 Yanmar diesel engine, fuel type and sulfur content not stated)	Prenatal exposure inhalation study (reproductive function) in F344 rats	DEP & fDEP (NO_2 also measured)	fDEP: Not stated DEP: 171 & 1,710µg/m^3 DEP exhaust (from GD7 to delivery) (exposure was to same volume of air)	fDEP and DEP exposed groups (both concentrations): ↓daily produced sperm, spermatids and Sertoli cells (96d after birth) and ↑ spermatids/Sertoli cells ratio and follicle-stimulating hormone levels As all exhaust-exposed groups showed almost the same reactions toward the inhalation, the gaseous phase must have included the responsible toxicants.	Not identified	fDEP: Not stated (NO_2 = 0.10ppm) DEP: 171µg/m^3 (NO_2 = 0.10ppm)

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Weldy et al 2014	TDE (Presumed) (Single-cylinder Yanmar diesel generator engine (YDG5500), fuelled with standard highway-grade number 2 diesel fuel)	Prenatal exposure inhalation study (intrauterine conditions, cardiovascular effects) in male and female C57Bl/6J mice	DEP (NO_2 also measured)	0 & 300µg/m^3 DEP exhaust (6h/d, 5d/wk, Embryonic Day 0.5 to Embryonic Day 17.5)	F0: ↑ embryo resorption, and promotes placental haemorrhage, focal necrosis, compaction of labyrinth vascular spaces, inflammatory cell infiltration and oxidative stress. F1: ↑ body weight, but ↓ blood pressure in adult male mice and ↑ susceptibility to pressure-overload induced heart failure	Not identified	300µg/m^3 (NO_2 = 0.06ppm)
Werchowski et al 1980a	TDE (Engine type, fuel and sulfur content were not stated).	Developmental inhalation study (teratogenic effects) in male and female albino Sprague Dawley rats	Diesel exhaust	10% diluted DEP exhaust (8h/d for day 6 to 15 of gestation)	No malformations nor other teratogenic effects were observed in the unborn rat foetus.	10% diesel exhaust	Not identified
Werchowski et al 1980b	TDE (Engine type, fuel and sulfur content were not stated).	Developmental inhalation study (teratogenic effects) in New Zealand albino rabbits	Diesel exhaust	10% diluted DEP exhaust (8h/d for day 6 to 18 of gestation)	No malformations nor other teratogenic effects were observed in the unborn rabbit foetus.	10% diesel exhaust	Not identified
Win-Shwe et al. 2008	TDE (presumed) 8L diesel engine J08C, Hino Motors (fuel type and sulfur content not stated)	Short-term inhalation study (spatial learning) in male BALB/c mice	Nano-rich DEP	0 & 148.86µg/m^3 nano-rich DEP exhaust with or without lipoteichoic acid (LTA) injections [DPE/LTE(+) or DPE respectively] (5h/d, 5d/wk, for 4 weeks)	DEP exposure alone did not affect to reach the hidden platform however mice from the DEP/LTA (+) group took a longer time to reach the hidden platform. ↑ relative mRNA levels of the NMDA receptor subunits and proinflammatory cytokines in hippocampus of DEP/LTA (+) group	DPE: 148.86µg/m^3 DPE/LTA(+): Not identified	DPE/LTA(+): 148.86µg/m^3
Win-Shwe et al. 2012	TDE (presumed) 8L diesel engine J08C, Hino Motors (fuel type and sulfur content not stated)	Sub-chronic exposure inhalation study (nervous system) in female BALB/c mice	DEP & fDEP (NO_2 also measured)	fDEP: 5.5µg/m^3 DEP: 35.5 & 122µg/m^3 (5h/d, 5d/wk, for 3 months) (unclear whether same volume of exhaust was used for both exposures, but likely to be the case)	Impaired spatial learning – increased escape latency from maze in DEP (high exposure) group only ↑ escaped latency and mRNA expression (NR2A) in the DEP (high concentration) exposed group ↑ mRNA expression (BDNF) in the DEP (high exposure) and fDEP exposed group	DEP: 35.5µg/m^3 (NO_2 = 0.16ppm)	fDEP: 5.5µg/m^3 (NO_2 = 0.47ppm) DEP: 122µg/m^3 (NO_2 = 0.48ppm)
Win-Shwe , et al. 2011	TDE (presumed) 8L diesel engine J08C, Hino Motors (fuel type and sulfur content not stated)	Sub-chronic exposure inhalation study (nervous system) in female BALB/c mice	DEP & fDEP (NO_2 also measured)	fDEP: 8.5µg/m^3 DEP: 47.8 & 129µg/m^3 (5h/d, 5d/wk, for 3 months) (unclear whether same volume of exhaust was used for both exposures, but likely to be the case)	Impaired novel object recognition and ↓ discrimination index in both fDEP and DEP (high concentration) exposed groups. ↓ expression of glutamate transporter EAAT4 also in both fDEP and DEP (high concentration) exposure groups. ↑ glutamic acid decarboxylase GAD65 in the hippocampus of DEP (high concentration) exposed mice. Prominent microglia activation in the hippocampal area of DEP (high cocnentration) exposed mice	DEP: 47.8µg/m^3 (NO_2 = 0.16ppm)	fDEP: 8.5µg/m^3 (NO_2 = 0.46ppm) DEP: 129µg/m^3 (NO_2 = 0.47ppm)
Win-Shwe et al. 2016	TDE (presumed) 8L diesel engine J08C, Hino Motors (fuel type and sulfur content not stated)	Pre and postnatal exposure inhalation study (social behaviour) in pregnant BALB/c mice (includes exposure to DEP and DEP with additional secondary organic aerosols (DEP-SOA))	DEP DEP-SOA (NO_2 also measured)	0, 113µg/m^3 DEP exhaust, 131µg/m^3 DEP-SOA exhaust or gas (5h/d, 5d/wk, until PND21)	DEP-SOA (F1 males): sociability and social novelty preference as well as social interaction were remarkably impaired. ↓ expression levels of oestrogen receptor-alpha, oxytocin receptor mRNAs and ↑ expression levels of HO-1 mRNAs and glutamate. Poor social novelty preference in DEP, DEP-SOA and gas exposed groups	Not identified	DEP: 113µg/m^3 (NO_2 = 0.43ppm) DEP-SOA: 131µg/m^3 (NO_2 = 0.99ppm)

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Win-Shwe et al. 2021	TDE (presumed) 8L diesel engine J08C, Hino Motors (fuel type and sulfur content not stated)	Pre and postnatal exposure inhalation study (autism) in pregnant BALB/c mice (includes exposure to DEP and DEP with additional secondary organic aerosols (DEP-SOA))	DEP DEP-SOA (NO_2 also measured)	0, 101µg/m^3 DEP exhaust, 118µg/m^3 DEP-SOA exhaust or gas (5h/d, 5d/wk, GD8 to PND8)	DEP-SOA-exposed male and female rats showed poor sociability and social novelty preference, socially dominant behaviour, and increased repetitive behaviour. ↓ exploration time for DEP and DEP-SOA exposed groups to Stranger 1 and Stranger 2, ↑ win% in DEP-SOA exposed male group and DEP and DEP-SOA exposed female group and ↑ repetitive behaviour in DEP and DEP-SOA exposed male group and DEP-SOA exposed female group. Serotonin receptor (5-HT(5B)) and brain-derived neurotrophic factor (BDNF) mRNAs were downregulated whereas interleukin 1 β (IL-β) and haeme oxygenase 1 (HO-1) mRNAs were upregulated in the prefrontal cortex of male and female rats exposed to DEP-SOA. ↓ mRNA expression (serotonin factors, Nign3, or BDNF) in DEP and DEP-SOA exposed male and female groups, ↑ mRNA expression (IL1β or HO1) in DEP and DEP-SOA exposed male and female groups. Glutamate concentration was also increased significantly in DEP-SOA-exposed male and female rats	Not identified	DEP: 101µg/m^3 (NO_2 = 0.58ppm) DEP-SOA: 118µg/m^3 (NO_2 = 1.14ppm)
Xu et al. 2009	TDE (Presumed) (Single-cylinder Yanmar diesel generator engine (YDG5500), fuelled with standard highway-grade number 2 diesel fuel)	Short to intermediate exposure inhalation study in male ApoE-deficient mice (with either scaffold implantation subcutaneously or hindlimb ischemia)	DEP	0, 1,000µg/m^3 DEP exhaust (6h/d, 5d/wk, for 2,5, or 8 wks)	↑ total cell counts in the scaffolds, aortic, and perivascular fat tissues. Enhanced macrophage infiltration and ↑ CD31 expression increased in the scaffold while ↓ endothelial nitric oxide synthase in the aortic wall. ↑ vessel volume in ischemic and non-ischemic hindlimbs. Induced capillary-like tube formation in endothelial cells *in vitro*, and capillary sprouting from aortic rings *ex vivo*. ↑ mRNA expression of vascular endothelial growth factor (VEGF) and hypoxia-inducible factor (HIF)-1α, and ↓prolylhydroxylase (PHD) 2 expression.	Not identified	1,000µg/m^3 DEP (NO_2 concentration not stated)
Yamagishi et al. 2012	TDE (presumed) 8L diesel engine J08C, Hino Motors (fuel type and sulfur content not stated)	Sub-chronic exposure inhalation study (reproductive developmental effects) in male Fisher rats	DEP & fDEP (NO_2 also measured)	fDEP: 3.1µg/m^3 DEP: 38 or 149µg/m^3 (5h/d, 5d/wk, for 1, 2, or 3M) (exhaust pre-diluted prior to exposure indicating exposure was to same volume of air)	↓ testosterone in testes (3 months) in fDEP exposed group only. ↑ testosterone in plasma in DEP (high concentration) and fDEP exposed groups. ↑ testosterone in testes in DEP (high concentration) exposed group at 1 month and ↓ testosterone in testes in fDEP exposed group at 3 months ↑ androstenedione in DEP (high concentration) exposed group (1 month only) ↓ CYP17α in fDEP exposed group at 1 and 2 months. Some changes in DEP (low concentration) exposed group however transient and not as numerous as in fDEP and DEP (high concentration) exposure groups.	DEP: 38µg/m^3 (NO_2 = 0.17ppm)	fDEP: 3.1µg/m^3 (NO_2 =0.51ppm) DEP: 149µg/m^3 (NO^2 = 0.53ppm)
Yokota et al. (2009)	TDE (presumed) 2369-cc diesel engine (Isuzu Motors, Ltd.) using a commercial light oil (sulfur content not stated)	Prenatal exposure inhalation study (neurological) in pregnant mice	DEP (NO_2 also measured)	1,000µg/m^3 DEP exhaust (8h/d, 5d/wk, for GD2 to GD17)	↓of locomotion and dopamine (DA) turnover in the striatum and nucleus accumbens. Maternally inhaled DEP might influence the development of central dopaminergic system and result in behaviour disorder.	Not identified	1,000µg/m^3 DEP (NO_2 = 0.23ppm)

Source	Diesel engine/exhaust type	Study Type	Pollutant	Concentrations (µg/m^3)	Effect observed	NOAEC/NOEC	LOAEC/LOEC
Yokota et al. (2013)	TDE (presumed) 2369-cc diesel engine (Isuzu Motors, Ltd.) using a commercial light oil (sulfur content not stated)	Short-term exposure inhalation study (neurological) in C57BL/6J mice	DEP (NO_2 also measured)	90µg/m^3 DEP exhaust (8h/d, for 28 days)	Changed expression levels of 112 genes and dysregulated genes were involved in inflammation and immune response. Background gene expression of the olfactory bulb of mice reared in a standard cage environment was changed by DEP, whereas there was no significant effect of DEP on gene expression levels of mice reared with environmental enrichment.	Not identified	90µg/m^3 DEP (NO_2 = 0.13ppm)

Acute and short term exposure = 28days exposure or less, Sub-chronic and intermediate exposure = >28days and < 6 months, long-term and chronic exposure + > 6 month exposure, DEP = diesel exhaust particulate, fDEP = filtered diesel exhaust, NO_2 = nitrogen dioxide, ↑ = increased, ↓ = decreased, GD = Gestational Day, NOEC = No observable effect concentration, NOAEC = No observable adverse effect concentration, LOEC = Lowest observable effect concentration, LOAEC = Lowest observable adverse effect concentration, PND = Post natal day, NTDE -= New Technology Diesel Engines, TDE = Traditional Diesel Engines, Pax6 = Paired box 6 (transcription factor, protein coded gene), Tbr1 = T-box brain 1 (transcription factor, protein coded gene), Tbr2 = T-box brain 2 (transcription factor, protein coded gene), Sp1 = Protein 1 (transcription factor, protein coded gene), TNFα = tumor necrosis factor alpha (inflammatory cytokines), Creb1 = CAMP Responsive Element Binding Protein 1, E0 = Embryonic day 0, NTS = Nucleus of the solitary tract (NTS), a key region in the medulla, IL-1β, IL-6, and IL-17A, = Interleukin1β, 6 or 17A (inflammatory cytokines), TNF-α = tumor necrosis factor alpha (inflammatory cytokines), iNOS = inducible nitric oxide synthase, CD36 = class B scavenger receptor, 8-OHdG = 8-hydroxydeoxyguanosine, BAL = bronchoalveolar lavage, BW = bronchial wash

Blue shaded rows identify those animal studies with information for DEP and fDEP considered in the report when evaluating whether the effects (adverse or biochemical) observed are related to the gaseous or particle component of diesel exhaust.

Bolded text in rows identify those long-term animal studies with three or more doses considered for identification of a departure point (e.g. NOAEC based on adverse effects).

1) Gould, T. Larson, T., Stewart, J., Kaufman, J.D., Slater, D., McEwen, N. (2008). *A controlled inhalation diesel exhaust exposure facility with dynamic feedback control of PM concentration*. Inhalation Toxicology, 20:1, 49-52,

2) NO_2 concentrations reported to range from ~0.35 to 3. 5 ppm, presumably consistent with diesel dilution hence in this assessment it was assumed that a NO_2 concentration of 0.35ppm corresponds to the low exposure group (100) and 3.5ppm corresponds to the high exposure group (1,000). As such, a NO_2 concentration of 1.05ppm is considered likely relevant for the middle exposure group (300 ÷ 100 x 0.35ppm = 1.05ppm).

3) It is not clear what the concentration in air is as DEP were collected in solution and then presumably administered as an aerosol. Hence the concentrations shown may be in the solution (and aerosol droplets) rather than in air.

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APPENDIX D

Summary of controlled human exposure studies with DEE (n=34)

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Andersen et al. 2019	10.3 (as black carbon or BC) (LOEC) NO_2: 54 (0.03ppm)	6 hours/day	3 days	29 self-reported healthy, non-asthmatic non-smokers (21-71 yrs)	Travel inside either diesel or electric trains (cross-over repeat measures, study participants acted as their own control)	Not Stated (NS) (diesel-powered train)	Lung function, cardiovascular function, effect biomarkers in blood.	↓ lung function; ↑ DNA strand breaks in PBMCs. No change in oxidative damage DNA, soluble cell adhesion molecules, acute phase proteins or urinary excretion of PAH metabolites.
Cardenas et al. 2021	300 (LOEC)	2 hours	1	13 healthy volunteers (26 ± 3.8 yrs)	Controlled exposure chamber (with intermittent exercise)	NS (diesel power generator)	DNA methylation	Altered DNA methylation in target bronchial epithelial cells.
Carlsten et al. 2007	0, 100, or 200 (as $PM_{2.5}$) (NOEC: 200) NO_2: 0.01-0.035ppm	2 hours	1	13 healthy volunteers, non-smokers (20.7-42.6 yrs)	Controlled exposure chamber (with intermittent exercise)	2002 model turbocharged direct-injection 5.9L Cummins B-series engine at load.	Fibrinolytic burden (D-dimer), platelet number, endothelial injury, inhibition of fibrinolytic pathway and inflammation.	No statistically significant changes in pro-thrombotic endpoints evaluated; no adverse effects reported.
Cliff et al. 2016	300 (as $PM_{2.5}$) (NOEC) or filtered air (FA)	2 hours	1 (Repeated after 4 weeks)	27 healthy non-smoking adults (19-49 yrs)	Controlled exposure chamber (with intermittent exercise)	NS (diesel power generator)	Neurotoxicity measurable using biomarkers of CNS; Inflammatory cytokines interleukin 6 (IL-6) and tumour necrosis factor (TNF-α).	A short-term exposure to DE amongst healthy adults does not acutely affect the systemic or CNS biomarkers.

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Clifford et al 2017	300 (as $PM_{2.5}$) or filtered air (FA) (LOEC)	2 hour	1 (Repeated after 4 weeks)	17 non-smokers with 47% of participants with asthma (20-46 years)	Controlled exposure chamber (with intermittent exercise)	NS (diesel power generator)	The effect of allergen and diesel exhaust exposure on bronchial epithelial DNA methylation.	Exposure to allergen alone, diesel exhaust alone, or allergen and diesel exhaust together (coexposure) led to significant changes in 7 CpG sites at 48 hours. This suggest that specific exposures can prime the lung for changes in DNA methylation induced by a subsequent insult.
Fountoulakis et al 2019	Exposure concentration not stated (as $PM_{2.5}$) or filtered air (FA)	2 hour	1	40 healthy subjects	Controlled exposure chamber (with intermittent exercise)	NS (diesel power generator)	Acute and short- term effects of diesel exhaust particles (DEPs) on endothelial function, vascular properties, inflammation.	Changes in biomarkers of endothelial function and vascular properties. Short-term exposure to diesel exhaust fumes may impair endothelial function, arterial wall properties, inflammation and fibrinolysis highlighting the harmful impact of particulate matter to cardiovascular system.

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Giles et al 2018	300 (as PM2.5) (LOEC) or filtered air (FA)	30 minutes	6 trials separated by a 7-day period	18 males non-smokers and without history of respiratory or cardiovascular disease	Controlled exposure chamber (with intermittent exercise)	NS (diesel power generator)	The effects of low- and high-intensity cycling with diesel exhaust (DE) exposure on pulmonary function, heart rate variability (HRV), fraction of exhaled nitric oxide (FeNO), norepinephrine and symptoms.	DE did not modify any effects of exercise intensity on HRV or norepinephrine. However, throat and chest symptoms were significantly greater immediately following DE exposure cf. FA. Healthy individuals may not experience greater acute pulmonary and autonomic effects from exercising in DE compared to FA; therefore, it is unclear if such individuals will benefit from reducing vigorous activity on days with high concentrations on particulate matter.
Gluck et al 2003	DEE Range between 31 and 60 (LOEC) – as diesel soot (details not provided) Benzo[a]pyrene range: 10 and 15 ng/m^3	5 years	8.4 hr/day; 42 hr/week	194 non-smoking customs officers	Travel inside diesel fuelled truck	TDE Heavy-goods vehicles with diesel engines	Adverse effects of chronic exposure to diesel engine emission (DEE) on respiratory mucous membranes.	Goblet cell hyperplasia with ↑ metaplastic and dysplastic epithelia and ↑in leukocytes found in DEE-exposed population. No evidence of progression of the cytopathologic changes. The changes were described as chronic inflammation of the nasal mucous membrane in the presence of chronic DEE exposure (chemical-induced rhinitis).

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Gould et al 2008	DEE: 100 and 200 (as PM2.5) FA: 2.9, 4.3, and 7.1	Describes exposure system only	Not applicable	Not applicable	Not applicable	2002 model year turbocharged direct-injection 5.9-L Cummins, Inc. (Columbus, IN), B-series diesel engine (model 6BT5.9G6) and a 100-kW generator (454ppm S diesel)	None (reports on the exposure system only)	While the exposure system does not entirely replicate diesel exhaust conditions in the atmosphere due to the relatively low ratio of nitrogen dioxide to total NOx, the fine particulate matter size distributions are quite similar to those of aged diesel exhaust. The facility enables study of the relationship between diesel exhaust and cardiovascular and respiratory health effects in human and animal models.
Hemmingsen et al 2015	276 (NOEC)	3h	4	18 healthy non-smoking subjects (9 males and 9 females) (40–66 yrs)	Controlled exposure chamber (with intermittent exercise) 22 m^3 volume	NS (passenger car)	Gene expression markers of inflammation, oxidative stress, and DNA repair.	3-h exposure to DE caused no genotoxicity, oxidative stress or inflammation in peripheral blood mononuclear cells (PBMCs).
Koch et al 2020	300 (as $PM_{2.5}$) (LOEC) or filtered air (FA)	1h	1	18 adults with EIB exercise-induced broncho-constriction (18-35 yrs)	Controlled exposure chamber (with intermittent exercise)	Transitional engine(5.5-kW diesel engine compliant with US EPA Tier 2, constant load of 500W)	Micro- and macrovascular response to physical activity after β2-agonist use while breathing diesel exhaust (DE) in individuals with exercise-induced bronchoconstriction.	Acute physical activity induced a vasodilatory response in the micro- and microvasculature in healthy adults, despite breathing DE. DE exposure was associated with significantly increased heart rate. This might be indicative of an increased sympathetic response to physical activity while breathing DE.

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Koch et al 2021	300 (as $PM_{2.5}$) (NOEC) or filtered air (FA)	1h	1	19 participants with EIB exercise-induced broncho-constriction (EIB) (22-33 yrs)	Controlled exposure chamber (with intermittent exercise)	Transitional engine(5.5-kW diesel engine compliant with US EPA Tier 2, constant load of 500W)	Respiratory responses in individuals with EIB when completing a cycling bout while being exposed to diesel exhaust (DE) or filtered air (FA) with and without the inhalation of salbutamol (SAL), a short-acting ß2-agonist.	Bronchodilation in response to salbutamol (SAL) and acute cycling was observed, independent of FA/DE exposure. DE ↑ ventilatory capacity by 2.4% but this did not affect dyspnoea. The use of salbutamol prior to moderate-intensity exercise when breathing high levels of DE, does not reduce respiratory function or exercise ventilatory responses for up to 60 min following exercise.
Kramer et al 2017	300 (as $PM_{2.5}$) (NOEC) 5 filtered air (FA) (the control for DE) NO_2 0.5 ppm NO 0.0087 ppm	2h	1	18 Atopic adults 19–49 yrs	Controlled exposure chamber followed by instilled allergen or saline.	EPA Tier-3 compliant 6.0kW diesel generator (with ULSD)	Study population following exposure to allergen and diesel exhaust (DE) by measuring serum and lung adiponectin, leptin, and resistin.	DE co-exposure with allergen showed correlations (not significant) between adiponectin/leptin ratio and FEV1 changes & airway responsiveness measures. DE did not augment allergen effect.

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Langrish et al 2011	300 (NOEC) filtered air (FA)	1 h	1	Healthy non-smokers	Controlled exposure chamber (with intermittent exercise)	NS (diesel power generator)	The role of nitric oxide (NO) in endothelium-dependent vascular dysfunction, increased blood pressure and arterial stiffness.	DE inhalation exaggerates the pressor and vasoconstrictor effects of systemic NO synthase inhibition, and increases plasma nitrite concentration, consistent with compensatory upregulation of basal NO production to counter enhanced consumption. In the presence of NO synthase inhibition, and absence of endogenous NO, DE inhalation does not cause further vasomotor dysfunction.

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Lucking et al 2011	320 (as PM2.5) (LOEC) 7.2 filtered DE (FA) NO2: 0.69ppm (DE) vs 3.44ppm (FA)	1h	1	19 healthy volunteers (mean age, 25±3 yrs)	Controlled exposure chamber	Volvo diesel engine (TD40 GJE, 4.0L, 4 cylinders) (S content 5-7mg/kg, PAH content 2-6%).	Cardiovascular function and thrombus formation followed by diesel exhaust inhalation.	Compared with FA, DE inhalation was associated with ↓ vasodilatation and ↑ex vivo thrombus formation under both low- and high-shear conditions. Particle trap markedly reduced DE particulate number (from 150 000 to 300 000/cm^3 to 30 to 300/cm^3) and mass and was associated with ↑ vasodilatation, ↓ thrombus formation, and an ↑ in tissue-type plasminogen activator release. A particle trap (diesel particulate filter–continuously regenerating trap) prevents several adverse cardiovascular effects of exhaust inhalation in men.
Lundback et al 2009	Approximately 350 (LOEC) or filtered air (FA)	1h	1	12 non-smoking, healthy men (mean age 26, range 21–30 yrs)	Controlled exposure chamber (with intermittent moderate exercise)	An idling Volvo diesel engine (Volvo, TD45, 4.5 L, 4 Cylinders, 680 rpm) using Gasoil E10	The effect of DE exposure on arterial compliance using a validated non-invasive measure of arterial stiffness.	Acute exposure to DE is associated with an immediate and transient ↑ in arterial stiffness (blood pressure & heart rate unaffected).

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Mills et al 2005	300 (as $PM_{2.5}$) (LOEC) NO2 1.6 ppm; NO 4.5 ppm; CO 7.5 ppm; total hydrocarbons,4.3 ppm; formaldehyde, 0.26 µg/m^3	1h	1	30 healthy men non-smokers between 20 and 38 yrs	Controlled exposure chamber (with intermittent exercise)	Assumed TDE (Idling lorry, details not provided)	The effects of DE inhalation on vascular and endothelial function in humans.	No differences in resting forearm blood flow or inflammatory markers after exposure to DE or air. DE blunted increase in forearm blood flow induced by bradykinin, acetylcholine and sodium nitroprusside, which persisted for 6 hours.
Mills et al 2011	348 (as $PM_{2.5}$) (LOEC) 6 filtered DE (FDE) <1 filtered air (FA) 70 pure carbon nanoparticulate NO2: 0.2ppm or <0.01ppm (FA)	2h	4 (at least 2-week washout between exposures)	16 healthy male non-smokers aged between 18 and 32 yrs	Controlled exposure chamber (with intermittent moderate exercise)	NTE Unloaded diesel engine (Deutz, 4 cylinder, 2.2 L, 500 rpm) using gas oil (EU Stage 5, EPA Tier 4). Filtered exhaust was generated in an identical manner, but the exhaust was passed through a highly efficient TE38 Teflon filter.	Forearm blood flow during intra-brachial bradykinin, acetylcholine, sodium nitroprusside and verapamil infusions.	Inhalation of DE and FDE ↑ systolic blood pressure but only DE attenuated vasodilation to bradykinin, acetylcholine, and sodium nitroprusside. Other treatments did not have effect on these parameters.

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Mudway et al 2004	108 (as PM_{10}) (NOEC) or filtered air (FA) NO_2 0.2 ppm Formaldehyde 43.5 µg/m3	2h	2 (least 3 weeks apart)	25 healthy non-smokers, 9 females, 16 males; mean age 24 years (19–42 yrs)	Controlled exposure chamber (with intermittent exercise)	TDE (Idling Volvo diesel engine, Volvo TD45, 4.5L, 4 cylinders, 1991, 680rpm).	Pulmonary inflammation, bronchoconstriction, increased airway reactivity, and oxidative stress in healthy subjects.	DE ↑ subjective symptoms & mild bronchoconstriction (marked inter-individual variability). No airway inflammation or antioxidant depletion was seen. Studied effect is not observed to any great extent in the airways of healthy subjects. This is explained by the antioxidant network at the air–lung interface in healthy subjects can deal with the oxidative challenge posed by DE at ambient concentrations.
Nightingale et al 2000	200 (as PM_{10}) (Minimal LOEC)	2 h	2 (after a 4-week washout period)	10 healthy, non-smoking volunteers (three males and seven females),(mean age 28 yr)	Controlled exposure chamber	TDE (Stationary diesel engine)	The inflammatory response to inhalation of diesel exhaust particulates (DEP) in normal volunteers.	There were no changes in cardiovascular parameters or lung function following exposure to DEP. ↑ in sputum neutrophils (9%) & myeloperoxidase but no change in inflammatory markers. Authors concluded exposure to DEPs at high ambient concentrations leads to an airway inflammatory response in healthy volunteers.
Peretz et al 2008	100 or 200 (as $PM_{2.5}$) (NOEC) or filtered air (FA)	2h	2 (at least 2 weeks)	16 healthy, non-smoking subjects (18–49 yrs)	Controlled exposure chamber	TDE (2002 model turbocharged direct-injection 5.9 l Cummins B-series engine).	Heart rate variability (HRV).	There was no observed consistent DE effect on the autonomic control of the heart in a controlled-exposure experiment in young participants.

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Pourazar et al 2004	300 (as PM_{10}) (LOEC) or filtered air (FA)	1h	2 (at least 3 weeks apart)	15 healthy, non-atopic non-smoking subjects (11 males, 4 females) mean age 24 years (range 21–28 yrs)	Controlled exposure chamber	NS	The epithelial response to DE in vivo, with particular reference to possible TH2 response, in non-atopic healthy subjects.	DE induced significant ↑ in IL-13 expression (no change in IL-10 or IL-18). The finding suggests an TH2-inflammatory response in the airways of non-atopic healthy individuals.
Rankin et al 2021	304 (as PM_{10}) (LOEC) or filtered air (FA) Total hydrocarbons 0.78 ppm NOx 3.42 ppm, NO2 0.02 ppm	1h	1	16 healthy, non-smoking men (aged 21–44 years).	Controlled exposure chamber	TDE Nonroad Volvo diesel engine (Volvo TD45, 4.5L, 4 cylinders, 680 rpm, manufactured in 1991) running on low sulfur SD10 (RF-06-03) diesel fuel	Muscle sympathetic nerve activity (MSNA), heart rate & respiration.	DE exposure ↑ MSNA bursts, no change to HRV indices. Study shows rapid modulation of the autonomic nervous system after exposure to DE, with an increase in MSNA.
Rudell et al 1999a	300 unfiltered DEE (1.3ppm NO2) (LOEC) <0.01 filtered air (FA) (<0.02ppm NO2) filtered DEE (conc NR) (1.2ppm NO2) diesel exhaust filtered with a ceramic particle trap	1h	1 each (3 weeks apart)	10 healthy never smoking subjects, eight men and two women, mean age (range) 27 (22–35 yrs)	Controlled exposure chamber	TDE (A new Volvo TD1F Intercooler model 1990, a six cylinder, four stroke, directly injected turbo-charged diesel engine, idling outdoors at 900rpm) (8ppm sulfur fuel)	The effect of diesel exhaust on broncho-alveolar cells and soluble components in normal healthy subjects.	DE exposure ↑ neutrophils & alveolar macrophage recruitment in airway lavage.

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Rudell et al 1999b	290 unfiltered DEE (LOEC) (NO2 0.7ppm) Filtered DEE ranged from 110-170 µg/m3 (NO2 0.3-0.7ppm) <30 µg/m3 filtered air (FA) (NO2 <0.02ppm)	1h	1 (once to DEE, once to DEE filtered with four different cabin air filters)	32 healthy non-smoking subjects (mean age 29, range 21–53, 15 men, 17 women)	Controlled exposure chamber	TDE The engine, a Volvo TD 45 model 1991, was connected to a water loaded engine dynamom-eter and it was operated at 1400 r/m (50% speed) and 30.6 kW (50% load). The engine had four cylinders, four strokes, and a direct injected turbocharger.	Subjective symptoms, nasal airway lavage, acoustic rhinometry, & lung function. .	No acute effects on NAL, rhinometry or lung function. No mutagenic effect in Ames test. ↑ subjective symptoms of eye irritation, smell & headache after exposure to unfiltered DEE & some filtered DEE. However, it is unclear if NO_2 contributed to these effects. The use of active charcoal filters, and a particle filter, clearly reduced the intensity of symptoms induced by diesel exhaust, however these exhausts also had the lowest NO_2 concentrations.
Salvi & Frew 1998	PM conc NR for DEENO2 2ppm, NO 4.5ppm, CO 7.5ppm formaldehyde 0.26µg/m^3.	1h	1	Healthy non-smoking subjects- details not provided.	Controlled exposure chamber (with intermittent exercise)	NS (diesel power generator)	Inflammatory effects of diesel exhaust on human airways via bronchoscopy.	Acute exposure to diesel exhaust was not associated with any significant change in the immediate lung function measurements. However, neutrophilic response was observed in peripheral blood, in BAL, and in bronchial epithelium and submucosa.
Sava et al 2013	300 (as $PM_{2.5}$) (NOEC) or filtered air (FA)	2h	2 (at least 2 weeks apart)	18 subjects with asthma	Controlled exposure chamber	NS	Airway responsiveness to metacholine, neuropeptides, and inflammatory markers in subjects with asthma exposed to DE.	No significant differences inflammatory markers. DE exposure augmented local levels of upper airway neuropeptides.

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Sibanda & Makaza 2019	Not stated.	3 months	Likely 8 hrs/d (workplace exposure)	36-year-old Zimbabwean had presented to her General Practitioner with asthma symptoms.	Diesel generator located near the office of the exposed subject.	Diesel generator located near the office of the exposed subject.	Allergen specific IgE antibody sensitization and asthma onset related to occupational exposure.	Atypical presentation of adult onset asthma in the absence of a history of either atopy or allergen specific IgE antibody sensitization resolved after relocating diesel generator and short course of oral corticosteroids.
Stiegel et al 2016	300 (NOEC) Ozone: 0.3 ppm NO2: 0.16ppm	2 h	4 exposure arms (at least 13 days apart)	15 healthy individuals, 18–55 yrs	Controlled exposure chamber (with intermittent exercise)	NS	Immune system response to DE exposure, O_3, and DE + O_3 co-exposure. Measured inflammatory cytokines & white blood cells.	Subtle cytokine response to all exposures, with more complex upon combined exposure. No change in white blood cell counts from DEE exposure alone. The associations between environmental exposures and cardiopulmonary effects are possibly mediated by inflammatory response mechanisms.
Thomson et al 2021	300 (as $PM_{2.5}$) (NOEC) or filtered air (FA)	2h Exposures: -FA with placebo -DEE with placebo -DEE with N-acetylcysteine (600mg)	1 (2 weeks washout period between each treatment)	19 subjects; mean age 28 (range 19–46 yrs), 14 with diagnosed asthma (7 females, 7 males), and 5 without (3 females, 2 males)	Controlled exposure chamber	NS (6.0-kW diesel generator operated to simulate on-road emissions).	Plasma cortisol levels considering effect modification by sex, asthma status, antioxidant gene variants, and antioxidant treatment.	DE exposure rapidly & transiently ↑ plasma cortisol. Degree of airway hyper-responsiveness was not associated with differential cortisol response to DE.

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Wierzbicka et al 2014	280 (as PM_1) (LOEC) (EC to total carbon 82%). 2 filtered air (FA) (Formaldehyde 400 µg/m3, acetaldehyde 200µg/m3, NO2 1.3ppm).	3h	2	18 healthy non-smokers (9 men and 9 women) of ages 40-66 (mean 51 yrs)	Controlled exposure chamber	NS (Idling, 900 rpm, Volkswagen Passat TDI (-98, 1900 cm3, 81 kW). Fuel sulfur content <10 ppm.	Detailed DE characteristics and symptoms during human chamber exposure.	DE properties vary to a great extent under the same DEP mass concentration and engine load. Eye irritation effects, most probably caused by aldehydes in the gas phase, as well as nose irritation were observed.
Meta-analyses or review papers or papers of interest								
Ghio 2012a	300 (as PM2.5) (LOEC)	Various (principally 2 hours)	Various (principally once)	The review summarises multiple studies with groups of healthy- non-smoking volunteers	Controlled exposure chamber (with intermittent exercise)	Likely various	Pulmonary and cardiovascular effects.	DE incites lung & systemic inflammation with a threshold concentration approximating 300 µg/m3. Systemic health consequences of DE exposure are considered consequent to the primary lung inflammation and include pro-thrombotic changes and cardiovascular disease. It appears that DEP is associated with some portion of the biological effect of DE.

Study reference	Exposure concentration DEE (µg/m^3)	Exposure duration	Exposure frequency	Population studied	Exposure method	Engine Classification	Health Effects studied	Findings
Ghio 2012b	300 (as PM2.5) (LOEC)	Various (principally 2 hours)	Various (principally once)	The review summarises multiple studies with groups of healthy- non-smoking volunteers	Controlled exposure chamber (with intermittent exercise)	Likely various	The review of the controlled human exposures to diesel exhaust and DEPs, and summary of the investigations into the associations between DEP exposure and airway inflammation.	Inflammation after diesel exhaust and DEP exposure is evident at higher concentrations only; there appears to be a threshold dose for DEPs approximating 300 µg/m^3. The lack of a biological response to DEPs at lower concentrations may reflect a contribution of gaseous constituents or interactions between DEPs and gaseous air pollutants to the human inflammatory response and function loss.
Vieira et al 2016a (Abstract only)	76.2-300 (unclear from abstract if effects were observed at low end of exposure range) (PM ranged from 76.2 ± 51.1 to 330 ± 12 µg/m3)	21 minutes – 2 hours	Not stated.	373 participants- A systematic review and meta-analysis of randomised controlled trials.	Controlled exposure chamber	NS	The short-term association between PM and acute cardiovascular consequences.	DE exposure attenuated vasodilation response, ↑ systolic blood pressure & lymphocyte & platelet count. No effect on heart rate, diastolic blood pressure, total white blood cell count or CRP levels. DE exposure in different concentrations is associated with surrogates of endothelial impairment and blood pressure elevation, which might be related to adverse cardiovascular events.
BC = Black carbon; NS = Not specified; PBMCs = peripheral blood mononuclear cells; LOEC = Lowest Observed Effect Concentration; NOEC = No Observed Effect Concentration. FA= filtered air; DE= diesel exhaust; TDE= Traditional Diesel Engine; NTDE=New Technology Diesel Engine; DEP= Diesel Exhaust Particles								

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APPENDIX E

PAH Toxicity Equivalency Factors (TEFs) and Potential Emissions in DEE

Table E1: PAH TEFs and Potential Emissions in DEE

PAH and nitro-PAH Compounds	Toxicity Equivalent Factor (TEF)		Khalek et al. 2011 2000 Technology Engine (mg/bhp hr)		Liu et al. 2010 2004 Technology Engine (mg/bhp hr)		Khalek et al. 2007 ACES Engines (mg/bhp hr)		Liu et al. 2010 2007 Engine (mg/bhp hr)	
	Value	Source	Amount emitted	BaP TEQ	Amount emitted	BaP TEQ	Amount emitted	BaP TEQ	Amount emitted	BaP TEQ
(Standard PAHs)										
Naphthalene	0.001	Fitzgerald 1998	0.4829	0.0004829	0.719	0.000719	0.0982	0.0000982	0.122	0.000122
Acenaphthylene	0.001	Fitzgerald 1998	0.0524	0.0000524	0.0305	0.0000305	0.0003	0.0000003	0.00218	0.00000218
Acenaphthene	0.001	Fitzgerald 1998	0.0215	0.0000215	0.0455	0.0000455	0.0004	0.0000004	0.022	0.000022
Fluorene	0.001	Fitzgerald 1998	0.0425	0.0000425	0.131	0.000131	0.0013	0.0000013	0.0129	0.0000129
Phenanthrene	0.001	Fitzgerald 1998	0.05	0.00005	0.0786	0.0000786	0.0055	0.0000055	0.0123	0.0000123
Anthracene	0.001	Fitzgerald 1998	0.0121	0.0000121	0.00738	0.00000738	0.0004	0.0000004	0.000862	0.000000862
Fluoranthene	0.01	Fitzgerald 1998	0.0041	0.000041	0.00431	0.0000431	0.0003	0.000003	0.00113	0.0000113
Pyrene	0.001	Fitzgerald 1998	0.0101	0.0000101	0.0117	0.0000117	0.0004	0.0000004	0.000979	0.000000979
Benzo(a)anthracene	0.1	NEPC 2013 B7	0.0004	0.00004	0.000586	0.0000586	0.00000005	0.000000005	0.0000632	0.00000632
Chrysene	0.1	NEPC 2013 B7	0.0004	0.00004	0.00105	0.000105	0.00000005	0.000000005	0.000123	0.0000123
Benzo(b)fluoranthene	0.1	NEPC 2013 B7	0.00015	0.000015	—	—	0.00000005	0.000000005	—	—
Benzo(k)fluoranthene	0.1	NEPC 2013 B7	0.00015	0.000015	—	—	0.00000005	0.000000005	—	—
Benzo(b,k,j)fluoranthene	0.1	NEPC 2013 B7	—	—	0.00024	0.000024	—		0.00000776	0.000000776
Benzo(g,h,i)fluoranthene	0.01	NEPC 2013 B7	—	—	0.000607	0.00000607	—		0.000258	0.00000258
Benzo(e)pyrene	1	Assumed	0.00015	0.00015	0.000232	0.000232	0.00000005	0.00000005	0.00000374	0.00000374
Benzo(a)pyrene	1	Fitzgerald 1998	0.00015	0.00015	0.0000797	0.0000797	0.00000005	0.00000005	0.00000613	0.00000613
Perylene	0.1	Fitzgerald 1998	0.00015	0.000015	—	—	0.00000005	0.000000005	—	—
Indeno(1,2,3-c,d)pyrene	0.1	NEPC 2013 B7	0.00015	0.000015	—	—	0.00000005	0.000000005	—	—
Dibenz(a,h)anthracene	1	NEPC 2013 B7	0.00015	0.00015	—	—	0.00000005	0.00000005	—	—

PAH and nitro-PAH Compounds	Toxicity Equivalent Factor (TEF)		Khalek et al. 2011		Liu et al. 2010		Khalek et al.		Liu et al. 2010	
			2000 Technology Engine (mg/bhp hr)		2004 Technology Engine (mg/bhp hr)		2007 ACES Engines (mg/bhp hr)		2007 Engine (mg/bhp hr)	
	Value	Source	Amount emitted	BaP TEQ	Amount emitted	BaP TEQ	Amount emitted	BaP TEQ	Amount emitted	BaP TEQ
Benzo(g,h,i)perylene	0.1	Fitzgerald 1998	0.00015	0.000015	0.0000724	0.00000724	0.00000005	0.000000005	0.0000168	0.00000168
Sum standard PAHs			0.678	0.00132	1.03	0.00158	0.107	0.000110	0.175	0.000218
% Standard PAHs in all PAHS			99.75%	93.27%	99.89%	99.58%	99.96%	98.12%	99.90%	99.91%
Nitro-PAHs										
1-Nitronaphthalene	0.001	Assumed (see naphthalene)	—	—	0.000361	0.000000361	—	—	0.0000858	8.58E-08
2-Nitronaphthalene	0.001	Assumed (see naphthalene)	—	—	0.000531	0.000000531	—	—	0.0000478	4.78E-08
2-Nitrofluorene	0.001	Assumed (see fluorene)	0.000065	0.000000065	—	—	0.0000009	9E-10	—	—
9-Nitroanthracene	0.001	Assumed (see anthacene)	0.0007817	7.817E-07	0.000192	0.000000192	0.0000031	3.1E-09	0.0000403	4.03E-08
2-Nitroanthracene	0.001	Assumed (see anthacene)	0.0000067	6.7E-09	—	—	0.000000005	5E-12	—	—
9-Nitrophenanthrene	0.001	Assumed (see phenanthrene)	0.0001945	1.945E-07	—	—	0.0000153	1.53E-08	—	—
4-Nitropyrene	0.1	Kelly et al. 2021	0.0000216	0.00000216	—	—	0.000000005	5E-10	—	—
1-Nitropyrene	0.1	Kelly et al. 2021	0.0006318	0.00006318	0.000055	0.0000055	0.00002	0.000002	0.000000125	1.25E-08
7-Nitrobenz(a)anthracene	0.1	Kelly et al. 2021	0.0000152	0.00000152	—	—	0.0000002	0.00000002	—	—
6-Nitrochrysene	11	Kelly et al. 2021	0.0000023	0.0000253	—	—	0.000000005	0.000000055	—	—
6-Nitrobenzo(a)pyrene	0.47	Kelly et al. 2021	0.0000038	0.000001786	—	—	0.000000005	2.35E-09	—	—
Sum nitro-PAHs			0.00172	9.50E-05	0.00114	0.000006584	0.0000395	2.10E-06	0.0001740	1.864E-07
% nitro-PAHs in all PAHS			0.25%	6.73%	0.11%	0.42%	0.04%	1.88%	0.10%	0.09%
Sum all PAHS			0.679	0.00141	1.032	0.00159	0.107	0.000112	0.175	0.000218

Red values indicate that the reported result was less than the limit of reporting. In this instance, 0.5x the limit of reporting was adopted consistent with risk assessments in Australia (enHealth 2012a)

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APPENDIX F

Summary of critiques and reanalyses of data presented in Vermeulen et al. (2014) meta-analysis

Following the publication of the DEMS data and Vermeulen et al. (2014) study, a number of reanalyses, critiques and responses to critiques ensued.

- Crump and van Landringham (2012) re-estimated yearly REC exposures in the DEMS study i) using data from mines to estimate the CO-REC relationship rather than assuming a linear relationship, ii) using a different method for assigning values to non-detect CO, and iii) taking account of statistical uncertainty to estimate bounds for REC exposures. Their analysis resulted in significantly different exposure estimates than the original DEMS study. The authors did not conduct reanalysis of health effect associations in this publication.
- Crump (2014) undertook a reanalysis of the data in the Vermeulen et al. (2014) study using all 5-year lags, instead of the combination of 5- and 15-year lags. The revised RR (95% CI) was not significant (0.38, -0.03-0.96)[42] with similar results obtained using a zero-year lag. It is noted the lower confidence interval for the RR is negative, which seems unusual. According to Crump (2014), "*There are other limitations of the analysis by Vermeulen et al. (2014): Garshick et al. (2012) employed a second measure of diesel exposure (exposure duration), which Vermeulen et al. did not account for in the analysis; and Vermeulen et al. used very crude exposure summaries (e.g., midpoints of exposure intervals)".*
- Vermeulen et al. (2014b) responded to the Crump (2014) critique stating that results for an analysis using a 5-year lag in DEMS were not published, although the 5-year lag had been included in analyses to examine changes in model fit as a function of lag. *"From those analyses (Silverman et al. 2012, Supplementary Figure 1), it is apparent that a 5-year lag showed the worst model fit of all lags (0-25 years); thus, it does not make sense to use this particular analysis as the primary exposure-response relation simply because the label "5-year lag" coincides with that of the other two studies."* The authors acknowledged that the interpretation of the risk function may be affected by differences in exposure between lag times. For this reason, they performed a sensitivity analysis that included different lags from each study; according to the authors, overall results changed only slightly. The authors also extended their earlier sensitivity analyses by including the unpublished 5-year lagged data from Silverman et al. noting, however, that the 5-year lagged data from the DEMS do not fit nearly as well as the 15-year lagged data. They found the 5-year lagged risk estimates for DEMS selected by Crump (2014) for his meta-analysis to be considerably lower than those of the two trucking studies and the alternative lag times for DEMS. Vermeulen et al. (2014b) also included three different regression lines based on the two trucking studies and one of three different sets of results for DEMS, obtained using the exposure data lagged 0, 5, and 15 years. All models are fitted using the full estimated covariance matrix to account for the correlation between categorical point estimates from the same study. They found the lowest meta-regression slope using the 5-year lagged exposure results from DEMS, a) to be higher than that reported by Crump using the variance estimates only; b) to be statistically significant (0.00065; 95% CI: 0.00028, 0.0010); and c) falling within their previous sensitivity analyses (Vermeulen et al. 2014).
- Subsequent reanalysis of the DEMS study was undertaken by Crump et al. (2015). Re-analysis used conditional logistic regression and adjusted for cigarette smoking in a manner similar to the original DEMS analysis. However, the reanalysis also included additional estimates of DEE exposure and adjustment for radon exposure. Without adjusting for radon, the results obtained by Crump et al. (2015) were similar to those in the original DEMS analysis: all but one of the nine DEE exposure estimates showed evidence of an association between DEE exposure and lung cancer mortality, with trend slopes differing only by about a factor of two. When exposure to radon was adjusted, the evidence for a DEE effect was greatly diminished, but was still present in some analyses that utilised the three original DEMS DEE exposure estimates. A DEE effect was not observed when the six alternative DEE exposure estimates were utilised, and radon was adjusted. No consistent evidence of a DEE effect was found among miners who worked only underground. OR (95%CI) for ever underground workers with cumulative exposure lagged 15 years were significant for some exposure cutoffs when not adjusted for radon exposure. A statistically significant OR with an apparent exposure response appears to have been observed for the following estimates:

[42] Note this RR appears to have been estimated using a different statistical model (log-regression) to that used by Vermeulen et al. (2014) and therefore the RR may not be directly comparable to that shown in Figure 6-2.

- DEMS_REC1: At exposures ≥ 383.5 µg/m^3.yrs
- DEMS_REC2: At exposures ≥ 318.2 µg/m^3.yrs
- DEMS_REC3: At exposures ≥ 157.1 µg/m^3.yrs
- REC1: At exposures ≥ 649.2 µg/m^3.yrs
- REC2: At exposures ≥ 556.5 µg/m^3.yrs
- REC3: At exposures ≥ 693.9 µg/m^3.yrs
- REC4: At exposures ≥ 329.8 µg/m^3.yrs
- REC5: At exposures ≥ 231.4 µg/m^3.yrs
- REC6: At exposures ≥ 204.9 µg/m^3.yrs

It is noted ORs were not significant when analysis was limited to workers who only worked underground (presumed to have higher DEE exposure). When examining the exposure cut-points (for cumulative or average REC) many of the ORs provided in Crump et al. (2015) are not statistically significant (their 95% CI overlap 1). Where they are significant, they do not appear to show a clear exposure-response except for 'ever underground' workers with cumulative exposure lagged 15 yrs (see above).

- Further reanalysis of the DEMS study was undertaken by Crump et al. (2016), who developed new estimates of REC exposures using historical data on use of diesel equipment, diesel engine horsepower (HP), mine ventilation rates, and the documented reduction in PM emissions per HP in diesel engines from 1975 through 1995. The new REC estimates were applied in a conditional logistic regression of the DEMS nested case-control data very similar to the one applied in the original DEMS analyses. The results of this reanalysis showed none of the trend slopes calculated using the new REC estimates were statistically significant ($p > 0.05$). The trend slopes were smaller by roughly factors of five without control for radon exposure and factors of 12 with control for radon exposure compared to those estimated in the original DEMS analyses. Also, the 95% confidence intervals for these trend slopes had only minimal overlap with those for the slopes in the original DEMS analyses. These results underscore the uncertainty in the precision of estimates of the potency of DEE in causing lung cancer based on analysis of the DEMS data due to uncertainty in estimates of exposures.
- Möhner (2016, 2018a) published critiques of the DEMS study in which they indicate there are some hints that point towards a bias in the DEMS results with respect to a healthy worker effect. They commented that from a methodological point of view the breakdown of the cohort into surface-only and 'ever-underground' sub-cohorts seems incorrect. Excluding the results based on the adjustment for work location, the authors indicated that the DEMS analyses do not show any noticeable risk increase with increasing REC exposure, a finding that has also been derived from the German potash miner cohort study. An approximation by unconditional logistic regression, based on the case–control data, yielded an OR of 0.92 (95%CI: 0.66–1.30) for miners who ever worked underground, adjusted for smoking (Möhner 2016). Möhner (2018a) indicated an association between lung cancer and cumulative REC exposure is not supported by the standardised mortality ratio (SMR) for 'ever-underground' workers which was lower than for surface workers (SMR = 1.21 and 1.33, respectively) when surface workers were the referent controls.
- Morfeld and Spallek (2015) performed extended re-analysis of the data used by Vermeulen et al. (2014) using different modelling approaches (fixed and random effects regression analyses, Greenland/Longnecker method) and explored the impact of varying input data (modified coefficients of Garshick et al. 2012, results from Crump et al. 2015 replacing Silverman et al. 2012, modified analysis of Möhner et al. 2013). The study authors were able to reproduce all the individual and main meta-analytical results from Vermeulen et al. (2014). Of all the meta-analyses Morfeld and Spallek (2015) performed, the evaluation of the data as used by Vermeulen et al. (2014) resulted in the highest risk estimates. However, the analysis by Morfeld and Spallek (2015) demonstrated a heterogeneity of the baseline relative risk levels between the three studies. Heterogeneity refers to a systematic difference in the baseline risk of the three studies (i.e. setting exposure

to zero). According to Morfeld and Spallek (2015), in other situations, authors typically tend to reject combinations of studies even with considerably less pronounced heterogeneity. They indicate that this uncertainty in the baseline level renders the use of the analysis by Vermeulen et al. (2014) problematic for risk estimates in the lower exposure region. The meta-coefficient estimated by Morfeld and Spallek (2015) was about 10–20 % of the main effect estimate in Vermeulen et al. (2014). The authors concluded: "*….the present re-analysis also revealed that the results of the meta-regression study by Vermeulen et al. [1] should not be used in any quantitative lung cancer risk evaluation without reservations, as the results vary significantly depending on the input data selected and the statistical methods used. This is particularly true for the low exposure region."*

- Recently, Vermeulen et al. (2020) themselves undertook a reanalysis of their original study to address two issues that have been raised in critiques of the DEMS study analysis (use of historical CO measurements to calibrate the exposure model and potential confounding by radon). Vermeulen et al. (2020) developed alternative REC estimates using models that did not rely on CO but rather on estimated use of diesel equipment, mine ventilation rates and changes in DEE emission rates over time. Validation of the new REC exposure estimates by Vermeulen et al. (2020) indicated that they overestimated historical REC by 200-400% (absolute differences >170–400 µg/m^3 REC and relative differences >100%), compared with only 10% for the original estimates. Exposure levels from these models were >1000 µg/m^3 for many jobs. Vermeulen et al. (2020) indicate that REC exposure levels >1000 µg/m^3 seem unrealistically high. Effect estimates for lung cancer using these alternative REC exposures or adjusting for radon typically changed by <10% when compared with the original estimates.

An independent panel reviewed the data from the DEMS study and the study by Garschick et al. (2012) and concluded both studies were well-designed and well-conducted studies and that each made considerable progress towards addressing a number of the major limitations that had been identified in previous epidemiological studies of DEE and lung cancer (HEI 2015b). Whilst the panel found that the studies have many strengths, they also noted that any effort at quantitative risk assessment will need to acknowledge some key uncertainties and limitations. These limitations related particularly to the need for metrics more specific to diesel, better models of historical exposures, and ultimately for quantitative estimates of historical exposures to DEE (HEI 2015b).

Möhner and Wendt (2017) reviewed the available epidemiological information and concluded that the currently published studies do not support the hypothesis of a causal relationship between DEE exposure and lung cancer risk, thus a reliable derivation of a quantitative exposure-response relationship is not possible at present. They do however suggest that a conservative lower bound threshold value could be derived from the German cohort study among potash miners. They note that an upper bound for the cumulative exposure of 2.5 mg/m^3.years REC seems to be sufficient to prevent a detectable increase of lung cancer risk. This value corresponds to an average annual cumulative exposure value of 0.05 mg/m^3 REC assuming a working life of 45 years (Möhner and Wendt 2017).

Sun et al. (2014) critically evaluated 42 cohort studies and 32 case-control studies from between 1970 to 2013 to examine the association between DEE exposures and lung cancer. They found that previous studies suffer from a series of methodological limitations, including design, exposure assessment methods and statistical analysis used. They noted that a lack of objective exposure information appears to be the main problem in interpreting epidemiological evidence. To facilitate the interpretation and comparison of previous studies, the authors created a job-exposure matrix (JEM) of DEE exposures based on around 4,000 historical industrial measurements. The values from the JEM were considered during interpretation and comparison of previous studies. Overall, they found neither cohort nor case-control studies indicate a clear exposure-response relationship between DEE exposure and lung cancer. They concluded that epidemiological studies published to date do not allow a valid quantification of the association between DEE and lung cancer, although such an association cannot be ruled out. They further note that causality of weak association is often difficult to establish, since it is susceptible to all forms of possible design bias (Sun et al. 2014).

The AIOH (2017) review concluded epidemiological outcomes of lung cancer and non-malignant respiratory disease associated with exposure to DPM remains unclear, due primarily to the absence of historical direct measurements of exposure. In addition, the evolution of NTDE, fuels and DPM emission controls increases uncertainty as to the applicability of any quantitative exposure-response coefficient (risk per unit of exposure) derived from exposures to TDE, and hence its application to a health-based exposure standard applicable to the current and future workforce (AIOH 2017). BauA (2017) also concluded the available epidemiological evaluations are currently not considered sufficiently reliable for a quantitative assessment.

APPENDIX G

Literature that was consulted but not cited in the body of the report (note some of these references have been cited in Appendices)

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