THE HEALTH CO-BENEFITS OF A CLEAN GROWTH FUTURE:

IDENTIFYING HEALTH CO-BENEFITS ASSOCIATED WITH LOW-CARBON INFRASTRUCTURE PROJECTS IN THE BUILDINGS, ENERGY AND TRANSPORT SECTORS

SEPTEMBER 2021

Smart Prosperity Institute

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September 2021

EXECUTIVE SUMMARY

Industry activities and infrastructure systems in the transportation, energy, and buildings sectors produce a variety of greenhouse gas emissions (GHG) and air pollutants that can lead to a wide range of adverse health impacts and contribute to climate change. Projects with the potential to reduce emissions of GHGs and pollutants can offer health benefits in the communities where they are implemented due to the reduction of emissions of these pollutants. Developing a more fulsome understanding of the co-benefits that could emerge from low-carbon projects is important to support investment decisions, and can further the economic case for low-carbon infrastructure projects.

This report serves as a resource for energy and climate policymakers to reference in order to better understand and identify the health co-benefits associated with low-carbon infrastructure projects in the transportation, energy and buildings sectors. Developed through a systematic literature review of recent scholarly research, this report identifies potential health impacts of exposure to pollution from these three emitting sectors.

This report identifies several environmental health benefits related to reducing pollution. Some pollutants have scientifically established relationships with the onset of diseases, and even mortality. Others are classified as risk factors, but the extent to which they impact human health is yet to be fully determined. Findings in this report are presented in decreasing order of scholarly consensus on the pollutants that have adverse health impacts. Pollutants with the most compelling evidence of adversely impacting health, as identified by scientific research, are presented first, followed by pollutants whose health impacts are less clear or require significantly more investigation.

There are four pollutants with strong evidence to adverse health impacts:

- Particulate matter 2.5/10;
- Nitrogen oxides (notably nitrogen dioxide);
- Sulphur oxides (notably sulphur dioxide); and,
- Ozone.

Particulate matter and ozone are non-threshold pollutants, meaning that there is no safe level of exposure. There is established evidence that any level of exposure to either of these pollutants offers considerable health risk, threatening the onset of cardiovascular and respiratory diseases, and an increased risk of mortality. Similar arguments can also be made about nitrogen dioxide and sulphur dioxide, two gases for which scholarship has established clear negative health associations. Overall, each pollutant above has strong links to adverse health impacts on the cardiovascular system, respiratory system, neurological system and a host of others.

This report also identifies four pollutants where evidence is inconclusive about health impacts, but identifies that they may be risk factors that contribute to worsening health. These four pollutants are:

- Carbon monoxide;
- Noise pollution;
- Persistent organic pollutants (POPs); and,
- Volatile organic compounds (VOCs).

Research evidence on their adverse health effects on different biological systems is inconclusive, or is open to dispute. Available evidence is outlined by biological system, identifying where studies have shown health benefits associated with reductions in pollutants that emerge as a result of implementing low-carbon infrastructure projects.

Overall, this research identified a number of key takeaways on how reductions in these pollutants can benefit human health:

- **The most prominent and strongly established evidence of health benefits from reducing air pollutants, which can be accomplished by implementing low-carbon infrastructure, are the significant positive impacts on cardiovascular and respiratory systems.** Across all pollutants where strong and mixed evidence exists, the incidence and severity of pollutant-related disease on these two systems is strong.

- **Reductions in air pollution can decrease incidence of all-cause and specific disease-related mortality.** Researchers have only begun to fully comprehend the extent to which air pollution is a causal factor of mortality. Some recent estimates suggest that fossil fuel combustion emits air pollutants that are linked to 10.2 million global premature deaths annually (Vohra, 2021). The argument that reducing air pollution has great potential to reduce pollutant-related deaths is backed by compelling scientific evidence.

- **It is likely that the implementation of low-carbon infrastructure may lead to even broader health benefits than previously thought.** Low-carbon infrastructure can reduce a wide range of pollutants. However, the full range of co-benefits originating from reductions in POPs as a result of low-carbon infrastructure projects in these three sectors is yet to be extensively investigated by research. This could further substantiate the health case for these investments.

- **More research is needed to identify how noise pollution impact biological systems, and how they can be reduced through low-carbon infrastructure projects.** Scholarly research have established robust evidence on the impact of many air pollutants on human health, and the co-benefits associated with reducing air pollution. However, more research is needed into other environmental health pollutants such as noise to improve the understanding of their health impacts, and better identify the causal relationship between a given pollutant and its effects on different organ systems.

Overall, this report considers only a sub-set of the overall health benefits that could accompany projects. Other health considerations emerging from the life-cycle impacts of disposal, how projects impact health equity, or those emerging from natural and nature-based solutions are not discussed in this report.

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LIST OF ABBREVIATIONS

ADHD	Attention Deficit Hyperactivity Disorder
CCOHS	Canadian Centre for Occupational Health and Safety
CO	Carbon Monoxide
COPD	Chronic Obstructive Pulmonary Disease
CVD	Cardiovascular Disease
DALY	Disability-Adjusted Life Year
EV	Electrical Vehicles
GHG	Greenhouse Gasses
NO	Nitric Oxide
NOx	Nitrogen Oxides
NO_2	Nitrogen Dioxide
PCB	Polychlorinated Biphenyls
PM	Particulate Matter
POP	Persistent Organic Pollutants
SO	Sulphur Oxide
SOX	Sulphur Oxides
SO_2	Sulphur Dioxide
TB	Tuberculosis
UHI	Urban Heat Island
VOC	Volatile Organic Compounds

INTRODUCTION

Canada's transition to net-zero emissions in the coming decades will offer a transformational change of direction for the country. Reaching Canada's 2030 greenhouse gas (GHG) emissions reduction targets will likely require $90 - $166 billion of investment in low-carbon infrastructure and technologies within the coming decade[1]. Every sector will need to reduce GHG emissions, especially the buildings, transportation and electricity infrastructure responsible for 47% of Canadian GHG emissions in 2018[2]. These three sectors are prominent because they are infrastructure and services present in every community across Canada, whose benefits are directly felt by all who drive or take public transit, live and work in buildings, and light and heat their homes.

Investments in low-carbon infrastructure in these three sectors offer opportunities to maintain these services and reduce greenhouse gas emissions. However, there are also a host of co-benefits that accompany these low-carbon projects. They are frequently discussed in climate policy circles as valuable and important, but have not historically been viewed as determinants when developing policies whose primary objective is to reduce GHG emissions[3]. Given Canada's ambitious climate targets, there is a need to better understand what co-benefits accompany specific projects to better identify the implications of a community investing in a given emissions reduction project versus another.

Identifying the co-benefits associated with different types of low-carbon projects offers an opportunity to meaningfully contribute to discussions around which projects may offer the greatest benefits within a given community.

One set of co-benefits that merits further consideration in decision-making are the co-benefits[4] to human health that emerge as a result of investments in low-carbon infrastructure

within the buildings, transportation, and energy sectors. As an example, modelling of car and vehicle fleet electrification has identified co-benefits from reductions in air pollution emissions that include decreases in premature mortality[5]. Identifying the co-benefits associated with different types of low-carbon projects offers an opportunity to meaningfully contribute to discussions around which projects may offer the greatest benefits within a given community. Incorporating this understanding can also help decision-makers in energy and climate policy advocate for a wider range of positive impacts that could accompany the projects they champion, adding a health lens to existing arguments of positive economic and environmental benefits.

This report synthesizes recent scholarship on a key set of benefits to human health from low-carbon infrastructure projects in the buildings, energy and transportation sectors. It is designed to be an easy-to-reference resource for energy and climate policymakers and decision-makers to better understand some of the potential health benefits that might accompany low-carbon infrastructure. By outlining how the reduction of environmental pollutants typically associated with carbon-intensive infrastructure projects is linked to health benefits, this report helps Candian policymakers identify a broader suite of advantages that accompany projects and better advocate for projects that help the economy, the environment, and benefit human health.

WHAT ARE HEALTH CO-BENEFITS?

Climate change has been called "the greatest threat to health of the 21st century", and its current and potential negative health impacts for Canadians are well-documented[6]. Health Canada[7] has identified six categories of negative impacts on health and well-being related to climate change[8]:

- Temperature-related morbidity and mortality that increase rates of heat and cold-related illnesses, occupational risks and rates of respiratory and cardiovascular disease;
- Weather-related natural hazards resulting in increased rates of social and mental stress, population displacement, and damaging public infrastructure;
- Reduced air quality, and increased exposure to air pollutants and allergens, can increase rates of respiratory disease and cardiovascular disease;
- Water-and-food-borne contamination can lead to intestinal illnesses and disorders;
- Higher exposure to ultraviolet rays can increase rates of skin damage, skin cancer and disturb immune function; and,
- Increased rates of vector-borne and zoonotic diseases as activity patterns of disease vectors such as mosquitoes, ticks and other animals change.

Due to the health-related risks climate change poses to individuals and communities, it is commonly understood that projects that adapt to climate impacts or reduce GHG emissions can also produce health benefits. If health benefits are not the primary objective of a policy, they can be considered co-benefits, defined here as the positive spillover effects associated with a particular action or measure aimed at accomplishing a goal. In the

case of health co-benefits, these are the ancillary positive health effects that result from policies, projects, or programs aimed at reducing GHG emissions, supporting greater environmental conservation, or supporting cleaner economic growth.

There are several health co-benefits associated with reducing greenhouse gas emissions, and they vary in scale and scope, contingent on time horizon and the geographic scale of impacts. Many of the health co-benefits associated with climate policies, identified in scholarly literature, include potential reductions in the severity or incidence of the adverse health impacts of a changing climate outlined above[9]. Others include reductions in the release of pollutants into the environment[10], potential reductions in disparities in health inequity between individuals[11], and improvements in mental health benefits across communities[12]. Identifying the health co-benefits that accompany projects can focus on any of these impacts, although this report examines only a subset of these potential co-benefits.

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WHICH HEALTH CO-BENEFITS ARE DISCUSSED IN THIS REPORT?

This report focuses on human health co-benefits associated with the reduction of pollutants, particularly due to the implementation of low-carbon infrastructure in the transportation, building, and energy sectors. This discussion is not to be thought of in terms of quantified estimates or savings associated with public health outcomes, but rather as how human health can improve by reducing pollution through the deployment of low-carbon infrastructure and technology. Such approach allows for a discussion that is sensitive to how individuals may have their health adversely impacted by pollution, and how interventions in the form of low-carbon infrastructure can have positive health impacts.

Projects and initiatives can improve community health, but to fully appreciate the scope of this impact, it is necessary to undertake a holistic assessment of the determinants of health, which takes into account individual, environmental, socioeconomic, and lifestyle factors[13]. These factors influence health outcomes, and they vary across communities, depending on local features such as neighbourhood design, transportation network use patterns, existing housing stock, and food systems, to name just a few[14]. These varying and interacting factors result in differential health experiences and disparities in health outcomes between individuals and communities. As such, the assessment of the health impacts of a given project is complex. It is difficult to establish credible associations without accounting for each

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community's specific context. This report does not replace this important assessment. It, however, allows an initial understanding of the potential of low-carbon infrastructure to improve community health.

It is important to note that this focus on how low-carbon projects may impact human health does not necessarily integrate a discussion of the life-cycle impacts from technology disposal that will vary based on waste management techniques. Given the variability of manufacturing processes, adopted waste management practices, and newly emerging scientific research on the topic, it is difficult to credibly connect a given health benefit to an entire infrastructure category or technology without accounting for this heterogeneity in technology design. These questions may be addressed in future work.

This report includes a discussion on the adverse health impacts of air, water, and noise pollutants. The decision to focus on these three types of pollutants was informed by Environment and Climate Change Canada's identification of criteria air contaminants (ammonia was excluded from this report because it is largely emitted from agricultural practices and less from urban settings) and a preliminary scan that raised compelling evidence that persistent organic pollutants, and noise pollution could also be potentially alleviated through the implementation of low-carbon infrastructure[15]. Based on a systematic review of academic scholarship examining this suite of pollutants, the report presents and organizes a range of research evidence on the co-benefits to human health associated with pollution reductions that might occur as a result of low-carbon infrastructure projects.

Report outline and structure

This report identifies a range of research evidence on the association of health co-benefits with reductions of specific pollutants (air, water, and noise) that could occur as a result of the implementation of projects in the buildings, energy and transportation sectors. This report is organized around pollutants, rather than projects, because it is difficult to credibly say that a specific health benefit emerges from a particular project without accounting for a broader set of contextual factors. One is an inability to account for how a given project will displace and/or induce emissions and pollutants that occur as a result of a project. This is apparent in the case of battery electric vehicles. There is research evidence that some of the adverse health impacts emerging from the use of internal combustion engines are mitigated or reduced when substituted for battery electric vehicles. However, it is difficult to make credible claims that one project is "better" for human health than another without accounting for factors like the emissions intensity of the electricity generated to power the batteries, how frequently a given solution is used, regional air quality, and the impacts of a project on different members of the community[16].

A second reason why it is difficult to tie individual projects to health benefits is that examining a project in isolation offers little sense of scale of potential impact, or how impacts evolve over time. Adopting a single battery electric vehicle is unlikely to have a notable effect on ambient air quality in a given region. Additionally, health impacts depend on the duration of exposure to a particular pollutant. The health impacts of long-term and short-term exposures can differ drastically in scope and severity. For this reason, any technology or project-specific examples cited in this report draw from specific examples or case studies conducted around how a given project impacts health in a specific context.

This report aims to answer the following questions about each pollutant examined:

- What does recent research evidence suggest about the association between environmental pollutants and human health impacts?
- What are the primary health pathways linked to adverse health impacts?

This report organizes both pollutants and their health effects based on the strength of evidence as reported by the literature reviewed: the pollutants with reported compelling evidence of health associations are presented first, followed by the pollutants with reported weaker health associations, and finally concluding by identifying pollutants where research evidence is mixed, or in need of greater research. The report thus begins with particulate matter and ends with noise pollution. Additionally, within each pollutant profile, adverse health impacts on various biological systems (cardiovascular, respiratory, reproductive, etc.) are presented in declining order based on the strength of available evidence.

This report has organized pollutants into two overarching sub-sections: Pollutants with strong evidence of adverse health impacts, and pollutants with the potential to increase the risk of adverse health impacts. Although this is not a perfect division, since some pollutants cross boundaries depending on the nature and strength of evidence available on their impacts on a given system, this approach allows for a more in-depth examination of pollutants with more compelling evidence, and highlights where evidence of the strongest links to human health exist. It also serves to distinguish between pollutants based on strength of evidence, identifying the pollutants whose reduction is strongly

identified with improvements in health outcomes, as per the literature reviewed. All pollutants will feature an examination of their effects on a range of biological systems, and a summary of some of the health co-benefits identified in the literature that accompany reductions in a given pollutant.

This report includes the following sections:

Pollutants with strong evidence to adverse health impacts:

- Particulate matter (PM) 2.5 and 10;
- Nitrogen oxides, notably nitrogen dioxide;
- Sulphur oxides, notably sulphur dioxide;
- Ozone[17].

For pollutants where strong association was identified between pollutants reductions and health benefits, this document will also report on:

  - Which subsectors in the electricity, transport, and buildings sectors are responsible for the highest emissions of this pollutant in Canada?
  - What is the geographic distribution of this pollutant by province?
  - What are some of the health co-benefits, as identified by recent scientific research, that have been associated with reductions in this pollutant?

Pollutants that potentially increase the risk of adverse health impacts*:*

- Carbon monoxide;
- Persistent organic pollutants;
- Volatile organic compounds;
- Noise pollution.

A note on multi-pollutant modelling

This report identifies some of the health consequences associated with individual pollutants. However, the combustion process that generates emissions of many air pollutants discussed in this report often emits multiple pollutants at once that interact with each other within ambient air. Occurrences of multiple pollutants alongside each other can lead to the creation of secondary pollutants, or result in joint effects that have adverse health impacts[18]. This is the case for all pollutants within this report, and policymakers looking to better understand how interactions between pollutants in ambient air specifically, or within emissions, impact human health are encouraged to use a technique called multi-pollutant modelling. This technique allows for the development of a more holistic understanding of how ambient air quality influences health[19].

This report focuses on identifying the adverse health effects associated with individual pollutants to offer insight into how each single pollutant affects human health, which is valuable given the breadth of evidence available and need to better understand the full suite of health outcomes linked to reductions. Accounting for any of these individually will support the business case for investing in low-carbon infrastructure by ensuring health benefits can be more clearly understood and considered. However, given that projects will typically reduce more than one pollutant, policymakers seeking to accurately represent the health outcomes associated with reductions through processes such as a Health Impact Assessment should consider the combinatorial effects on human health of reducing more than a single pollutant through one policy or project[20].

POLLUTANTS WITH STRONG EVIDENCE TO ADVERSE HEALTH IMPACTS PARTICULATE MATTER

What is particulate matter?

Particulate matter (PM) is composed of small solid fragments or liquid matter of varying size, shape, and chemical composition[21]. The nomenclature defines PM according to the size of matter: PM_{10} is 10 micrometres (μm) or smaller and is referred to as coarse PM, while $PM_{2.5}$ is referred to as fine PM and is approximately 2.5 μm. While there are other types of PM[22], the evidence presented in this report primarily covers these two types. PM can either be emitted by natural or anthropogenic sources, or it can be formed secondarily through atmospheric chemical reactions involving nitrogen oxides, sulfates, and hydrocarbons in the presence of sunlight and water vapour[23]. $PM_{2.5}$ is also an important component of smog[24].

Particulate matter is both a primary (directly emitted as a result of activities) and secondary (formed as a result of reactions in

ambient air) pollutant. Emissions sources for PM in this report focus on where it is directly emitted, but health considerations are relevant for PM regardless of how it occurs in the atmosphere.

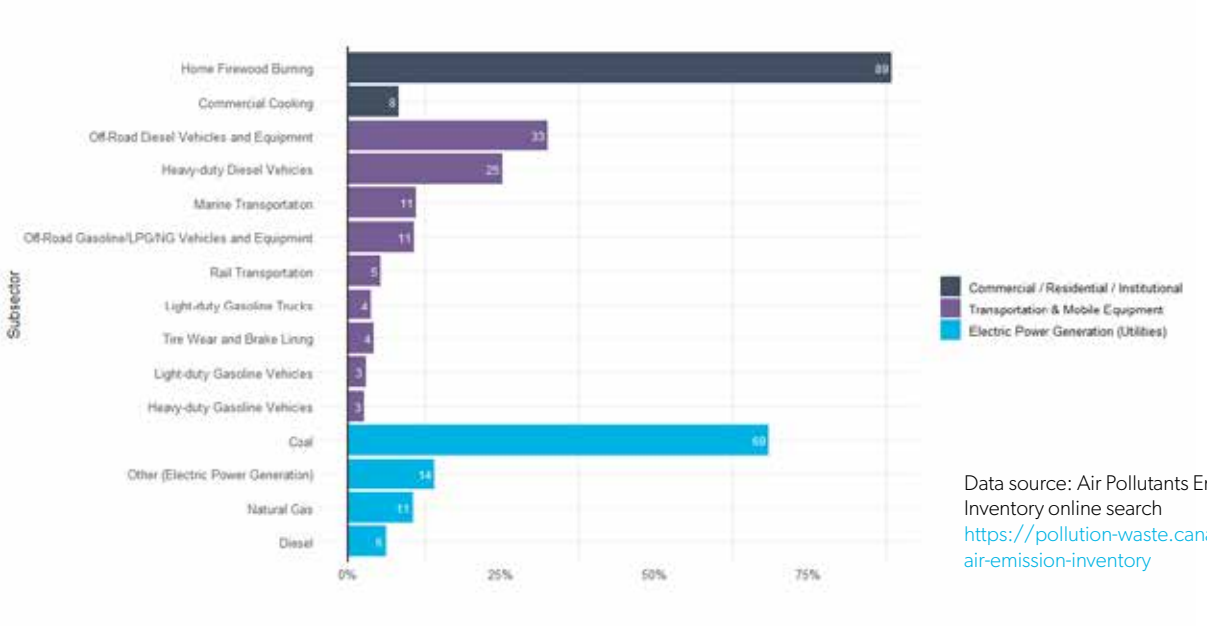
Sources of particulate matter

All forms of road transportation that use fossil fuels produce PM[25]. Pollution comes from vehicles producing diesel engine emissions, light-duty vehicles, and motor vehicle exhaust[26]. Emissions can also arise from re-suspended road dust, tire wear, or brake wear[27]. Concentrations of pollution tend to be higher near-road sites and in highly trafficked areas[28].

Electricity production from power plants or power stations for electricity or heat production generate large amounts of PM due to the burning of fossil fuels, including coal[29]. Coal combustion, compared to renewables or natural gas, produces higher rates of $PM_{2.5}$[30].

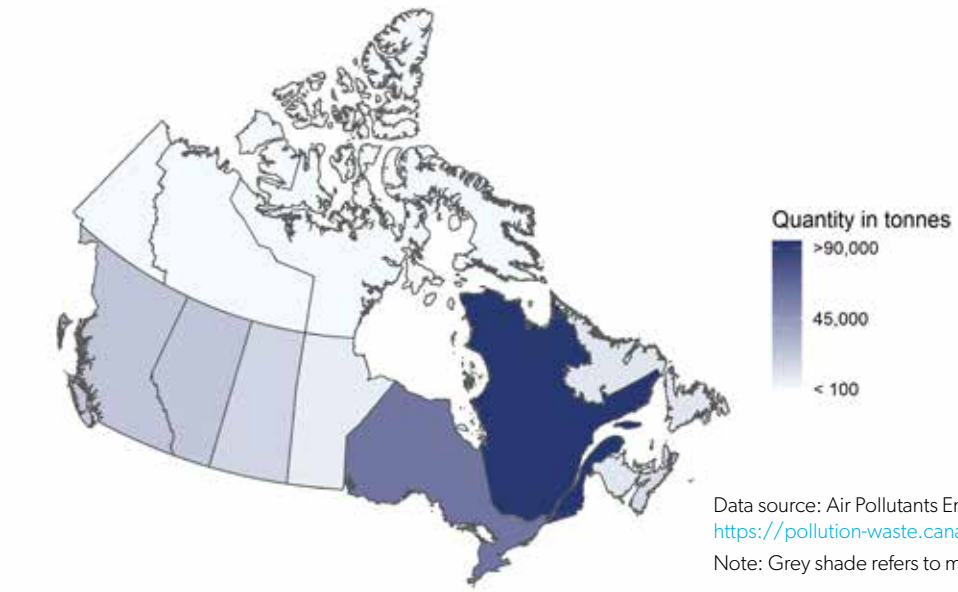
Industrial or residentials areas also account for PM. PM_{10} emissions can result from industrial and domestic fossil fuel combustion[31]. In residential areas, stoves and furnaces generate PM, with home heating being a key contributor[32]. Heavily polluting industries like coal mining and the chemical industry, or industrial processes are also sources of PM[33], although this report does not discuss industrial emissions.

Figure 1: The largest sub-sectoral contributions of particulate matter 2.5 across Canada in the buildings, electricity and transportation sectors; Canada-wide values for 2018



Data source: Air Pollutants Emissions Inventory online search https://pollution-waste.canada.ca/air-emission-inventory

Figure 2: The largest regional contributions of particulate matter across Canada in the buildings, electricity and transportation sectors; Values for 2018



Data source: Air Pollutants Emissions Inventory online search https://pollution-waste.canada.ca/air-emission-inventory.
Note: Grey shade refers to missing data.

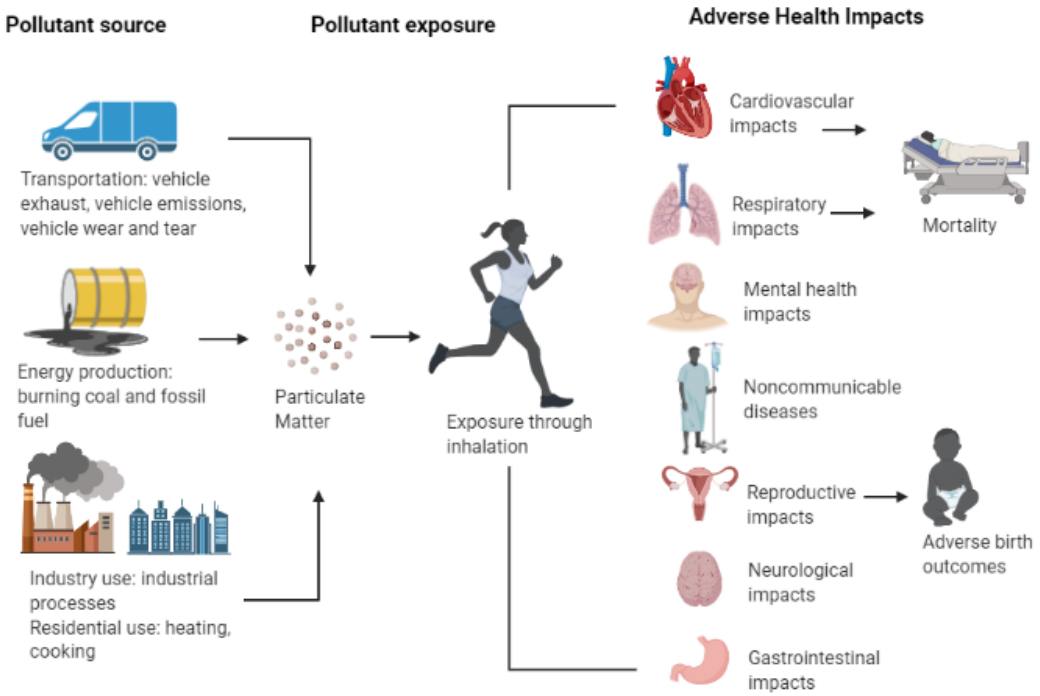
Primary health impacts

A common proxy to air pollution, PM is extremely harmful to human health at any level of exposure[34]. Particulate matter negatively impacts:

- Cardiovascular systems;
- Respiratory systems;
- Mortality;
- Non-communicable diseases;
- Neurological system;
- Mental health;
- Reproductive system;
- Gastrointestinal system.

Coal combustion, compared to renewables or natural gas, produces higher rates of PM2.5.

Figure 3: Pathways of particulate matter and some of the adverse health impacts it can cause.



Analysis of adverse health impacts

Cardiovascular system

Research evidence suggests that exposure to PM, even if independent of other pollutants[35], increases the risk of cardiovascular diseases, which potentially results in either morbidity and mortality[36]. $PM_{2.5}$ is strongly associated with hypertension, arrhythmias, myocardial infarction, congestive heart failure, and cardiovascular mortality[37]. PM is a non-threshold substance, meaning that there are no safe levels of exposure[38]. As such, short-term exposures increase the risk of heart failure, ischemic heart disease, cardiopulmonary morbidity, and acute myocardial infarction, which ultimately may lead to spikes in hospitalization[39]. Long-term exposure has been associated with atherosclerosis, hypertension, and ischemic heart disease[40]. Additionally, exposure to PM has been positively associated with increased risk for stroke, coronary events, unstable angina, and non-fatal heart attacks[41].

Biological pathways for cardiovascular disease include translocating matter into the bloodstream to induce clot formation, matter causing systemic inflammation or matter interfering with the central nervous system, all of which can manifest an array of cardiovascular diseases[42]. Overall, there is compelling evidence on the adverse health impacts of PM on the cardiovascular system.

Respiratory system

PM has serious impacts on human lungs. Exposure to $PM_{2.5}$ significantly increases the risk of chronic obstructive pulmonary disease (COPD)[43], which is ultimately associated with an increase in hospitalizations and COPD mortality[44]. Exposure to PM_{10} is observed to significantly decrease the quality of life for COPD patients and impair lung functionality, which may also lead to mortality[45]. Factors that stimulate inflammation, and the production of reactive oxidative molecules that can cause cellular damage, are possible pathways to COPD, although research is limited[46].

There is also compelling research linking exposure to PM to the development or exacerbation of asthma[47]. Higher concentrations of $PM_{2.5}$ and PM_{10} are associated with higher rates of asthma hospitalizations[48]. $PM_{2.5}$ and PM_{10} is also proportionally related to asthma symptom severity, contingent on factors such as time of day, level of exposure, and proximity of pollutant source[49]. Some research evidence suggests that PM-associated asthma and poorer lung functionality may be worse in children, even in low concentrations[50]. The respiratory health of children is of concern because of their immature lungs; they may be most susceptible to the effects of outdoor ambient pollution exposure as they spend time performing vigorous activities outdoors[51]. PM is also associated with cough, phlegm, wheezing, and shortness of breath[52]. Other respiratory diseases include impaired lung function, bronchitis, constricted airways, and lower respiratory infections, like tuberculosis[53].

Research evidence suggests that exposure to PM, even if independent of other pollutants, increases the risk of cardiovascular diseases, which potentially results in either morbidity and mortality.

PM particles access lungs by depositing in the bronchioles or alveoli to cause local inflammation and oxidative stress, or penetrating the alveolar regions and use the gas exchange area as an entry point into blood. PM_{10} is too large to penetrate deep into the respiratory tract and enter circulation, however, it is still associated with respiratory diseases and diseases in other organs[54]. A mechanism by which this may happen is through local inflammation which can lead to subsequent systemic inflammation[55]. Overall, PM is often strongly associated with adverse respiratory consequences.

Mortality

There is strong research evidence to suggest that PM contributes to premature death, excess mortality and all-cause mortality; overall, the pollutant is a significant risk factor for death[56]. Both $PM_{2.5}$ and PM_{10} are attributed to non-accidental deaths, increase of cardiovascular mortality, and increased risk of respiratory death[57].

For instance, the risk of ischemic heart disease and cardiovascular diseases increases from either excessive PM_{10} exposure or from regular inhalation of PM[58], which ultimately are positively associated with death. Additional research suggests that lung cancer, COPD, and reduced respiratory function as mortality risks are strongly associated with exposure to elevated levels of PM[59], although there is some debate around how significantly PM_{10} is associated with COPD-related mortality[60].

Length of exposure is an important factor in PM-related mortality. Short-term exposure has been related to increases in daily total mortality, increases to overall respiratory death, risk of mortality for people with chronic morbidities, and a possible association between PM_{10} and coronary heart disease mortality[61]. Chronic or long-term exposure is also associated with an increase in natural cause mortality[62].

Some emitting sources are related to PM-related mortality more frequently. Notably, several studies attribute mortality risk to $PM_{2.5}$ from transport emissions due to traffic density and diesel fuel combustion[63]. Overall, there is strong evidence to suggest the impacts of short-term and long-term exposure of $PM_{2.5}$ on mortality[64].

Noncommunicable diseases

Several jurisdictions classify $PM_{2.5}$ as a human carcinogen[65]. $PM_{2.5}$ and PM_{10} are associated with increased risk of cancer, most notably, lung cancer risk[66], although there is inconclusive evidence on the strength of correlation between the incidence of lung cancer and exposure to $PM_{2.5}$ and PM_{10}[67]. The most common types of cancers related to $PM_{2.5}$ emissions generally affect the lungs, trachea, and bronchi[68]. Several studies associate exposure to PM with primary lung cancers, lung adenocarcinoma, and an increased risk of lung cancer mortality[69]. A Canadian study provides evidence that different compounds in PM increases the risk of development of adenocarcinoma and small cell lung cancer[70]. For example, diesel and transition metals found in PM are associated with chronic cancer risk[71].

A number of studies explore the connection between type-2 diabetes, insulin resistance, and obesity in children with exposure to PM[72]. There is some evidence to suggest that PM induces inflammation, which impairs insulin responses, increasing the risk for diabetes[73].

Neurological system

Cognitive decline and impairment have been linked to PM exposure[74], although research evidence has been inconclusive about how PM impacts cognitive ability. Some studies argue that PM bears an indirect impact on cognitive functions while others describe a significant negative association[75]. PM has been noted as a risk factor for cognitive decline and dementia pathogenesis[76].

Emerging evidence suggests that $PM_{2.5}$ exposure during pregnancy has long-term impacts on nervous system development, including neurodevelopmental impairment, central nervous system or neuro-behavioural disorders in children, decreased sleep efficiency, altered sleep duration, and spatial memory dysfunction[77]. Some studies indicate that prenatal exposure to $PM_{2.5}$ is a risk factor for autism spectrum disorder[78].

Biological mechanisms have yet to be fully established, however, some research suggest that $PM_{2.5}$ may cause inflammation to the neurological system in a dose-dependent manner[79]. Overall, the evidence is inconclusive in demonstrating strong correlations and associations, but the evidence that links PM to cognitive decline and neurodevelopment impacts is compelling.

Mental health

Some studies explore the association between exposure to PM and mental health conditions such as psychosis morbidity (e.g., schizophrenia), anxiety, depression (e.g., major depressive disorder), suicidality, and attention deficit hyperactivity disorder (ADHD).

Although research evidence to connect psychosis, specifically schizophrenia, to PM exposure is limited[80], studies have established a significant association between PM, major depressive disorder and psychological distress[81]. The length of exposure is a significant factor that may exacerbate the mental health impact of PM[82]. Prenatal exposure has been linked to behaviour difficulties, but there is not enough evidence to solidify this hypothesis[83].

Some researchers have identified PM_{10} exposure as a risk factor for suicide, particularly in high concentrations. They also suggest that underlying physical or mental comorbidities increase the risk of suicide when people are exposed to PM pollution[84], but there is yet to be a biological understanding for this relationship.

While fine PM is noted to have "the most consistent statistically significant associations with mental health disorders"[85], the overall research evidence is inconclusive, and factors such as age, length of exposure, and underlying comorbidities impact the strength of association.

Reproductive system

The relationship between adverse birth outcomes and exposure to PM is generally unclear. Some studies have explored associations between PM and either pre-term birth or low birth weight; evidence for both of which is inconclusive, ranging from either inconsistent to positive and significant association[86]. Differences may arise due to varying factors such as exposure levels, the timeframe of exposure (e.g., trimester), and how the 'preterm' was defined for measurement[87]. Certain birth defects may have stronger relationships with PM. For example, some research provide statistically significant evidence that exposure to PM_{10} is associated with congenital cardiovascular defects, but the biological mechanisms for these adverse birth outcomes are yet to be elucidated[88]. In terms of the links between PM and the reproductive system, research evidence suggests that traffic-related pollution, including PM as a pollutant, may be connected to a reduction of fertility rates[89]. However, evidence is insufficient to suggest that PM has direct impacts on the reproductive system.

Gastrointestinal system

The effects of PM on the gastrointestinal system lacks conclusive evidence to propose a relationship. Some research postulates positive associations with colon cancer, Crohn's disease, and appendicitis. A biological explanation for the manifestation of appendicitis includes PM potentially triggering an inflammatory response in the body[90]. More research is required to identify the impacts of PM on the gastrointestinal system.

Core benefits of reducing particulate matter

Reducing PM emissions has a wide range of public health and air quality benefits[91]. Research provides extensive evidence that reducing PM is associated with avoided premature death[92] and increased life expectancy[93]. Some studies have even established that life expectancy increased by 7-9 months for every 10 $\mu g/m3$ concentration decrease of PM over 20 years[94]. Emission reductions could reduce cardiovascular deaths, especially PM_{10} decreases[95]. Cardiovascular complications like non-fatal heart attacks, stroke, acute myocardial infarction, stroke mortality, or ischemic heart disease mortality, and respiratory conditions such as asthma-related emergency room visits, COPD, COPD mortality and respiratory illnesses are also avoided following a decrease in PM through air pollution or emissions control[96].

Some case studies indicate that there is the potential to avoid premature births if PM concentrations are reduced. For instance, the closure of the Utah Valley Steel Mill (August 1986 – September 1987) led to a significant drop in PM_{10} pollution levels and a subsequent reduction in the likelihood of delivering prematurely[97]. PM reduction also brings great benefits for lung function in children, reduced risk of childhood asthma, and reduced acute respiratory inflammation[98]. Lung growth has also been demonstrated to be faster in children living in areas experiencing greater $PM_{2.5}$ and nitrogen dioxide (NO_2) decline[99]. Those with underlying chronic conditions face greater respiratory and cardiac emergency and hospital admissions, which aggravated by higher $PM_{2.5}$ concentrations in low-income neighbourhoods. A decline of PM levels in these neighbourhoods would therefore offer greater benefit in health outcomes[100].

Shifting to green mobility and a zero emissions scenario has been modelled to contribute to PM_{10} reduction and decrease incidence of myocardial infarctions and lung cancer cases[101]. Car and fleet electrification contributes to a decline in PM emissions, and subsequent health co-benefits[102]. Some modelling projections indicate that the adoption of electric vehicles (EV) decreases premature mortality; the greater the percentage of electrification in transport, the greater the decrease in mortality[103]. However, the benefits of $PM_{2.5}$ reduction with EV adoption vary by region, adoption rates, season, and the power generation sources used to charge EVs[104]. Hydrogen vehicles are also an option for realizing health co-benefits. Modelling demonstrates that 100% instantaneous replacement of on-road vehicles from

fossil fuel to hydrogen could result in between 3710 - 6350 avoided deaths per year due to $PM_{2.5}$ concentration changes[105].

Renewable energy projects also generate health co-benefits. Solar integration reduces air pollution, and modelling suggests that it may significantly avoid premature deaths[106]. There is less exposure to pollutants like particulate matter, sulphur dioxide, volatile organic compounds with energy efficiency of green buildings which reduce greenhouse gas emissions[107].

Investments in more energy efficient buildings are associated with several health benefits including avoided morbidity, acute bronchitis, emergency department visits for asthma, asthma exacerbation, upper and lower respiratory symptoms, non-fatal heart attacks, respiratory hospital admissions, and lost work days or restricted activity days[108]. A case study suggests that the removal of wood-heater use results in a decline in PM pollution, followed by a subsequent fall in cardiovascular and respiratory related deaths[109].

Nitrogen Oxides

What are nitrogen oxides?

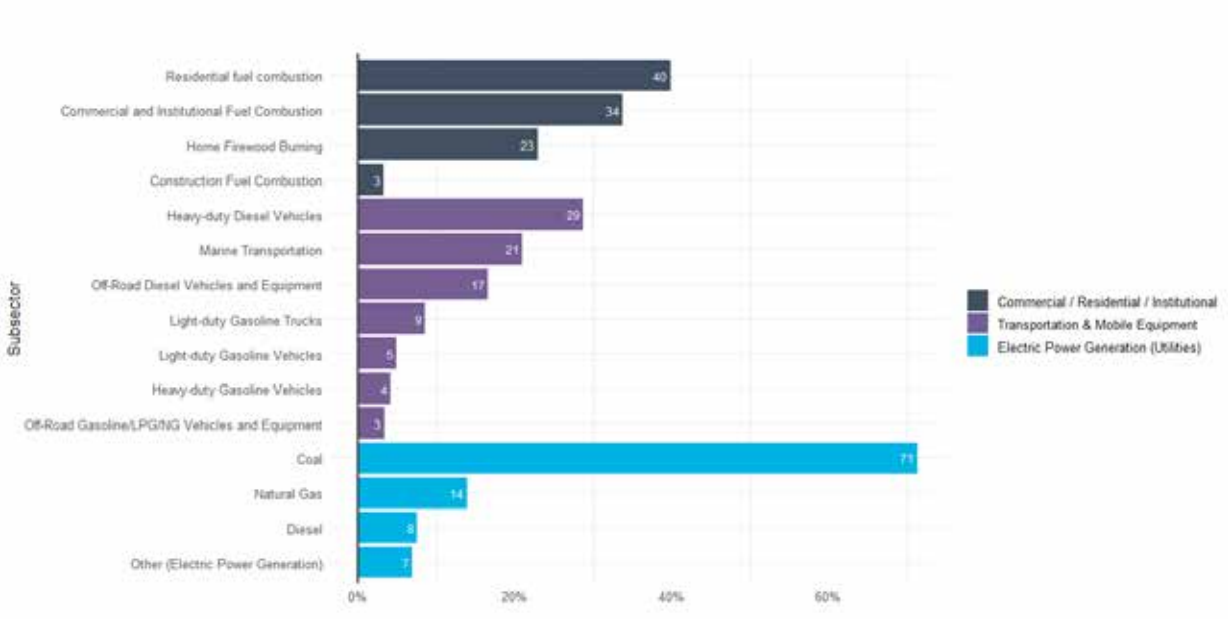
Nitrogen oxides (NOx) are a class of greenhouse gases, formed primarily from the liberation of nitrogen, that includes nitric oxide (NO) and nitrogen dioxide (NO_2). The former is a colourless gas not considered to be harmful to human health, but the latter is a brown, hazardous odour gas that can result in adverse health impacts[110]. During fuel combustion, nitrogen is emitted into the atmosphere, where it combines with oxygen producing nitrogen dioxide[111]. In Europe, for example, of all NOx emissions, 40% come from road transport, 21% from energy production and distribution, and 14% from commercial, institutional, and residential sources[112]. This report focuses mainly on NO_2 as it poses significant risks to human health. Nitrogen oxides are a primary emission source, but are not classified as a non-threshold pollutant. This means that adverse health impacts only occur once exposure occurs past a certain threshold.

Sources of nitrogen dioxide

The main emitters of NO_2 are transportation, electricity production, heat production, and home cooking[113]. Forms of road transportation that use fossil fuels generate NO_2, primarily through motor vehicle exhaust from gasoline and diesel-combustion[114]. In Canada, NO_2 emissions from diesel exhaust exceeded the annual ambient air quality standard in 2020, in urban areas both located near major roads and 150m away[115].

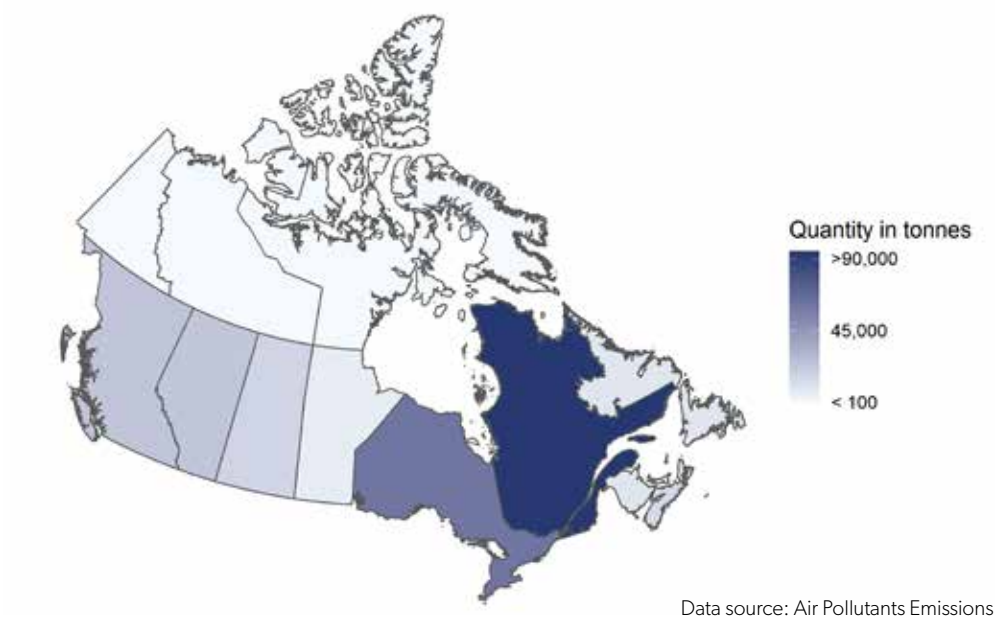
Electricity production, notably stationary emitter such as power plants, buildings and built-infrastructure assets that rely on fossil fuel combustion for electricity and heating, also generate NO_2[116]. Energy intensive sectors are important culprits for the emission of NO_2, especially the power, thermal production, and supply industry, the non-metallic mineral production industry, and the ferrous metal smelting industry[117].

Figure 4: The largest sub-sectoral contributions of nitrogen oxides across Canada in the buildings, electricity and transportation sectors; Canada-wide values for 2018



Data source: Air Pollutants Emissions Inventory online search https://pollution-waste.canada.ca/air-emission-inventory

Figure 5: The largest regional contributions of nitrogen oxides across Canada in the buildings, electricity and transportation sectors; Values for 2018



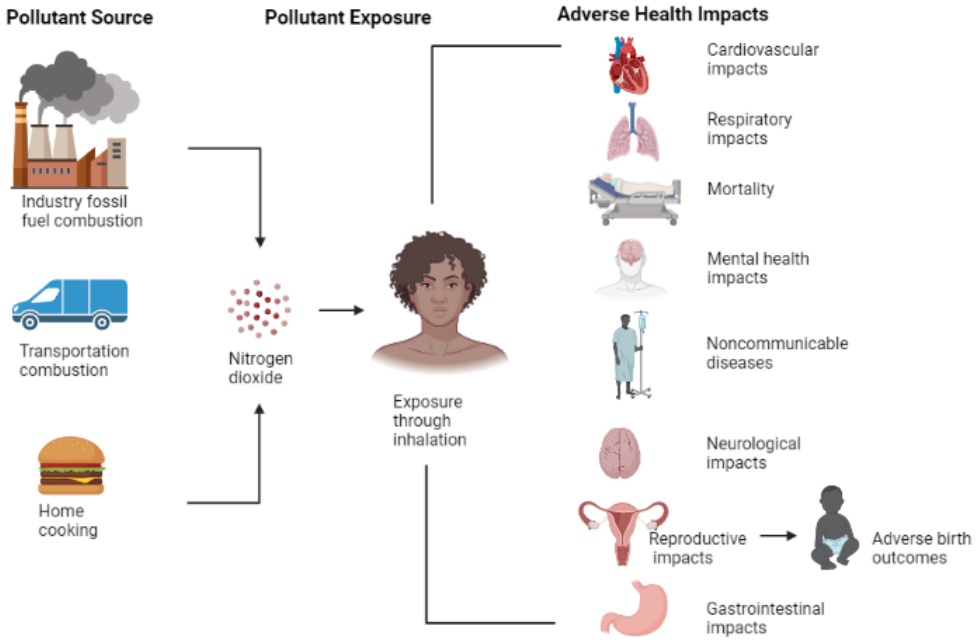
Data source: Air Pollutants Emissions Inventory online search https://pollution-waste.canada.ca/air-emission-inventory.
Note: Grey shade refers to missing data.

Primary health impacts

Nitrogen oxides, notably nitrogen dioxide, negatively affects:

- Cardiovascular systems;
- Respiratory systems;
- Mortality;
- Mental health;
- Non-communicable diseases;
- Neurological system;
- Reproductive system;
- Gastrointestinal system.

Figure 6: Pathways of nitrogen dioxide and some of its associated adverse health impacts



Analysis of adverse health impacts

Cardiovascular system

There is compelling research evidence that exposure to NO_2 is positively associated with cardiovascular disease and mortality[118]. NO_2 is associated with COPD, coronary heart disease, hypertension, and stroke[119]. Some studies have found that even shorter exposure to NO_2 increases the risk of heart failure, emergency hospitalizations, hypertension, and mortality[120]. Meanwhile, longer exposures may be related to stroke, ischemic heart disease, and cancers[121]. The typical pathway involves inhalation of this gaseous pollutant, which then circulates throughout the body through the bloodstream[122]. A potential downstream pathway is the increased risk of oxidative stress due to anoxia, the absence of oxygen, particularly in people with cardiovascular disease, which indirectly increases the risk of cardiovascular death[123].

It is important to note that although many studies claim a positive association between exposure to NO_2 and cardiovascular morbidity and mortality, others question the strength of this relationship between NO_2 and mortality. This is because the evidence of an association between NO_2 and adverse health impacts can be confounded by PM[124]. This means that, despite the abundance of evidence to prove the relationship between $PM_{2.5}$ and cardiovascular ailments and mortality, some scholars claim that the actual impacts of NO_2 are unclear because it is rarely collected independently of PM and ultrafine particles[125]. Others argue that NO_2 is a superior surrogate for vehicular combustion than PM or ultrafine particles, significantly generating greater risks for adverse health impacts[126].

Respiratory system

Both short-term and long-term exposures to NO_2 have been positively associated with multiple respiratory health consequences, such as chest tightness, asthma, wheezing, persistent cough and phlegm, allergic rhinitis, shortness of breath, emphysema, COPD, pneumonia, chronic bronchitis, cancers, and mortality[127]. Renewable energy projects that result in reduction of NO_2 emissions have shown to reduce the incidence of chronic bronchitis and fewer respiratory deaths[128]. Childhood exposure to NO_2 has been positively associated with reduced lung function as a result of stunted lung growth[129].

Many studies have found statistically significant evidence that associates ambient NO_2 with increased incidence of tuberculosis (TB), particularly in short-term exposure to NO_2[130]. NO_2 is recognized as a significant risk factor of active TB and both short-term and long-term exposures can result in higher rates of TB mortality[131]. However, some other studies argue that either the correlation between NO_2 exposure and TB is only true in multi-pollutant studies or is not statistically significant[132].

Short-term exposure to NO_2 is positively associated with increased risk of mortality from respiratory diseases[133]. Short-term also exacerbates existing respiratory ailments because the inhalation of NO_2 impairs the function of phagocytes, cells that protect the body from harmful foreign particles, contributing to airway inflammation and possible long-term respiratory symptoms and diseases[134].

Long-term exposure to ambient NO_2 is a risk factor to lung cancer and adenocarcinoma because NO_2 is not highly soluble, making it easy to get trapped in small airways of the lungs, increasing its potency and impact on the immune cells and inflammation[135]. Although there is a large quantity of evidence that support the positive association of NO_2 exposure and an increased risk of cancers, there are some sources that have not found substantial evidence to support this relationship[136]. Mixed evidence supports the claim that NO_2 is an oxidative stressor in the respiratory system, but it is not likely that NO_2 is solely responsible for the negative health impacts[137]. Research suggests that multipollutant exposure is often responsible for the most severe adverse respiratory health impacts[138].

Mortality

Research evidence suggests that exposure to NO_2 is positively associated with premature death, respiratory mortality, cardiovascular mortality, and cerebrovascular mortality[139]. Research indicates that air pollution from vehicular sources, including NO_2, should be considered a factor of increased mortality in urban regions of Canada[140]. Increases in NO_2 concentrations are also positively associated with a rise in hospital admissions and all-cause mortality[141]. All-cause mortality can be understood as all of the deaths that occur in a particular population regardless of cause[142]. Short-term and long-term exposure has been associated with increased cardiovascular mortality from different health conditions includingcardiovascular disease, ischemic heart disease, and circulatory system diseases[143]. Some researchers even claim that long-term exposure to NO_2 increases the risk of more devastating health impacts[144]. There is some mixed evidence correlating the impacts of NO_2 exposure with respiratory health[145]. Respiratory disease death and lung cancers do not have a significant statistically association with long-term or short-term NO_2 exposure[146]. However, other studies have found that short-term exposure to NO_2 is positively associated with respiratory-related mortality[147]. There is significant research evidence that cardiorespiratory-related mortality is robustly associated with short-term and long-term exposure to NO_2[148]. Long-term exposure to NO_2 is also positively associated with increased risk of cerebrovascular mortality[149].

Mental Health

The evidence of the relationship between exposure to NO_2 and mental health is inconclusive. Some researchers find limited statistical significance for this relationship, while others claim that increased levels of NO_2 are associated with the likelihood of developing a mental health disorder (e.g., depression, ADHD, conduct disorder, anxiety, suicide)[150]. Controlling for socioeconomic status, familial covariates, smoking, and childhood depressive symptoms, some researchers found that exposure to NO_2 has, among the air pollutants discussed

in this report, one of the most significant positive associations with psychological distress, depression, and anxiety[151]. Some sources explore a connection between suicide and exposure to NO_2, but the biological explanation of the positive association between NO_2 exposure and completed suicide is not yet clear[152]. However, some studies indicate that exposure to gaseous pollutants through inhalation can potentially trigger systemic neuronal inflammation and oxidative stress and induce anxiety and depressive-like behaviour[153]. Recent evidence indicates that NO_2 impacts cognitive function[154]. For instance, some scholars indicate that children who have been exposed to NO_2 have an increased risk of harmful behavioural problems and require psychiatric medication[155].

Non-communicable diseases

The evidence connecting exposure to NO_2 and the occurrence of non-communicable diseases is mixed. Long-term exposure to NO_2 has been connected to lung cancer, adenocarcinoma, and squamous cell carcinomas[156]. This is often related to close proximity to high traffic roads, where the concentration of NO_2 is significantly higher, resulting in an increased risk of cancer[157]. Exposure to NO_2 has also been associated with diabetes and irregular levels of glucose[158].

Transitioning to vehicles that are electric, powered by hydrogen, or hybrids has the capacity to substantially decrease the concentration of NO_2 in urban areas.

Neurological system

There is limited research that explores the relationship between NO_2 and the neurological system. However, researchers who have explored this topic have found a relationship between NO_2 exposure and the neurological system, particularly, a positive association with memory impairment, dementia, Parkinson's disease, Alzheimer's disease, and cerebrovascular mortality[159]. When NO_2 is inhaled, it triggers an inflammatory immune response, which is the common way pollutants, including NO_2, damage human organs[160]. NO_2 activates the microglia, which are the immune system in the brain, which can lead to the development and aggravation of key proteins resulting in a number of neurodegenerative diseases, including Parkinson's disease[161].

Reproductive system

Long-term and short-term exposure to NO_2 have both been associated with adverse reproductive health impacts. Research evidence on this topic is inconsistent, mixed, and limited. However, there is some evidence to suggest that exposure to NO_2 is positively associated with higher incidences of infertility, preterm birth, lower live birth rates, low birth weight, and fetal loss in early pregnancy[162]. It has been concluded that there are no significant associations between long-term and short-term exposure to NO and maternal mortality or pregnancy related deaths[163]. The manner through which exposure to NO_2 can affect reproductive processes is not well understood. However, it is hypothesized that exposure to air pollution activates cytokines, resulting in placental inflammation[164]. Placental inflammation can limit the transplacental nutrient exchange, thereby limiting fetal growth and resulting in other adverse pregnancy outcomes[165].

Gastrointestinal system

Some researchers propose a connection between short-term exposure and increased incidence of inflammatory disease, such as appendicitis, Crohn's disease, and irritable bowel disease[166]. They argue that NO_2 triggers an inflammatory immune response, resulting in the development of the aforementioned conditions[167]. However, more research is required in order to establish the impacts of NO_2 exposure on the gastrointestinal system.

Core benefits of reducing nitrogen oxides

There is strong research evidence that indicates a number of human health co-benefits associated with the reduction of NOx emissions. This report identified core benefits associated with the implementation of low-carbon infrastructure, particularly in regards to the transport sector, renewable energy technologies, and residential building retrofits.

Decarbonizing the transport industry through improved fuel efficiency and transitioning from diesel and gasoline powered vehicles limits the emissions of pollutants like NO_2[168]. Transitioning to vehicles that are electric, powered by hydrogen, or hybrids has the capacity to substantially decrease the concentration of NO_2 in urban areas[169]. A study conducted in the United Kingdom indicates that with electric or alternative fuel powered vehicles comprising a substantial proportion of transport, there would be a significant reduction in NO_2 emissions, resulting in human health benefits[170]. This study found that the introduction of alternative fuel and electric-powered vehicles resulted in a 51% decrease of NO_2 over one year[171]. Two of the main co-benefits of the introduction of electric vehicles to human health is decreased mortality and increased life expectancy due to the reduction of NO_2 emissions[172]. It has also been argued that the introduction of programs to reduce the pollutants produced by the heaviest emitting trucks would be both cost effective and beneficial to human health[173]. Limiting NO_2 emissions from trucks can avoid mortality, thereby reducing the healthcare costs of premature death[174]. Guided by the 'Four Rs' (retrofit, repair, relocate, or retire) heavy emitting trucks could be taken off the roads or retrofitted into low-emitting trucks, resulting in a decreased output of NO_2 of approximately 40%[175].

The transition to non-emitting energy sources will reduce the reliance on coal and natural gas as energy sources[176]. The implementation of low-carbon energy infrastructure decreases emissions of nitrogen oxides, which benefits human health by improving cardio-pulmonary conditions, reducing the number of premature deaths, and diminishing the number of hospital visits due to air pollutant induced cardiovascular and respiratory diseases[177]. An example of this benefit can be found in a densely populated area in Northern China, where the transition to renewable projects decreased NO_2 emissions substantially, resulting in an average of 2.3 fewer premature deaths per 1.6 million people per year[178]. While this finding is relatively small, it is indicative of a relationship, and identifies that further research to quantify health benefits would be valuable. The renewable energy projects also resulted in the improved cardio-pulmonary conditions of the affected populations, thus resulting in fewer hospitalizations for cardiovascular and respiratory illnesses, and a decrease of chronic bronchitis cases per year[179].

Residential building retrofits lead to greener buildings with lower output of pollutants like NO_2. One study in New Zealand indicates that residents with sinusitis symptoms or hypertension saw their symptoms improve by 5% and 14%, respectively, as a result of upgraded buildings[180]. This same study also reported that young children that live in buildings that are retrofitted are less likely to be underweight than those whose residences see no change[181]. The positive health impacts of building retrofits also translate to positive economic impacts, such as savings due to avoided climate damages and health-related costs[182].

Sulphur Oxides

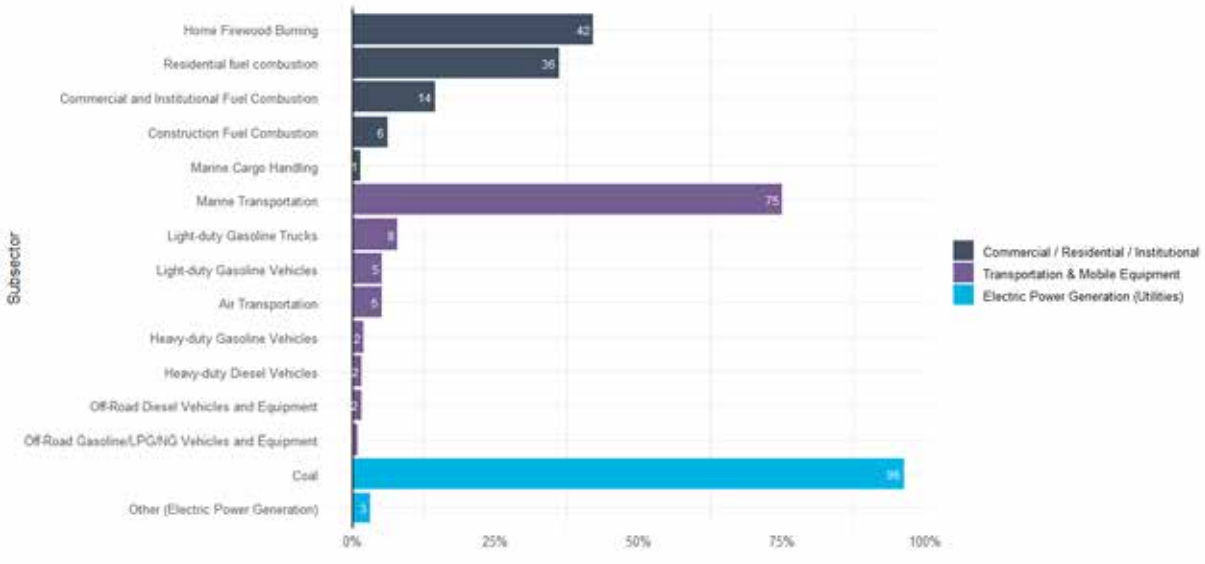
What are sulphur oxides?

Sulphur oxides (SOx) are a class of greenhouse gases, formed primarily from the release of nitrogen, that includes sulphur oxide (SO) and sulphur dioxide (SO_2)[183]. Although other sulphur oxides are present in the atmosphere (e.g., SO_3), SO_2 presents significant hazardous risk to human health[184]. SO_2 is a colourless, gaseous substance that is both naturally and anthropogenically made[185]. SO_2 is classified by the Canadian Centre for Occupational Health and Safety (CCOHS) as a compressed and extremely toxic gas, with a suffocating odor[186]. SO_2 is produced from burning fossil fuels (e.g., coal and oil) and its main anthropogenic sources are vehicular transportation, power and heat generation[187]. SO_2 is a primary emission source, and is not a non-threshold pollutant.

Sources of sulphur dioxide

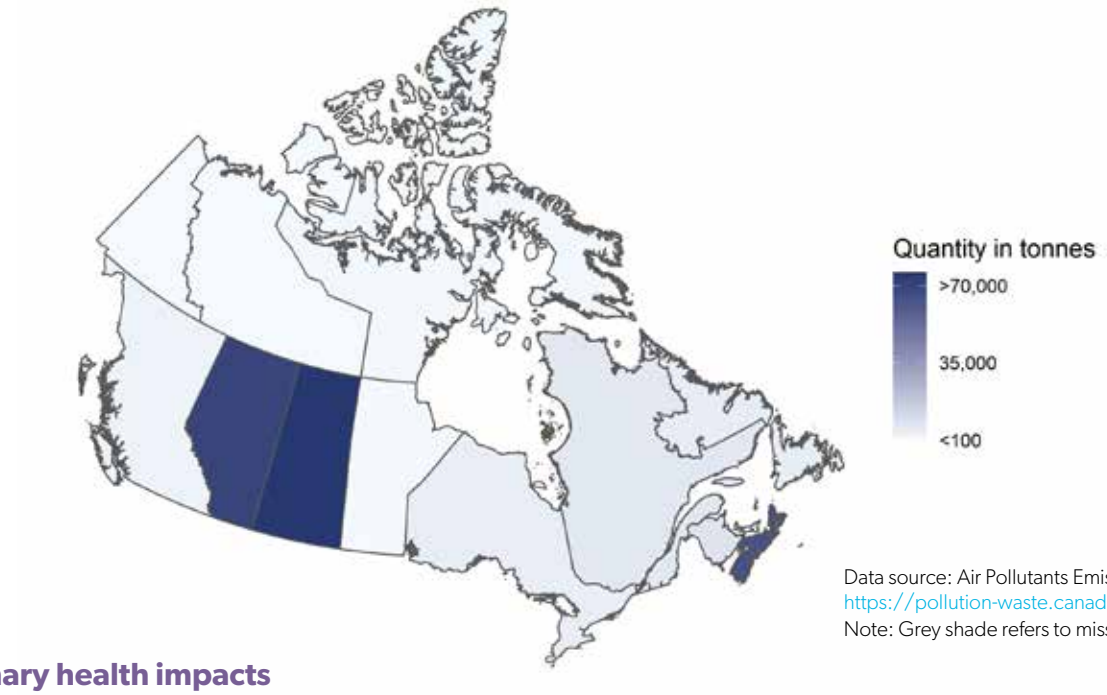
Anthropogenic SO_2 emissions are released by the combustion of fossil fuels, the smelting of ores, and burning of coal for indsutrial heat of power[188]. SO_2 is seen to be a major air pollutant, particularly in highly populated areas that rely on combustion of coal and fossil fuels for transportation, electricity, and heat production[189]. In urban areas, the most common emitter of SO_2 is road traffic, and therefore high concentrations of SO_2 are present in high traffic density regions. Industrial fossil fuel combustion and the use of residential fire wood also contribute to emissions of SO_2[190]. In the US, for example, the electricity and transportation sectors account for 96% of SO_2 emissions[191].

Figure 7: The largest sub-sectoral contributions of sulphur dioxide across Canada in the buildings, electricity and transportation sectors; Canada-wide values for 2018



Data source: Air Pollutants Emissions Inventory online search
https://pollution-waste.canada.ca/air-emission-inventory

Figure 8: The largest regional contributions of sulphur dioxide across Canada in the buildings, electricity and transportation sectors; Values for 2018



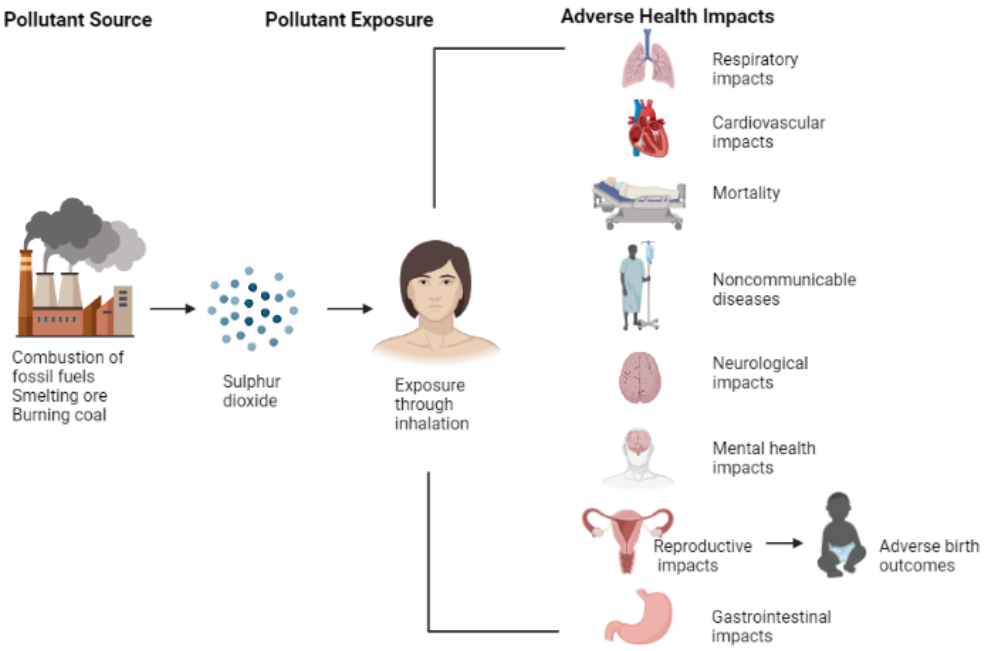
Data source: Air Pollutants Emissions Inventory online search https://pollution-waste.canada.ca/air-emission-inventory.
Note: Grey shade refers to missing data.

Primary health impacts

Sulphur oxides, notably sulphur dioxide, negatively affects:

- Respiratory systems;
- Cardiovascular systems;
- Mortality;
- Reproductive system;
- Neurological system;
- Gastrointestinal system.
- Non-communicable diseases;
- Mental health.

Figure 9: Pathways of sulphur dioxide and some of its associated adverse health impacts



Analysis of negative health effects

Respiratory system

SO_2 is a significant irritant to the respiratory system and this irritation acts a bronchoconstrictor, and induces systemic inflammation and oxidative stress[192]. SO_2 impacts both the growth and the function of the lungs[193]. Medium-term exposure to SO_2 is positively associated with an increased incidence of bronchoconstriction, COPD, hospital admissions for COPD, and COPD-related mortality[194]. Short-term exposure to SO_2 also increases the risk of intrinsic asthma and related hospitalizations. Intrinsic asthma is asthma that is caused by specific, non-allergen triggers (such as SO_2) inhaled into the respiratory tract[195]. Some evidence suggests that short-term exposure is positively associated with the development of childhood asthma[196].

Short-term exposure to SO_2 is associated with a significant increase in the incidence of TB and TB-related mortality[197]. This is even more worrisome if one considers that SO_2 exposure is also linked to the development of drug-resistant tuberculosis[198]. Indeed, air pollution-related tuberculosis is more strongly associated with SO_2 than any other air pollutant[199]. The respiratory tract is the main biological system impacted by SO_2. However, it is also the gateway to the rest of the body as the gas enters the systemic circulation via the bloodstream from the respiratory tract[200].

Cardiovascular system

Multiple studies provide evidence of a positive association between long-term and short-term exposure to ambient concentrations of SO_2 and reduced cardiopulmonary function. For instance, exposure to SO_2 increases the risk of hypertension[201], stroke mortality, cardiovascular disease[202], ischemic heart disease, and cerebrovascular mortality[203]. This is because SO_2 is inhaled into the respiratory tract before it spreads to the blood circulation, leading to breathing difficulties, hypoxia, and as a result, indirectly impacting heart function and leading to an increased risk of CVD mortality[204]. Short-term exposure is positively associated with increased risk of myocardial infarction[205], coronary heart disease[206], angina and coronary artery diseases[207].

Mortality

Exposure to SO_2 is positively associated with all-cause mortality and overall respiratory, cardiovascular, and cerebrovascular mortality[208]. Although there is mixed evidence concerning the strength of the association between SO_2 and all-cause mortality, research evidence suggests that there is a positive relationship between short-term and long-term exposure to SO_2 and all-cause mortality[209]. Long-term effects include mortality risk, reduced lung function, airway remodelling and increase in respiratory symptoms[210]. There is also evidence linking long-term exposure to SO_2 to lung cancer mortality[211]. Short-term and medium-term exposure to high concentrations of SO_2 is positively connected to increased risk of tuberculosis-infection[212]. High concentrations of SO_2 increases the risk of stroke and cardiovascular mortality[213]. There is a possible association between SO_2 and cerebrovascular mortality, however, evidence is not strong due to a paucity in studies and insignificant statistical relationships[214].

Reproductive system

Exposure to high concentrations of SO_2 is strongly associated with reproductive issues including increased prevalence of miscarriages, stillbirths, low birth weight, difficulties conceiving, and decreased quality of sperm[215]. There is a positive association between the short-term, medium-term and long-term exposure to SO_2, pregnancy mortality and fetal loss in early pregnancy[216]. Research evidence indicates that short-term exposure to high concentrations of SO_2 negatively impacts the probability of conception[217]. Some scholars found a significant association between exposure to ambient SO_2 and low birth weight[218]. It is not well-understood why long-term and short-term exposure to SO_2 and air pollution in general can cause adverse pregnancy outcomes[219]. However, some experts argue that the inhalation of gaseous pollutants like SO_2 can trigger placental inflammation and oxidative stress, which can inhibit transplacental nutrient exchange[220]. Although there is mixed evidence concerning the relationship between SO_2 and sperm health, several studies have also indicated a significant relationship between SO_2 exposure and sperm[221]. Other scholars suggest that exposure to SO_2 can decrease sperm concentration, total motility, sperm quality, and sperm count[222]. Recent experimental evidence demonstrates the potential of SO_2 to disrupt reproductive function[223].

Neurological system

The impacts of SO_2 exposure on the neurological system have not been adequately explored to establish a strong positive association. However, some studies have found that long-term exposure to SO_2 can result in impaired memory, Parkinson's disease, or risk of cerebrovascular mortality[224]. Experimental results suggest that co-exposure of multiple pollutants, including SO_2, is what could cause the aforementioned negative impacts on the neurological system[225].

Gastrointestinal system

The impacts of short-term or long-term exposure to SO_2 on the gastrointestinal system have not been adequately substantiated and therefore lack the necessary evidence to postulate a significant association. However, some studies suggest that short-term exposure to SO_2 can increase the prevalence of appendicitis, acute diarrheal disease, intestinal diseases, and irritable bowel disease[226].

Noncommunicable diseases

There is significant research evidence linking long-term exposure to SO_2 to increased incidence of lung cancer and lung cancer mortality[227]. It has been found that proximity to heavy-traffic roads

increases concentration of SO_2, therefore long-term proximity to heavy-traffic roads also increases the risk of lung cancer and lung cancer mortality[228].

Mental health

There is not enough evidence to adequately support a positive association between SO_2 exposure and mental health problems. Recent evidence suggests a relationship between SO_2 exposure and an increased likelihood of suicide, but the biological explanation is yet to be developed[229]. The most commonly proposed pathway, which also applies to multiple other gaseous pollutants, is that the inhalation of ambient particles triggers neuronal inflammation and oxidative stress, which can induce anxiety and depressive behaviours[230]. It is therefore biologically plausible that pollutants like SO_2 play a role in the increased risk of mental health problems and suicide attempts[231].

Core benefits of reducing sulphur dioxide

Reducing SO_2 emissions provides an array of human health co-benefits, especially in sectors such as buildings and electricity. A transition from coal and other fossil fuels to renewable energy and alternative clean fuel sources can prevent an increase in SO_2 emissions[232]. Upgrading the transportation system and prioritizing the implementation of renewable sources of energy can result in a reduction in SO_2 emissions, leading to multiple health co-benefits, such as reduced mortality rates, fewer cases of chest discomfort, and decreased incidence of respiratory diseases[233]. A study in a densely populated region of Northern China indicates that the introduction of renewable energy projects reduced all-cause mortality and hospitalizations for SO_2 exposure-related respiratory and cardio-pulmonary diseases by approximately 40%[234].

The implementation of programs focused on reducing the emissions of pollutants like SO_2, and prioritizing alternatives to heavy emitting sources, results in multiple health co-benefits, such as a reduction in respiratory symptoms like shortness of breath and discomfort in chest[235]. An illustrative example is a policy adopted in Hong Kong that limited the quantity of sulphur content of fuel oil used for vehicles and power plants, resulting in a 45% decrease in SO_2 concentrations and significant reductions in annual rates of all-cause mortality, respiratory mortality, and cardiovascular mortality[236].

Improving energy efficiency in buildings has been proven to reduce SO_2 emissions, resulting in fewer adverse health impacts of SO_2 exposure including, asthma, respiratory symptoms, and mortality[237]. Residential building retrofits improve the overall health of residents as they see a reduction in respiratory and cardiovascular symptoms, improved sinusitis, hypertension, and asthma symptoms[238].

Ozone

What is ozone (O_3)?

Ozone is classified as a secondary pollutant, meaning it is formed from atmospheric and chemical reactions to other pollutants[239], such as atmospheric reactions between NOx gases and volatile organic compounds (VOC) in the presence of sunlight, and is usually found in the lower atmosphere[240]. Conditions conducive to ozone accumulation include vehicle emissions that produce NOx and VOCs[241], increases of atmospheric visibility and solar intensity corresponding with a decrease in ambient carbonaceous aerosols (e.g., soot)[242], and presence of particulate matter[243]. Ambient ozone concentrations tend to be higher during summer because NOx and VOC reactions occur in warmer weather; winter months can see high ozone concentrations in areas experiencing high NOx and VOC emissions[244]. Ozone is also a non-threshold pollutant, meaning exposure at any level is considered a health risk.

Sources of ozone

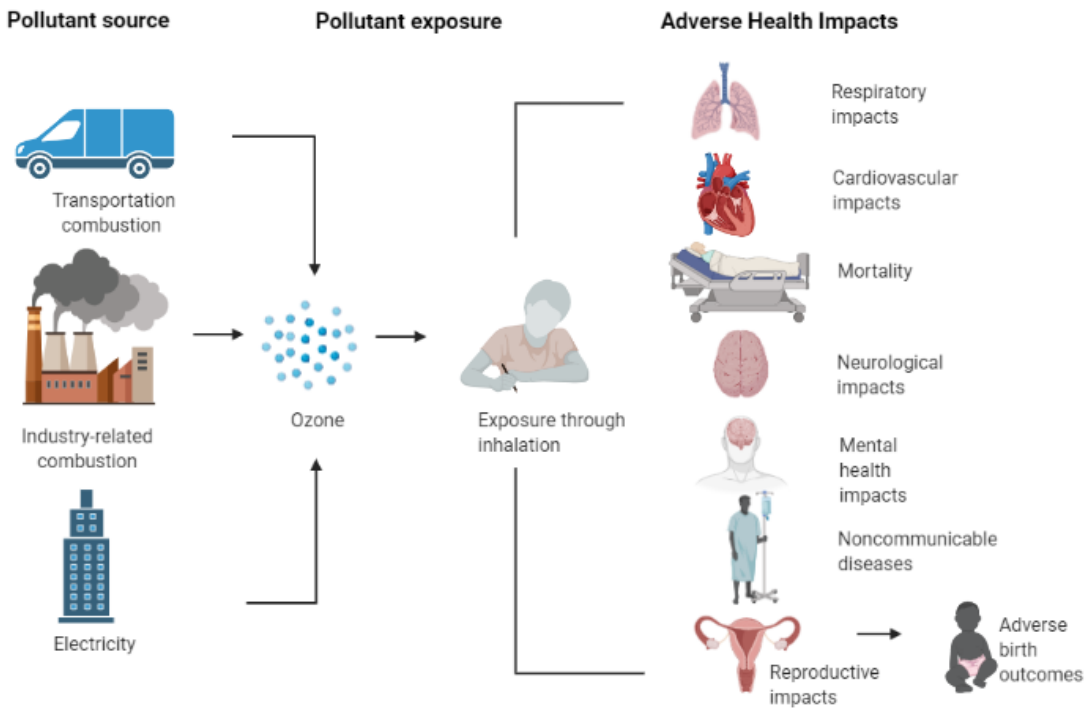
Ozone is a secondary pollutant originating from traffic- and industry-related combustion processes, meaning that evaluating the sources of ozone in Canada require assessing the sources of pollutants that react to form ozone [245]. Light-duty vehicles are sources of primary and secondary pollutants that can generate ground-level ozone[246]. Vehicle emissions from cars, trucks, and buses can produce high amounts of NO_x and VOCs which are conducive to ozone production and accumulation[247]. Ozone tends to have higher concentration rates further away from high traffic sources or city centers [248]. In residential areas, high levels of electricity consumption also increases ozone pollution[249]. Electricity demand is closely correlated with higher ozone concentrations[250]. Finally, indoor sources of ozone include stoves and furnaces[251, 252].

Primary health impacts

Ozone negatively affects:

- Respiratory systems;
- Cardiovascular systems;
- Mortality;
- Neurological system;
- Mental health;
- Non-communicable diseases;
- Reproductive system;

Figure 10: Pathways of ozone and some of the adverse health effects it can cause



Analysis of negative health effects

Respiratory system

Ozone is consistently associated with a decline in respiratory function[253]. Ozone also has associations with various health consequences like COPD, impaired lung development, and mortality, commonly recognized health impacts of ozone include increased occurrences of respiratory disease, induced asthma attacks, reduced lung function, and breathing problems [254].

Ozone is cited as a strong respiratory irritant[255]. Even short-term exposure increases the risk of respiratory diseases, upper and lower respiratory tract illness (which can manifest in low level air pollution areas), respiratory tract infection, wheezing, respiratory tract injury, systemic inflammation, and coughing[256]. Research also provides compelling evidence of a strong relationship between ozone exposure and risk of COPD[257], even following short-term exposure. There is also a potential association with pulmonary inflammation[258].

Long-term exposure to ozone is a risk factor for asthma development, with potentially strong associations[259]. Warm seasons are more likely to observe asthmatic outcomes[260]. Children are disproportionately impacted by ambient pollution exposure, especially ozone; it is well documented that pollution exacerbates asthma in children and can "increase airway oxidative stress and airway inflammation in asthmatic children"[261].

The disproportionate impact may have to do with children spending a larger amount of time outdoors[262]. Asthmatic patients are more sensitive to the effects of ozone, and ozone exposure leads to inflammation, which can further exacerbate the severity of asthma[263].

Long-term exposure to ozone is associated with lung function deficits or abnormal lung development, especially in children[264]. This may have to do with the inhalation of a higher dose per body mass, as well as the fact that children's lungs are still developing[265]. Short-term exposure in children has also been shown to be associated with decreased lung function[266].

In residential areas, high levels of electricity consumption also increases ozone pollution. Electricity demand is closely correlated with higher ozone concentrations. Finally, indoor sources of ozone include stoves and furnaces.

Airway inflammation is the most common biological explanation of how ozone impacts the lungs. As seen in human and animal models, ozone can travel to distal airways and impact alveolar lining fluid, and react with cellular membranes to generate oxidative stress and induce immune responses[267].

Cardiovascular system

Researchers have also linked ozone to acute coronary events, cardiac arrests, cardiovascular disease, cardiovascular hospital admissions, and cardiovascular mortality from preceding coronary events like heart failure, stroke or ischemic heart disease[268]. There are strong and often significant associations between ozone exposure and cardiovascular diseases, stroke, myocardial infarction, ischemic stroke, even over a short period of exposure[269]. Although the relationship between ozone and cardiovascular disease can be attenuated in multi-pollutant models, the association still remains significant[270].

After controlling for co-pollutants, some researchers have raised evidence that short-term exposure has significant, positive associations with mortality from "cardiovascular, dysrhythmia, cardiometabolic, and ischemic heart disease"[271]. There is also research evidence that ozone is associated with blood pressure, however this must be interpreted with caution as hypertension cannot be diagnosed after a short-period of time exposure[272]. Some other research has identified a consistent association between short-term ozone exposure and heart failure hospitalization[273].

Therefore, there is robust evidence that ozone is associated with several adverse cardiovascular impacts.

Cardiovascular impacts arise from systemic inflammation or oxidative stress, which could affect cardiac function, vascular function, autonomic tone, or hemostasis[274].

Mortality

There is a clear and significant established link between ground level ozone concentration and death[275]. Short-term changes in ozone levels have been reported to be associated with short-term changes in death and increased mortality[276]. While congestive heart failure mortality and ozone are reported to have no association, there is a strong association with myocardial infarction history[277]. While short-term and long-term ozone is often linked to cardiovascular mortality[278], the evidence associating long-term ozone exposure to cardiovascular mortality is still contradictory and therefore limited in confirming a strong link[279].

Ozone is associated with mortality resulting from respiratory diseases including COPD, lower respiratory infection, and lung cancer[280]. However, the research evidence suggesting associations between ozone and mortality from respiratory diseases can be mixed[281].

While the biological mechanisms by which ozone affects mortality have yet to be elucidated, toxicology studies outline decreased heart rate, blood pressure, cardiac output, and blood pressure to be potential pathways[282]. Other pathways may be due to systemic and pulmonary inflammation leading to thrombus (clot) formation or activation of neural reflexes and changing of cardiac or vascular function[283].

Neurological system

There is emerging evidence suggesting that ozone impacts the neurological system[284]. Ozone and cognitive decline shared a mixed relationship[285]. Development and progression of conditions, such as dementia, depend on complex interactions between different environmental and lifestyle risk factors and genetic characteristics[286]. The severity of impact also changes according to the cognitive test used. Sometimes, genetic factors exacerbate the impacts that ozone exposure may have on cognitive decline[287]. Induced inflammatory responses in the lungs due to acute or chronic exposure to ozone may also lead to memory deterioration and neuronal morphology changes[288]. Recent studies have related ozone exposure to neurodegenerative disorders like Parkinson's disease and Alzheimer's disease[289].

A proposed biological explanation is that ozone triggers immune inflammatory responses, which then impacts the circulatory system, and subsequently reaching the neurological system[290]. This is plausible as "air pollution has been proven to cause stress hormone increases and to alter metabolic behavior" in addition to having been "shown to cross the blood-brain barrier"[291]. Further research is required to uncover and specify the strength of linkages between ozone and neurological effects.

Mental health

There is not enough evidence to link mental health to ozone exposure. Some scholars, however, suggest that ozone is significantly associated with the prevalence of symptoms of depression, anxiety, and psychological distress[292]. Others claim that long-term exposure to ozone in multipollutant models with PM_{10} and NO_2 has a mixed association with depressive symptoms[293]. Only one paper commented on a significant increase in hospital admission for psychoses on hot days for higher ozone and PM_{10} concentrations[294].

Noncommunicable diseases

The evidence is insufficient to establish a relationship between noncommunicable diseases and ozone exposure. There are some single-pollutant and mixed-pollutant studies linking ozone to cancer (e.g. lung and squamous cell). The strength of association can be attenuated with interaction with different pollutants[295]. Some studies have indicated significant association between diabetes and ozone exposure, as well as appendicitis and short-term ozone exposure[296].

Reproductive system

The evidence associating ozone exposure to birth outcomes and reproductive effects is inconclusive. There is a weak association between ozone exposure and birth outcomes[297]. One study observed an increase in the number of preterm births within the last two months of pregnancy due to ozone exposure[298]. The mechanisms of adverse pregnancy are not well understood, but some scholars suggest that inflammation during pregnancy may cause adverse impacts for both the pregnant person and developing fetus[299].

Core benefits of reducing ozone

The health benefits of reducing ozone include decrease in premature mortality, avoided deaths, morbidity, hospital admissions, respiratory emergency room visits, respiratory hospital admissions, acute respiratory symptoms, asthma exacerbations, acute myocardial infarctions, and emergency room visits for asthma symptoms[300].

Introducing electric vehicles can lead to substantial health co-benefits. It is estimated that electrification of at least 17% of light-duty vehicles and 8% of heavy-duty vehicles will lead to widespread reductions of 1 ppb of ozone and 0.5μg m^{-3} of fine particulate matter ($PM_{2.5}$)[301]. Ozone reduction was demonstrated across different scenarios. In reducing ambient O_3 and PM, air quality increases which can lead to decreases in premature mortality annually, ranging from 170 to 7548, depending on the scenario[302]. Other health benefits from ozone reduction may be relatively modest because the burden of mortality from vehicular sources are higher for $PM_{2.5}$[303].

POLLUTANTS WITH THE POTENTIAL TO INCREASE RISK OF ADVERSE HEALTH IMPACTS

This section identifies emergent pollutants that are identified as being potential risk factors for adverse health impacts within the literature. For each pollutant in this section, the details included are those commonly available in scholarly research. The shorter overall write-up of each section is indicative of a lack of sufficient evidence to make credible claims of negative health impacts, or health co-benefits associated with low-carbon infrastructure projects.

Carbon Monoxide

What is carbon monoxide?

Carbon monoxide (CO) is a gas usually formed during fossil fuel combustion when fuels undergo incomplete combustion[304] It is one of the most common and widely distributed air pollutants. The gas has no smell, colour, or taste, and it is poorly soluble in

water[305]. Outside urban areas, one of the main sources of CO include plants, oceans and oxidation of hydrocarbons which can also give rise to ambient concentrations[306].

Sources of carbon monoxide

Traffic-related combustion is a high source of CO, but industry-related combustion processes can also be a source of this pollutant[307]. Road transportation produces nearly 22% of all emitted CO[308]. Cars, buses, and gasoline-powered vehicles emit CO[309]. CO tends to be 40%-50% higher near road-sites[310]. Compared to gasoline engines, diesel engines emit lower levels of ozone but are still important contributors to atmospheric particulate pollution[311]. Fossil fuel burning, wood burning, or home heating can also directly emit CO[312].

Primary health impacts

Carbon monoxide may negatively affect:

- Cardiovascular systems;
- Respiratory systems;
- Non-communicable diseases;
- Mortality;
- Gastrointestinal system
- Mental health and the neurological system;
- Reproductive system;

Analysis of negative health effects

Cardiovascular system

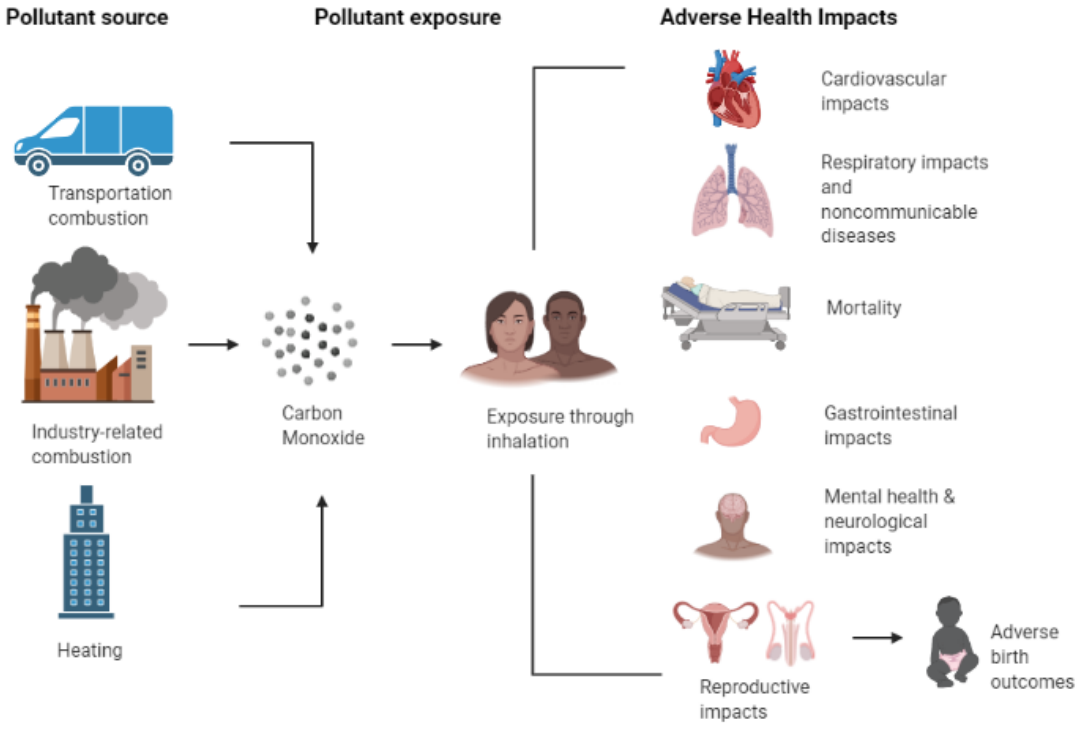
Literature presents compelling evidence that CO impacts the cardiovascular system resulting in cardiovascular disease incidence, mortality, and morbidity[313]. Studies indicate that CO is related to heart failure, increased risk of acute myocardial infarction, ischemic heart disease, and stroke[314]. There may also be an association between CO and unstable angina in those with underlying coronary artery diseases but this requires further exploration[315]. CO is significantly associated with cardiovascular mortality[316]. Even incremental increases of CO are related to cardiovascular disease mortality[317].

There are two proposed ways to explain how CO may result in cardiovascular diseases. Adverse outcomes can arise directly through inhalation and subsequent entry into blood circulation, or by acting on the respiratory system and causing breathing difficulties which could impact heart function[318]. The cardiovascular system can be impacted by CO binding to hemoglobin on red blood cells instead of oxygen[319]. This can lead to a loss of oxygen and outcomes like ischemia, hypoxia, and cardiovascular disease[320].

Respiratory system and non-communicable diseases

CO is significantly associated with different health outcomes in the respiratory system[321]. A number of studies have identified strong or significant associations between CO exposure and

Figure 11: Pathways of carbon monoxide and some of its adverse health impacts



higher rates of asthma hospitalization, hospitalization from acute exacerbation of COPD, and pneumonia hospitalization[322]. An association between CO exposure and decreased lung function and childhood asthma incidence has also been identified[323].

CO is associated with lung cancers like adenocarcinoma[324]. There is also a recognition that CO is a risk factor for tuberculosis incidence and exacerbation of tuberculosis resistance[325]. Emerging evidence suggests that CO, in addition to other pollutants ($PM_{2.5}$, PM_{10}, SO_2, NO_2, lead, VOCs), is significantly correlated with California's COVID-19 epidemic[326].

Overall, research evidence suggests a significant association between respiratory outcomes and CO exposure, but there has yet to be an explanation on the precise biological pathways through which CO impacts the respiratory system.

Mortality

Incremental increases of CO are related to a rise in overall deaths and all-cause mortality, disproportionately impacting urban populations[327]. There are direct, significant correlations between CO and respiratory disease mortality[328]. A gender stratified analysis reveals a potentially higher association with mortality impact attributable to traffic-related air pollution of CO in men compared to women[329]. Multipollutant interactions between CO and other pollutants, specifically NOx and SO_2, have been suggested to be positively and significantly correlated to mortality from various diseases in the respiratory and cardiovascular systems[330]. There is a potential relationship between infant mortality and cerebrovascular mortality, although this requires further exploration[331]. Overall, there is compelling evidence to suggest an association between mortality and CO.

Gastrointestinal system

Some research evidence suggests that CO impacts the gastrointestinal system. There is a modest association with enteric disease and potentially inflammatory bowel syndrome and ulcerative colitis[332]. The last two conditions seem to be more associated with youth under 25 and in mixed pollutant models[333]. CO and SO_2 is potentially a positively correlated with acute diarrheal disease[334]. The biological pathways to explain these impacts are yet to be elucidated. Overall, the evidence to suggest strong associations on impacts on the gastrointestinal system is mixed to weak.

Mental health and the neurological system

Some scholars suggest an association between CO and schizophrenia, but further research is required to verify the strength of this relationship[335]. There is potentially an association between increased annual exposure to CO and Alzheimer's disease[336]. CO exposure may also aggravate Parkinson's disease; however, this was explored in the context of a multipollutant model and isolating the degree of impact is therefore difficult[337]. Inflammation is a suggested biological pathway contributing to these conditions[338]. CO has also been identified as a risk factor for cognitive decline, autism, and dementia[339]. Further research is required to outline the impacts of CO on mental health and the neurological system.

Reproductive system

Adverse birth outcomes like preterm birth, pregnancy induced hypertensive disorders, and low birth weight are claimed to be associated with CO exposure[340]. Adverse birth outcomes may result from inflammation in the placenta[341]. There is some evidence supporting an association between CO and premenstrual symptoms, as well as semen quality[342]. Overall, the evidence tying reproductive outcomes to CO exposure is weak.

Core benefits of reducing carbon monoxide

There is limited evidence to suggest that reductions in CO constitute substantive identified co-benefits to human health when occurring as a direct result of low-carbon infrastructure projects. Some research evidence indicates that the implementation of energy retrofits improved conditions like sinusitis and hypertension. The improvement of these two health factors is also connected to improved energy efficiency and heating[343]. However, more research is needed to identify health co-benefits that may emerge as a result of adoption of low-carbon technologies or construction of low-carbon projects.

Persistent Organic Pollutants (POPs)

What are persistent organic pollutants?

Persistent organic pollutants (POPs) are chemicals that are resistant to environmental degradation through natural processes (e.g., biological, chemical, and photolytic)[344]. Due to POPs persistence, these chemicals can accumulate in the environment, resulting at times in negative impacts on the health of humans and the environment[345]. POPs have become a matter of global concern because of their ability to withstand environmental degradation[346]. The most common forms of POPs are industrial chemicals, organochlorine pesticides, and polychlorinated biphenyls (PCB)[347]. POPs are also frequently unintentional by-products of multiple industrial processes[348].

Sources of persistent organic pollutants

POPs are produced anthropogenically[349]. POPs can be pesticides, industrial chemicals (e.g., polychlorinated biphenyls and polybrominated diphenyl ethers), pharmaceuticals, and solvents[350]. Polychlorinated biphenyls are often used in transformers as heat exchange agents, heat transfer fluids, pesticide extenders, and hydraulic lubricants[351]. Polybrominated diphenyl ethers are found in many household items as they are used as flame retardant.[352]

Findings below are related to studies on the impacts of POPs as a result of infrastructure projects in the buildings, transportation, and enregy subsectors. It is recognized that the creation of POPs arises from more than infrastructure projects in these three sectors, and future work may take additional sources into account.

Primary health impacts

POPs may negatively affect:

- Reproductive systems;
- Neurological systems;
- Non-communicable diseases and cardiovascular systems.

Analysis of negative health effects

Reproductive system

The association between exposure to POPs and adverse health impacts on the reproductive system is still emerging, and the available research is quite limited. Some sources have indicated moderate associations. For instance, some studies indicate that POPs exposure is associated with reproductive issues and the contamination of breast milk and placental transfers, as mothers easily transfer POPs to the fetus through milk, placenta, adipose tissue, and blood[353].

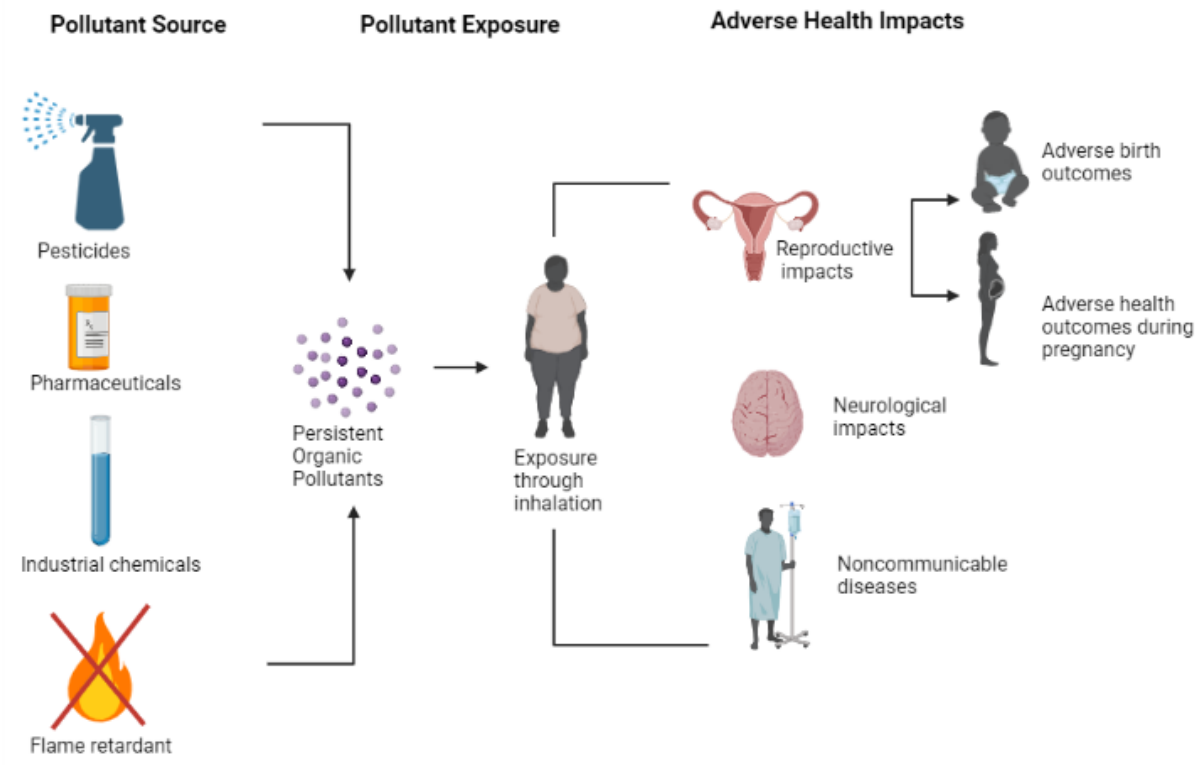
Neurological system

It has been suggested that exposure to POPs can result in nervous system damage, but the literature on this is quite limited[354]. Animal studies that have explored the relationship between POP exposure and adverse neurological health impacts have found that long-term exposure can result in neurodevelopmental anomalies, as POPs are seen to be extremely neurotoxic[355].

Noncommunicable diseases and cardiovascular system

The methodology used in this literature review has unveiled limited research evidence connecting POPs exposure and the development of non-communicable diseases. Some sources have identified exposure to POPs as a risk factor to breast cancer and type 2 diabetes[356]. Several other studies and authoritative sources categorize POPs as carcinogens and important endocrine disrupting factors. As such, caution must be made when assessing the impacts of POPs on the cardiovascular system and its relation with the onset of noncommunicable diseases, as POP is a class of highly toxic substances that can cause a wide array of health issues.

Figure 12: Pathways of POPs and some of its associated adverse health impacts



The impacts of POPs on cardiovascular health have not been adequately researched and the available literature on this relationship is inconclusive.

Core benefits of reducing persistent organic pollutants

Evidence exploring the human health co-benefits of reducing the flow of persistent organic pollutants is extremely limited, with insufficient information available to adequately discuss any potential health benefits from the reduction of POP airsing from the adoption of low-carbon infrastructure projects.

Volatile Organic Compounds (VOCs)

What are volatile organic compounds?

Volatile organic compounds (VOCs) are anthropogenic chemicals that are extremely toxic air contaminants[357]. They are sometimes referred to as indoor organic pollutants as they primarily impact indoor air quality[358]. Typically, VOCs are used in everyday household products such as, wax, varnishes, furniture, cleaning solutions, pesticides, building materials, office equipment, markers, glue and paint[359]. Each of these household VOCs can be released in use or while in storage[360]. Common VOCs are benzene, propane, and isobutane[361].

Sources of volatile organic compounds

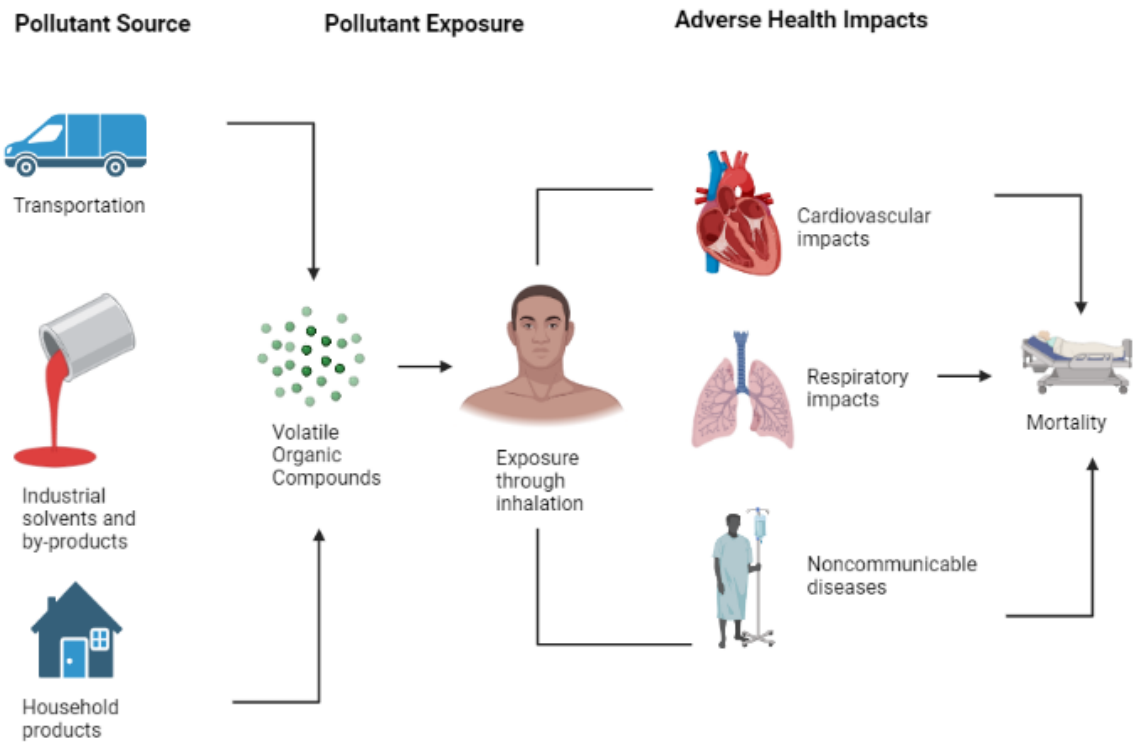
VOCs have low water solubility, high vapour pressure, and can be emitted as gases from certain solids or liquids[362]. Generally, VOCs are produced through fuel combustion in internal combustion engine vehicles, or through evaporative emissions[363]. VOCs are typically components of hydraulic fluids, dry cleaning agents, petroleum, and paint thinners[364]. VOCs are also often produced in the manufacturing of paints, refrigerants, and pharmaceuticals[365]. VOCs can also be industrial solvents or by-products of chlorination of water, where the VOC chloroform is produced[366]. VOCs are regularly found in contaminated ground-water[367].

Primary health impacts

VOCs may negatively affect:

- Cardiovascular system;
- Mortality;
- Non-communicable diseases;
- Respiratory system;
- Urological system.

Figure 13: Pathways of VOCs and some of its associated adverse health impacts



Analysis of negative health impacts

Cardiovascular system

The cardiovascular effects of exposure to VOCs are sparsely documented[368]. Short-term exposure of VOCs, specifically alkyne and benzene, increase risk of emergency hospitalizations for heart failure[369]. Long-term exposure to VOCs has been associated with cardiovascular dysfunctions and heart rate variability because of systemic inflammation and arrhythmia[370].

Mortality

There is limited research exploring the relationship between VOCs exposure and mortality. Some sources have inferred that exposure to VOCs are positively associated with all-cause, cardiovascular, cerebrovascular, and infant mortality[371]. Occupational studies have found that long-term exposure to VOCs, especially benzene and styrene, is associated with increased risk of mortality[372].

Noncommunicable diseases

Multiple studies have identified VOC exposure as a risk factor to the development of various forms of cancer[373]. Indeed, the International Agency for Research on Cancer classifies multiple VOCs, like benzene, as human carcinogens[374]. Benzene, specifically, is associated with an increased incidence of leukemia and childhood leukemia[375]. VOC, through the formation of reactive oxidative species, may play a role in the development of cancer risk; benzene has also been noted to increase the risk of lung cancer[376]. Formaldehyde from combustion sources, cigarette smoke, and off-gassing furniture, has also been positively associated with lung cancer and leukemia[377].

Respiratory system

Exposure to VOCs has been positively associated with adverse respiratory health outcomes, including respiratory symptoms and hospitalization for respiratory diseases because VOCs induce inflammation in the respiratory tract[378].

Urological system

The adverse urological health outcomes of exposure to VOCs have not been adequately researched to infer a relationship. However, few sources have linked VOC exposure and kidney regression[379].

This literature review did not identify research evidence that connects VOC exposure to the reproductive, gastrointestinal, or neurological systems.

Core benefits of reducing volatile organic compounds

Projects, programs, and policies that seek to reduce VOC emissions, specifically projects related to transportation and residential building retrofits, can potentially result in co-benefits to human health. The vast majority of VOC emissions come from exhaust fumes from motor vehicles, therefore the introduction of programs aimed at changing transportation behaviours (e.g., walking, cycling, running) is likely to reduce exposure to VOCs relative to other forms of travel. It has been argued that this decrease in exposure reduces the incidence of coronary heart disease, hypertension, stroke, and diabetes[380].

Retrofits that improve energy efficiency and heating have shown to greatly reduce residents' exposure to many air pollutants, including VOCs[381]. Buildings retrofitted to improve energy efficiency improve asthma symptoms, shaken baby syndrome symptoms, sinusitis symptoms, hypertension, and mental health of people living in these buildings[382]. Although it has been suggested that residential retrofits improve human health through the reduction of VOC exposure, the findings were not able to clearly identify the magnitude of the impact of retrofits[383].

The vast majority of VOC emissions come from exhaust fumes from motor vehicles, therefore the introduction of programs aimed at changing transportation behaviours (e.g., walking, cycling, running) is likely to reduce exposure to VOCs.

Noise Pollution

What is noise pollution?

Noise pollution is regular and consistent exposure to elevated, anthropogenic sound and is associated with adverse health impacts on humans[384]. Noise pollution is emitted primarily from transportation, including internal combustion engine vehicles, ships, trains, and aircrafts [385]. Motor vehicles and highly trafficked areas produce the highest levels of noise pollution[386]. Traffic-related noise is most prominent in highly populated urban areas, particularly at peak travel times in the morning and evening rush hours[387].

Sources of noise pollution

The main emitter of noise pollution is transportation (e.g., internal combustion engine vehicles, trains, planes, and trucks)[388]. It has been postulated that the intensity of the noise pollution and the source are particularly important as they may increase the severity of human health impacts[389]. The impact of exposure to noise pollution on mental health varies depending on the intensity of the sound. It has also been argued that the severity of mental health problems varies greatly depending on the source of the noise pollution[390]. For example, heavy vehicles (e.g., trucks and busses) and uneven flow of traffic have led to higher degrees of annoyance and anxiety[391]. It has also been suggested that because noise pollution and air pollution are emitted by many of the same sources (e.g., motor vehicles), noise pollution is considered to be a confounder of air-pollution related adverse health outcomes[392].

Primary health impacts

Noise pollution may negatively affect:

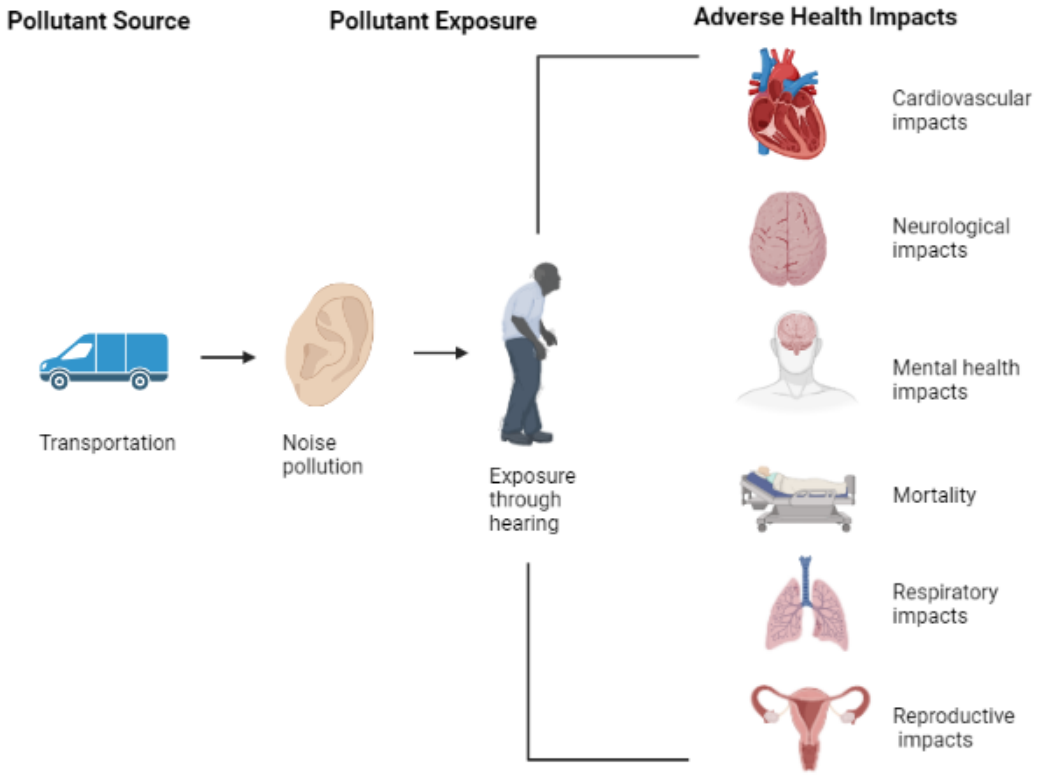
- Cardiovascular system;
- Neurological system;
- Mental health;
- Mortality;
- Respiratory system;
- Reproductive system.

Analysis of negative health impacts

Cardiovascular system

The impacts of noise pollution on human health have not been thoroughly studied, and many sources indicate the need for greater exploration of this topic to develop a more complete and nuanced understanding of this relationship. Exposure to noise is positively associated with elevated blood pressure, cardiovascular disease hypertension, coronary heart disease, sudden cardiac death, ischemic heart disease including stroke and myocardial infarction, and increased need and use of cardiovascular medication[393]. It has been argued that noise pollution exposure may trigger the same responses in the human body, for example systemic inflammation and oxidative stress, as that of air pollution[394]. Noise pollution can have negative impacts on cardiovascular function that are independent of sleep quality and noise sensitivity[395]. One identified indirect pathway is that noise pollution impacts the central nervous system, resulting in a number of related adverse health outcomes, including a rise in heart rate and stress hormones (adrenalin, noradrenaline, and cortisol), thus placing large amounts of stress on the systems and organs in the human body[396]. It has also been argued that nighttime traffic noise may impact the strength of one's immune system as a result of sleep disturbances, increased blood pressure, and vascular dysfunction[397].

Figure 14: Pathways of noise pollution and some of its associated adverse health impacts



Neurological system

Some studies have identified harmful impacts of noise pollution exposure on the neurological system[398]. One explanation is that noise directly impacts human health through the central nervous system, where there is an immediate interaction of noise with the acoustic nerve[399]. This direct pathway is activated by an instantaneous interaction of the acoustic nerve with structures of the central nervous system, resulting in elevated levels of stress hormones through the activation of the hypothalamic-pituitary-adrenal axis[400]. An indirect pathway is the cognitive perception and reaction to the noise, the following cortical activation, and its relation to emotional responses[401]. This indirect pathway in turn represents the cognitive perception of the sound, and its subsequent cortical activation and is related to emotional responses[402]. Both direct and indirect pathways can cause physiological stress responses involving the hypothalamus and the overall nervous system, which results in a cascading effect on multiple other systems, including cerebrovascular, respiratory, and cardiovascular[403]. Long-term exposure to noise pollution has been positively associated with an increased risk of stroke, particularly in populations over the age of 65[404]. The overall impact of traffic noise on cognition and neurological diseases (e.g., vascular dementia, Parkinson's disease, Alzheimer's disease) is still unclear and requires further exploration[405].

Mental Health

Evidence of the relationship between exposure to noise pollution and mental health issues is inconclusive, with some sources finding strong associations and others identifying questionable statistical results or no association[406]. Noise pollution poses a complex and different kind of exposure from air pollution, as generally populations are aware of their exposure, unlike with environmental contaminants where exposure is often unnoticed[407]. This exposure can be related to the development of mental health problems, including behavioural problems, cognitive decline, anxiety, and depression[408]. This is because the recognition of exposure can trigger annoyance and stress, which translates into neurological responses, resulting in depression, anxiety, and other mental health disorders[409]. Short-term and long-term exposure to noise pollution are both associated with annoyance with elevated noise levels in proximal populations[410]. Proposed explanations of how noise pollution exposure impacts mental health include a direct pathway that takes the form of sleep disturbances, and an indirect pathway that takes the form of noise annoyance[411]. Sleep disturbances may increase levels of stress and the development of mental health conditions like behavioural and emotional disorders, anxiety, and depression[412], whereas noise annoyance may lead to the development of cardiovascular and respiratory disease, as well as mental health issues[413]. The harmful effects of exposure to noise pollution have been more closely related to the mental health of children and adolescents, however, further research is required to understand the biological reasons[414]. It is suggested that both the direct and indirect pathways through which noise pollution impacts mental health lead to an acute physiological response that causes an elevation in the production of stress hormones[415].

What about heat stress?

Recent record-breaking heat waves in Canada and abroad have brought attention to the impact of excess heat on human health. Although excess heat is not a pollutant, thus falling out of scope of this report, it can both adversely impact health and accelerate the production of ground-level ozone, a non-threshold pollutant[426]. Heat is generated in urban areas due to urbanization wherein natural landscapes are gradually replaced with urban surfaces like roads, buildings and other structures made of concrete, asphalt, or tarmac[427]. These materials absorb heat while reducing evapotranspiration and surface permeability, further trapping heat. Energy use in buildings and vehicles can also add heat to surroundings through air pollution[428]. A cumulation of these factors lead to the Urban Heat Island (UHI) effect, which is the result of urban spaces absorbing and retaining heat[429]. Due to this effect, air pollution levels increase, raising subsequent risks of experiencing heat-related illnesses or mortality[430].

The use of cool roofs, a potential low-carbon infrastructure project for buildings, can provide great benefits through heat reduction[431]. Cool roofs use reflective materials to reduce absorption of solar energy which can reduce the UHI effect[432]. By implementing cool roofs in urban areas, air temperatures are reduced resulting in cooler daytime temperature[433]. Heat stress can also be reduced through the reduction of air pollution by redesigning transportation infrastructure or through better urban planning[434]. Some health co-benefits of projects like the aforementioned may include avoided mortality due to UHI and heatwaves, in addition to reduced cardiovascular and respiratory mortality, especially in elderly populations[435]. The greatest benefits can be reaped in urban centres where there is a high density of buildings and traffic[436].

Mortality

Exposure to noise pollution is strongly associated with all-cause mortality and results in an increase in disability-adjusted life years (DALYs) annually[416]. It has been argued that the impacts of noise pollution on human health is comparable to that of radon and second-hand smoke[417]. Many studies have identified a strong association between short-term exposure to noise pollution and cardiovascular mortality, specifically from ischemic heart disease, sudden cardiac death, and myocardial infarction[418]. It has also been reported that there is a strong association between short-term exposure to noise pollution and respiratory mortality, diabetes-related mortality, cerebrovascular mortality[419].

Respiratory system

There are few studies that have explored the respiratory outcomes of long-term and short-term exposure to noise pollution. Two pioneering studies that explored this relationship identified a correlation between noise pollution exposure, asthma, and chronic bronchitis in children[420]. However, it was postulated that these findings were confounded by exposure to NO_2 and SO_2[421].

Reproductive system

The impacts of long-term and short-term noise pollution exposure on reproduction have not been adequately explored and the evidence that has been found to date is questionable[422]. Overall, the few studies that have been conducted to date have found that, to some extent, noise pollution has no harmful effect on birthweight, congenital abnormalities, and preterm births[423].

Core benefits of reducing noise pollution

There is limited evidence that substantiates human health co-benefits of noise pollution reduction. However, one source found that introducing low-carbon policies, such as advancing vehicle electrification technologies (e.g., electric, hydrogen, fuel cell, or hybrid cars), have the benefit of reducing noise pollution as well as meeting climate change goals[424]. Electric vehicles are significantly quieter, and often have noise barriers and quieter tires that alleviate noise pollution in highly trafficked areas[425].

Advancing vehicle electrification technologies (e.g., electric, hydrogen, fuel cell, or hybrid cars), have the benefit of reducing noise pollution as well as meeting climate change goals.

CONCLUSION

This report presents the potential health consequences arising from exposure to an array of pollutants, as well as the potential health co-benefits of reducing pollution brought on by the implementation of low-carbon infrastructure projects. Energy and climate policymakers and decision-makers alike may reference this report support arguments about possible human health co-benefits associated with low-carbon infrastructure. The information detailed stands as a reference and resource to further advocate for health considerations in environmental and clean economy efforts.

While health co-benefits include a broad range of ancillary benefits (from improvements to the healthcare system to the shift to a more active and healthier lifestyle), this report focuses on co-benefits that have direct physical or mental health outcomes. This analytical focus allows the understanding of the potential impacts of low-carbon projects on the health of members of a specific community.

Three sectors that play key roles in local built environments - buildings, energy, and transportation – were chosen in the analysis in order to advance understanding and provide key insight on how the implementation of low-carbon infrastructure might benefit the health of local communities. The report contributes to advancing the discussions regarding how low-carbon infrastructure project can improve the air quality of a community and health outcomes alongside their broader contributions to emissions reductions.

Key messages

Exposure to air, water, and noise pollution can result in an increased severity and occurence of diseases and conditions targeting various physiological systems in the human body. Consequently, mortality, adverse birth outcomes and other negative health impacts can be observed in communities following prolonged exposure. The reduction of these pollutants is desirable from both environmental and health perspectives, and the implementation of low-carbon infrastructure provides a viable option to reduce air pollution, which provides a range of potential health co-benefits.

The literature on the impacts of air pollution on health, and the available evidence on the health co-benefits of reducing air pollution are vast, as this report shows. Some noteworthy evidence unveiled by this report are:

- **The implementation of low-carbon infrastructure offers a wide potential to improve air quality and, consequently, improve human health.**

- **The most prominent and strongly established evidence of health benefits from reducing air pollutants through, which can be accomplished by implementing low-carbon infrastructure, are the significant positive impacts on cardiovascular and respiratory systems.** Across all pollutants where strong and mixed evidence exists, the incidence and severity of pollutant-related disease on these two systems is strong.

- **Reductions in air pollution offer an opportunity to reduce occurrrence of all-cause and specific disease-related mortality.** Researchers have only begun to fully comprehend the extent to which air pollution is a risk factor for mortality. Some recent estimates suggest that fossil fuel combustion emits air pollutants that are linked to an annual 10.2 million global premature deaths (Vohra, 2021). The argument that reducing air pollution has a great potential to reduce pollutant-related deaths is backed by strong scientific evidence.

- **There is a strong possibility that the implementation of low-carbon infrastructure may lead to even broader health benefits than previously thought.** Low-carbon infrastructure can reduce a wide range of pollutants such as POPs. However, the full extent of the project-related co-benefits originated from reducing POPs is yet to be extensively investigated. Greater research is needed to identify the role that low-carbon infrastructure can play in reducing POPs and VOCs, which have negative health impacts. This could further substantiate the health case for these investments.

- **Greater research is needed into other environmental health pollutants, such as noise, to improve the understanding of their health impacts on different organ systems.** Scholarly studies have established robust evidence on the impact of many air pollutants on human health, and the co-benefits associated with reducing air pollution. The strongest available evidence is associated with $PM_{2.5}$/ PM_{10}, NO_2, SO_2 and ozone. However, more research is needed into other environmental health pollutants to improve the understanding of their health impacts, and better identify the strength of the relationship between a given pollutant and its effects on different organ systems.

This report also offers a high-level picture of the current distribution of pollutants by province or territory, in addition to the sub-sectors within buildings, energy and transportation sectors that are primarily responsible for the release of pollutants. This allows the identification of sectors and specific target areas to focus on, in which low-carbon infrastructure projects become an appealing option to reduce emissions and realize health co-benefits.

While this report considers a subset of health co-benefits that accompany projects, it is important to acknowledge that there are other co-benefits that can still be considered in the assessment of low-carbon infrastructure projects, such as health equity considerations, the life cycle impacts of projects resulting from disposal, or nature-based solutions and green spaces.

APPENDIX 1: RESEARCH METHODOLOGY

The literature review that underpins this report followed state of the art methodological techniques[437] to identify the most recent and robust evidence on human health co-benefits associated with a set of low-carbon infrastructure projects. The review was conducted by three researchers, all following pre-established protocols developed with the assistance of university librarians to allow uniformization of evidence collection and analysis.

The majority of studies collected were peer-reviewed articles. Occasionally, health guidelines and reports from grey literature were also collected. The large majority of these materials were sourced from six bibliographic citation databases, four of which are specialized in biomedical, pharmaceutical and environmental sciences :

- Medline: A bibliographic database including work related to medicine and health care.
- EMBASE: A biomedical and pharmaceutical bibliographic database.
- APA PsycInfo: A database to source bibliographic materials on the mental health impact of pollutants.
- GreenFile: A multidisciplinary database covering environmental science studies.
- Scopus: A comprehensive citation database covering several subject disciplines.
- Web of Science: Like Scopus, a comprehensive citation database covering several subject disciplines.

In total, more than 280 articles, systematic reviews, and reports were collected through the three-step search strategy outlined below :

1. **A preliminary scan**: A precursor to the systematic collection of bibliographic evidence, the rationale behind this preliminary scan was to identify key concepts used in scholarly research, as well as the characteristics and factors related to health co-benefits of low-carbon infrastructure that are important to address in a more systematic review. The findings from this preliminary scan informed the selection of key search terms used in the second step, as well as the development of the coding structure used in the analysis of all documents retrieved[438].
2. **A systematic search**: This step involved a search strategy to identify and collect bibliographic material that would allow uncovering international evidence, and producing evidence-based statements to guide decision-making. The following eligibility criteria guided the selection of bibliographic sources in the systematic review: a) research focusing on pollutants originating from fossil-fuel combustion sources, b) research identifying and describing human health outcomes of pollutants from carbon emissions. The selection of sources was decided by a careful examination of the title and abstract of the work. If the material proved unfit for the purposes of the review during the analysis phase, the material was excluded from the dataset and its removal was recorded and justified.
3. **Snowballing search technique**: In a third step, reviewers collected and examined the references from the material collected above to fill in remaining gaps and identify missed systematic reviews.

To manage the material retrieved, all citations were uploaded to the reference management software Zotero, and the document files were centralized in a shared folder. The bibliographic material was analyzed with the qualitative analysis software NVivo 12. A pre-established code structure was shared among all reviewers to ensure data is extracted and presented in a structured and consistent way. The coding structure was pretested before sharing among the reviewers in order to avoid perception bias. Having received the coding structure, all reviewers conducted a pilot-test of codes. A group debriefing assessment prior to the review of bibliographic material attested the reliability and validity of codes. To ensure consistency of understanding and use of the coding structure among all reviewers, the review protocol listed the definition and the rationale of all codes used in the analysis of the bibliographic material.

For reproducibility and transparency purposes, the coding structure and review protocol are available upon request.

APPENDIX 2: GLOSSARY OF TERMS

Cardiovascular system: The cardiovascular system is composed of the heart and blood vessels[439]. The heart pumps blood throughout the body using blood vessels as a network of tubes to deliver blood to organs and tissues[440].

Gastrointestinal system: The gastrointestinal system consists of the luminal and hepato-biliary-pancreatic parts[441]. The luminal section is more commonly referred to as the digestive tract and mainly involves the degradation and absorption of food[442]. The hepato-biliary-pancreatic includes the liver, salivary glands, and pancreas all of which produce important substances to assist with digestion and absorption of food[443].

Mental health: Mental health moves beyond just the absence of mental disorders and encompasses "a state of wellbeing" where individuals are able to (1) realize their abilities, (2) cope with the stressors, (3) work productively, and (4) contribute to their communities[444]. An interplay between social, psychological, and biological factors can determine the level of mental health a person experiences at a given time[445].

Mortality: Mortality refers to death[446]. In public health, it relates to the number of deaths resulting from a specific health event[447].

Neurological (nervous) system: The neurological system, more commonly referred to as the nervous system, is structurally divided into the central nervous system (CNS) and peripheral nervous system (PNS)[448]. The CNS, containing the brain and spinal cord[449]. The other nervous structures existing outside of the CNS composes the PNS[450]. Functionally, the nervous system is divided into the somatic part, which receives and responds to information external to the body, and the visceral part, which detects and responds to information from within the body[451].

Noncommunicable diseases: This class of diseases including cancer and diabetes, that are the leading cause of global mortality. Several modifiable factors like poor diet, obesity, and lack of physical activity, can accelerate the development of noncommunicable diseases[452].

Reproductive system: The reproductive system consists of the reproductive tract and is accompanied by interactions with hormonal glands within the body[453]. It mainly serves as a system to facilitate puberty, reproduction, and fetal development[454]. This report also included birth outcomes under this category.

Respiratory system: The respiratory system mainly concerns the lungs which participates in controlling breathing, gas exchange, speech, air filtration, and other metabolic activities[455]. In addition to retaining oxygen and exhaling carbon dioxide, the lungs employ defenses mechanisms to protect the body from taking up harmful particles[456].

Urological system: This system mainly focuses on the anatomy and functions of the urinary tract, inclusive of the kidneys and the male reproductive system[457].

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4 The term health co-benefits is defined in the "What are health co-benefits?" section of this report.
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6 The Lancet, 2020.
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8 This is not a comprehensive list of all health-related impacts from climate change in Canada. Others include increased rates of allergens, potential increased rates of cancer, reductions in overall mental health and well-being, and socio-economic impacts that negatively amplify existing health inequities, to name a few (CAPE, 2019).
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ACKNOWLEDGEMENTS

This report was written by Aline Coutinho, Harshini Ramesh, and Madeleine McGillivray. Editing and writing support was provided by Harshini Ramesh and Sushant. This report would like to thank Health Canada, Maryam Shekarrizfard of the Atmospheric Fund, and two anonymous reviewers, for providing insight and guidance to this research report. Responsibility for this final product is Smart Prosperity Institute's alone, and should not be assigned to any reviewers, interviewees or any external party. Being interviewed or reviewing this report does not mean endorsement, and any errors remain the authors responsibility.