Human Health Risk Assessment for Diesel Exhaust

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AQBAT	Air Quality Benefits Assessment Tool
AURAMS	A Unified Regional Air Quality Modelling System
BC	black carbon
CD	census division
CEPA 1999	*Canadian Environmental Protection Act, 1999*
CO	carbon monoxide
COPD	chronic obstructive pulmonary disease
CRF	concentration–response function
DE	diesel exhaust
DEMS	Diesel Exhaust in Miners Study
DEP	diesel exhaust particles
EC	elemental carbon
EPA	Environmental Protection Agency
HDDV	heavy-duty diesel vehicle
HEC	human equivalent concentration
HRV	heart rate variability
IHD	ischemic heart disease
LDDV	light-duty diesel vehicle
LDGV	light-duty gasoline vehicle
LOAEL	lowest-observed-adverse-effect level
MOVES2010a	Motor Vehicle Emission Simulator version 2010a
NAPS	National Air Pollution Surveillance
NH_3	ammonia
NO	nitric oxide
NO_2	nitrogen dioxide
NOAEL	no-observed-adverse-effect level
NOx	nitrogen oxides
O_3	ozone
OC	organic carbon
PAH	polycyclic aromatic hydrocarbon
PM	particulate matter
$PM_{2.5}$	fine particulate matter
ppb	parts per billion
ppbv	parts per billion by volume
SO_2	sulphur dioxide
TWBL	tire wear and brake lining
UFP	ultrafine particulate matter
US EPA	United States Environmental Protection Agency
VOC	volatile organic compound

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The report *Human Health Risk Assessment for Diesel Exhaust* is a comprehensive review and analysis of the potential adverse health effects associated with diesel fuel use in Canada. The report focuses on diesel exhaust (DE) emissions from on-road and off-road vehicles (excluding rail and marine applications) and targets impacts resulting from general population exposures. Part A includes a review of diesel fuels, engines and emissions, a review of exposure to DE and an evaluation of the health effects associated with DE exposure. Part B presents a quantitative analysis of the population health impacts associated with the contribution of DE to criteria air contaminant concentrations in Canada.[1]

This report does not address the health risks of diesel fuel itself, which is under review as part of the Chemicals Management Plan of the Government of Canada and will be reported elsewhere.

Part A – Human health risk assessment for diesel exhaust

1 Diesel fuels, engines and emissions

Diesel fuels are mixtures of hydrocarbon compounds composed of 12–20 carbon atoms. The composition of diesel fuel varies depending on factors such as crude oil feedstocks, production processes and blending practices. Regulations and fuel quality standards prescribe the fuel composition. The *Canadian Environmental Protection Act, 1999*[2] (CEPA 1999) includes various regulations pertaining to motor vehicle fuels, including the *Sulphur in Diesel Fuel Regulations*[3] and the *Renewable Fuels Regulations*.[4] The *Sulphur in Diesel Fuel Regulations* set a maximum sulphur content of 15 parts per million (ppm; or 15 mg of sulphur per kilogram of fuel) in on-road and off-road diesel fuels. The *Renewable Fuels Regulations* require a 2% renewable content by volume in diesel, which is based on an annual national average of the whole diesel pool. Motor vehicle fuels produced or imported for the Canadian market must also meet specifications developed and published by the Canadian General Standards Board. CAN/CGSB 3.517-2007, Automotive (On-Road) Diesel Fuel, is the standard for automotive low-sulphur diesel fuel intended for use in high-speed diesel-powered engines (e.g. intercity trucks). CAN/CGSB 3.6-2010, Off-Road Diesel Fuel, is the standard for diesel fuels that are suitable for use in off-road and stationary diesel engines. These variables and requirements influence the physical and chemical fuel parameters that subsequently affect exhaust emissions.

Diesel fuels are used in compression ignition internal combustion engines (i.e. diesel engines). Diesel engines, characterized as fuel efficient and durable, are widely used for on-road and off-road applications.[5] Successive regulations have been adopted to ensure that vehicles and engines meet increasingly more stringent emission standards. Specific emission standards apply to new engines or vehicles for their intended useful lives. Since 1988, partly because of the integrated North American

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[1] The content of this report is based on information presented in a comprehensive supporting document prepared by Health Canada.

[2] laws-lois.justice.gc.ca/eng/acts/C-15.31/

[3] laws-lois.justice.gc.ca/eng/regulations/sor-2002-254/index.html

[4] laws.justice.gc.ca/eng/regulations/SOR-2010-189/

[5] Off-road applications include, for example, construction, mining, farming and forestry equipment.

market for vehicles, engines and fuels, the general approach in Canada regarding engine and vehicle emission standards has been one of harmonization with the United States Environmental Protection Agency's (US EPA) federal standards.

Environment Canada regulates emissions from engines other than those used in aircrafts, locomotives and commercial marine vessels (for which Transport Canada has the authority) under CEPA 1999. The *On-Road Vehicle and Engine Emission Regulations*[6] came into effect in January 2004 and aligned with the US EPA's federal emission standards.[7] These regulations apply to all on-road vehicles, regardless of fuel type. Regulations targeting heavy-duty vehicles are essential for controlling DE emissions, as most of the heavy-duty vehicles are powered by compression engines. Notably, the Environment Canada and US EPA regulations include emission standards for particulate matter (PM) and nitrogen oxides (NOx) for on-road heavy-duty truck engines of model year 2007 and later, which reduce PM emission limits by 90% and NOx emissions by 95% compared with previous standard levels. These reductions were achieved through the use of emission control technologies, such as diesel particulate filters, exhaust gas recirculation and selective catalytic reducers. Regulations adopted for light-duty vehicles are also important, although diesel vehicles represent only 2–3% of the light-duty vehicle fleet.

Emissions from off-road engines were not federally regulated prior to CEPA 1999. Before the development of regulations targeting off-road engines, Environment Canada signed an agreement with engine manufacturers for the supply in Canada of engines meeting the US EPA Tier 1 standards starting with the 2000 model year. The Canadian *Off-Road Compression-Ignition Engine Emission Regulations*,[8] adopted in February 2005, introduced emission standards for model year 2006 and later diesel engines used in off-road applications.[9] The Canadian *Regulations Amending the Off-Road Compression-Ignition Engine Emission Regulations*[10] came into force on January 6, 2012. They impose stricter standards and new requirements starting with engines of the 2012 and later model years (i.e. Tier 4 standards).

Whereas fuel-related regulations have immediate impacts on emissions for the entire targeted mobile source fleet, it may take more than 20 years to fully benefit from the adoption of new engine or vehicle standards, because older, more polluting vehicles remain in use for many years.

Emissions from diesel vehicles may originate from several sources, such as combustion (i.e. exhaust), mechanical wear (e.g. tires, brakes) and fugitive releases. Exhaust emissions are generally the dominant source of emissions and were the focus of this assessment. The composition of the exhaust emission mixture is dependent on several factors, such as fuel characteristics and additives, lubricants, engine and vehicle technologies, emission control devices and environmental conditions. PM and NOx emissions are the main concerns associated with diesel engines because of their association with health impacts and their relatively high emission levels compared with spark ignition gasoline engines.

Diesel PM generally consists of fine particulate matter ($PM_{2.5}$) and ultrafine particulate matter (UFP), which are released directly or formed secondarily via gaseous precursors in exhaust and evaporative

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[6] laws-lois.justice.gc.ca/eng/regulations/sor-2003-2/index.html
[7] US Tier 2 program for new light-duty vehicles and trucks and medium-duty passenger vehicles; Phase 1 and Phase 2 programs for new heavy-duty vehicles and engines.
[8] laws-lois.justice.gc.ca/eng/regulations/SOR-2005-32/
[9] These regulations covered the US EPA Tier 2 and Tier 3 standards for off-road diesel engines.
[10] www.gazette.gc.ca/rp-pr/p2/2011/2011-12-07/pdf/g2-14525.pdf

emissions.[11] It is estimated that close to 90% of the particles (PM number basis) emitted from diesel engines are ultrafine particles. In urban areas, vehicle emissions are generally the most important source of UFP. The small size of diesel exhaust particles (DEP) implies a very large surface area on a per mass basis with a potential to adsorb large amounts of compounds, including organic and sulphur compounds. Identifying and quantifying the variety of chemical species found in DEP are challenging. The operational definitions of elemental carbon (EC) and organic carbon (OC) offer a practical way of analyzing the carbon fraction of the complex matrix of compounds. EC is associated with soot formed during combustion and is found in the particulate phase. EC is often used synonymously with black carbon (BC).[12] OC is associated with condensed-phase compounds (e.g. acids, alcohols, aldehydes, alkanes and alkenes) on the soot core. OC compounds in the atmosphere partition between the gas and particulate phases, depending on the vapour pressure of the compounds, the quantity and type of ambient PM and environmental conditions.

NOx refers to a group of seven gaseous compounds that includes nitric oxide (NO) and nitrogen dioxide (NO_2). Following combustion, NOx is generally dominated by NO, which is gradually converted to NO_2 following its release. With newer diesel vehicles, the initial fraction of NO_2 in emissions appears to be increasing compared with the older diesel fleet. Newer vehicles emit more of their NOx as NO_2 as a result of their exhaust after-treatment systems, which convert NO to NO_2 for oxidation purposes.

The historical data suggest that the observed decline in diesel emissions through the years is a result of changes in engine technology due to increasingly more stringent emission standards and higher fuel quality (e.g. lower fuel sulphur content). Traditional diesel exhaust – that is, emissions from on-road model year engines up to 2006 – and new technology diesel exhaust – that is, emissions from 2007 and later on-road model year engines encompassing a variety of different diesel engine technologies and after-treatment system configurations – differ significantly for regulated and unregulated compounds. New technology diesel exhaust emissions show considerable reductions (more than 70%) across a broad spectrum of compounds, such as EC, metals, NOx, PM, UFP and volatile organic compounds (VOCs). Further, the use of new technologies has considerably altered the physical and chemical characteristics of PM and gaseous DE emissions.

As mentioned previously, diesel engines and vehicles are very durable and will remain in service for extended periods. The current diesel fleet is composed of old and new vehicles designed and required to meet different emission standards. The rate of fleet turnover determines the fraction of vehicles from each model year currently in use, and this must be considered when conducting an assessment of DE emissions.

2 Exposure to diesel exhaust

DE emissions contain many gaseous and particulate compounds, including carbon monoxide (CO), NOx, $PM_{2.5}$, UFP, polycyclic aromatic hydrocarbons (PAHs) and many semi-volatile organic compounds and VOCs. Given that one third of Canadians live within 250 m of a major road and that diesel engines are pervasive on major roadways and in urban areas of Canada, it is reasonable to assume that most

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[11] For regulatory purposes, exhaust particulates are currently defined as all solid or liquid matter collected at temperatures of 47°C ± 5°C on a filter surface.

[12] BC is an optical term and is also referred to as soot or light-absorbing carbon. BC includes EC and OC compounds capable of absorbing light efficiently at different wavelengths. For DE, EC is considered the major contributor to light absorption, and for this reason, EC and BC are sometimes used interchangeably for DE.

Canadians are regularly exposed to DE. Owing to the complex and variable nature of DE in space and time and the fact that many of its constituents are also emitted by other sources, quantifying human exposure to DE has been challenging, both for general population exposures and in occupational settings.

Researchers have considered a number of surrogate markers of DE in ambient air and of DE exposure. Ideally, surrogates should 1) have diesel as the principal source of atmospheric emissions, 2) vary with other constituents of DE over time, 3) be accurately measurable at low concentrations and 4) reasonably approximate personal exposure to DE. To date, the DE surrogates identified are not without limitations.

NOx, especially NO and NO_2, have been commonly used as a surrogate for traffic-related exposures owing to a rich monitoring database and the large contribution of mobile sources to NOx emissions. However, in Canada, the diesel fleet and the gasoline fleet emit roughly equal amounts of NOx, and roadway exposures cannot be explicitly associated with DE. NOx is therefore not considered an appropriate marker for general population exposures to DE.

PM, which can be measured based on different characteristics, such as mass, particle number, size fractions and chemical composition, is a potential surrogate for DE. Mobile sources are responsible for about one quarter of $PM_{2.5}$ emissions in Canada, and DE contributes about half of that. However, the use of PM as a surrogate for general population exposure to DE has several limitations, including the many sources of primary and secondary $PM_{2.5}$, transformation of DEP in the atmosphere, temporal changes in both the quantity and the composition of DEP emissions associated with new technologies and fuels, and the significant proportion of semi-volatile $PM_{2.5}$ components.

Carbonaceous material represents a significant fraction of PM content and mass. The carbon composition of PM (i.e. EC and OC), particularly EC, has been used as a surrogate for DE emissions in analyses of urban environments. Although EC is not a unique tracer for DE, the analysis of PM carbon fractions has been used to differentiate between PM emissions from diesel and gasoline vehicles and to allocate a fraction of particulate emissions to each fuel type. Typically, gasoline vehicle emissions have been reported to have high concentrations of OC fractions, whereas on-road diesel sources have shown some association with high EC concentrations. BC has also been used as a proxy for DE exposure, as diesel engines are considered a main source of BC in urban areas. Factors that may confound the link between DE and BC include contributions from high-emitting gasoline vehicles and the long-range transport of air pollutants.

Air pollutant concentration data in Canada are available from the National Air Pollution Surveillance (NAPS) program. However, data collected at central site monitors like those of the NAPS network are of limited use for the estimation of population exposures to DE, as they lack the resolution necessary to capture both the temporal and spatial variability of DE pollutants. Studies have investigated concentrations of traffic-related air pollutants or DE surrogates in near-roadway environments and how pollutant concentrations evolve in space and time. Generally, urban populations are exposed to higher traffic pollutant concentrations compared with populations in rural areas. Further, pollutant concentrations in microenvironments influenced by traffic (e.g. in-vehicle and roadside) are higher than concentrations reported at background and indoor locations. Although the fraction of the day spent in traffic-influenced microenvironments may be limited (e.g. 6% of day is spent in-vehicle on average in Canada), the higher pollutant concentrations in these microenvironments can result in an elevated contribution to daily exposure (e.g. 21% for BC). At this time, estimates for Canadian population

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exposures to DE or DEP are limited by the absence of an explicit DE surrogate and uncertainties affecting exposure characterization.

3 Health effects of diesel exhaust

This report provides a detailed review of the scientific literature with the objective of identifying and characterizing the human health effects associated with exposure to emissions from diesel engines. Given the very large body of literature addressing this issue, findings from previously published health risk assessments by the California Environmental Protection Agency (California EPA 1998) and the US EPA (2002) were used as a starting point in this review. A detailed review of studies published between January 1, 2000, and June 15, 2012, is provided and evaluated within the context of the previous information. Only studies pertaining to DE or DEP were reviewed, and studies on the health effects of individual DE constituents, traffic pollution in general or ambient $PM_{2.5}$ were not considered for review.

3.1 Weight of evidence for determination of causality

Although substantial epidemiological research has focused on the potential role of DE in the development of lung cancer, many studies have targeted other cancers and non-cancer effects. In addition, studies in humans, experimental animals and in vitro models have investigated the toxicological effects of DE under controlled conditions and the mechanisms by which DE may exert its toxic effects. Studies were reviewed individually, and the overall weight of evidence was evaluated to assess the causal role of DE in the development of specific adverse health effects, including carcinogenicity and respiratory, cardiovascular, immunological, reproductive, developmental and central nervous system effects. Causality determinations are categorized as 1) causal relationship, 2) likely to be a causal relationship, 3) suggestive of a causal relationship, 4) inadequate to infer a causal relationship and 5) not likely to be a causal relationship (Table 1). In weighing the evidence, consideration is given to the following criteria of causal inference: biological plausibility, temporal sequence, consistency of the association, coherence, biological gradient and strength of the association.

A summary of the weight of evidence causality determinations is provided in Table 2, and details of the evaluations are provided in the following sections.

3.1.1 Carcinogenicity

The epidemiological database includes significant new studies investigating the role of DE exposure in the development of lung cancer, as well as updates of previously studied cohorts of US railroad workers and trucking industry workers. The magnitude of the risk estimates is low, with the majority between 1.1 and 3. Importantly, several recent studies include quantitative estimates of personal DE exposure for each of the exposed workers, based on respirable EC or total carbon. Exposure–response relationships were noted in some, but not all, studies, including in a large pooled analysis of 11 case–control studies, among railroad workers, truck drivers and non-metal miners, and in a population-based study. The recently published US Diesel Exhaust in Miners Study (DEMS), which includes a large cohort analysis and nested case–control study, is given significant weight in this review, as it successfully addressed key limitations in the pre-existing database. The DEMS provides strong evidence of an association between DE exposure and lung cancer, and updates of studies in trucking industry workers and railroad workers along with a large pooled analysis provide additional strong support. Overall, consistently elevated lung cancer risks in association with occupational exposure to DE have been reported by many research groups, for many occupations, using multiple study designs and various exposure metrics, and

Table 1. Weight of evidence for determination of causality

Relationship	Description
Causal relationship	Evidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures. That is, the pollutant has been shown to result in health effects in studies in which chance, bias and confounding could be ruled out with reasonable confidence – for example: a) controlled human exposure studies that demonstrate consistent effects; or b) observational studies that cannot be explained by plausible alternatives or are supported by other lines of evidence (e.g. animal studies or mode of action information). Evidence includes replicated and consistent high-quality studies by multiple investigators.
Likely to be a causal relationship	Evidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures, but important uncertainties remain. That is, the pollutant has been shown to result in health effects in studies in which chance and bias can be ruled out with reasonable confidence, but potential issues remain – for example: a) observational studies that show an association, but co-pollutant exposures are difficult to address and/or other lines of evidence (controlled human exposure, experimental animal or mode of action information) are limited or inconsistent; or b) animal toxicological evidence from multiple studies from different laboratories that demonstrates effects, but limited or no human data are available. Evidence generally includes replicated and high-quality studies by multiple investigators.
Suggestive of a causal relationship	Evidence is suggestive of a causal relationship with relevant pollutant exposures, but is limited because chance, bias and confounding cannot be ruled out – for example, at least one high-quality epidemiological study shows an association with a given health outcome, but the results of other studies are inconsistent.
Inadequate to infer a causal relationship	Evidence is inadequate to determine that a causal relationship exists with relevant pollutant exposures. The available studies are of insufficient quantity, quality, consistency or statistical power to permit a conclusion regarding the presence or absence of an effect.
Not likely to be a causal relationship	Evidence is suggestive of no causal relationship with relevant pollutant exposures. Several adequate studies, covering the full range of levels of exposure that human beings are known to encounter and considering susceptible subpopulations, are mutually consistent in not showing an effect at any level of exposure.

Adapted from US EPA (2009)

Table 2. Summary of causal determinations for exposure to diesel exhaust

Outcome	Acute/chronic DE exposure	Causality determination
Carcinogenicity	Chronic	Causal (lung cancer) Suggestive (bladder cancer) Inadequate (other cancers)
Respiratory effects	Acute	Causal
	Chronic	Likely
Cardiovascular effects	Acute	Likely
	Chronic	Suggestive
Immunological effects	–	Likely
Reproductive and developmental effects	–	Suggestive
Central nervous system effects	Acute	Suggestive
	Chronic	Inadequate

controlling for key potential confounders. Therefore, it is considered unlikely that the observed results are due solely to chance, bias or confounding.

There has been a significant amount of new research examining the potential role of DE in bladder cancer development in human populations. A large pooled analysis and three meta-analyses consistently presented small but significant risk estimates (1.1–1.4) associated with DE exposure. However, several factors limit the interpretation of the overall findings, including a lack of measured DE exposure in the studies and the fact that most risk estimates were derived for occupational groups exposed to a mixture of vehicle emissions rather than to DE alone. Other cancers have also been studied in relation to DE exposure, and a small number of studies have reported an increase in markers of genotoxicity in workers exposed to DE; however, those databases remain limited.

Both the particulate and gaseous phases of DE contain known or suspected carcinogens, and the very small size of DEP contributes to their efficient delivery to the deep lung. In addition, there is extensive evidence that DE is mutagenic and genotoxic in experimental animals and in cell culture. It is therefore biologically plausible that DE as a mixture is carcinogenic in humans. Although lung tumours have been observed in multiple studies in rats following long-term exposure to DE, the evidence suggests that this is the result of a chemically non-specific and species-specific response to overloading of the lung with particles. This occurs only at very high DE exposures (≥ 2 mg/m^3 DEP), at which point clearance mechanisms are overloaded, resulting in chronic inflammatory responses and leading to tumour induction. This mechanism is not considered to be relevant to general population exposures to DE and lung cancer development. Animal models have not been sensitive to lung cancer development at lower DE concentrations, which has been similarly observed following tobacco smoke exposure.

The overall literature, including studies pertaining to in vitro, experimental animal and human exposures to DE, presents a coherent body of evidence indicating that DE is carcinogenic to humans. Based on sufficient evidence of a causal relationship between DE exposure and lung cancer in occupational studies, substantial supporting evidence from toxicological studies establishing the mutagenicity and genotoxicity of DE and the fact that DE contains known human carcinogens, **it is concluded that there is sufficient evidence of a causal relationship between DE exposure and lung cancer.** Based on limited epidemiological evidence supporting a causal relationship between DE exposure and bladder cancer, substantial supporting evidence from toxicological studies establishing the mutagenicity and genotoxicity of DE and the fact that DE contains known human carcinogens, **it is concluded that the evidence is suggestive of a causal relationship between DE exposure and bladder cancer.** Based on the small number of studies addressing individual cancer types and the equivocal results published to date, **it is concluded that there is inadequate evidence to infer a causal relationship between DE exposure and the development of individual cancers other than lung cancer and bladder cancer.**

3.1.2 Respiratory effects

Studies examining the impact of DE on respiratory health outcomes in human populations have focused on occupationally exposed workers and general population groups exposed to traffic-related DE. These studies provide evidence of increased risk of various adverse health effects in association with DE exposure, such as asthma, pulmonary function decrements and chronic obstructive pulmonary disease (COPD). Several studies have reported increased risk of chronic respiratory symptoms, such as wheeze, in infants and children in relation to traffic-related DE exposure, suggesting potential enhanced vulnerability of this age group. Risk estimates for respiratory outcomes were relatively low, with values up to about 2, and several exposure–response relationships were observed. The major limitations of most studies are the lack of quantitative DE exposure assessment and the potential for general population DE exposure estimates to include exposures from other sources owing to the lack of a unique DE marker.

Many controlled human exposure studies have reported that short-term DE exposure was associated with respiratory symptoms, altered lung function, respiratory inflammation and respiratory oxidative response. DE exposure was associated with increased measures of airway resistance, indicative of bronchoconstriction, in healthy and asthmatic individuals. In addition, both DE and DEP can induce a range of inflammatory responses in human airways.

Studies in animals indicate that short-term DE exposure can cause an increase in airway resistance and reactivity and that short- and long-term DE exposures can result in respiratory inflammation. In addition, many studies have demonstrated that DE and DEP exposures can lead to pulmonary injury in laboratory animals and to cytotoxic effects in cell culture.

Overall, **it is concluded that there is sufficient evidence of a causal relationship between acute DE exposure and adverse respiratory health outcomes**, based on clear evidence of adverse respiratory symptoms, decrements in lung function and inflammatory responses from multiple controlled human exposure studies and supporting evidence of enhanced airway responsiveness and respiratory inflammation from toxicological studies. However, it should be noted that the adverse respiratory health outcomes were observed at high DE exposure levels. Furthermore, the most plausible mechanism of action for adverse acute respiratory effects is likely irritation of the respiratory tract, leading to inflammatory responses.

It is concluded that there is sufficient evidence that the relationship between chronic DE exposure and adverse respiratory health outcomes is likely to be causal, based on epidemiological studies, supporting long-term animal toxicological studies and panel studies. Epidemiological studies indicated various adverse respiratory symptoms, increased risk of COPD, increased risk of asthma development in children and some evidence of pulmonary function decrements in association with DE exposure. The epidemiological studies included multiple study designs and provided some evidence of an exposure–effect relationship. Although the characterization of DE exposure and co-exposure to other combustion emissions in epidemiological studies remain a challenge, the use of surrogate DE markers has improved the quantitative DE exposure assessment. Multiple studies conducted in animals show consistent adverse respiratory effects resulting from chronic DE exposure. In addition, there is limited evidence of pulmonary function decrements and pulmonary inflammation in sensitive subpopulations from the panel studies. The most plausible mechanism of action for adverse chronic respiratory effects is likely irritation, which is supported by the inflammatory and immunological responses, as well as histopathological findings in the long-term animal studies.

3.1.3 Cardiovascular effects

The association between DE exposure and adverse cardiovascular effects is a relatively new area of research. Increased risks of ischemic heart disease (IHD) and myocardial infarction were demonstrated in some occupational groups exposed to DE, with risk factors below 2. Exposures have been based on job category only, and hence exposure–response relationships have not been evaluated. Results for an association between DE exposure and cerebrovascular disease were equivocal. Studies of potentially susceptible populations exposed to DE at ambient levels have reported alterations in cardiac function in relation to DE exposure.

The effects of short-term exposures to DE in the general population have been studied in exposure chambers. Studies have reported changes in vasomotor function in healthy individuals, as well as increases in prothrombogenicity effects, but not in blood coagulability. There is only minimal evidence

of effects on cardiac function, blood pressure and atherosclerosis, and further investigation in this area is warranted.

Studies with laboratory animals support some of the findings in humans. Animal studies provide evidence that DE and DEP can have adverse effects on cardiac function, resulting in decreased heart rate variability (HRV), arrhythmia, ischemia and decreased contractility. Studies using models of chronic ischemic heart failure reported that DE and DEP exposures may further impair cardiac function in this sensitive subgroup. In addition, short- and medium-term DE exposures in animals impair normal vasoreactivity, and medium-term exposures resulted in increased thrombogenicity and reduced blood coagulability. Inconsistent effects on blood pressure were reported. There is some evidence that DE exposure in experimental animals results in systemic inflammation and oxidative stress in the cardiovascular system and that DE exposure may enhance the progression of atherosclerosis in animals predisposed to this disease.

Overall, **it is concluded that there is sufficient evidence that the relationship between acute DE exposure and adverse cardiovascular health outcomes is likely to be causal**, based on changes to vasomotor function and some evidence of prothrombogenicity effects in controlled human exposure studies. In addition, there is supporting evidence from toxicological studies, based on the observations of decreased cardiac contractility, altered HRV, ischemia and altered vasomotor function in experimental animals.

It is concluded that the evidence is suggestive of a causal relationship between chronic DE exposure and adverse cardiovascular health outcomes, based on epidemiological studies and supporting long-term animal toxicological studies. In general, there is limited evidence of an increased risk of cardiovascular disease and of exposure–response relationships in the recent epidemiological studies. Toxicological studies provide supporting evidence of perturbations in a number of adverse cardiovascular endpoints that are due to DE exposure, including potential progression of atherosclerosis. Although the mode of action for adverse cardiovascular health outcomes in relation to DE exposure has not been elucidated, the toxicological studies aid in identifying potential key events for cardiovascular pathogenesis.

3.1.4 Immunological effects

Immunological effects were investigated in a limited number of studies among children exposed to DE from traffic-related sources. One out of two studies found evidence of increased risk of allergic sensitization to outdoor allergens and increased immunoglobulin E in association with DE exposure metrics, and another study found a potential synergistic effect of DE exposure and indoor endotoxin levels on risk of persistent wheeze. The magnitude of risk estimates was about 2–6, but confounders were not fully addressed.

Controlled human exposure studies have shown that DEP can have an adjuvant effect – an effect that has been associated with the organic content of DEP – with several allergens in healthy individuals. This is consistent with the observations from epidemiological studies. In addition, individual susceptibility to an increased allergic response may be influenced by genotype. However, low DE exposure concentrations did not augment immunological responses to an allergen in asthmatic subjects.

Results from toxicological studies support the effects seen in humans. In laboratory animals, co-exposure to DE or DEP with an allergen has an adjuvant effect, exacerbating allergic inflammation and

inducing airway hyperreactivity; this provides a possible link to development and exacerbation of allergic asthma. In addition, several studies demonstrated that DE or DEP exposure increased susceptibility to bacterial and viral pathogens in laboratory animals and cell culture, with multiple effects on host defence systems.

Overall, **it is concluded that there is sufficient evidence that the relationship between DE exposure and immunological effects is likely to be causal**, based on the evidence from multiple controlled human exposure studies, which demonstrated an adjuvant effect of DE or DEP with different allergens and a potential role for DE in allergic airway disease and allergic sensitization, as well as limited evidence of reduced viral clearance. Supporting toxicological studies demonstrated exacerbation of allergic responses, increased sensitization to allergens and reduced host defence in animals. Furthermore, there is limited epidemiological evidence based on the increased risk of asthma in children exposed to DE, which is associated with a T helper type 2 immune response.

3.1.5 Reproductive and developmental effects

Studies examining reproductive and developmental effects of DE exposure in humans are very limited.

Multiple studies in rodents have demonstrated that DE and DEP can exert adverse effects on the male reproductive system. Exposure of adult animals resulted in changes in hormone levels, sperm characteristics and testicular tissue. Prenatal exposure to whole or filtered DE led to functional, histological and hormonal effects on the male reproductive system, indicating that the gaseous component of DE was involved. Non-linear dose–response relationships were observed, often with adverse effects present at low and medium, but not at high, exposure levels. There is more limited evidence of effects on the female reproductive system. Several studies have investigated potential developmental effects of prenatal or perinatal DE exposure in laboratory animals and have reported alterations in neurodevelopment, neurobehaviour, immunological outcomes and changes in deoxyribonucleic acid.

Overall, **it is concluded that the evidence is suggestive of a causal relationship between DE exposure and reproductive and developmental effects**, based on consistent evidence of adverse male reproductive effects from multiple animal studies and limited supporting evidence from a single cross-sectional study. Although the mechanisms of adverse reproductive and developmental effects have not been fully elucidated, DE exposure has been shown to result in alterations in hormone levels, nuclear receptor activation and gene expression in toxicological studies. These mechanisms of action may play a role in the observed histopathological changes in male and female reproductive systems, altered sperm morphology parameters, delayed sexual maturation and developmental neurotoxicity in animals.

3.1.6 Central nervous system effects

A limited number of studies in humans, experimental animals and cell cultures have investigated the potential effects of DE exposure on the nervous system. Neurophysiological symptoms associated with DE exposure were reported in epidemiological and controlled human exposure studies. Indications of altered neurological activity were noted in human and experimental animal studies. Also, oxidative stress was induced in neuronal cells exposed to DEP.

It is concluded that the evidence is suggestive of a causal relationship between acute DE exposure and central nervous system effects, based on acute neurophysiological symptoms in overexposed workers

in a case study and in subjects exposed to high DE levels in a controlled human exposure study. In addition, changes to frontal cortex activity were observed in the controlled human exposure study. Toxicological studies provide supporting evidence for an effect of DE exposure on spatial learning and memory deficits, as well as alterations in neurotransmitter levels and gene expression in the brains of offspring of dams exposed during pregnancy, which may have resulted from either single or repeated exposures.

It is concluded that there is inadequate evidence to infer a causal relationship between chronic DE exposure and central nervous system effects, based on limited evidence from potential perturbation of the parasympathetic nervous system due to observed HRV alterations in the panel studies. Furthermore, there is limited supporting evidence of neurotoxic effects in the developmental neurotoxicity study, as well as in vitro evidence of DEP-induced oxidative stress to neuronal cells and potential changes to brain capillaries due to oxidative stress.

3.2 Quantitative risk assessment

Evidence from the studies evaluating the impacts of diesel emissions on human health was also considered for use in quantitative risk assessment for the derivation of a cancer risk estimate as well as chronic exposure and short-term exposure guidance values for non-cancer health effects. It is recognized that Canadian population exposures to DE or DEP cannot be estimated at this point in time; however, the guidance values represent the current state of the science and may be of use in the future.

A summary of the quantitative risk assessments is provided in Table 3, and details are provided in the sections below.

Table 3. Summary of quantitative risk assessments

Health outcome	Risk/guidance value	Critical effect
Cancer	N/A[a]	
Non-cancer – chronic exposure	5 µg/m^3	Respiratory – inflammation, histopathological and/or functional changes
Non-cancer – short-term exposure	10 µg/m^3	Respiratory – increased airway resistance and inflammation

[a] See section 3.2.1.

3.2.1 Cancer

Within the current document, Health Canada has not evaluated the reviewed studies for use in a quantitative exposure–response analysis of lung cancer risk with DE exposure. Health Canada will consider the report of the Health Effects Institute's Diesel Epidemiology Project Panel[13] to inform any future activities relating to quantitative risk assessment analysis of DE. This expert panel was tasked with evaluating the potential utility of the existing epidemiological studies for estimation of cancer risk associated with DE exposure. It is considered that the observations made in rat bioassays of lung tumour induction due to DE exposure are not directly relevant to unit risk quantification owing to a number of factors, including the apparent species specificity of the observed effect, the very high levels of DE exposure required to solicit those effects and the role of particle overload in tumour development.

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[13] www.healtheffects.org/Workshops/DieselWorkshop2014/DieselEpiWorkshop2014.html

3.2.2 Chronic exposure to diesel exhaust and non-cancer effects

It is recognized that it would be preferable to use human data for characterization of the exposure–response relationship between DE and non-cancer health effects and for derivation of a chronic exposure guidance value. However, the current body of epidemiological literature is not considered adequate for this purpose. Consequently, evidence from studies in experimental animals was evaluated in lieu of epidemiological data. Using a traditional risk assessment framework and approach, the following methods were used to estimate a chronic exposure guidance value for DE.

For non-cancer health effects, DEP was chosen as the basis for development of a chronic exposure guidance value, for several reasons. Toxicological studies have demonstrated DEP to be the main causative agent of many of the health effects associated with DE exposure. Removal of the particulate component of DE resulted in fewer or less severe health effects. The DEP component of exhaust contains compounds known to be hazardous to human health, and DEP contributes to ambient PM, which is also known to be harmful to human health. Lastly, DEP is typically the parameter used to set experimental exposure levels.

For chronic exposure to DE, respiratory effects were chosen as the critical health effect for determination of a guidance value. The literature database for respiratory health effects is the most fully developed among the non-cancer health effects, with a consistent exposure–response relationship observed in several animal species in chronic exposure studies, and effects are observed at lower exposure concentrations compared with other effects. Additionally, epidemiological studies provide corroborative evidence that respiratory health effects are a pertinent health outcome for human exposures.

Considering the total body of literature on respiratory health effects, the no-observed-adverse-effect level (NOAEL) from the study by Ishinishi et al. (1986, 1988) was chosen as the point of departure for development of a chronic exposure guidance value. Five multi-dose, chronic exposure studies in rats were identified in the literature as candidates for guidance value development, with each reporting dose-dependent effects in the respiratory tract, including inflammation, histopathological changes and/or functional changes. Of the different studies, the lowest-observed-adverse-effect level (LOAEL) from each was within a narrow range of exposure concentrations, indicating a consistency in effect. From these candidate studies, the highest NOAEL was reported by Ishinishi et al. (1986, 1988). From this study, the NOAEL of 0.46 mg/m^3 DEP was chosen as the point of departure.

To develop a chronic exposure guidance value, dosimetric modelling was performed using the Multi Path Particle Dosimetry model. The model was used to estimate a human equivalent concentration (HEC) based on the point of departure concentration derived from the animal studies (Ishinishi et al. 1986, 1988). The HEC for a 70-year lifetime exposure was estimated to be 0.12 mg/m^3 DEP. An uncertainty factor of $10^{0.4}$ was applied to reflect potential toxicodynamic differences in animal to human extrapolation, and an additional uncertainty factor of 10 was applied to account for sensitive individuals in the human population. Based on the HEC of 0.12 mg/m^3 DEP and applying a composite uncertainty factor of 25 ($10^{0.4} \times 10$), a guidance value of 5 µg/m^3 DEP (0.12 mg/m^3 / 25) was derived. This chronic exposure guidance value is consistent with values previously developed by the World Health Organization, the US EPA and the California EPA.

Using a traditional risk assessment approach, the chronic exposure guidance value derived herein is an estimate of the concentration of DEP to which the general population, including sensitive subgroups,

may be exposed for a lifetime without the likelihood of appreciable harm from non-cancer effects. It is important to note that the recent epidemiological literature provides evidence of respiratory health effects associated with DE exposure, including increased risk of wheeze and asthma in children. The characterization of DE exposure is limited in these studies; therefore, it is uncertain if effects occur at levels below the chronic exposure guidance value. Further research is required to improve DE exposure quantification for the general population, which would allow for better understanding of the exposure–response relationships and characterization of population health risks associated with chronic DE exposure.

3.2.3 Short-term exposure to diesel exhaust and non-cancer effects

As per the discussion above regarding the chronic exposure guidance value, it would similarly be preferable to use epidemiological data for large populations for characterization of the exposure–response relationship between short-term DE exposure and non-cancer health effects. However, the current data are not deemed adequate for this purpose, and evidence from controlled human exposure studies was used instead. Using a traditional risk assessment framework and approach, the following methods were used to estimate a short-term exposure guidance value for DE.

Controlled human exposure studies were reviewed to determine the critical effect and point of departure. These studies provide a body of evidence that short-term exposure to DE can induce biological effects, with respiratory or cardiovascular health effects most often evaluated. Review of these studies indicated that respiratory endpoints are the most sensitive, with effects demonstrated at lower concentrations than for other types of endpoints. In three studies conducted with healthy and/or mildly asthmatic participants, increased measures of airway resistance were observed at 100 μg/m^3 DEP for a 2 h exposure period (Mudway et al. 2004; Riedl et al. 2012; Stenfors et al. 2004). Additionally, three studies reported respiratory inflammation in healthy subjects exposed to 100 μg/m^3 DEP for 2 h (Behndig et al. 2006, 2011; Stenfors et al. 2004). Respiratory inflammation was not noted in asthmatic subjects. Based on the consistency of results across multiple studies, a LOAEL of 100 μg/m^3 DEP was chosen as the point of departure for a short-term exposure guidance value.

Given that the studies used to select the point of departure included evaluation of a potentially sensitive subgroup (subjects with mild asthma), an uncertainty factor of 10$^{0.5}$ was applied to account for further susceptibility in the population due to age, disease status or genetic factors. To account for extrapolation from a LOAEL to a NOAEL, an uncertainty factor of 10$^{0.5}$ was applied, because the respiratory effects observed at the LOAEL are mild and reversible. This resulted in a composite uncertainty factor of 10. From the point of departure of 100 μg/m^3 DEP, a short-term exposure (2 h) guidance value of 10 μg/m^3 DEP (100 μg/m^3 / 10) was derived. Previous assessments of DE did not derive a short-term exposure guidance value.

Using a traditional risk assessment approach, the short-term exposure guidance value derived above is an estimate of the concentration of DEP to which the general population, including sensitive subgroups, may be exposed for up to 2 h without the likelihood of appreciable harm from non-cancer effects. However, large-scale epidemiological studies examining the acute effects of DE in the general population would likely provide a better understanding of the exposure–response relationships and characterization of population health risks associated with short-term DE exposure.

3.2.4 Guidance values for diesel exhaust and PM$_{2.5}$ health effects

It is important to note that DE is a major contributor to ambient PM$_{2.5}$ in Canada and that both DE and ambient PM$_{2.5}$ are mixtures that contain combustion emissions and many of the same constituents. Extensive epidemiological research into the population health effects of ambient PM$_{2.5}$ and the associated exposure–response relationships has provided evidence that PM$_{2.5}$ causes cardiorespiratory health effects with no indication of a threshold of effect in the population (e.g. respiratory hospital admissions and mortality). Importantly, the epidemiological studies of PM$_{2.5}$ examine very large populations and thus include a wide range of susceptibilities and health states that cannot be easily or adequately examined in laboratory settings. It is unclear at this time how DE contributes to the observed health effects of the larger mixture of ambient PM$_{2.5}$. In addition, owing to limitations in the DE exposure surrogates available for use in general population studies, it has not been possible to evaluate the population health impacts of DE as effectively as has been done for PM$_{2.5}$. In this document, Health Canada has evaluated the body of evidence regarding the health effects of exposure to DE or DEP specifically. The guidance values derived herein, which are traditionally based on the assumption that thresholds exist for the adverse health endpoints in the current database for DE/DEP, reflect an appropriate evaluation of the available data. However, it is recognized that further research may reveal a role for DE in the observed non-threshold population health effects of ambient PM$_{2.5}$ and better characterize the exposure–response relationships of DE health effects.

4 Conclusions

Internationally, the potential health effects of DE exposure have long been recognized, and great effort has resulted in substantial reductions in diesel emissions, including in Canada. A key accomplishment has been the introduction of stringent emission regulations for new diesel vehicles and engines, resulting in improved engine and emission control technologies in both the off-road and on-road diesel fleets. In addition, the quality of diesel fuel used in on-road, off-road, rail, marine and stationary engines has improved, particularly in terms of the sulphur content. Some jurisdictions have undertaken additional initiatives to mitigate in-use diesel engine emissions and human exposure to them, such as inspection and maintenance programs, retrofit and scrappage programs, idling restrictions and the establishment of low-emission zones. However, the Canadian in-use diesel fleet is still dominated by engines pre-dating the most recent emission standards.

Diesel-powered vehicles are pervasive on major roadways and in urban centres in Canada. It is reasonable to assume that most Canadians are regularly exposed to DE. Because of the variable and complex nature of DE and the fact that DE constituents are emitted by other pollution sources, it has been difficult to quantify general population exposure to DE. Several surrogates have been used to represent DE, all of which have had their limitations, and the respirable fraction of EC is considered to be one of the better options used to date.

This risk assessment considered the reviews and conclusions of the California EPA (1998) and the US EPA (2002) human health risk assessments for DE and provided detailed review of the health effects literature published since 2000. The available information supports the conclusion that DE emissions have direct effects on human health.

The newly published health studies along with supporting evidence from work published prior to 2000 provide sufficient evidence to conclude that DE is carcinogenic in humans and is specifically associated with the development of lung cancer. Although the risk estimates are generally small, the population

health risks are considered to be significant given the ubiquitous presence of DE emissions in Canada. The evidence is also suggestive that DE may be implicated in the development of cancer of the bladder in humans, but further research is required to allow definitive conclusions to be drawn. A limited number of studies have investigated other cancers in association with DE exposure, but the evidence is inadequate to draw conclusions regarding causality. Overall, these conclusions are consistent with the categorization of DE as a human carcinogen (Group 1) by the International Agency for Research on Cancer (Benbrahim-Tallaa et al. 2012; IARC 2013).

Regarding non-cancer health effects and the potential causal role of DE in their development, a number of conclusions are drawn from the existing literature. The evidence supports a causal relationship between acute exposure to DE at relatively high concentrations (a LOAEL of 100 $\mu g/m^3$ was identified for mildly asthmatic subjects) and effects on the respiratory system, including increases in airway resistance and respiratory inflammation. Under conditions of chronic exposure, DE exposure is likely to be causal in the development of respiratory effects. The evidence suggests that the toxicity of DE is associated with irritation of the respiratory tract, eliciting an immunological response via inflammatory processes. It was concluded that DE exposure is likely to be causal in the development of adverse cardiovascular outcomes following acute exposure and in the development of adverse immunological responses. The evidence reviewed is suggestive of a causal relationship between DE and 1) adverse cardiovascular outcomes following chronic exposure, 2) adverse reproductive and developmental effects and 3) central nervous system effects following acute exposure to DE. Currently, there is inadequate evidence to draw conclusions regarding the potential neurological impacts of chronic DE exposure.

Based on traditional risk assessment methodologies and with regard to general population exposures, a short-term exposure guidance value of 10 $\mu g/m^3$ and a chronic exposure guidance value of 5 $\mu g/m^3$ have been derived based on DEP to protect against adverse effects on the respiratory system. The available evidence indicates that respiratory effects occur at lower concentrations of DE than those associated with other non-cancer adverse effects, and so these guidance values are considered protective against the non-cancer health impacts of DE exposure. However, it is recognized that there have not been adequate large-scale epidemiological studies of non-cancer effects associated with either short-term or chronic DE exposure to conclusively characterize the exposure–response relationships. More research is needed to elucidate this and to evaluate the potential role of DE in the observed non-threshold population health effects of $PM_{2.5}$.

In general, it has been shown that sensitive subpopulations, such as the elderly, children and asthmatics, can be at greater risk of adverse respiratory effects due to DE exposure. Exposure of the elderly and asthmatics to traffic-related DE has been shown to increase respiratory inflammation. Also, pulmonary function decrements have been demonstrated in asthmatics exposed to traffic-related DE. Furthermore, traffic-related DE exposure in children has been implicated in potential asthma development later in life. The guidance values for short-term and chronic DE exposure presented above account for the enhanced sensitivity of subgroups in the population.

Overall, it is concluded that DE is associated with significant population health impacts in Canada, and efforts should continue to further reduce emissions of and human exposures to DE.

5 Key uncertainties and gaps

The epidemiological evidence along with supporting information regarding the genotoxicity and mutagenicity of DE and the known presence of human carcinogens in DE clearly indicate that DE is a

human carcinogen. However, the specific mode of action by which DE results in lung cancer in humans remains to be elucidated. In addition, it is unclear why cancer has not been consistently reported in animal models with a mode of action with relevance to humans, although it is noted that animal models have been similarly insensitive to the induction of lung tumours by mainstream tobacco smoke. These factors represent uncertainty in the database.

DE is a complex mixture of hundreds of chemicals, and the component or components of DE that are the most relevant toxicologically in the development of lung cancer or other health effects have not yet been identified. The evidence suggests that DE-related carcinogenicity is associated with the organic compounds adsorbed to the DEP. Evidence for several other health endpoints (e.g. respiratory and immunological outcomes) suggests that the toxicity of DE is associated predominantly with the particulate content.

The most appropriate metric for DE exposure remains unknown. Respirable EC is the best exposure surrogate used to date; however, it is not expected that EC is the toxicologically active component. Identification of the toxicologically relevant exposure metric(s) would allow for improved exposure–response quantification and estimation of general population risks.

Efforts to identify one or more exposure metrics for DE and to generate population exposure estimates would help to fill an important gap. Owing to the lack of an exposure metric specific to DE, it has been difficult to conduct large-scale general population epidemiological studies to investigate the health effects of DE specifically, limiting our understanding of the impacts of DE on potentially sensitive populations and our ability to estimate risk levels and to investigate the presence or absence of thresholds of effects. Such studies would provide an improved understanding of the exposure–response relationships for cancer and non-cancer effects associated with both short-term and chronic DE exposure and help to elucidate the potential role of DE in health effects associated with exposure to $PM_{2.5}$.

Although there have been some studies targeting effects in the elderly, children and asthmatics, in general, more information is required regarding the effects of DE in potentially sensitive subpopulations, including during pregnancy.

Characterization of historical exposures to DE in occupational and general population studies remains difficult, and, in spite of robust efforts to address this in occupational studies in particular, some level of uncertainty in the exposure estimates remains.

There is a general lack of human exposure–response information for chronic non-cancer health effects associated with DE exposure. The human guidance value for chronic inhalation exposure to DEP derived in this document was based on results from animal studies, introducing a level of uncertainty.

For other non-cancer health effects (e.g. immunological) to be considered as a critical effect of DE exposure, further epidemiological and toxicological evaluations to establish a consistent exposure–response relationship and mode of action would be required. For example, the role of DEP in allergic sensitization is a growing area of research, and the potential link to the increasing prevalence of asthma and allergy in the population warrants further evaluation.

Canadian population exposures to DE or DEP have not been characterized, and efforts in other jurisdictions, such as the United States, include substantial uncertainty.

Health Canada recognizes that there has been substantial evolution in diesel engine design and emission after-treatment, largely driven by new engine emission regulations over time. In addition, diesel fuel quality has changed to meet the technical requirements of the engines. Of key significance, new technology on-road heavy-duty diesel vehicles (HDDVs) since 2010 meet drastically reduced PM and NOx emission standards in North America. DE exposures in the epidemiological and toxicological studies reviewed in this document were mostly derived from older technology engines. Although the magnitude of emissions from new technology diesel engines is reduced, emissions may also change both physically and chemically, and it remains unclear as to how this will affect the toxicological properties of DE as a mixture and the health risks of DE exposure. In addition, because of the durability of diesel engines, it is expected that older engines will continue to contribute substantially to DE exposure of Canadians.

Part B – Health impacts assessment of diesel exhaust

As part of this assessment, efforts were made to quantify the population health impacts associated with the contribution of DE to criteria air contaminant concentrations in Canada. The analysis of population health impacts was conducted in a stepwise manner with the use of computer simulation tools to 1) estimate emissions from the Canadian diesel fleet, 2) estimate the impact of those emissions on ambient concentrations of criteria air contaminants across the country and 3) estimate population health impacts resulting from the incremental contribution of DE to air pollution levels. This was undertaken for calendar year 2015, and results were assessed on a national, provincial/territorial and regional basis. This analysis is complementary to the traditional risk assessment approach presented in Part A of this document.

6 Mobile source diesel emissions in Canada

The assessment of population health impacts resulting from exposure to air pollutants associated with the use of diesel fuel in Canada begins with an analysis of the pollutants released to the atmosphere. The national emission inventory was developed by Environment Canada for the year 2015, based on the best available data and tools. Emissions were projected for all source categories except open and natural sources,[14] with special consideration given to on-road and off-road[15] mobile diesel sources. Emission scenarios were defined in this analysis to estimate the contribution to Canadian pollutant emissions from on-road diesel emissions and the contribution from on-road and off-road diesel emissions combined. All Canadian engine emission standards and fuel regulations projected for 2015 were considered. The sulphur content of ultra low sulphur diesel was set at 10 ppm in the model to be more representative of current Canadian fuels.

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[14] Open source emissions are dispersed over large areas in a non-point manner, such as dust from farms, construction operations and paved and unpaved roads. As some of these sources involve the use of diesel vehicles and equipment, it is reasonable to expect that a fraction of these emissions, especially particulates, would be affected by the removal of diesel-related emissions. Allocating open source emissions to specific activities and sources was not currently feasible. Open source emissions were not affected by the scenarios investigated for this project.

[15] Note that rail and marine sources, which also consume diesel fuels, were not considered in off-road diesel applications to reflect the assumptions of the current assessment.

The 2015 Canadian mobile source emission inventory was generated from three different models: the NONROAD2012C model for off-road applications,[16] MOBILE6.2C for the light-duty on-road fleet and Motor Vehicle Emission Simulator version 2010a (MOVES2010a) for on-road HDDVs. These models, initially developed by the US EPA, were modified to reflect Canadian conditions (e.g. vehicle population and age distribution, vehicle emission standards, meteorological conditions and fuel characteristics). Estimates were available for exhaust, fugitive and evaporative emissions of air pollutants on a regional and provincial or territorial basis.[17] The models provided emission rates for a series of pollutants, such as NOx, PM, sulphur dioxide (SO_2) and VOCs, which were then combined with vehicle activity data. Emission factor ratios based on the SPECIATE data set from the US EPA were added for compounds that were not explicitly modelled (e.g. formaldehyde, acetaldehyde and benzene). Greenhouse gases were not considered.

Vehicle or engine population data were evaluated with respect to population numbers, fuel use and geographical distribution. For example, whereas HDDVs represent 68% of the heavy-duty class for the on-road fleet, light-duty diesel vehicles (LDDVs) account for only 3% of the on-road light-duty fleet. HDDVs and LDDVs are responsible for 94% and 6%, respectively, of the on-road diesel fuel consumption. For off-road applications, engines used in agricultural, mining, construction and oil sands operations, which represent only 10% of the fleet, are responsible for 80% of off-road diesel fuel use in Canada. In terms of geographical distribution, HDDVs are generally limited to major roadways and truck routes, LDDVs are concentrated in populated areas and off-road diesel applications are distributed in urban and rural or less populated regions. Consequently, the influence of the different diesel fleets on pollutant emissions and concentrations is expected to vary across Canada.

Table 4 shows on-road and off-road diesel pollutant emissions by province or territory. Canadian emission estimates are also included for different emission source categories. Lastly, the contributions from on-road and off-road diesel sources to Canadian emissions are presented. On-road diesel sources contribute significantly to on-road NOx, PM, SO_2 and VOC emissions. For example, on-road diesel sources are responsible for 72% of on-road $PM_{2.5}$ emissions and 54% of on-road NOx emissions. Off-road diesel sources also contribute considerably to total off-road emissions, with respective estimates of 37%, 43% and 46% for ammonia (NH_3), NOx and $PM_{2.5}$.

However, the contribution from on-road and off-road diesel sources to national pollutant emissions is limited to 2%, except for NOx at 23%. This suggests that although on-road and off-road diesel sources emit a considerable fraction of on-road and off-road source emissions, respectively, they contribute only minimally to total emissions of most air pollutants. It is important to recognize that the values presented in Table 4 are national averages and do not account for the spatial distribution of emissions across Canada. Although the portion of the national emission inventory associated with mobile source diesel emissions is limited, mobile source diesel emissions are relatively concentrated in populated areas, and their influence on urban air pollutant concentrations and human health is expected to be significant.

On-road HDDVs are associated with greater NOx emissions than on-road and off-road gasoline mobile source emissions combined (not shown). In comparison, on-road LDDV emissions are considerably less

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[16] The off-road diesel applications considered by the model include, for example, agricultural, mining and construction equipment.

[17] On-road diesel emissions included tire wear and brake lining (TWBL) emissions. TWBL emissions were not available for off-road applications, as they are not routinely generated by NONROAD2012C. TWBL emissions contribute only to particulate emissions.

Table 4. On-road and off-road diesel pollutant emissions by province or territory in comparison with Canadian emission estimates for mobile source and all emission source categories, for the year 2015

	CO	NH$_3$	NOx	PM$_{10}$	PM$_{2.5}$	SO$_2$	VOCs
Province or territory	On-road and off-road[a] diesel emissions (t)[b]						
Alberta	59 063	152	126 626	8 897	8 380	226	12 586
Ontario	36 812	144	72 532	5 766	5 169	105	8 551
Quebec	30 243	106	64 921	4 658	4 192	156	6 358
Saskatchewan	16 696	47	39 796	2 570	2 407	58	3 274
British Columbia	13 087	55	28 420	1 971	1 710	30	2 809
Manitoba	5 492	20	14 149	796	697	24	1 139
New Brunswick	5 021	17	11 160	742	664	30	1 111
Nova Scotia	4 618	14	9 544	686	631	22	1 000
Newfoundland and Labrador	3 144	10	8 210	474	447	45	672
Northwest Territories	1 372	3	2 946	192	186	5	317
Prince Edward Island	714	2	2 056	96	88	3	125
Yukon	201	1	541	32	30	2	42
Nunavut	29	0	90	4	4	0	6
Total (Canada)	176 492	572	380 989	26 885	24 604	707	37 992
Canadian source category	Canadian emissions (t)						
On-road HDDVs	59 597	301	164 183	8 711	6 885	301	13 166
On-road LDDVs	3 910	41	2 736	347	249	19	1 483
Off-road diesel[a]	112 984	230	214 070	17 828	17 471	387	23 343
On-road mobile – all fuels	3 133 371	22 980	308 072	15 032	9 865	1 889	155 666
Off-road mobile – all fuels[a]	2 225 190	623	493 968	39 900	37 964	7 710	232 192
All sources[c]	7 621 595	490 829	1 680 058	6 683 732	1 252 862	969 184	1 679 326
Canadian source category[d]	Contribution of mobile source diesel use to Canadian emissions (%)						
On-road diesel as % of on-road mobile (all fuels)	2.0	1.5	54.2	60.3	72.3	16.9	9.4
Off-road diesel as % of off-road mobile (all fuels)[a]	5.1	36.9	43.3	44.7	46.0	5.0	10.1
On-road and off-road[a] diesel as % of total emissions[c]	2.3	0.1	22.7	0.4	2.0	0.1	2.3

CO: carbon monoxide; HDDVs: heavy-duty diesel vehicles; LDDVs: light-duty diesel vehicles; NH$_3$: ammonia; NOx: nitrogen oxides; PM$_{xx}$: particulate matter with an aerodynamic diameter of xx μm or less; SO$_2$: sulphur dioxide; TWBL: tire wear and brake lining; VOCs: volatile organic compounds

[a] Excluding locomotive and marine applications.

[b] On-road PM emissions include TWBL emissions. TWBL emissions contribute only to particulate emissions, notably PM$_{10}$. Off-road diesel emissions do not include TWBL emissions.

[c] All emission source categories except open and natural sources

[d] For this section of Table 4, the contributions were determined by comparing the on-road and/or off-road diesel source emissions with the selected Canadian source category. For example, off-road diesel sources contribute 5.1% to total off-road CO emissions; on-road diesel sources contribute 2.0% to total on-road CO emissions; and on-road and off-road diesel sources contribute 2.3% to CO emissions from all sources.

than on-road light-duty gasoline vehicle (LDGV) emissions (e.g. equivalent to approximately 10% of PM$_{2.5}$ and 2% or less of CO or NOx emissions from on-road LDGVs). On-road HDDV PM emissions are 2–3 times higher than PM emissions estimated for on-road gasoline vehicles. VOC emission levels are relatively limited for diesel vehicles compared with emissions from gasoline vehicles. Nonetheless, as diesel-related emissions of VOCs and other pollutants (e.g. CO, PM$_{2.5}$, SO$_2$) tend to be localized near populations (e.g. within urban centres and along major truck routes), DE emissions potentially influence population exposure to several air pollutants.

The spatial distribution of on-road heavy-duty diesel emissions reflects the regional and provincial/territorial distribution of on-road HDDV populations. The combined emissions from Ontario, Quebec, Alberta and, to a lesser extent, Saskatchewan are responsible for most of the Canadian on-road HDDV pollutant emissions. The highest share of LDDV emissions is allocated to Alberta and Quebec.

Off-road diesel emissions are very high in Alberta, being responsible for nearly 40% of Canadian off-road NOx, $PM_{2.5}$ and VOC emissions. In particular, the oil sands region of Alberta is associated with very high emission levels.

7 Effects of on-road and off-road diesel emissions on air quality

The Canadian emission inventory developed for the current assessment was used as input for air quality model simulations with the source-oriented model A Unified Regional Air Quality Modelling System (AURAMS). AURAMS is a prognostic tool that integrates meteorological data, emission data and specific algorithms to simulate the diffusion, transport and chemical transformation of gases and particles in the atmosphere. It is used to predict how atmospheric concentrations change if emission rates or input scenarios are modified. AURAMS is used by Environment Canada to study the formation of tropospheric ozone (O_3), PM and acid deposition in North America, in support of air quality policy and management decisions for Canada.

The objective of the current analysis was to evaluate the impact that diesel emissions have on air quality in Canada. A sensitivity analysis technique was used wherein air quality was modelled under three scenarios: 1) with the full Canadian emission inventory, 2) with on-road diesel emissions removed from the Canadian inventory and 3) with on-road and off-road diesel emissions removed from the Canadian inventory.[18] The scenarios were based on 2015 emission projections. The AURAMS model estimated ground-level concentrations of individual air pollutants under each scenario, for each grid cell of a national domain. The air quality differences between the full emission inventory scenario and the scenarios with diesel emissions removed were assumed to represent the impact of diesel emissions in Canada.

The AURAMS modelling was initially conducted over a continental 45 km grid domain. Two nested regional domains that overlap to cover the 10 Canadian provinces were run using piloting files from the 45 km grid. The outputs from the regional domains were merged to obtain national coverage on a 22.5 km grid. AURAMS generated results for the following annual concentration metrics: 1 h daily maximum for CO and O_3; 8 h running average daily maximum for O_3; and 24 h daily average for CO, NO_2, O_3, PM_{10} and $PM_{2.5}$. Summer 1 h daily maximum O_3 (i.e. May–September) results were also generated.[19] Modelling results are presented as ground-level concentrations over grid cells and Canadian census divisions (CDs). Concentrations from individual 22.5 km grid cells were used to generate the following figures.

The discussion herein focuses on O_3 and $PM_{2.5}$, which are constituents of smog, and NO_2. O_3 is not emitted in DE but is formed from gaseous precursors found in DE emissions – notably, NOx and VOCs. The local concentration of NOx influences whether the atmosphere in a particular region is either a source or a sink of O_3. Atmospheric conditions also influence O_3 concentrations.

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[18] Similar scenarios were not assumed for the United States or Mexico.
[19] O_3 formation is generally higher in summer than in winter as a result of meteorological conditions (e.g. increased solar radiation).

7.1 Impacts associated with diesel emissions

7.1.1 Ozone

Diesel emissions are associated with both increases and decreases in O_3 concentrations across Canada. Increases of 1–5% in O_3 concentrations cover economically active and populated areas – for example, most of the Windsor–Québec corridor, the area along the St. Lawrence River and the southern portion of the Prairies. Figure 1 shows the absolute contribution of on-road diesel emissions to summer 1 h daily maximum O_3 concentrations. Positive variations indicate that diesel emissions contribute to an increase in O_3 concentrations, whereas negative variations suggest that diesel emissions lead to a decrease in O_3 concentrations. Absolute contributions are generally estimated at 0.01–1.00 parts per billion by volume (ppbv), although increases of 1.00–2.78 ppbv (highest value reported near Regina) are projected downwind of major urban centres (e.g. east of Montréal).

Within large urban centres, on-road diesel emissions are associated with reductions in O_3 concentrations of 5% or more. These correspond to decreases of 0.01–5.76 ppbv, with the maximum variation being reported in Vancouver. Decreases in urban areas are likely related to elevated NOx emissions from on-road diesel sources, which decrease O_3 levels via O_3 scavenging and atmospheric radical removal by NO and NO_2, respectively.

For O_3, the air quality effects associated with on-road and off-road diesel emissions combined are larger than the effects of on-road diesel emissions only. On-road and off-road diesel sources contribute 1–5% to summer O_3 concentrations over most of the southern half of Canada. Contributions of 5% or more to O_3 concentrations are noted over large areas of Alberta, Saskatchewan and Quebec. Reductions of 5% or more in O_3 concentrations are associated with on-road and off-road diesel emissions in most of the large urban centres, but they are more spatially limited than reductions associated with on-road emission results (e.g. compare Figure 2 with Figure 1). Although the exact reason for this observation is uncertain, it is possible that on-road and off-road diesel emissions combined do not have the same influence on the NOx/VOC equilibrium in urban grid cells as do on-road emissions alone. As a result, decreases in O_3 are observed in fewer grid cells, presumably only in those with higher NOx to VOC ratios (i.e. urban cores).

Figure 2 shows that on-road and off-road diesel emissions are associated with an increase in summer 1 h daily maximum O_3 levels of 0.01–0.50 ppbv across a large part of Canada, in particular across areas with low populations. Increases of 0.50–2.00 ppbv affect the Lower Fraser Valley region of British Columbia, the Prairies, the Windsor–Québec corridor and areas along the St. Lawrence River, whereas contributions of 2.00–8.44 ppbv are modelled over vast areas around Calgary, Edmonton, Saskatoon, Regina and Montréal. Decreases in O_3 levels are limited to grid cells corresponding to large urban centres. A decrease of 7.63 ppb is modelled in Vancouver.

7.1.2 Fine particulate matter

Based on the modelling, on-road diesel emissions contribute 0.1–5% to annual daily $PM_{2.5}$ concentrations across Canada. Contributions of 1–5% are modelled in the Lower Fraser Valley area of British Columbia, the southern part of the Prairie provinces and the Windsor–Québec corridor and along the St. Lawrence River. Increases of 5–10% are also observed in some urban areas (e.g. Vancouver, Edmonton, Winnipeg and Montréal), where on-road diesel emissions are presumably concentrated and more important. Areas between and around urban centres are also affected by on-road emissions, likely
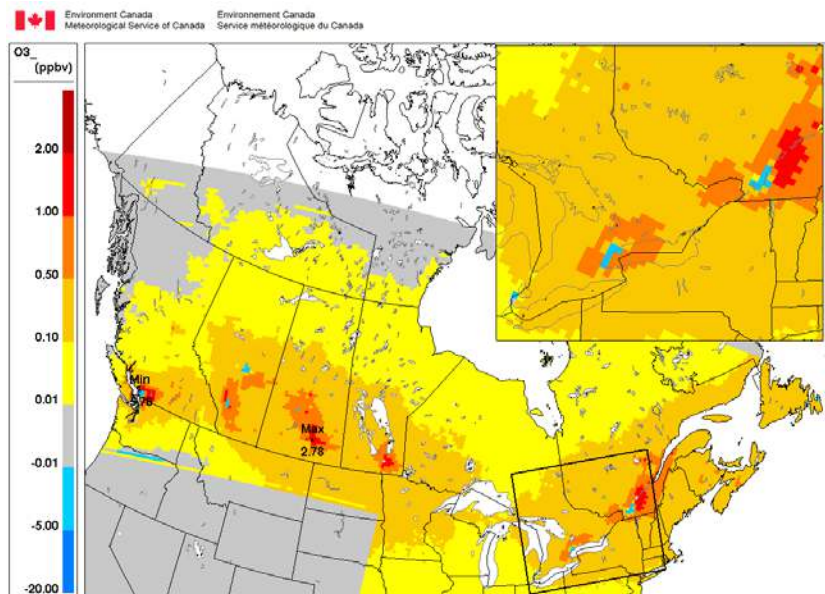
Figure 1. Absolute contribution to summer 1 h daily maximum O_3 concentrations associated with on-road diesel emissions in Canada in 2015

Figure 2. Absolute contribution to summer 1 h daily maximum O_3 concentrations associated with on-road and off-road diesel emissions in Canada in 2015

owing to the transportation of goods on major roads connecting population and economic centres. In terms of $PM_{2.5}$ composition, on-road diesel emissions potentially contribute 10–50% of the EC content by mass (maximum contribution reported in Edmonton).

Figure 3 shows that on-road diesel emissions contribute 0.01–0.50 µg/m^3 to $PM_{2.5}$ concentrations in the populated and economically active regions and 0.50–1.35 µg/m^3 in urban centres (maximum contribution reported in Edmonton).

Figure 3. Absolute contribution to annual daily mean PM$_{2.5}$ concentrations associated with on-road diesel emissions in Canada in 2015

On-road and off-road diesel emissions increase PM$_{2.5}$ concentrations by 0.10–10% across Canada. Contributions of 5–10% are observed in the Lower Fraser Valley area of British Columbia, most of the Prairie provinces as well as an area located southeast and east of Montréal. Contributions to PM$_{2.5}$ concentrations higher than 10% are also reported in Calgary and Edmonton, in addition to a few grid cells in Saskatchewan. On-road and off-road diesel emissions influence 25–50% of the EC fraction content of PM$_{2.5}$ mass in urban and economically active areas. In Edmonton, at least 50% of the EC fraction appears to derive from on-road and off-road diesel emissions.

Figure 4 shows the absolute contribution from on-road and off-road diesel emissions to annual daily mean PM$_{2.5}$ concentrations. The Lower Fraser Valley region of British Columbia, the southern half of Alberta and Saskatchewan and the Windsor–Québec corridor are associated with contributions of 0.10–1.00 µg/m^3. On-road and off-road diesel emissions from agricultural and mining (e.g. oil and gas) activities seem to have a considerable effect on air quality in the Prairie provinces. In the larger urban centres, on-road and off-road diesel emissions increase PM$_{2.5}$ concentrations by 1.00–2.65 µg/m^3 (maximum contribution reported for Calgary).

7.1.3 Nitrogen dioxide

On-road diesel emissions contribute 0.01–50% to NO$_2$ concentrations across Canada. Higher contributions (10–50%) are projected around the more populated areas, as well as in areas where sources of NO$_2$ emissions other than on-road diesel vehicles (e.g. industrial) are possibly limited, such as New Brunswick, Prince Edward Island and the Saguenay-Lac-St-Jean and Bas-Saint-Laurent regions of Quebec. In absolute terms, on-road diesel emissions contribute 0.01–0.50 ppbv to NO$_2$ concentrations in the more populated and economically active regions and 0.50–4.62 ppbv in urban centres (maximum contribution reported in Montréal; Figure 5).

On-road and off-road diesel emissions are responsible for a minimum of 1–10% of NO$_2$ concentrations across Canada. Contributions of 10–50% are estimated over large regions. Nonetheless, in absolute
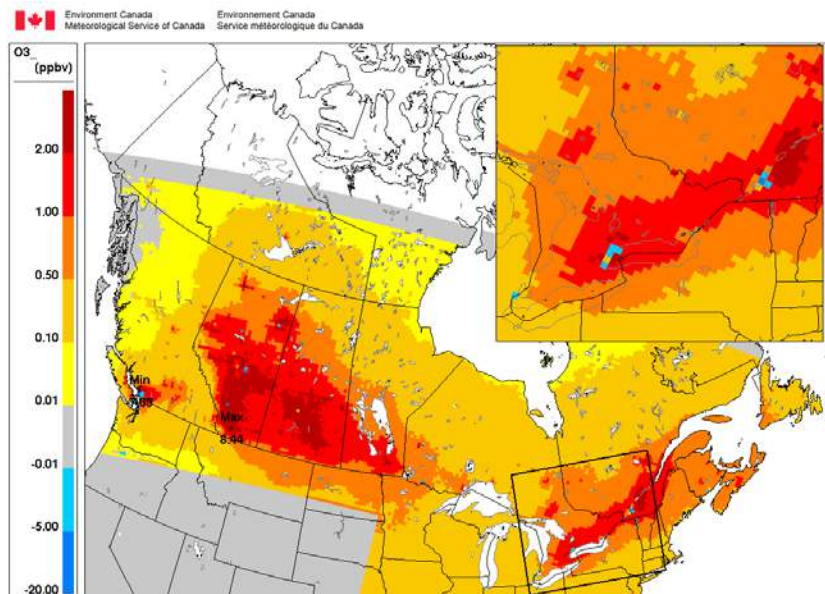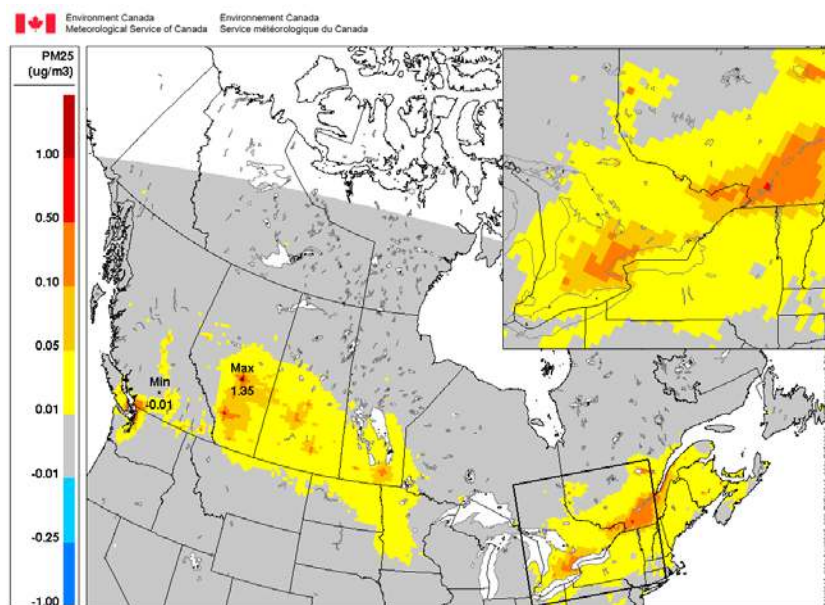
Figure 4. Absolute contribution to annual daily mean PM$_{2.5}$ concentrations associated with on-road and off-road diesel emissions in Canada in 2015
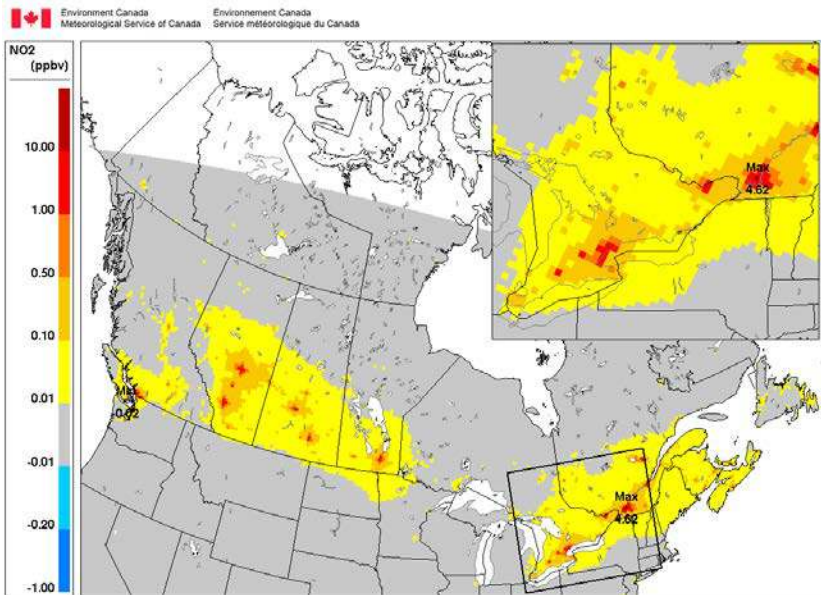
Figure 5. Absolute contribution to annual daily mean NO$_2$ concentrations associated with on-road diesel emissions in Canada in 2015

terms, on-road and off-road diesel emissions are generally associated with an increase of less than 1 ppbv (Figure 6).

A comparison of Figures 5 and 6 reveals the relative importance of off-road diesel NO$_2$ emissions in urban and rural areas. Large urban centres, intensive agricultural regions and important mining regions (e.g. Alberta oil sands) correspond to increases of 1–8.04 ppb (maximum contribution reported in Calgary).
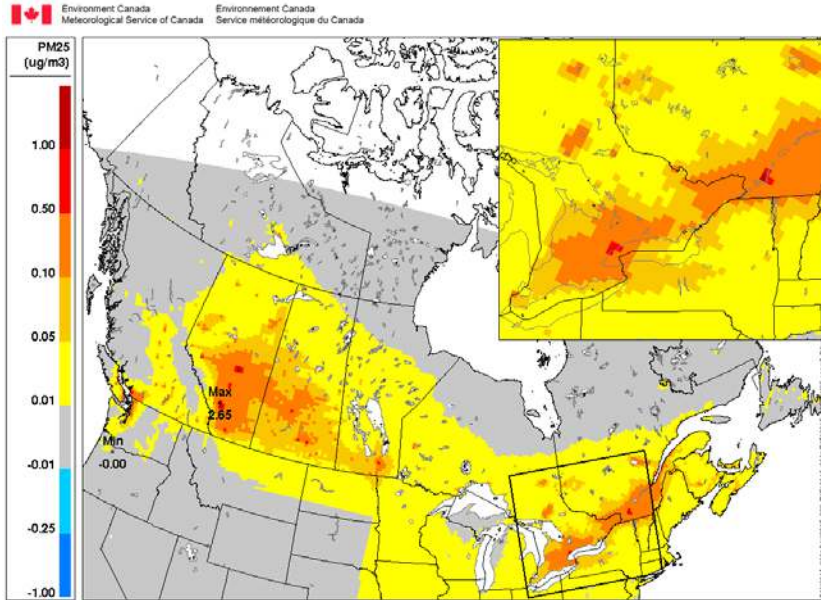
24

Figure 6. Absolute contribution to annual daily mean NO_2 concentrations associated with on-road and off-road diesel emissions in Canada in 2015
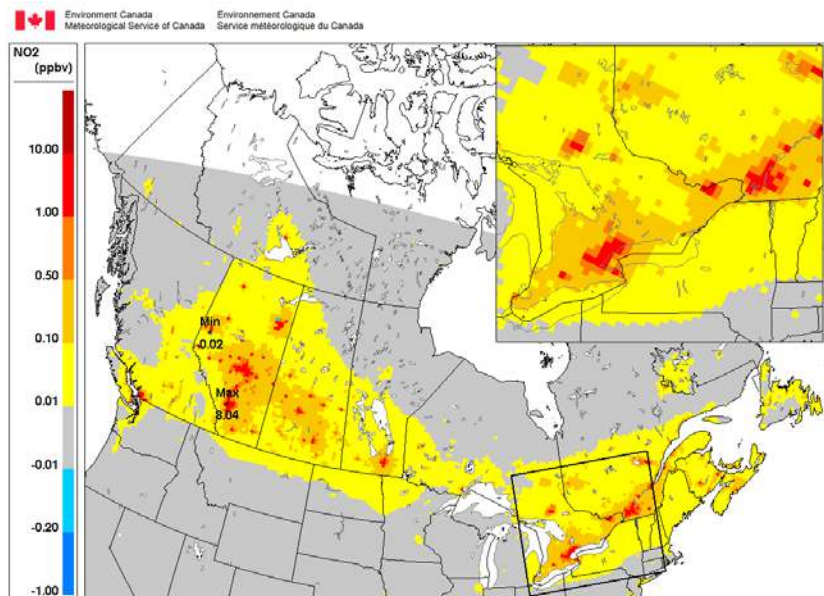
7.1.4 Other pollutants

Diesel emissions contributed minimally to SO_2 concentrations across Canada as a result of the use of ultra low sulphur diesel in on-road and off-road diesel applications. Diesel emissions showed limited contributions to CO concentrations. Diesel emissions also had a limited influence on VOC concentrations, contributing 2.21 ppbv or less even in urban centres where most of the diesel emissions are released.

7.2 Results by Canadian census division

The air quality impacts of DE were also estimated at the CD level for CO, NO_2, O_3, $PM_{2.5}$ and SO_2. The CD-based results are used in the Air Quality Benefits Assessment Tool (AQBAT) presented below. Results from the 22.5 km grid modelling were area-weighted for each CD based on the 2006 Census of Canada.[20] Generally, DE contributions to air pollutant concentrations per CD are lower than those at the grid cell level owing to the area weighting. The more extreme values observed in a single or a few grid cells tend to lessen when averaged across larger areas. This is especially the case outside of major urban areas, where CDs cover large areas.

The national mean of the area-weighted CD concentrations represents the average change in concentration across all CDs, irrespective of the population that resides in each CD. An alternative

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[20] Area-weighted concentrations for each CD were determined by summing the product of a grid cell concentration and the area of that grid cell occupied by the CD, for all grid cells intersecting with the CD. For example, if three grid cells intersect with a CD, its concentration (C_d) is determined by Equation 1:

$$C_d = (A_{d1} \times C_{g1} + A_{d2} \times C_{g2} + A_{d3} \times C_{g3}) \div A_d \qquad \text{(Equation 1)}$$

where A_{dx} is an intersection area between the model grid squares and the CD, C_{gx} is the concentration of the grid cell gx and A_d is the area of the CD.

approach to estimating a national average from the CD results is to use a population-weighting method. This method estimates the average exposure concentration for an individual within a geographic unit. When averaged across larger geographic units, CDs with high populations have more influence or weight than CDs with low populations. Population-weighted concentrations for all provinces and territories were determined by summing the product of a CD concentration and the fraction of the provincial population that is included in that CD, for all CDs in a province.[21] From the provincial and territorial population-weighted results, the same method is used to estimate the population-weighted national average. The national and provincial mean population-weighted results for $PM_{2.5}$, NO_2 and summer O_3 are provided in Table 5.

Table 5. Impact of mobile source diesel emissions on modelled national and provincial mean population-weighted concentrations of $PM_{2.5}$, NO_2 and summer O_3 in Canada in 2015[a,b,c]

Region [population]	Modelled mean population-weighted concentration [On-road diesel impact (%) / On-road and off-road diesel impact (%)]		
	$PM_{2.5}$ ($\mu g/m^3$)	NO_2 (ppb)	Summer O_3 (ppb)
Canada [36 101 253]	5.80 [2.8 / 5.3]	4.77 [17.2 / 26.5]	40.72 [−0.2 / 1.7]
Alberta [3 995 100]	4.80 [2.3 / 6.9]	4.39 [10.2 / 24.1]	45.07 [0.7 / 6.4]
British Columbia [4 888 059]	4.61 [3.9 / 5.7]	3.64 [20.9 / 26.4]	39.64 [−1.1 / −1.0]
Manitoba [1 300 664]	4.02 [5.2 / 6.6]	5.28 [32.8 / 36.9]	37.85 [1.2 / 3.5]
New Brunswick [768 857]	2.26 [1.1 / 2.1]	0.58 [18.7 / 28.0]	35.51 [1.0 / 2.1]
Newfoundland and Labrador [510 193]	2.00 [0.3 / 0.4]	0.44 [6.7 / 12.4]	31.88 [0.5 / 1.2]
Nova Scotia [967 182]	2.78 [0.6 / 1.4]	0.67 [13.4 / 23.3]	35.66 [1.0 / 2.1]
Ontario [14 127 882]	6.45 [2.1 / 4.8]	5.20 [13.7 / 24.2]	44.24 [0.1 / 1.7]
Prince Edward Island [148 740]	3.49 [0.8 / 1.4]	0.58 [24.8 / 34.2]	34.98 [1.1 / 2.2]
Quebec [8 212 175]	7.65 [3.6 / 5.9]	6.56 [21.1 / 29.3]	35.79 [−1.4 / −0.2]
Saskatchewan [1 067 999]	2.27 [2.1 / 4.7]	1.09 [15.4 / 24.7]	39.43 [1.6 / 5.4]

NO_2: nitrogen dioxide; O_3: ozone; $PM_{2.5}$: fine particulate matter or particulate matter with an aerodynamic diameter of 2.5 µm or less; ppb: parts per billion
[a] Concentration values rounded to two decimals; percent values rounded to one decimal.
[b] For O_3 concentrations, summer is defined as the period from May to September.
[c] Values for the Northwest Territories, Nunavut and Yukon are not shown. The values were generally very low and difficult to interpret.

On-road and off-road diesel emissions combined contribute approximately 2 times more to $PM_{2.5}$ concentrations than on-road diesel emissions alone. This suggests that off-road and on-road diesel emissions, individually, have a roughly similar impact on concentrations of $PM_{2.5}$ in Canada. On-road and off-road diesel emissions also increase NO_2 concentrations by 1.5 times compared with on-road diesel emissions alone. Thus, on-road diesel emissions appear to have more influence on ambient NO_2 concentrations than off-road diesel emissions, possibly because of the spatial distribution of those emissions. However, as NOx emissions are involved in multiple photochemical reactions, a linear scaling of NO_2 concentrations between scenarios is unlikely. Nonetheless, regional variations are observed. For example, the data provided in Table 5 suggest that on-road diesel emissions have relatively more impact

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[21] For example, if a province or territory includes three CDs, the following formula would apply to determine its population-weighted concentration (C_{pw}):

$$C_{pw} = (CD_{d1} \times CD_{pop1} \div PT_{pop}) + (CD_{d2} \times CD_{pop2} \div PT_{pop}) + (CD_{d3} \times CD_{pop3} \div PT_{pop}) \qquad \text{(Equation 2)}$$

where CD_{dx} is the concentration of CDx, CD_{popx} is the population of CDx and PT_{pop} is the population of the province or territory.

in British Columbia and Manitoba, whereas off-road diesel emissions have relatively more impact in Alberta.

The national population-weighted summer O_3 concentration estimate associated with on-road diesel emissions is negative (i.e. –0.2 % or –0.06 ppb), whereas the national estimate for on-road and off-road emissions is positive (i.e. 1.7 % or 0.69 ppb). The negative result reflects the fact that on-road diesel emissions are expected to decrease summer O_3 concentrations in a few highly populated CDs, which include a large fraction of the Canadian population. Owing to non-linear processes that affect O_3 concentrations, it is not possible to accurately differentiate the contributions from on-road and off-road diesel emissions individually within the current analysis. The change from a negative to a positive contribution cannot be allocated to off-road diesel emissions. Rather, the positive value reflects the contribution from emissions equal to the combined on-road and off-road contributions. Overall, the results suggest that both on-road and off-road diesel emissions affect O_3 concentrations across Canada and that the magnitude and direction of their contributions to O_3 levels are dependent on environmental conditions and non-linear atmospheric chemical mechanisms.

8 Health impacts of on-road and off-road diesel emissions

Population health impacts associated with exposures to CO, NO_2, O_3, $PM_{2.5}$ and SO_2 resulting from diesel fuel use were estimated using the AQBAT, a Health Canada model that estimates annual population health and welfare benefits or costs associated with changes in ambient concentrations of pollutants in Canada. The health effects of these air pollutants are well documented in the scientific literature, and diesel applications contribute to both primary emissions and secondary production of these pollutants in the atmosphere. The health effects assessed in the AQBAT include both morbidity and mortality outcomes.

For this analysis, the AQBAT was used to estimate the incremental health impacts associated with the difference in air pollutant concentrations between the diesel scenarios modelled with AURAMS, as described in section 7. Specifically, health impacts associated with the air pollutant concentration differences between the scenario with the full emission inventory and the scenario with either the on-road diesel emissions or the on-road and off-road diesel emissions removed were estimated. The population health impacts are assumed to represent the effect of diesel emissions in Canada. Health impacts were estimated for individual geographic areas, represented by 288 CDs of varying geographical and population sizes.[22]

The AQBAT includes health impact information for $PM_{2.5}$, O_3, CO, NO_2 and SO_2 in the form of concentration–response functions (CRFs) derived from published peer-reviewed epidemiological studies pertaining to Canadian and other populations. A CRF is a probabilistic value expressed as the average per capita excess risk of an adverse outcome (e.g. asthma symptom days) per unit increase in ambient pollutant concentration (e.g. per 1 $\mu g/m^3$ of $PM_{2.5}$). CRFs, which are input as distribution functions to reflect the uncertainty of the estimates, are used to estimate the magnitude of health impacts resulting from a specified change in air quality. The calculations used in the AQBAT to estimate the number of excess health outcomes attributable to a change in air pollutant concentrations are based on the baseline risk of each outcome in the population, population counts, the CRFs and the change in air pollutant concentrations. The health outcomes in the AQBAT are considered to have no threshold for

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[22] Population estimates for the 2015 AQBAT runs were based on projections prepared by Statistics Canada.

effect (i.e. effects are assumed to occur at all levels of exposure). The health endpoints, their acute or chronic nature, the associated CRFs and the applicable population group(s) (e.g. age-specific groups) are pre-defined within the AQBAT and represent Health Canada–endorsed values drawn from the health science literature.

The AQBAT also includes economic valuation estimates for the health outcomes considered in the model. Economic valuation estimates consider the potential social, economic and public welfare consequences of the health outcomes, including medical costs, reduced workplace productivity, pain and suffering, and the effects of increased mortality risk. The dollar value per health outcome is entered as a distribution in the AQBAT, reflecting the uncertainty in the estimates. For example, the central value provided for premature mortality is $6.5 million (low and high values of $3.5 million and $9.5 million, respectively; probability weight factor of 50% for the central value and of 25% for the low and high values).

To estimate the incremental health costs or benefits across the Canadian population associated with diesel emissions in 2015, two runs were simulated to address the following questions: 1) What are the annual health impacts associated with on-road diesel emissions in Canada? and 2) What are the annual health impacts associated with on-road and off-road diesel emissions in Canada? The analysis focused solely on variations in pollutant emissions during vehicle use and excluded emissions from upstream activities, such as diesel fuel production, storage and transport.

8.1 National estimates of population health impacts from diesel fuel use

Results of the AQBAT simulations for the 2015 national diesel use scenarios are presented in Table 6. Mean incremental health risks associated with diesel emissions are presented for individual health endpoints. Economic valuation estimates are the mean estimates, expressed in 2013 Canadian dollars. Negative values indicate a benefit in terms of health outcomes and monetary valuation associated with diesel emissions, whereas positive values reflect a cost. The values are interpreted as the incremental population health impacts associated with diesel emissions in Canada.

8.1.1 On-road diesel sources

Based on the AQBAT simulations, it is estimated that on-road diesel emissions contribute 320 premature mortalities in 2015 (2.5th and 97.5th percentiles equal 110 and 740, respectively; Table 6). Chronic exposure IHD mortality (170 outcomes or 51% of all mortalities), acute exposure mortality (85 outcomes or 26% of all mortalities) and chronic exposure lung cancer mortality (50 outcomes or 15% of all mortalities) account for the majority of mortalities. Incremental premature mortalities are driven by mortality risk associated with chronic exposure to $PM_{2.5}$ (250 premature mortalities) and acute exposure to NO_2 (150 premature mortalities). The large effects associated with $PM_{2.5}$ and NO_2 concentrations concur with the emission data presented previously, which demonstrated that mobile diesel sources contributed significantly to $PM_{2.5}$ and NO_2 emissions. It also shows that relatively small changes in $PM_{2.5}$ concentrations (generally less than 1 $\mu g/m^3$; see Figure 3) can have considerable impacts on Canadian population health.

On-road diesel emissions are associated with reductions in premature mortalities caused by exposure to annual O_3 (68 avoided acute premature mortalities) and summer O_3 (9 avoided chronic premature mortalities). Diesel emissions can decrease O_3 concentrations in areas where ambient NOx

concentrations are elevated compared with VOC concentrations. In the case of annual O_3, on-road diesel emissions increase health impacts in 260 CDs and decrease impacts in 25 CDs.[23] The overall O_3 effect associated with on-road diesel emissions is positive, because the 25 CDs, which included Greater Vancouver, Toronto and Montréal, represent 48% of the Canadian population. On-road diesel emissions

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[23] Results were available for 285 of the 288 CDs in Canada.

Table 6. National count estimates by health endpoint and total cost estimates associated with diesel emissions, based on results from the AQBAT for Canada in 2015

Health endpoint[a]	Pollutant	On-road diesel emissions[b]	On-road and off-road diesel emissions[b]	Difference[c]
Mortality[d]				
Acute exposure mortality[e]	CO, SO$_2$	1	2	1
Acute exposure mortality[f]	NO$_2$	150	230	80
Acute exposure mortality	O$_3$	−68	−33	35
Chronic exposure respiratory mortality	O$_3$ summer[g]	−9	55	64
Chronic exposure cerebrovascular mortality	PM$_{2.5}$	13	25	12
Chronic exposure COPD mortality	PM$_{2.5}$	17	32	15
Chronic exposure IHD mortality	PM$_{2.5}$	170	310	140
Chronic exposure lung cancer mortality	PM$_{2.5}$	50	94	44
Total chronic mortality[c]	O$_3$ summer, PM$_{2.5}$	240	520	280
All mortalities[c]	All pollutants	320 [110; 740]	710 [330; 1500]	390
Morbidity[d]				
Acute respiratory symptom days	O$_3$ summer, PM$_{2.5}$	880 000	2 200 000	1 400 000
Adult chronic bronchitis cases	PM$_{2.5}$	360	680	320
Asthma symptom days	O$_3$ summer, PM$_{2.5}$	62 000	170 000	110 000
Cardiac emergency room visits	PM$_{2.5}$	37	70	43
Cardiac hospital admissions	PM$_{2.5}$	28	53	25
Child acute bronchitis episodes	PM$_{2.5}$	1 500	3 000	1 400
Elderly cardiac hospital admissions	CO	5	8	3
Minor restricted activity days	O$_3$ summer	−11 000	110 000	120 000
Respiratory emergency room visits	O$_3$ summer, PM$_{2.5}$	76	340	260
Respiratory hospital admissions	O$_3$ summer, PM$_{2.5}$	15	67	52
Restricted activity days	PM$_{2.5}$	490 000	940 000	450 000
Costs[c] ($ thousands)				
All mortalities	All pollutants	$2 300 000 [$690 000/ $5 600 000]	$5 100 000 [$2 100 000/ $11 000 000]	$2 800 000
All endpoints	All pollutants	$2 500 000 [$870 000/ $5 800 000]	$5 500 000 [$2 500 000/ $12 000 000]	$3 000 000

AQBAT: Air Quality Benefits Assessment Tool; CO: carbon monoxide; COPD: chronic obstructive pulmonary disease; CRF: concentration–response function; IHD: ischemic heart disease; NO$_2$: nitrogen dioxide; O$_3$: ozone; PM$_{2.5}$: fine particulate matter or particulate matter with an aerodynamic diameter of 2.5 μm or less; SO$_2$: sulphur dioxide

[a] Unless otherwise specified, CRFs reflect an exposure to the pollutant at any time during the year.

[b] Counts represent mean estimates from the multiple iterations; counts and costs for all mortalities and all endpoints include [2.5th; 97.5th] percentiles.

[c] Total or difference may not calculate as expected because of rounding.

[d] Counts of health outcomes and valuation estimates ($ thousand) are rounded to the nearest integer with a maximum of two significant figures. Costs are in 2013 Canadian dollars.

[e] Premature mortalities associated with acute exposure affect all ages, whereas other premature mortality endpoints apply only to adults.

[f] Although the AQBAT includes a CRF for acute exposure mortality associated with NO$_2$ exposure, it is not assumed to reflect a causal relationship. Rather, NO$_2$ may be acting as a surrogate for a specific component of the air pollution mixture, such as vehicle exhaust emissions.

[g] May–September only.

Table 7. Comparison of national and provincial or territorial premature mortality counts for pollutants associated with diesel emissions, based on results from the AQBAT for Canada in 2015

Region [population]	Contribution to premature mortality – counts[a]							
	On-road diesel emissions				On-road and off-road diesel emissions			
	NO_2	$PM_{2.5}$	O_3[b]	All pollutants	NO_2	$PM_{2.5}$	O_3[b]	All pollutants
Canada [36 101 253]	150	250	−77	320	230	460	21	710
Alberta [3 995 100]	7	14	2	23	16	42	38	96
British Columbia [4 888 059]	18	34	−15	37	22	50	−15	57
Manitoba [1 300 664]	12	12	−2	23	14	15	4	34
New Brunswick [768 857]	1	1	2	3	1	2	4	7
Newfoundland and Labrador [510 193]	0	0	1	1	0	0	1	2
Northwest Territories [45 541]	0	0	0	0	0	0	0	0
Nova Scotia [967 182]	1	1	2	4	1	2	5	8
Nunavut [34 101]	0	0	0	0	0	0	0	0
Ontario [14 127 882]	49	77	−21	110	86	170	12	270
Prince Edward Island [148 740]	0	0	0	1	0	0	1	1
Quebec [8 212 175]	64	110	−50	120	89	170	−41	220
Saskatchewan [1 067 999]	1	2	4	7	2	5	13	20
Yukon [34 760]	0	0	0	0	0	0	0	0

[a] Counts represent mean estimates of premature mortality. Counts are rounded to the nearest integer and given to a maximum of two significant figures. All pollutants also include premature mortalities associated with CO and SO_2.
[b] Acute and chronic exposure premature mortalities combined.

are also associated with a considerable number of acute respiratory symptom days, asthma symptom days and restricted activity days.

The total economic value of the incremental health outcomes associated with on-road diesel emissions in 2015 is approximately $2.5 billion (2.5th and 97.5th percentiles equal $870 million and $5.8 billion, respectively). Premature mortalities alone are valued at $2.3 billion (Table 6). Although the monetary value associated with morbidity endpoints is low compared with mortality endpoints, the morbidity outcomes represent a significant impact on Canadian population health.

Benefits or costs associated with on-road diesel emissions are not distributed equally across provinces/territories and CDs owing to several key factors, such as population density, vehicle fleet composition and size, and baseline air quality. The highest costs and counts are associated with the most populated provinces, such as Alberta, British Columbia, Ontario and Quebec (Table 7), as well as with CDs corresponding to large urban centres. For example, on-road diesel emissions are generally associated with greater health impacts in the CDs of Montréal (CD2466), Toronto (CD3520) and Greater Vancouver (CD5915). In addition, on-road diesel emissions are estimated to have high population health impacts relative to population size in the province of Quebec (e.g. 120 premature mortalities from all pollutants in Quebec compared with 110 for Ontario). Possible reasons for this observation are population density and geographic distribution, vehicle fleet characteristics and atmospheric transport of traffic emissions. In fact, it is likely that urban centres in southwestern Quebec (including Laval, Longueil and Montréal) are affected by on-road emissions released locally and regionally along the Windsor–Québec corridor.

8.1.2 On-road and off-road diesel sources

Diesel emissions from on-road and off-road sources combined are associated on average with incremental health impacts of approximately 710 premature mortalities in 2015 (Table 6). Chronic exposure IHD mortality (310 outcomes or 43% of all mortalities), acute exposure mortality (200 outcomes or 28% of all mortalities) and chronic exposure lung cancer mortality (94 outcomes or 13% of all mortalities) account for the majority of mortalities. Chronic and acute effects are responsible for approximately 73% and 27% of total mortalities, respectively, for both simulations. $PM_{2.5}$ concentrations account for most of the chronic mortalities, whereas NO_2 and O_3 concentrations account for most of the acute mortalities.

Incremental mortality risks are associated with chronic exposure to $PM_{2.5}$ (460 premature mortalities) and summer O_3 (55 premature mortalities) and acute exposure to NO_2 (230 premature mortalities) and CO (1 premature mortality). On-road and off-road diesel emissions are associated with a notable increase in chronic exposure respiratory mortality compared with on-road diesel emissions alone (9 avoided mortalities for on-road diesel emissions compared with 55 mortalities for on-road and off-road diesel emissions; Table 6). On-road and off-road diesel emissions are associated with a decrease in acute O_3 exposure (33 avoided premature mortalities). Overall, on-road and off-road diesel emissions are associated with O_3-related exposure mortalities (acute and chronic combined). This is in contrast to on-road diesel emissions, which were linked to reductions in O_3-related premature mortalities.

The total economic value of the health outcomes for on-road and off-road diesel emissions is approximately $5.5 billion (2.5th and 97.5th percentiles equal $2.5 billion and $12 billion, respectively), largely due to premature mortalities (valued at $5.1 billion). On-road and off-road diesel emissions are also associated with considerable costs related to morbidity outcomes: acute respiratory symptom days ($2 300 000), adult chronic bronchitis cases ($280 000 000), asthma symptom days ($12 000 000) and restricted activity days ($61 000 000).

The greatest impacts are located in the more populated provinces and CDs. On a provincial basis, the greatest premature mortality impacts for all pollutants associated with diesel emissions are observed in Ontario (Table 7), whereas $PM_{2.5}$ and NO_2 impacts are similarly high in Ontario and Quebec. For summer O_3 chronic respiratory exposure mortalities, on-road and off-road diesel emissions increase counts in Alberta, Ontario and Saskatchewan (24, 22 and 7 premature mortalities, respectively), but show benefits in Quebec (4 avoided premature mortalities). For acute O_3 exposure mortalities, benefits are observed in Quebec, Ontario and British Columbia (37, 12 and 11 avoided premature mortalities, respectively), whereas impacts are observed in Alberta and Saskatchewan (15 and 6 premature mortalities, respectively). Approximately 28% of the Canadian population is included in 13 CDs that are associated with a decrease in annual O_3 concentrations. In contrast to on-road diesel emissions, on-road and off-road diesel emissions increase O_3 concentrations in the province of Alberta (i.e. additional health impacts). On a CD basis, on-road and off-road diesel emissions are associated with the highest premature mortality counts for $PM_{2.5}$ and NO_2 in Montréal, for O_3 in Calgary and for all pollutants in Toronto. On-road and off-road diesel emissions are associated with the most avoided O_3-related premature mortality counts in Montréal.

8.1.3 Comparison between the AQBAT simulations

On average across Canada, the combined pollutants associated with on-road and off-road diesel emissions are associated with 2.2 times more premature mortalities than on-road diesel emissions

alone. This ratio suggests that off-road and on-road diesel emissions contribute equally to population health effects or that off-road diesel emissions contribute slightly more than on-road diesel emissions. The results for $PM_{2.5}$-related health impacts lead to similar observations (average ratio of 1.9 for all mortalities across Canada), whereas NO_2-related health effects differ less between simulations (average ratio of 1.5 for all mortalities across Canada). A comparison of health impacts from on-road and off-road diesel emissions with those for on-road diesel emissions only (Table 6) reveals that $PM_{2.5}$-related impacts approximately double (e.g. chronic exposure COPD mortality and child acute bronchitis episodes), whereas impacts linked to both $PM_{2.5}$ and summer O_3 approximately triple (e.g. asthma symptom days and respiratory hospital admissions). For endpoints associated with summer O_3 only, on-road diesel emissions are linked to benefits, whereas on-road and off-road diesel emissions are associated with disbenefits (e.g. chronic exposure respiratory mortality and minor restricted activity days). As O_3 is a secondary atmospheric pollutant that is influenced by precursors emitted in DE, non-linear effects are not unexpected.

Off-road diesel emissions have a greater influence on health outcomes relative to on-road diesel emissions in Alberta and Saskatchewan, as reflected by ratios for all pollutants of 4.2 and 2.9, respectively, between simulations (Table 7). This concurs with the higher off-road vehicle populations in Alberta and Saskatchewan. The low values modelled for the territories and Atlantic provinces do not allow comparative analyses.

The maximum differences in premature mortality counts between the two scenarios are observed in large urban centres, such as Toronto and Montréal. The large apparent impacts of off-road emissions are in large urban centres, owing to the confluence of high emissions and high population density. The atmospheric transport of off-road diesel source emissions that are released upwind of urban centres (e.g. in agricultural or mining areas) can also contribute significantly to urban air pollution. Off-road diesel emissions have relatively more influence in Calgary and Edmonton than in other urban centres, highlighting the importance of the off-road diesel fleet in those areas.

In all provinces and territories, on-road and off-road diesel emissions are associated with higher O_3-related premature mortalities or lower avoided premature mortalities than on-road diesel emissions alone. Differences in the O_3-related effects between the scenarios are concentrated and highest in urban centres, such as Montréal, Toronto, Calgary and Edmonton. The combination of on-road and off-road diesel emissions is associated with increased summer O_3 levels in several Canadian urban centres, except in the Montréal and Toronto areas. Although the exact causes of the differences among urban centres are unclear, it is likely that the importance of the gasoline fleet, the nature of industrial activities and environmental factors influence the resulting contribution from diesel emissions. Caution must be exercised when interpreting the results, owing to the non-linearity of O_3 formation. The increase in premature mortalities and other health endpoints estimated for on-road and off-road diesel emissions in comparison with on-road diesel emissions cannot be attributed to off-road diesel emissions alone. The estimates represent the influence from on-road and off-road emissions combined.

8.2 Discussion

The results suggest that off-road and on-road diesel emissions, individually, have a similar influence on health impacts. This comparison includes inherent uncertainties, as outlined previously. The $PM_{2.5}$ results suggest that on-road and off-road diesel emissions have roughly equivalent impacts on population health, although off-road diesel $PM_{2.5}$ emissions are much higher than on-road diesel $PM_{2.5}$ emissions (see Table 4). However, the populations exposed to off-road emissions are likely smaller (a

large fraction of off-road emissions being allocated to rural and less populated areas) than those exposed to on-road diesel emissions (mainly in urban centres), which decreases the magnitude of the effect on population health on a per emission basis. Further, as secondary $PM_{2.5}$ formation is not linear, it is not possible to determine the contribution from off-road diesel emissions to $PM_{2.5}$-associated health outcomes with high certainty without additional modelling runs. Similar limitations must be considered when interpreting the NO_2 results, as NO_2 emissions are involved in photochemical reactions leading to the formation of both O_3 and $PM_{2.5}$.

Concentrations of O_3 are dependent on precursor emissions from all sources and are sensitive to photochemical conditions and regimes (e.g. relative ambient concentrations of NOx and VOCs). Comparing the two AQBAT runs does not provide a clear indication of the effects associated with off-road diesel emissions, as the difference ignores the influence of non-linear photochemical reactions that affect ambient concentrations of several pollutants, including O_3 and secondary $PM_{2.5}$. For example, as shown in Table 7, on-road diesel emissions are associated with 77 avoided O_3-related premature mortalities, whereas on-road and off-road diesel emissions are linked to 21 additional O_3-related premature mortalities. It would be misleading to conclude, based on these results, that off-road diesel emissions, via their impact on O_3 levels, are responsible for 98 premature mortalities. In fact, the 21 additional premature mortalities reflect how a change in emissions equivalent to those from on-road and off-road diesel applications affects ambient O_3 concentrations. Although on-road diesel emissions appear more beneficial than off-road diesel emissions in terms of O_3-related health effects, it is more likely that adding off-road emissions into the equation affects the NOx/O_3 equilibrium that is responsible for the avoided premature mortalities with on-road diesel emissions.

The magnitude of health impacts varies regionally. In general, health effects associated with diesel emissions are expected to be larger in more populated CDs (e.g. Montréal, Toronto and Greater Vancouver) and in areas where diesel activity is more important (e.g. Alberta oil sands region), unless other significant variables exist. Additional factors that may influence the human health impact estimates include the relative importance of diesel emissions in comparison with emissions from other sectors, such as industry, gasoline and open sources. For example, diesel emissions in an area dominated by industrial emissions may not have the same impact as diesel emissions on a diesel-dominated roadway. Further, the impact of DE emissions may also depend on background air concentrations, which are influenced in part by long-range transport of pollutants, local or regional sources of emissions (e.g. industrial and institutional facilities, power production) and meteorology.

9 Conclusions

The air quality scenarios modelled with AURAMS and the AQBAT were selected in order to provide an indication of potential air quality and health impacts associated with diesel fuel use in on-road and off-road applications in Canada. On-road and off-road diesel applications are responsible for substantial levels of pollutant emissions. Compared with other mobile sources, diesel vehicles and engines contribute significantly to NO_2 and $PM_{2.5}$ emissions, whereas gasoline mobile sources contribute the majority of CO and VOC emissions. Diesel source emissions are notably important in large urban areas, such as Greater Vancouver, Toronto and Montréal, where a large fraction of the Canadian population resides. Diesel emissions are also important along major trucking routes and roadways connecting major cities (e.g. Windsor–Québec corridor), as well as in agricultural and mining areas (e.g. Alberta). The characteristics of the mobile fleet and the dominating economic sectors in a particular region determine the influence of diesel emissions. The concentration of diesel emissions in specific geographic areas leads to distinct air quality impacts across Canada.

Diesel emissions are estimated to contribute significantly to ambient concentrations of NO_2, $PM_{2.5}$ and O_3. The air quality modelling results show that on-road diesel emissions contribute significantly to air pollutant concentrations in urban and economically active areas and along major transportation routes. Off-road diesel emissions, which are more widely distributed than on-road diesel emissions, affect air quality in both rural and urban areas. The combination of on-road and off-road emissions leads to greater air quality impacts in the largest Canadian urban centres, notably Greater Vancouver, Edmonton, Calgary, Winnipeg, Toronto and Montréal. Off-road diesel emissions also have a relatively large impact in less developed areas characterized by few other sources of pollutant emissions (e.g. remote mining communities).

Based on the current health impact analysis, on-road and off-road diesel emissions result in significant and substantial population health impacts and societal costs in Canada via the contribution of DE to ambient concentrations of criteria air contaminants. The modelling undertaken estimates that on-road diesel emissions are associated with 320 premature mortalities for 2015 (valued at $2.3 billion), with 65% and 35% of the estimated mortalities attributable to $PM_{2.5}$ and NO_2, respectively. On-road and off-road diesel emissions are associated with 710 premature mortalities (valued at $5.1 billion), with 65%, 32% and 3% of the estimated mortalities being attributable to $PM_{2.5}$, NO_2 and O_3, respectively. Diesel emissions are also associated with significant numbers of acute respiratory symptom days, restricted activity days, asthma symptom days, hospital admissions, emergency room visits, child acute bronchitis episodes and adult chronic bronchitis cases across Canada. Results from the AQBAT simulations for the current assessment suggest that on-road and off-road emissions each contributed approximately equally to health impacts. The results also indicate that both on-road and off-road diesel applications have significant health impacts in major Canadian urban centres. Diesel emissions have higher health impacts in the most populated provinces, such as British Columbia, Alberta, Ontario and Quebec, and in the most populated CDs, which correspond to the Greater Vancouver, Calgary, Winnipeg, Toronto and Montréal areas. The greatest air quality impacts are also observed in those areas. Overall, it is concluded that efforts should continue to further reduce emissions of DE in Canada, particularly in areas with large populations.

10 Key uncertainties and gaps

Estimating the population health impacts associated with a fuel use scenario is the final step in a sequential process that is preceded by an evaluation of mobile source emissions and the impact of these emissions on air quality. The modelling conducted in the current assessment made use of the best available tools and data for Canadian scenarios. For practical reasons, assumptions were required at each step, which introduced uncertainties in the analysis. Estimates of health effects from on-road and off-road diesel emissions were also influenced by the uncertainties in each of the previous steps in the analysis (i.e. emissions and air quality modelling).

Limitations and uncertainties in estimating future emissions from mobile and other sources include modelling assumptions and simplifications. Uncertainty in modelling or imperfect spatial and temporal predictions arise for a variety of reasons, such as simplified algorithms, limited vehicle fleet data and errors in forecasting future economic activity. Additional uncertainties are associated with the use of three distinct models to estimate emissions from different segments of the mobile source population. Although these models share comparable methodologies, they can provide different results for air pollutants of interest. To assess some of the limitations, modelling data are analyzed in parallel to available air quality, meteorological, and engine or vehicle emission testing data to provide some perspective for emission modelling results and to corroborate results with as many sources of

information as possible. This is important, as assumptions made during the estimation of mobile source emissions are reflected in subsequent stages of the analysis (e.g. air quality modelling) and may affect the final conclusions.

Air quality models aim to accurately reproduce atmospheric processes that influence the fate and transport of pollutants. Nonetheless, the use of approximations is required for practical purposes, and the approximations used must be carefully considered when interpreting the results. Examples of modelling limitations and uncertainties include the quality of the meteorological and emission data, the horizontal grid resolution, the precision of spatial surrogates and the accuracy of the numerous and complex atmospheric reactions.

For example, the impact of diesel emissions on air quality is estimated for each 22.5 km grid cell, which cannot accurately represent the very local and likely higher impacts expected near major roadways. In addition, a limited number of pollutants are explicitly addressed in AURAMS. As a result, the contribution of diesel emissions to local levels of several pollutants of interest, such as UFP or individual air toxics (e.g. PAHs), is not estimated. Spatial surrogates are used to allocate pollutant emissions in different geographic locations across Canada, especially for non-point sources such as mobile source emissions. Although road network, traffic and demographic data, for example, are considered suitable proxies for part of the mobile fleet, there are limitations to this method.

The interpretation of the results was also limited by the fact that compensating effects were indistinguishable with the sensitivity analysis approach that was selected. For example, a reduction in on-road diesel NOx emissions affects the rate of O_3 formation per quantity of NOx released from other sources, such as off-road diesel emissions. Hence, NOx emissions from other sources could be producing O_3 more efficiently compared with the reference scenario, and this would lead to a mischaracterization of the contribution from on-road diesel emissions to air pollutant concentrations. This is one reason why the contribution from off-road diesel emissions to air pollutant concentrations cannot be determined simply by comparing the on-road with the on-road and off-road diesel use scenarios. The method shows only the absolute or marginal change in pollutants associated with a variation in emissions. Lastly, air quality models do not include algorithms to reflect all atmospheric reactions. This is especially important for secondary pollutants, such as secondary aerosols, secondary VOCs and O_3, that originate from atmospheric reactions. As a result of this limitation, mischaracterization of secondary pollutants is a possibility.

The quantitative health impact analysis is based on differences in ambient concentrations of $PM_{2.5}$, O_3, NO_2, CO and SO_2 and established CRFs for morbidity and mortality outcomes associated with these pollutants. Although the CRFs are derived from peer-reviewed sources and are endorsed by Health Canada, they include some inherent uncertainties, which are reflected in the AQBAT output as distributions for the health outcomes. The AQBAT includes a limited number of CRFs related to criteria air contaminants. Although other health outcomes associated with exposure to these pollutants (e.g. reproductive and developmental outcomes) are reported in the literature, not all health effects of air pollution are adequately quantified to be included in the analysis. As the AQBAT model does not include all pollutants, such as VOCs, the health and economic impacts associated with on-road and off-road diesel emissions are likely underestimated. As more robust pollutant-specific health data become available, the AQBAT will improve with the addition of more specific CRFs.

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