Major Human Health Impacts of the Kinder Morgan Trans Mountain Pipeline Expansion

A Report for BROKE and NOPE

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1 Executive Summary

1.1 Introduction & Background

The health and environmental impacts of fossil fuel use are extensive and varied. This report examines some important impacts on human health of the Kinder Morgan Trans Mountain Expansion Project (TMEP). The most profound human health impacts of the proposed expansion have been administratively removed from consideration by the National Energy Board, those being from the contribution this large fossil energy project will make to the global problem (with local impacts) of increased greenhouse gases. Therefore, this report will only discuss other health impacts that are also underestimated in the Trans Mountain (TM) proposal and accompanying human health risk assessment. The primary health outcome of concern is childhood leukemia arising from potential benzene exposure as a result of both routine operations at West Ridge Terminal and a pipeline or tanker spill.

Over the past year of work leading up to this report, several requests were made to local health authorities to generate a new risk assessment based upon less ideal conditions than those outlined by TM. The Fraser Health Authority, Vancouver Coastal Health, Metro Vancouver, BC Centres for Disease Control and the BC Cancer Agency were all asked to contribute. Due to the resources required, the organizations did not feel equipped to carry out a new risk assessment so this has not been done. In the two rounds of Information Requests, TM would not provide the necessary information regarding model inputs to adequately access the human health risk assessment (HHRA) undertaken as part of the proposal. Citing proprietary aspects of the modelling, the TMEP effectively circumvents important scrutiny of the model inputs and therefore its conclusions lack validation.

In 2013, the oil and gas company, Kinder Morgan, proposed a pipeline expansion project for their Trans Mountain Pipeline that connects Strathcona County Alberta (near Edmonton), to Burnaby British Columbia (BC) (Trans Mountain, 2014). This expansion project would increase the pipeline capacity from 300,000 barrels of oil per day to 890,000 barrels of oil per day transported to the BC coast (Trans Mountain, 2014). The proposed route for this expansion includes 987 kilometres of new pipeline carrying a mix of crude oil, refined and semi-refined products together in the same line (Kinder Morgan, 2014; Trans Mountain Pipeline ULC, 2014). A portion of this pipeline expansion is proposed to run through the geographically sensitive landscape of Burnaby Mountain and surrounding residential area to the Westridge Marine Terminal at the heavily populated areas of Metro Vancouver around Burrard Inlet.

The review was sponsored, in part, by North Shore NO Pipeline Expansion (NS NOPE) and Burnaby Residents Opposing Kinder Morgan Expansion (BROKE). Both the City of Burnaby and communities along the North Shore will be directly affected by the expansion project via pipeline construction through Burnaby Mountain, increased tanker traffic, spill risk and corresponding emissions in Burrard Inlet.

1.2 Methods & Organization of the Report

The authors first conducted a comprehensive search for relevant literature available in the public domain. They then employed several key frameworks and theories (a population health framework, risk assessment and future climate projections) to assess the potential impact of the TMEP on human health. A Population Health Framework is foundational to health promotion and protection. It is used to examine the complex interactions and linkages between the social, physical, economic and political factors that affect health, as well as the biological factors that contribute to health and illness. Risk Assessment is a quantitative framework for identifying and evaluating levels of risk that are associated with adverse events or hazards (Frumkin, 2010). In undertaking this review, the authors sought to answer three questions: what can happen? how likely is it to happen? and, what are the consequences if it does happen?

For human health risk assessment, it is crucial to consider the population exposed to the particular hazard in question. This includes the number of individuals and any vulnerabilities they may have (such as age, concomitant disease risk and genetic susceptibility to the hazard). The TM HHRA uses spill and emission scenarios with lower risk than would likely be felt in Burrard inlet and surrounding communities if the scenarios were chosen where the bulk of the population lives.

We have chosen to focus on the acute and chronic health effects of exposure to two chemical toxicants found in crude oil and diluted bitumen (dilbit): benzene and 1,3-butadiene. Both have long been known as harmful to human health and are recognized worldwide as known human carcinogens. Further, as described in Section 4 of this report, there is clear evidence connecting adverse health outcomes with exposure to these chemicals, particularly in vulnerable populations. Of particular concern is the risk of childhood leukemia. This outcome is inadequately accounted for by the TMEP human health risk assessment (HHRA).

The report also addresses other health impacts that would have large attributable risk to the populations who live in the regions around the proposed pipeline expansion. Section 5 of the report describes the potential impact of two plausible worst-case scenarios of the TMEP: operations at Westridge Marine Terminal during extreme weather conditions and following a

Second Narrows marine spill. Both scenarios would have detrimental effects on the residential population within the potentially affected areas including impacts on health, quality of life, real estate values, fire hazard, economic impacts, environmental impacts and more.

1.3 Potential Impacts on Human Health if the TMEP Goes Ahead

Benzene and 1,3-butadiene are chemical toxicants found in crude oil and dilbit. Both are Class 1 (most potent) human carcinogens with no known safe threshold of exposure. An increase in exposure to benzene and 1,3-butadiene, as is likely with the expansion of the TM pipeline and cuold have significant health impacts on the population. This could lead to increased healthcare costs and burden of disease among the exposed community. Our regulators need to focus on further reducing today's emissions not adding to the health burden. Section 4 of the report summarizes the major health effects associated with exposure to benzene and 1,3-butadiene.

1.3.1 Health Effects of Exposure to Benzene

Exposure to benzene can lead to number of health effects depending upon the route of exposure, the amount and the duration of exposure time, as well as the age of the individual exposed and whether or not there are pre-existing medical conditions. Acute exposure to benzene can cause dizziness, drowsiness, rapid/irregular heartbeat, loss of consciousness, tremors, convulsions, and death. Chronic exposure to low levels of benzene can cause destruction of the red blood cells (aka haematotoxicity), toxicity to the immune system (aka immunotoxicity), and the formation of tumours (aka neoplasia). Benzene and the BTEX[1] family of chemicals have also been linked to developmental and reproductive issues. These include: low sperm counts and sperm abnormalities; and, neurological, psychological, and behavioural abnormalities and neural tube defects in infants as a result of maternal exposure.

Epidemiological studies have shown that there is an association between prenatal exposure to benzene and the development of childhood hematopoietic[2] cancers such as leukemia. Benzene has been observed in foetal cord blood with levels similar to or higher than levels found in the maternal blood. The implication of this is that benzene can cross the placental barrier during pregnancy and can be transported from mother's blood to foetal blood.

The chronic condition of most concern in relation to the TMEP is acute myeloid leukaemia (AML). AML is a type of cancer, which affects the blood stem cells in bone marrow. AML is the most common childhood cancer and is the leading cause of death from cancer in children.

[1] BTEX is the acronym for benzene, toluene, ethylbenzene and xylene.

[2] The hematopoietic system is the system responsible for the formation of blood cells.

However, its incidence rate also increases with age and, as a result, more adults die of this disease than children (Jemal *et al*, 2002).

1.3.2 Health Effects of Exposure to 1,3-Butadiene

Butadiene is classified as Group 1, *carcinogenic to humans*, by the International Agency for Research on Cancer (IARC, 2012). Studies on human exposure to 1,3-butadiene demonstrate links to leukemia, cancer of haemolymphatic organs, adverse cardiovascular and respiratory effects, and damage to the central nervous system. Acute exposure to 1,3 butadiene by inhalation can lead to irritation of the eyes, nasal passages, throat, and lungs (Environmental Protection Agency, 2009; Carpenter *et al*, 1944; Larionov *et al*, 1934). At very high levels of exposure, inhalation can lead to blurred vision, fatigue, headache, and vertigo. Exposure through skin contact can cause a sensation of coldness followed by a burning sensation, which may have the potential for frostbite.

1.3.3 Potential Impacts on the Health of Vulnerable Populations

When examining the potential health impacts of the TMEP, it is important to consider whether there are populations that are more vulnerable to the risk of exposure. Vulnerable populations can include those who are economically disadvantaged, racial and ethnic minorities, low-income families, the elderly, the homeless, those with chronic health conditions (including severe mental illness), those who live in remote communities or who have difficulty accessing certain services and those with genetic susceptibility as described in this report. The following may also enhance these vulnerabilities: race, ethnicity, age, gender, sexual orientation, socioeconomic status, and genomic instability (AMJC, 2006).

Although it is important to note that individuals in all age groups could be affected by the pipeline expansion project, there are a few groups in particular that are at greater risk – namely, children, women of childbearing age, and older adults. The needs of these populations are serious and exposure to environmental contaminants can be debilitating or detrimental to their health.

Children have a particular physiological vulnerability to air pollution due to their immature organs, narrower airways, developing lungs and respiratory system, behaviour and lack of knowledge (Natural Resources Defense Council (NRCD), 1997; Health Canada, 2011). Children are often more active than adults and have greater respiratory rates due to their smaller size. As a result, irritation caused by pollutants or environmental contaminants that may only produce minor responses in adults can result in potentially more significant respiratory obstruction in children. Children are also at higher risk than adults for AML, the primary health outcome of interest in this report.

Environmental contaminants can have significant health consequences for women of childbearing age, and, in particular, expectant mothers. The foetus can be exposed to a number of chemical contaminants that enter through the placenta, causing developmental abnormalities, including to the central nervous system whose development is particularly sensitive (Health Canada, 2011). Depending on which stage of the pregnancy exposure occurs at, there can be detrimental health and developmental impacts for both the mother and neonate (Health Canada, 2011).

Older adults are more likely to suffer from comorbidities (i.e., having one or more chronic conditions) making them more susceptible to environmental hazards. Even those in good overall health are more vulnerable than the average population (Health Canada, 2011). According to Health Canada, their bodies also contain a "lifetime worth of environmental contaminants, many of which can remain in their systems for decades". This higher "body burden" of environmental contaminants, potential cumulative effects, and general susceptibility to disease puts older adults at increased risk to hazards in the environment.

According to the 2011 census, approximately 20% (or 44,250) of the Burnaby population and 24% (20,860) of the North Shore population consist of children and youth under 19 years of age. Approximately 9.3–10.4% of the population in both municipalities is under 10 years. Approximately 30% (34,055) of the Burnaby population and 20% (16,915) of the North Shore population consist of females of childbearing age between 20–39 years. Approximately 13.8% (30,900) of the Burnaby population and 15.5% (13,130) of the North Vancouver district population consist of adults over the age of 65 years.

1.3.4 Potential Impacts on Human Health of an Oil Spill

It is not implausible that an oil spill could occur in Burrard Inlet if the TMEP goes ahead. Since its construction in the 1950's, the TM pipeline has reported approximately 82 spills to the National Energy Board of Canada, 13 of which have been within the last 10 years. These spills have released a mix of crude oil, oil waste, natural gas, and isooctane into the environment and surrounding communities (Trans Mountain, 2014).

As shown in Section 5.3 of the report, a number of epidemiological studies on human health after a marine oil spill also describes acute toxic and psychological effects. These include headaches, eye and throat irritations, breathing difficulties, nausea, vomiting, and skin rashes (Rodriguez-Trigo *et al*, 2007; Sim *et al*, 2010). In addition, both asthma and negative mental health experiences have been directly associated with past oil spills and have significant population attributable risk (Sim *et al*, 2010; Lyons *et al*, 1999). Although asthma and negative

mental health outcomes are considered less severe than cancer or reproductive outcomes and are potentially less costly to the healthcare system on a case-by-case basis, these illnesses are more widespread among the exposed populations and can greatly impact the larger community's quality of life and well being.

1.4 Key Conclusions

We conclude that the proposal does not adequately assess the human health risks posed by the proposed expansion. Information to understand the human health impacts in the community surrounding the terminal and the exit for ships through First and Second Narrows needs to be generated in order to better understand the impacts outlined in this report. This information should include a focus on those most vulnerable to the effects of petroleum products transported by TM, namely the developing foetus, young children and those with genetic susceptibility to carcinogens such as benzene.

Accidents happen and they appear to happen frequently at TM installations. The risk of a significant spill by TM has been amply demonstrated twice in Burnaby in the past eight years. Though not TM, the April 2015 tanker spill in English Bay is only the latest in a string of bad events all of which could have been much worse. The increased pipeline and tanker traffic is likely to increase human health risk. Such a risk is unacceptable to the communities likely to be affected.

Lastly, it is critical to the integrity of the public process to recognize the impact this project has on climate change and to understand the project risks in terms of future climate projections. The authors of this report acknowledge climate change as a serious public health concern that will affect many generations in the future. This is of paramount importance and is a factor that is completely ignored in the TMEP application.

2 Introduction

2.1 Background

In 2013, the oil and gas company, Kinder Morgan, proposed a pipeline expansion project for their Trans Mountain Pipeline that connects Strathcona County Alberta (near Edmonton), to Burnaby British Columbia (BC) (Trans Mountain, 2014). This expansion project would increase the pipeline capacity from 300,000 barrels of oil per day to 890,000 barrels of oil per day transported to the BC coast (Trans Mountain, 2014).

The proposed route for this expansion includes 987 kilometres of new pipeline carrying a mix of crude oil, refined and semi-refined products together in the same line (Kinder Morgan, 2014; Trans Mountain Pipeline ULC, 2014). A portion of this pipeline expansion is proposed to run through the geographically sensitive landscape of Burnaby Mountain and surrounding residential area to the Westridge Marine Terminal at Burrard Inlet. If the proposed expansion is approved, this will become one of the largest pipelines on BC's coast, and the one located within the densest human population. The intersection of an urban setting and a high volume pipeline has the potential to result in significant, devastating health and environmental impacts.

The authors have been contracted to examine some of the important impacts of the TMEP in Greater Vancouver. The primary focus of this review was the following:

1. to determine the current standing of the Kinder Morgan Trans Mountain Expansion Project,
2. to undertake an in-depth review of the major chemicals of concern: benzene and 1,3-butadiene and their health impacts
3. to describe alternate and plausible case scenarios of oil spills and related health outcomes; and
4. to provide recommendations for and areas of concern regarding the inadequacy of the TMEP application.

The review was sponsored, in part, by North Shore NO Pipeline Expansion (NS NOPE) and Burnaby Residents Opposing Kinder Morgan Expansion (BROKE) for the public comment process on the TMEP application pertaining to these residential areas. Both communities will be directly affected by the expansion project via pipeline construction through Burnaby Mountain and/or increased tanker traffic, spill risk and both these corresponding emissions and the routine operations emission from expanded ship and barge loading in Burrard Inlet.

2.2 Methods

2.2.1 Literature Search

A comprehensive search for relevant literature available in the public domain was conducted. This search included both peer-reviewed articles and limited grey literature from oil and gas corporations. Sources used to conduct the search were primarily from the Internet: Kinder Morgan Trans Mountain Pipeline Expansion Application, Google Scholar, Pub Med, Web of Science, Statistics Canada, Environment Canada, and the Agency for Toxic Substances and Disease Registry reports. The snowball technique was applied to identify other primary sources relevant to the topic of interest. Key search terms are included in Section 10.1.

2.2.2 Literature Review and Evaluation

The authors employed several key frameworks and theories that are foundational to the project: a population health framework, risk assessment and future climate projections.

A Population Health Framework is foundational to health promotion and protection (see Section 10.2). It is used to examine the complex interactions and linkages between the social, physical, economic and political factors that affect health, as well as the biological factors that contribute to health and illness. This approach "is a unifying force for the entire spectrum of health system interventions - from prevention and promotion to health protection, diagnosis, treatment and care" (Public Health Agency of Canada, 2013). Environmental health is one aspect of this framework and includes risk assessment, our secondary framework.

Risk Assessment is a quantitative framework for identifying and evaluating levels of risk that are associated with adverse events or hazards (Frumkin, 2010). In the field of environmental health, risk assessments primarily focus on health impacts that may occur from chemical exposures by living or working in a particular environment (Frumkin, 2010). According to Kaplan (1997), risk assessment seeks to answer three questions:

1. What can happen?
2. How likely is it to happen? and
3. What are the consequences if it does happen? (Frumkin, 2010, p. 1038).

Environmental health risk assessments are often referred to as a combination of 'science and judgment' (National Research Council, 1994). They synthesize existing scientific evidence to provide a basis for sound decision-making that will influence specific policy issues and regulations (Frumkin, 2010, p. 1038). As one of the underlying frameworks for this report, risk assessment concepts and key terms will be referred to throughout. For human health risk

assessment, it is crucial to consider the population exposed to the particular hazard in question. This includes the number of individuals and any vulnerabilities they may have (such as age, concomitant[3] disease risk and genetic susceptibility to the hazard.

2.3 Organization of the Report

In preparing the report, the authors focussed on two prevalent chemical toxicants found in the Trans Mountain pipeline, namely benzene and 1,3-butadiene, and the health risks associated from both chronic and acute exposure. Benzene and 1,3-butadiene are both constituents of transported crude oil and were selected as the primary focus of this report based on clear evidence connecting adverse health outcomes with exposure, particularly in vulnerable populations. Both compounds have long been known as detrimental to human health and are recognized worldwide as known human carcinogens.

After describing the health impacts of exposure to benzene and 1,3-butadiene, the report addresses other health impacts that would have large attributable risk to the populations who live in the regions around the proposed pipeline expansion. The review also draws on health effects evidence gathered from communities proximal to recent oils spills, such as the 2007 Hebei Spirit oil spill in Taean, Korea, and the community impacts still emerging from the Deep Water Horizon oil spill in the Gulf of Mexico.

This report also identifies two potential worst-case scenarios of the TMEP. The first describes daily operations at Westridge Marine Terminal during extreme weather conditions and the second outlines the potential impact to Greater Vancouver area of a Second Narrows marine spill. Both scenarios would have detrimental effects on the residential population within the potentially affected areas including impacts on health, quality of life, real estate values, fire hazard, economic impacts, environmental impacts and more. The report focuses on the risk of childhood leukemia because this is inadequately accounted for by the TMEP human health risk assessment (HHRA). In our opinion, the TM HHRA uses spill and emission scenarios with significantly lower risk than would be felt in Burrard inlet and surrounding communities.

[3] A concomitant disease risk is a disease that occurs or exists at the same time as another.

3 Background

3.1 The Kinder Morgan Trans Mountain Project

3.1.1 The Current Pipeline

In 1953, KM constructed an 1150 kilometre Trans Mountain Pipeline connecting Edmonton, Alberta to the coastal ports of Burnaby, British Columbia. The Westridge Marine terminal in Burnaby BC was constructed in 1957. It is used to load different kinds of fuel oil onto the Aframax or Panamax class tankers and has been used for exporting crude oil by the Trans Mountain pipeline from Alberta to California and Asia. Aframax are medium sized tankers, 245m in length, with a carrying capacity of 70,000 – 100,000 metric tonnes or 750,000 barrels (Maritime Connector, undated). In addition, jet fuel is unloaded from tankers and barges at the terminal (Volume 4 section B of Trans Mountain Pipeline Expansion application). We have chosen to primarily focus on Westridge Marine terminal due to the vast array of changes that would result at this terminal in the proposed pipeline expansion.

At present, the Westridge Terminal contains one dock with one berth. The routine emissions from this loading include benzene, 1,3 butadiene and other volatile carcinogenic compounds with no known safe risk threshold. Currently, based on current market demands, five vessels and three barges are loaded with crude oil and one to two barges are unloaded at this dock each month. There are also three storage tanks holding approximately 290,000 bbl of jet fuel reserves that are then transported via pipeline to Vancouver International Airport.

3.1.2 The Proposed Expansion

In 2013, TM proposed a pipeline expansion project for their Trans Mountain Pipeline that connects Strathcona County, Alberta (near Edmonton) to Burnaby, British Columbia (Trans Mountain, 2014). The proposed route for this expansion includes 987 kilometres of new pipeline carrying a mix of crude oil, refined and semi-refined products together in the same line (Kinder Morgan, 2014; Trans Mountain Pipeline ULC, 2014) to the Westridge Marine Terminal at Burrard Inlet. A portion of this pipeline expansion is proposed to run through the geographically sensitive landscape of Burnaby Mountain and surrounding residential area. The expansion project would increase the pipeline capacity from 300,000 barrels of oil per day to 890,000 barrels of oil per day (Trans Mountain, 2014).

According to the TMEP plans, the Westridge Marine terminal will be modified so that it is able to load three Aframax-sized tankers from the current one, further expanding construction into Burrard Inlet (Kinder Morgan, undated). In addition, a small dock would be incorporated with

multiple berths to accommodate tugboats, pilot boats, spill response vessels and equipment, and boom boats. See Section 10.3 for TM's graphic representation of Westridge expansion based on preliminary designs. Additionally, there will be two added delivery lines connecting Burnaby terminal to Westridge Marine Terminal bringing the total to three delivery lines (Kinder Morgan, undated).

With the pipeline expansion, the vessel calling is expected to rise from its present state of 3 percent to 14 percent of the total traffic moving through the Port Metro Vancouver. Due to increasing market demand of crude oil, primarily diluted bitumen, modifications are expected to be made at dock and foreshore terminal of Westridge Marine terminal to serve approximately 34 Aframax tankers every month with no reported changes in number of loading and unloading barges. The modifications to the foreshore terminal include the following: excavation of undesirable elements and installation of the infill materials with densification of the existing infill regions, excavation of the area leading to the terrace for Vapour Recovery Unit process tanks, construction of an access road system and other associated modifications. The proposed layout of the terminal has been "conditionally" approved by Port Metro Vancouver representatives, the Pacific Pilotage Authority and BC Coast Pilots, (Volume 4 section B), though it has not received necessary air quality permits. Were expansion to go forward hazardous emissions into the populated areas surrounding the terminal would increase.

3.2 Products Transported by the Pipeline

Table 1: Products Transported in Trans Mountain Pipeline

Product Type	Example	Description
Refined Petroleum	Gasoline, Diesel, Iso-Octane	Refined
Synthetic Crude Oil	Processed Bitumen	Semi-refined
Light Crude Oil	Conventionally sourced crude oil	Un-refined
Heavy Crude Oil	Diluted Bitumen	Un-refined

(Products Shipped in Pipeline, Trans Mountain, 2014)

These products are carried in the same pipeline through a process called 'batching' (Trans Mountain, 2014). See Figure 1, below, for an illustration of the batching process. Products (which include crude oil, refined, and semi-refined products) are added to the pipeline in a particular sequence based on density and other characteristics so as to reduce "product interface" or mixing (Trans Mountain, 2014).

Figure 1: Kinder Morgan Batching Technique

Bitumen is a form of crude oil extracted from the Canadian oil and tar sands as a semi-liquid material at room temperature. It contains various volatile organic compounds (VOCs), "such as benzene, toluene, ethylbenzene, xylenes, and polycyclic aromatic hydrocarbons (PAHs)" (Kim, Park, Choi, et al, 2013, p.1). Because components of extra heavy oil are often naturally mixed with bitumen (resulting in a very viscous tarlike substance), it must first be diluted in order to move through the pipelines (Government of Canada, 2013). Diluent is light petroleum distillate and a by-product of natural gas that is added to bitumen to decrease the viscosity and density of the product and to aid in transportation (Crosby et al, 2013). This new compound of bitumen plus diluent is more commonly referred to as "dilbit". According to a report by Environment Canada, Fisheries and Oceans Canada, and National Energy Resources, "typically, dilbit consists of blends of 20% to 30% condensate and 70% to 80% bitumen (Crosby et al, 2013; Crude Quality, 2013)." It contains benzene in varying quantities depending upon the source.

The TMEP Application and projected spill modelling use TM's primary oil composition commonly referred to as the Cold Lake Winter Blend (CLWB). According to the TMEP Application, Cold Lake Winter Blend diluted bitumen (dilbit) is expected to remain the main product transported in the expanded pipeline. This blend of oil and diluent contains a high level of volatile organic compounds (VOCs) that are released as vapours from the marine terminal and tankers during loading operations (Trans Mountain Pipeline ULC, 2014, p. 3-3).

3.3 Main Contaminants of Concern

As noted above, crude oil contains a wide range of volatile organic compounds. This report focusses specifically on benzene and 1,3-butadiene. According to Section 11 of the *Canadian Environmental Protection Act* (CEPA), both of these compounds fulfill all the requirements of being a toxic material:

> "...a substance is toxic if it is entering or may enter the environment in a quantity or concentration or under conditions
>
> a. having or that may have an immediate or long-term harmful effect on the environment;
> b. constituting or that may constitute a danger to the environment on which human life depends; or

c. constituting or that may constitute a danger in Canada to human life or health.

(Health Canada, 1993)

Information on the chemical composition, sources and pathways of human exposure is set out below. A high level description of the health effects of exposure is provided in Section 4 of this report. Detailed information on the mechanisms underlying these health effects is provided in Sections 10.4 and 10.5.

3.3.1 Benzene

3.3.1.1 Chemical Composition

Benzene is a colorless liquid with a sweet odour. It is classified as an aromatic organic compound (hydrocarbon) with the molecular formula C_6H_6. Structurally, it has six unsaturated carbon atoms chemically bonded together in a hexagonal ring with alternating single and double bonds. Each carbon atom has one hydrogen atom attached to it. It evaporates quickly into the atmosphere, is slightly soluble in water and highly soluble in organic solvents because of its non-polar properties. The boiling point of benzene is 80.1 degree Celsius and the melting point is 5.5 degree Celsius (Carey, 2014).

3.3.1.2 Sources of Exposure

Benzene is an important component of gasoline (1-4%) due to its high octane number, which gives the high compression rates for the fuel to prevent knocking (Kirk *et al*, 1983). On average, benzene content in premium and regular unleaded gasoline is 2.15% by weight or 1.76% by volume (Madé, 1991). The quantities of benzene in diluent are similar.

Major sources of benzene exposure from human activities include emissions from automobiles, automobile refuelling process and industrial emissions. Exposure to benzene occurs from direct dermal contact at workplace settings where benzene is used as a manufacturing component, inhalation from contaminated air, consumption of contaminated groundwater resulting from the leakage of underground storage tanks, landfills, or deposits of natural gas. The petroleum industry is one of the most important sources of benzene emissions. These emissions can arise during the processing of petroleum products, coking of coal, and during the production of toluene, xylene and other aromatic compounds used in petroleum industry. According to the Agency for Toxic Substances and Disease Registry (ATSDR), emission rates tends to be higher in areas of high automobile traffic, near gas stations, storage tanks and other facilities involving storage and distribution of petrol (ATSDR, 1991).

About 10% percent of benzene emissions are from gasoline where levels of benzene are restricted to be lower than 1% by weight set by US Environmental Protection Agency. Government of Canada regulations on benzene have prohibited the sale of gasoline with more than 1.5% benzene by volume (Environment Canada, 2014). If the expansion project goes ahead, a higher level of benzene can be expected to be present in the atmosphere since the increase in pipeline infrastructure releases would be added to the level of benzene already released into the environment through gasoline. This is could lead to a significant increase in human health risk because there is no safe threshold for the carcinogenic effects of benzene.

3.3.1.3 *Human Exposure Pathways*

Benzene can enter the human body through three pathways: inhalation, ingestion and dermal. Approximately 50% of the human exposure route for benzene is passed through the lungs and a very small amount is absorbed through the skin (Agency for Toxic Substances and Disease Registry, 1989).

3.3.2 1,3-Butadiene

3.3.2.1 Chemical Composition

1,3-Butadiene is also known as butadiene, biethylene, erythrene, divinyl, vinylethylene, and pyrrolylene (PubChem, undated). At room temperature, butadiene is a colourless gas with a mild aromatic or gasoline-like odour (PubChem, undated). It has the molecular formula C_4H_6 and a molecular weight of 54.09 g/mol. Butadiene has a low boiling point of -4^{o}C, a very low water solubility, and a vapour density greater than air.

3.3.2.2 Sources of Exposure

Anthropogenic sources (i.e., due to human activity) of concern for human exposure to butadiene include the following (Hughes *et al*, 2001):

- fugitive and combustion emissions from pipelines, pump stations, and storage terminals, during both construction and operations.
- internal combustion engines, which produce butadiene from incomplete combustion. with the amount generated depending on the composition of fuel, type of engine, and emission controls used (Hughes *et al*, 2001)
- butadiene-producing industries, such as plastics, petroleum, coal, and chemicals
- prescribed forest burning, cigarettes, waste incineration, and spillage.

Exposure to butadiene is highest in occupational settings (IARC, 2012). Several industries are important, including petroleum refining, production of purified butadiene monomer, production of butadiene-based rubber, plastic polymers, rubber, and plastic products.

Based on estimates by CAREX in the European Union (see Table 2, on the next page), in the top 10 industries, approximately 31,600 workers were exposed to butadiene from 1990 to 1993 (CAREX, 1999). The National Occupational Exposure Survey estimates that approximately 52,000 workers were exposed in the US (NIOSH, 1990).

Table 2: Estimated numbers of workers exposed to 1,3-butadiene in the European Union

Industry, occupational activity	
Manufacture of industrial chemicals	8300
Manufacture of rubber products	7100
Manufacture of plastic products not elsewhere classified	7000
Petroleum refineries	2200
Construction	1600
Manufacture of other chemical products	1300
Education services	700
Manufacture of transport equipment	700
Wholesale and retail trade and restaurants and hotels	600
Manufacture of machinery except electrical	500
TOTAL	31600

From CAREX (1999)

(Source: Hughes *et al*, 2001)

Non-occupational exposure to butadiene may occur in the surrounding vicinity of point sources. This can include gas volatilization, vehicle emissions, structural, wood, or brush fires, and cigarette smoking (IARC, 2008). Several surveys have been conducted in Canada to measure ambient concentration of butadiene. In one surveillance program, butadiene was detected in 80% of 9168 24-hour samples from 47 distributed sites across Canada from 1989 to 1996 (Hughes *et al*, 2001). The mean concentration was 0.3ug/m^3; the mean urban concentration was 0.4ug/m^3; the 50th percentile was 0.21 ug/m^3; the 95th percentile was 1.0 ug/m^3; the maximum concentration measured was 14.1 ug/m^3. Another survey collected 1611 outdoor air samples from 25 sites within 14 cities, towns, and rural locations in Ontario between 1990 and 1994 (Steer, 1996). The mean concentration of butadiene was 0.1 µg/m^3 and the maximum was 1.7 µg/m^3. Areas near industrial point sources of butadiene show higher concentrations at a maximum of 28 mg/m^3 and a mean of 0.2 mg/m^3 (Hughes *et al*, 2001).

In Canada, there is one manufacturer of butadiene located in Sarnia, Ontario (Environment Canada, 2000). It is estimated that 120 kilotonnes of butadiene was produced in 2001 with a domestic consumption of 87 kilotonnes (Camford Information Services, 1999). In 2012, 23 major industries that use butadiene – petroleum refineries, chemical manufacturing facilities, and one diamond mine – released a total of 21 tonnes of butadiene into the environment, disposed of 0.045 tonnes of butadiene, and recycled 0.006 tonnes of butadiene at off-site facilities (CAREX Canada, from The National Pollutant Release Inventory).

3.3.2.3 Human Exposure Pathways

There are multiple pathways for human exposure to 1,3-butadiene (Hughes et al, 2001). Inhalation is the predominant route of exposure; however, it may also be absorbed through the

skin or ingested. Butadiene is a highly volatile gas and is primarily released into the air (hence the dominance of exposure through inhalation). Butadiene is not expected to persist in the air as it undergoes rapid destruction from photo-initiated reactions. Butadiene is also in water and soil, but is not expected to be absorbed in substantial amounts due to rapid volatilization back into the air.

4 Health Effects of Exposure to Benzene and Butadiene

Both benzene and 1,3-butadiene are Class 1 (most potent) human carcinogens with no known safe threshold of exposure. An increase in exposure to benzene and 1,3-butadiene, as is likely with the expansion of the TM pipeline, can have significant health impacts on the population. This could lead to increased healthcare costs and burden of disease among the exposed community. This section of the report summarizes the major health effects associated with exposure to benzene and 1,3-butadiene. A detailed description of the relevant mechanisms is provided in Sections 10.4 and 10.5.

4.1 Benzene

Exposure to benzene can lead to number of health effects depending upon the route of exposure, the amount and the duration of exposure time, as well as the age of the individual exposed and whether or not there are pre-existing medical conditions. According to the World Health Organization (2010), “human exposure to benzene has been associated with a range of acute and long term adverse health effects and diseases, including cancer and aplastic anemia”. Benzene is considered to be a “non-threshold toxicant”. This means that there are chances of occurrence of adverse effects if exposed to any level of benzene (Health Canada, 1993).

4.1.1 Acute Health Effects

Acute health effects result from the immediate exposure to the toxicant and are generally severe and dangerous. However, once the toxicant is removed from the setting, these effects can subside (Helmenstine, 2014; Marcus, 1990). Acute effects of exposure to benzene result from accidents or misuse of benzene.

Exposure to lower levels of benzene (700-3000 ppm) can cause dizziness, drowsiness, rapid/irregular heartbeat, loss of consciousness, tremors, and convulsions. Brief exposure (5-10 minutes) to high levels of benzene (10,000–20,000 ppm) can lead to death and 7500 ppm for 30 minutes is considered dangerous for life (ATSDR, 2007).

Direct contact of eyes and skin with benzene can cause irritation, reddening and blistering of the skin and even damage to the cornea. If foods or beverages contaminated with benzene are consumed, the following symptoms can occur within minutes to hours: vomiting, irritation of the stomach, sleepiness, and coma (ATSDR, 2007). The single oral dose for acute effects to occur is 8.8 g which can cause vomiting, staggering gait, shallow and rapid pulse, somnolence, delirium, pneumonitis, and collapse (Sandmeyer, 1981).

4.1.2 Chronic Health Effects

Chronic health effects result from the long-term exposure to a toxic substance. Chronic exposure to low levels of benzene can cause destruction of the red blood cells (aka haematotoxicity), toxicity to the immune system (aka immunotoxicity), and the formation of tumours (aka neoplasia). Three kinds of bone marrow defects are linked to benzene exposure: aplastic anemia, chromosomal defects and cancer. The primary target for red blood cell changes is the bone marrow (where the stem blood cells differentiate into specific blood cells). Research suggests that the cells at higher risk of benzene toxicity are rapidly proliferating blood cells (Marcus, 1990). Several blood-related problems resulting from benzene exposure are reported such as pancytopenia, aplastic anemia, thrombocytopenia, granulocytopenia, lymphocytopenia and leukaemia. (Note: these diseases may be interlinked and not a discrete disease entity.) The specific effects are determined by the dose, the length of exposure and the stage of stem cell development affected (Galton, 1986).

Benzene exposure has been linked to changes in bone marrow, resulting in changes in humoral and cell-mediated immunity. Kipen *et al* (1989) and Yin *et al* (1987) have reported a decrease in circulating immune system cells (which could be subdivided into B and T lymphocytes depending upon whether they target the antigen directly or they require other cells to present the antigen to them to fight infections) and other blood elements with levels of benzene exposures from 15-75 ppm. Benzene is considered a human clastogen (Huff *et al* 1989). A clastogen is an agent that can cause disruption or breakage of chromosomes leading to chromosome rearrangements, deletion, or addition.

A specific chronic condition caused by exposure to benzene is the devastating disease in children and adults, acute myeloid leukaemia.

4.1.2.1 Acute Myeloid Leukaemia

Acute Myeloid Leukaemia (AML) is a type of cancer, which affects the blood stem cells in bone marrow. Specifically, it occurs in the “myeloid” line, which gives rise to white blood cells, to platelets (in some cases) and to red blood cells. In this case, the bone marrow is unable to differentiate the stem cells into the myriad of mature functional blood cells. This results in an

increased number of premature cells, which can pathologically differentiate into any kind of blood cell depending upon the environment it is developing in. These cells can proliferate at a high rate and begin to outnumber the normal blood cells in the bone marrow, disrupting the further production of normal blood cells.

According to World Health Organization criteria, a diagnosis of AML is established when the blood or bone marrow contains 20% or more of leukemic myeloblasts (Harris *et al*, 1999). AML is the most common childhood cancer and is the leading cause of death from cancer in children. However, its incidence rate also increases with age and, as a result, more adults die of this disease than children (Jemal *et al*, 2002). In rare cases, the first symptoms of AML might cause the formation of a solid leukemic mass (called chloroma) outside the bone marrow. It is possible for a person to show no symptoms and for AML to be diagnosed during a routine blood test (Abeloff, 2004). See Section 10.5 for a graphic representation of mechanisms of how AML occurs.

As an example of benzene's ability to cause leukemia, a recent study reported that living in close proximity to a gas station (a prime source of benzene) might be associated with childhood leukemia (Brosselin *et al*, 2009). Details of this study's findings are presented in Section 10.4.4.

4.1.3 Reproductive Effects (Pregnant Women, Neonates and Developmental Risks)

Epidemiological studies have shown that there is an association between prenatal exposure to benzene and the development of childhood hematopoietic[4] cancers such as leukemia. Benzene has been observed in foetal cord blood with levels similar to or higher than levels found in the maternal blood. The implication of this is that benzene can cross the placental barrier during pregnancy and can be transported from mother's blood to foetal blood (Dowty *et al*, 1976). This placental transfer has also been shown in studies of mice exposed to various levels of radiolabeled benzene (0, 5, 10, 20 ppm benzene from day 6 to day 15 during gestation). All concentrations of benzene caused changes in red blood forming cells. The study observed a number of changes in both the red- and white blood forming cells following exposure in utero and again in adulthood. The authors concluded that, at the current occupational exposure limit, in utero exposure to benzene causes changes in the hematopoietic system of mice. These changes could persist into adulthood leading to an increased risk of leukemia (Keller & Snyder, 1986).

Benzene and the BTEX[5] family of chemicals, associated with unconventional natural gas development, have been linked to developmental and reproductive issues (Webb *et al*, 2014).

[4] The hematopoietic system is the system responsible for the formation of blood cells.

[5] BTEX is the acronym for benzene, toluene, ethylbenzene and xylene.

Studies of mice exposed to these chemicals reveal that embryonic hematopoietic stem cells, isolated from mouse yolk sacs, are more susceptible to benzoquinone metabolite (1-10uM) than adult bone marrow stem cells (Zhu *et al*, 2013). Though benzoquinone affected both, lower concentrations decreased viability, proliferation and differentiation of embryonic stem cells. Embryonic stem cells were also more susceptible to DNA double stranded breaks than adult bone marrow stem cells.

Long term exposure of male rubber manufacturing workers to benzene and other BTEX chemicals is associated with low sperm counts and sperm abnormalities (including, but not limited to, morphological and chromosomal). A 1998 study by De Celis *et al* show low sperm counts alone or in combination with other abnormalities in 50% of workers compared to 8% of workers not exposed to chemicals (De Celis *et al*, 1998). Xing *et al* (2010) found that workers exposed to the US permissible limit of benzene exposure in Chinese factories had elevated sperm abnormalities of chromosomes X, Y, and 21. These abnormalities are associated with increased risk of childhood leukemia, as up to 40% of children with acute lymphocytic leukemia (ALL) have these genetic abnormalities. Both paternal and maternal benzene exposure may increase the risk of leukemia.

4.1.4 Neurological Effects

Maternal exposure to solvents such as benzene has been associated with neurological, psychological, and behavioural abnormalities and neural tube defects in infants. In human epidemiological studies of women living in Texas between 1999 and 2004, mothers from census tracts where the ambient levels of benzene exposure ranged from low-medium (0.45–0.98 ug/m^3) to medium-high (1.52–2.86 ug/m^3) had positive associations with spina bifida. The highest levels of benzene exposure were associated with more than double the incidence of spina bifida (Lupu, 2011).

In mice studies, Lo Pumo *et al* (2006) found that pups had decreased motor skills and high depressed learning outcomes compared to controls after maternal exposure to benzene at 15 days gestation (the critical period for development). Toluene, another off-gassed by-product in BTEX, is known to interfere with both motor and cognitive processes. The evidence suggests that benzene is likely to exert its effects in a similar manner.

A few epidemiological studies have found an association between maternal exposure to air pollution and autism spectrum disorders. One particular study by Von Ehrenstein *et al* (2014) sought to identify which environmental toxicants were more responsible than others by examining cases of autism at 3.5 and 5km from air quality monitoring stations in California.

Elevated levels of maternal exposure to benzene and 1,3-butadiene were very high in their list of associations.

4.2 1,3-Butadiene

Studies on human exposure to 1,3-butadiene demonstrate links to leukemia, cancer of haemolymphatic organs, adverse cardiovascular and respiratory effects, and damage to the central nervous system.

4.2.1 Acute Health Effects

Acute exposure to 1,3 butadiene by inhalation can lead to irritation of the eyes, nasal passages, throat, and lungs (Environmental Protection Agency, 2009; Carpenter *et al*, 1944; Larionov *et al*, 1934). At very high levels of exposure, inhalation can lead to blurred vision, fatigue, headache, and vertigo. Exposure through contact with skin can cause a sensation of coldness followed by a burning sensation, which may have the potential for frostbite. Tests show that butadiene has low acute toxicity in rats and mice.

4.2.2 Chronic Health Effects

Like benzene, the chronic health effects of exposure to 1,3-butadiene include cardiovascular disease and cancer.

4.2.2.1 Cardiovascular Disease

One large study examined a cohort of 12,110 male workers employed at eight styrene-butadiene polymer manufacturing plants in the United States and Canada from 1943 to 1982 (Matanoski *et al*, 1990). An analysis of mortality data show a significant excess in mortality from arteriosclerotic heart disease, but only among black maintenance workers as compared to the general population. Though the study found that mortality for specific diseases differed by work area in the manufacturing plants, excess mortality from arteriosclerotic heart disease occurred among blacks regardless of where they worked the longest.

4.2.2.2 Cancer

Butadiene is classified as Group 1, *carcinogenic to humans*, by the International Agency for Research on Cancer (IARC, 2012). A detailed review of epidemiological studies on butadiene and cancer in humans can be found in Volume 100F of the International Agency for Research on Cancer (IARC) Monograph series (IARC, 2012). IARC reviewed three large cohort studies on workers in the butadiene-monomer industry (Ward *et al*, 1995; Divine & Hartman, 2001; Tsai *et*

al, 2001) and two large cohort studies on workers in the styrene-butadiene rubber industry (McMichael *et al*, 1974, 1976; Meinhardt *et al*, 1982; Matanoski & Schwartz, 1987; Matanoski *et al*, 1990, 1993). In all but one of the butadiene-monomer cohort studies, excess mortality from leukemia was observed among workers exposed to butadiene. These cohorts were later combined and further examined for association between mortality and exposure to butadiene (Delzell *et al*, 1996; Macaluso *et al*, 2004; Graff *et al*, 2005; Sathiakumar *et al*, 2005; Delzell *et al*, 2006; Cheng *et al*, 2007). The results confirm excess mortality from leukemia seen in approximately 17,000 male workers from eight different styrene-butadiene rubber manufacturing facilities in Canada and the USA.

IARC notes that the diagnosis and classification of lymphatic and hematopoietic malignancies is complex and can change over time (IARC, 2012). Though excess mortality from leukemia is seen among workers who have been employed for more than ten years and work in the most highly exposed areas of plant facilities, the most recent updates to the cohort studies show only slightly elevated mortality from leukemia (Sathiakumar *et al*, 2005; Delzell *et al*, 2006; Cheng *et al*, 2007). A significant exposure-response relationship for butadiene to leukemia was observed, and was independent of exposure to benzene, styrene, and dimethyl-dithiocarbamate (Delzell *et al*, 2006; Cheng *et al*, 2007). No safe threshold for the leukemia risk has been found.

A study in South-Eastern Texas assessed the impact of ambient air concentrations of butadiene and benzene on incidence of lymphohaematopoietic cancer in children (Whitworth *et al*, 2008). The study included 977 cases of childhood lymphohaematopoietic cancer and used the Environmental Protection Agency's (EPA) estimates of butadiene and benzene for 886 census tracts. Elevated rates of leukemia were found for the census tracts with highest levels of butadiene and benzene. An analysis examining the impact of both exposures simultaneously shows that the effects of butadiene and benzene are independent of each other so they could be either additive or synergistic in their effects.

5 Risk Assessment of Kinder Morgan Pipeline Expansion

5.1 What are the Risks to Human Health if the Pipeline Expansion Goes Ahead?

The principal risks examined in this report are the risks due to fire and from a catastrophic oil spill. It is likely that the proposed expansion would also increase the day-to-day emissions from loading and processing at Westridge terminal and Burnaby Mt. tank farm. In our opinion, these risks were inadequately considered by the HHRA performed for the TMEP. Harmful emissions into Burrard inlet should be decreased given our understanding of the carcinogenicity of these

emissions. There is no justification for increasing emissions as proposed by the expanded pipeline and transport.

5.1.1 Risk of Fire

Diluent, and benzene and butadiene are all extremely flammable. The Handbook of Toxic and Hazardous Chemicals and Carcinogens states: “because of low flash point, 1,3-butadiene’s fire and explosion hazard may be more serious than its health hazard” (Sittig, 1991).

TM’s plans to increase the number of oil tanks at the existing tank farm on Burnaby Mountain has caused significant concern to the Burnaby Fire Department (O’Neil, 2014). The Burnaby Fire Department has warned that adding 14 larger storage tanks to the existing 13 tanks increases the risk of a catastrophic fire because of the higher number of tanks within a confined area. This can lead to a scenario involving a potential explosion resulting in toxic fumes and molten crude rolling towards residents and a nearby school. Further, because of climate change caused by burning fossil fuels, the risk of forest fires is increasing on Burnaby Mountain as the forest has longer and hotter dry periods (Pacific Climate Impacts Consortium: Plan2Adapt. http://www.pacificclimate.org/analysis-tools/plan2adapt, accessed 22May15).

The Deputy Fire Chief and former emergency management consultant specializing in tank-fire suppression in the Alberta oil sands, Chris Bowcock, states that were a tank fireball to ignite one of the many nearby tall trees, they expect every tree on the mountain to burn (Chris Bowcock, personal communication March 2015). The City of Burnaby recognizes that TM will need to rely on the city’s Fire Department for an emergency given their lack of personnel for hands-on fire protection (Moreau, 2014). TM has responded by stating that they will be providing "more detailed emergency response plans for the expansion" in the future, though that is not going to change their reliance on the Fire Department in the case of emergencies (Moreau, 2014). The National Energy Board (NEB) has rejected demands from the B.C. government that TM’s emergency management plans need to be fully disclosed (Hoekstra, 2015). This has led Premier Christy Clark to state publicly on the Kamloops radio station CHNL that the province cannot allow the TMEP to go ahead without meeting the government’s five conditions (Hunter, 2015). The high risk to humans and the environment is compounded by the lack of communication between responsible parties on Burnaby Mt. regarding the emergency response plans.

5.1.2 Risk of an Oil Spill

One recent example of the detriments that oil spills can have on nearby communities as well as substantial economic clean-up costs is the Kalamazoo River incident, the largest inland oil spill in US history (Parker, 2014). On July 25, 2010, a 6-foot break in Enbridge Energy’s Line 6B

connecting Griffith, Indiana to Sarnia, Ontario led to over 3.3 million litres of Canadian dilbit entering the Kalamazoo River, 80 miles upstream from Lake Michigan (Ellison, 2014; Dollhopf & Durno, 2011).

This pipeline carries a similar crude oil mix as the TM pipeline, of Cold Lake Crude Oil (with benzene diluent) from Western Canada (Dollhopf & Durno, 2011). As such, the Kalamazoo spill represents a parallel of the realities Burnaby and the North Shore of Vancouver could face in a similar scenario.

> Eighty miles of shoreline and adjacent lands (e.g., overbank areas) were assessed and cleaned. Submerged oil was assessed and recovered at over 25 locations. Over 100 residents were displaced and relocated in response to air quality (benzene) concerns (p.1)…the county health departments issued a drinking water advisory for water wells located within 200 feet of the affected waterways. (Dollhopf & Durno, 2011, p.6)

Three years after the spill (approximately 2 years after the 56 km stretch of river reopened to the public) and 1.2 billion dollars in clean-up costs later, the US EPA estimated that approximately 684,000 litres of bitumen remain in the river due to its sinking properties in fresh water (CBC, 2013). This meant that normal clean-up techniques and equipment were of little use to clean up the remainder of crude oil and dredging of the river was the only foreseeable solution. The $1.2 billion figure includes $551.6 million for response personnel and equipment, $227 million for environmental consultants, and $429.4 million on professional, regulatory and other costs (Ellison, 2014). Recent filings report an additional $89.5 million to the $1.2 billion due to other unforeseen costs (Parker, 2014). Many private class action lawsuits were filed against Enbridge, some of which have been addressed others still on hold four years after the spill (Parker, 2014). Several local residents have stated that their neighbourhoods have changed radically after the spill – with many of the residents having moved away after the spill (Paris, 2013).

One of the major concerns regarding this relatable case scenario is the lack of pipeline maintenance and responsibility from Enbridge Energy LLP that could have prevented the pipeline rupture in the first place.

> Records show that where the pipeline defect occurred [had been] <u>detected at least three times</u> before the pipeline ruptured, in 2005, 2007, and 2009, according to documents Enbridge filed with PHMSA [Pipeline and Hazardous Materials Safety Administration]... But each time, Enbridge decided it wasn't significant enough to require repairs… ten days before 6B ruptured, Enbridge <u>applied to PHSMA for another extension</u>. It asked for an additional two and a half years to decide whether 6B should be repaired or replaced." (McGowan & Song, 2012, p. 5-6)

A similar event would be devastating for the densely populated shoreline communities in and around Burrard Inlet.

5.2 Previous Spills Along the Existing Pipeline

Since its construction in the 1950's, the TM pipeline has reported approximately 82 spills to the National Energy Board of Canada, 13 of which have been within the last 10 years. These spills have released a mix of crude oil, oil waste, natural gas, and isooctane into the environment and surrounding communities (Trans Mountain, 2014). Most recently, in 2007, the residents of Burnaby witnessed over 234,000L of crude oil released from the Trans Mountain pipeline after a construction crew punctured the pipeline during roadway construction. According to the BC Ministry of Environment, during a 25-minute period the 30 metre geyser of oil sprayed into the air contaminating approximately 50 homes, property, and a section of the Barnett Highway.

> "Subsequently the oil seeped into the surrounding soil, storm drains, sewer lines and along other down gradient pathways. The oil moving through the storm drain system eventually reached the marine waters of Burrard Inlet below the spill site where it began to spread further with wind and tides" (BC Ministry of Environment, 2007, para 2).

This event and others like it including worker fatalities at KM facilities in California are evidence of how volatile and unpredictable environmental disasters can be as well as the amount of damage that can occur in a short time.

5.3 What will be the Health Consequences of an Oil Spill?

5.3.1 Evidence from Epidemiological Studies

Many epidemiological studies on human health after a marine oil spill describe acute toxic and psychological effects. Research found population reports of headaches, irritation of the throat, and itchy eyes after the MV Braer oil spill (Campbell *et al*, 1993). A later study by the same authors found that symptoms in the exposed group two weeks after the incident showed more tiredness and fever, but fewer throat, skin, eye irritations, and headaches (Campbell *et al*, 1994).

A study on people who joined in the cleanup work after the Nakhodka oil spill (in the Sea of Japan, 1997) identified a number of risk factors for development of similar symptoms, including being female, the number of working days on cleanup activities, direct exposure to oil products, history of hypertension, and history of low back pain (Morita *et al*, 1999). Other studies on cleanup workers after the Erika oil spill (off the Coast of France, 1999) also found duration of

work as a risk factor for similar health problems including lumbar pain, migraine, dermatitis, ocular irritation, respiratory problems, and nausea (Schvoerer *et al*, 2000). Later studies on this population found increased risk for developing skin irritations and dermatitis for people who had been in bare-handed contact with oil, but no significant increase in risk to developing cancers (Baars, 2002; Dor *et al*, 2003).

Research on cleanup workers after the Prestige, a 'small' Aframax ship, broke apart in a storm off of Spain in 2002 showed a higher prevalence of lower respiratory tract symptoms and significantly higher DNA damage with higher exposure time to volatile organic compounds from oil (Zock *et al*, 2007; Laffon *et al*, 2006; Perez-Cadahia *et al*, 2006 and 2007). Those who used proper health protective devices during work had lowered frequency of health problems (Suarez *et al*, 2005). For the Tasman Spirit that ran aground off of Karachi, Pakistan in 2003, studies showed that a person's perception of health impact decreased with the distance of the person's residency from the spill site (Janjua *et al*, 2006) suggesting a dose-response to the adverse effects.

5.3.2 Evidence from Case Studies

Both asthma and negative mental health experiences have been directly associated with past oil spills and have significant population attributable risk (Sim *et al*, 2010; Lyons *et al*, 1999). Population attributable risk is a measure of the proportion of cases that would not occur in a population if the factor (e.g., an oil spill) was eliminated or never occurred (Frumkin, 2010). As noted in the above examples, other acute symptoms can include headaches, eye and throat irritations, breathing difficulties, nausea, vomiting, and skin rashes (Rodriguez-Trigo *et al*, 2007; Sim *et al*, 2010)

Although asthma and negative mental health outcomes are considered less severe than cancer or reproductive outcomes and are potentially less costly to the healthcare system on a case-by-case basis, these illnesses are more widespread among the exposed populations and can greatly impact the larger community's quality of life and well being. One of the best documented examples of these negative health outcomes is from the Hebei Spirit spill off the coast of Korea.

5.3.2.1 Hebei Spirit Oil Spill

In 2007, an oil tanker known as the Hebei Spirit, spilled 10,900 tons of crude oil 8 km off the coast of Taean, Korea contaminating 167 km of the coastline (Kim, *et al*, 2013; Jung *et al*, 2013). Within the last few years, research has been published assessing the long-term burden of disease from this spill. Several of these studies identified asthma and respiratory impairments

as major health outcomes (Kim *et al*, 2013; Gwack *et al*, 2012; Jung *et al*, 2013). A substantial population attributable risk for these outcomes was observed.

A study conducted by Jung *et al* (2013) evaluated respiratory effects on 436 children living in the area exposed to the Hebei Spirit oil spill. They compared respiratory health outcomes in children living within 2 km of contaminated coastline to those in children living more than 2 km from the contaminated coastline. Children living within 2 km of the contamination showed significant respiratory health impacts 18 months after the spill. These impacts included a significantly lower forced expiratory volume in one second (FEV1), an increased prevalence of ever having asthma, and airway hyper-responsiveness. The prevalence of asthma and wheezing was twice as high as the Korean national average. What is most notable in this study is that in contrast to previous studies of asthmatic symptoms and impaired lung function in adults participating in oil spill clean-up (where symptoms were reversible within one year), there were prolonged health impacts on these children 18 months post spill (Jung *et al*, 2013). Apart from long-term respiratory impairments, the authors noted: “oil spill exposure can also affect the development or aggravation of acute irritant-induced asthma” (Jung et al, 2013).

Among six diseases that Kim *et al* (2013) identified in their study on the Hebei Spirit oil spill, asthma appeared to be the most prominent disease burden (with 6.5 times higher prevalence than the total burden of asthma in South Korea), followed by post-traumatic stress disorder (PTSD) and rhinitis. They also assessed the economic burden of disease from increased cases of allergic rhinitis attributed to the oil spill. Although it was a small population affected by the spill, the direct cost and lost productivity were estimated to be an additional cost of $243,170 per year (Kim *et al*, 2013). These results suggest that although the population affected may have been small, the health and economic outcomes are not negligible (Kim *et al*, 2013).

5.3.2.2 Sea Empress and Exxon Valdez Oil Spills

In February of 1996, an oil tanker known as the Sea Empress spilled approximately 72,000 tons of crude oil along the southwest Wales coastline. Lyons *et al* (1999) conducted a retrospective cohort study to assess the acute physical and psychological impacts of this oil spill among the exposed population. The study found that “living in areas exposed to the crude oil spillage was significantly associated with higher anxiety and depression scores, worse mental health; self reported headache, sore eyes, and sore throat” (Lyons et al, 1999, p. 306).

A second major epidemiological study was undertaken following the 1989 Alaskan Exxon Valdez oil spill to identify the effects of PTSD and anxiety on approximately 600 men and women one year after having been exposed to the spill (Palinkas *et al*, 1993). The authors found that exposed individuals were “3.6 times more likely to have generalized anxiety disorder, 2.9 times

more likely to have posttraumatic stress disorder, and 2.1 times more likely to obtain a high score on the depression scale" (Rodriguez-Trigo *et al*, 2007; Palinkas *et al*, 1993). Women and First Nations people exposed to this event were particularly vulnerable to experiencing depressive symptoms one year after the oil spill (Palinkas *et al*, 1993).

Cultural differences played an important role in perception of psychological damage following the Exxon Valdez oil spill. A low level of family support and impact on subsistence activities were significantly associated with PTSD in indigenous Alaskan, but not in Euro-Americans (Palinkas *et al*, 2004). A separate study reported increased levels of expectations for out-migration, social disruption, and psychological stress immediately following the Exxon Valdez oil spill, though it started to diminish a year after (Gill & Picou, 1998).

6 Who is Most at Risk From Exposure to Benzene and 1,3-Butadiene?

6.1 Vulnerable Populations

According to the American Journal of Managed Care (2006), vulnerable populations can include those who are economically disadvantaged, racial and ethnic minorities, low-income families, the elderly, the homeless, those with chronic health conditions (including severe mental illness), those who live in remote communities or who have difficulty accessing certain services. These kinds of vulnerabilities may be enhanced by race, ethnicity, age, gender, sexual orientation, socioeconomic status, genetic susceptibility, and others (AMJC, 2006). Genetic susceptibilities include genomic instability and diminished DNA repair mechanisms that contribute to cancer risk as well as variations in the ability to detoxify foreign substances in the body. Babies in utero seem to be particularly vulnerable to these effects from benzene and 1,3-butadiene on the human genome. The detailed mechanisms are discussed in detail in Section 10.4.3. These effects occur at low doses such as might be experienced after a series of hot days with stagnant air in Burrard inlet.

Vulnerabilities can be assessed by three main domains – the physical, psychological, and social (AMJC, 2006). This report focusses on the physical vulnerabilities of specific groups as it pertains to their health and wellbeing. Although it is important to note that individuals in all age groups could be affected by the pipeline expansion project, there are a few groups in particular that are at greater risk. These include children, women of childbearing age, and older adults.

As described below, the needs of these populations are serious and exposure to environmental contaminants can be debilitating or detrimental to their health. These groups were identified for their specific vulnerabilities. However, this list is not all encompassing and it is important to

acknowledge the vast heterogeneities within our community and what this means in the case of an environmental disaster such as an oil spill or increased air contaminant.

6.1.1 Children

Children have a particular physiological vulnerability to air pollution due to their immature organs, narrower airways, developing lungs and respiratory system, behaviour and lack of knowledge (Natural Resources Defense Council (NRCD), 1997; Health Canada, 2011). Irritation caused by pollutants or environmental contaminants that may only produce minor responses in adults can result in potentially more significant respiratory obstruction in children. Children are often more active than adults and have greater respiratory rates due to their smaller size. This suggests they breathe a proportionately greater volume of air and inhale more pollutants per pound of body weight than grown adults (NRCD, 1997; Health Canada, 2011). Based on the height of small children and their play habits (for example, crawling and/or rolling), younger children are more likely to be exposed to pollutants or aerosols that are heavier than air and that tend to concentrate closer to ground level (NRCD, 1997; Health Canada, 2011).

As noted elsewhere in this report, children are at higher risk than adults for acute myelogenous leukemia (AML), the primary health outcome of interest in this report. The figure on the next page (Table 4 of the Brosselin *et al* study) illustrates that babies exposed in utero carry the highest risk.

Figure 2: Table of results from the Brosselin et al (2009) study

Table 4

Acute childhood leukaemia and residence adjoining a garage or petrol station by leukaemia subtype: acute lymphoid leukaemia (ALL) and acute myeloblastic leukaemia (AML)

	ALL					AML				
	Cases (n =647)		Controls (n =1681)		OR* (95 CI)	Cases (n =100)		Controls (n =1681)		OR* (95 CI)
	No	%	No	%		No	%	No	%	
Any period of exposure										
Garage or petrol station	67	10.3	107	6.4	1.6 (1.9 to 2.3)	7	7.0	107	6.4	1.1 (0.5 to 2.5)
Garage only	37	5.7	65	3.8	1.5 (0.9 to 2.3)	3	3.0	65	3.8	0.8 (0.2 to 2.5)
Petrol station only	16	2.4	21	1.2	2.0 (1.0 to 4.0)	3	3.0	21	1.2	2.5 (0.7 to 8.8)
Garage and petrol station	14	2.1	21	1.2	1.8 (0.9 to 3.5)	1	1.0	21	1.2	0.8 (0.1 to 6.2)
Missing	0	0.0	6	0.3		0	0.0	6	0.3	
Garage or petrol station by period										
Intrauterine only	9	1.4	4	0.2	6.0 (1.8 to 19.5)	2	2.0	4	0.2	8.3 (1.4 to 49.4)
Childhood only	16	2.5	19	1.1	2.3 (1.2 to 4.5)	2	2.0	19	1.1	2.2 (0.5 to 9.8)
Both	38	5.9	77	4.6	1.3 (0.9 to 1.9)	2	2.0	77	4.6	0.4 (0.1 to 1.7)
Missing	5	0.8	13	0.8		1	1.0	13	0.8	

*OR estimated by unconditional logistic regression models including the stratification variables, age and gender, and number of children <15 years of age living in the household.

6.1.2 Women of Childbearing Age (20-39 years)

As we know from previous studies, environmental contaminants can have significant health consequences for women of childbearing age, and, in particular, expectant mothers. The foetus can be exposed to a number of chemical contaminants that enter through the placenta, causing developmental abnormalities, including to the central nervous system whose development is

particularly sensitive (Health Canada, 2011). Depending on which stage of the pregnancy exposure occurs at, there can be detrimental health and developmental impacts for both the mother and neonate (Health Canada, 2011). These effects are only compounded by ubiquitous exposures to other neurotoxins such as mercury, lead, and polychlorinated biphenyls (PCBs), and persistent organochlorine pollutants (POPs) which can all have negative intergenerational health effects on neural development, immune system development and other systems through gestational exposures (Thompson & Boekelheide, 2013; Hansen et al, 2014; Strom et al, 2014). With little known about multiple chemical exposures among childbearing-aged women, this population is of considerable concern because of the potential impact on mother and child (Thompson & Boekelheide, 2013; Strom *et al*, 2014).

6.1.3 Older Adults, Adults over the age of 65 years and Individuals with Chronic Disease

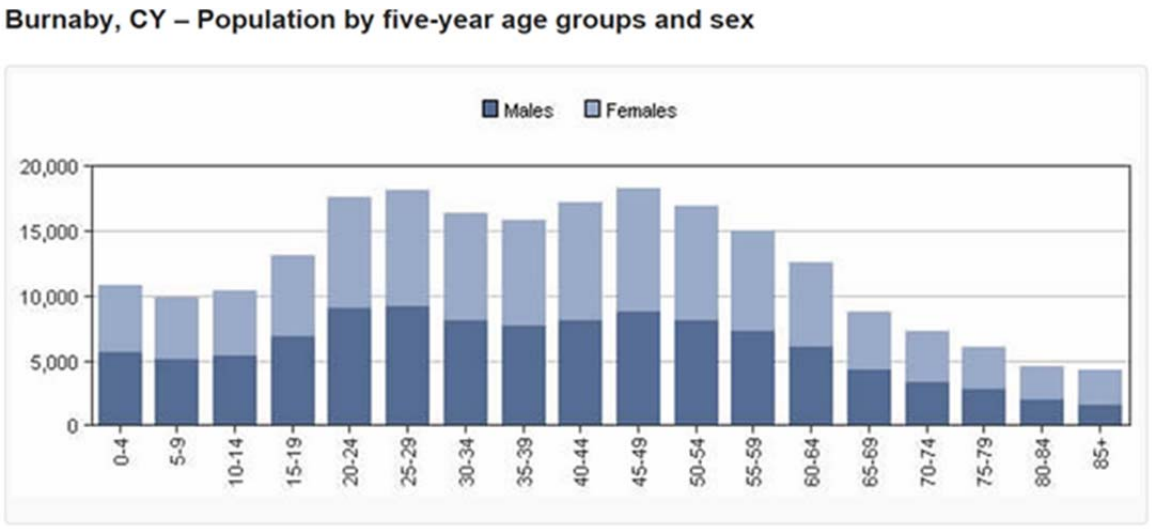
Older adults are more likely to suffer from comorbidities (i.e., having one or more chronic conditions) making them more susceptible to environmental hazards. Even those in good overall health are more vulnerable than the average population (Health Canada, 2011). According to Health Canada, their bodies also contain a "lifetime worth of environmental contaminants, many of which can remain in their systems for decades". This higher "body burden" of environmental contaminants, potential cumulative effects, and general susceptibility to disease puts older adults at increased risk to hazards in the environment.

6.2 Specific Municipalities Affected by the Pipeline Expansion

6.2.1 City of Burnaby

The City of Burnaby is the third most populated city in British Columbia after Vancouver and Surrey. According to the 2011 census, the population of Burnaby was 223,218 individuals, an increase of 10.1% from the 2006 census, which compares to the national average growth of 5.9%. The following graph (and the corresponding table in Section 10.6) from Statistics Canada depict the age and sex distribution of the population of Burnaby. It can be seen from Figure 3 that the median age in Burnaby is 39.8 years, with the female majority falling within the childbearing age range (20-44 years).

Figure 3: City of Burnaby Population in 2011 (Source: Statistics Canada)

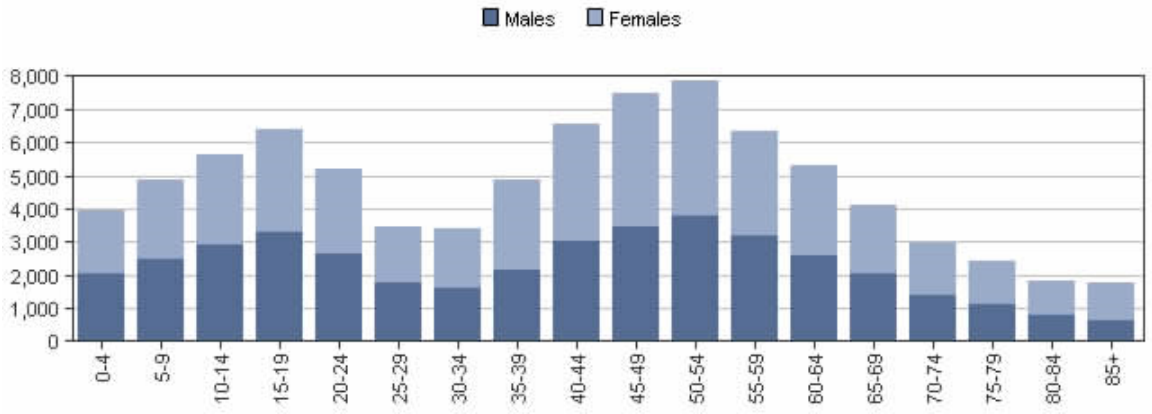


In considering the impact of a possible environmental disaster on Burnaby, it is important to consider its population density. With a land area of 90.61 square km, the population density in 2011 was 2,463.5 persons per square km. In contrast, the overall provincial population density was estimated to be 4.8 persons per square km. With approximately 12,318 people within 5 square kilometres, the impact that an oil spill would have on this residential area is vast.

6.2.2 District of North Vancouver

The following graph (and the corresponding table in Section 10.6) from Statistics Canada depict the age and sex distribution of the population of District of North Vancouver. Note the older median age of 43.3 years, with 15.5% of the population aged 65 and over (compared to the national percentage of 14.8%).

Figure 4: District of North Vancouver Population in 2011 (by five-year age groups and sex)



6.2.3 Vulnerable Populations in the City of Burnaby and the District of North Vancouver

According to the 2011 census, approximately 20% (or 44,250) of the Burnaby population and 24% (20,860) of the North Shore population consist of children and youth under 19 years of age. Approximately 9.3–10.4% of the population in both municipalities is under 10 years. Approximately 30% (34,055) of the Burnaby population and 20% (16,915) of the North Shore population consist of females of childbearing age between 20–39 years. Approximately 13.8% (30,900) of the Burnaby population and 15.5% (13,130) of the North Vancouver district population consist of adults over the age of 65 years.

7 Discussion

7.1 Air Quality Issues

The Air Quality Health Index (AQHI) is a rating that is reported in B.C. that predicts the level of air quality health risk based on a mixture of pollutants (B.C. Air Quality, undated). The AQHI is reported on a colour scale and number scale from 1 to 10, and labels health risk as low, moderate, high, or very high. It predicts health risk over the next 36 hours based on monitoring data collected on particulate matter (PM2.5 and PM10), ground-level ozone (O3), and nitrogen dioxide (NO2). Up-to-date information can be viewed in real time online (B.C. Air Quality, undated).

Air quality monitoring is conducted by the British Columbia provincial government, Metro Vancouver, and industry where required by permit. There are approximately 150 stations throughout B.C. monitoring for air contaminants including carbon monoxide (CO), nitrogen dioxide (NO_2), ozone (O_3), particulate matter (PM2.5 and PM10), sulphur dioxide (SO_2), hydrogen sulphide (H_2S), volatile organic compounds (VOCs) and others (B.C. Air Quality, undated).

As shown in Figure 5 (on the next page), Metro Vancouver operates a network of 27 air quality monitoring stations (Fraser Health, 2013). Of these, only six monitor VOCs: Port Moody, Chilliwack, Burnaby South, Burnaby-Burmount, Burnaby North, and Richmond-Airport (Fraser Health, 2013). Two stations, Burnaby North (T24) and Burnaby-Burmount (T22), are adjacent to petroleum industry facilities. The monitoring stations that measure VOCs collect samples in canisters, which are analyzed for up to 177 different VOCs. See Section 10.7 for Air Quality Monitoring Stations in Metro Vancouver.

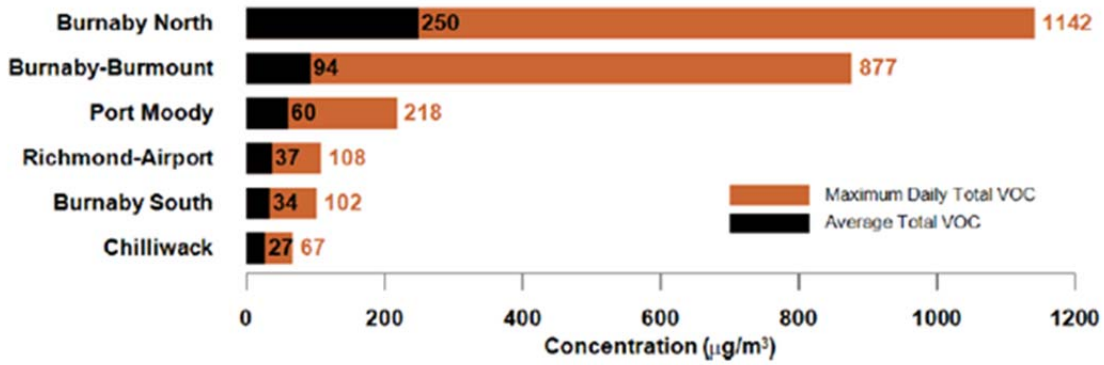
Figure 5: Lower Fraser Valley AIr Quality Monitoring Network

(Source: Fraser Health, 2013).

7.1.1 Air Quality Measurements in Municipalities of Interest

Figure 6, below, shows that highest average total VOC were measured at the Burnaby-North station, which is near industrial sources at the Burrard Inlet (Fraser Health, 2013). The highest daily concentration was also measured here on January 27, 2011.

Figure 6: Total VOC Monitoring in Metro Vancouver (Source: Metro Vancouver, 2011)

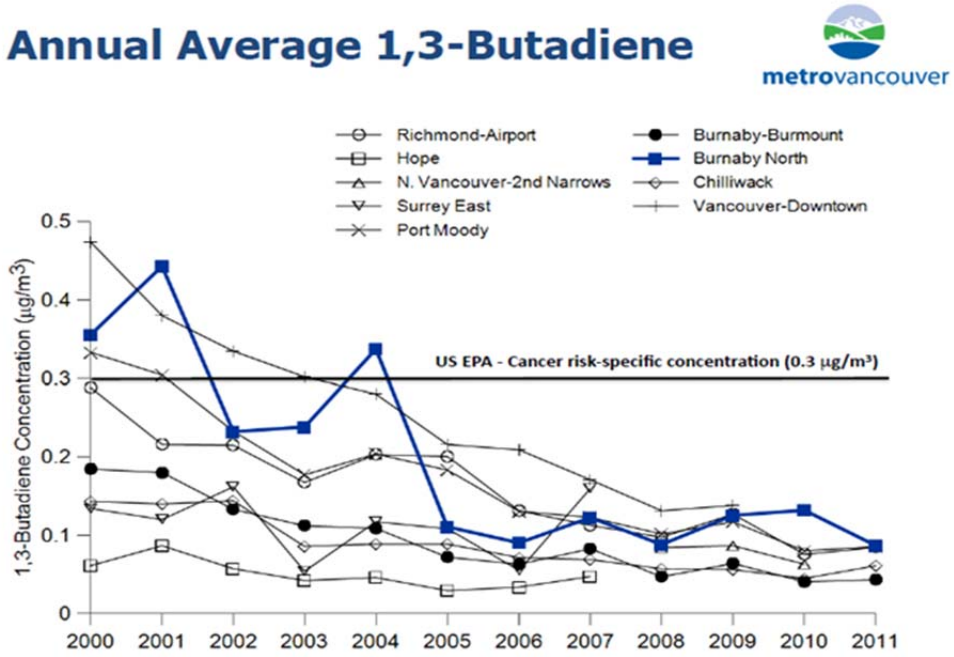


This figure demonstrates the already high burden of pollutants faced by Burnaby North and Burmount residents. The proposed pipeline expansion would further compound this problem.

Figure 7, below, shows the annual average butadiene concentration in Metro Vancouver between 2000-2011. Annual average concentrations have decreased substantially since 2000

and have been below the US EPA cancer-risk specific concentration of 0.3ug/m^3 since 2004 (Fraser Health, 2013).

Figure 7: Annual Average 1,3-Butadiene Concentrations in Metro Vancouver



(Source: Fraser Health, 2013)

7.1.1.1 Particulate Matter (PM) and Sulphur Dioxide Measurements

The air pollutants from TMEP would of course be added on to current levels. Monitoring by Fraser Health Authority and Metro Vancouver show that currently an important hazardous air pollutant, fine particulate matter (PM 2.5), is already elevated at the Burnaby South monitoring station with hourly measures to 6.1ug/m^3. The maximum 24-hour rolling average is currently at 24ug/m^3. The upper regulatory limit is 25ug/m^3. (See 2013 MetroVan report http://www.metrovancouver.org/Search/Pages/results.aspx?k=LFV%20AQ%20monitoring)

The hourly objective for sulphur dioxide was exceeded at the Burnaby-Capitol Hill station in 2013 for a total of 2 hours during January 20 and 21. The 24-hour objective was exceeded at this station for a total of 3 hours on January 21. It is thought that the exceedances were caused by a combination of poor dispersion conditions along with emissions from marine vessels and the Chevron refinery. During this time there were stagnant weather conditions, which limited dispersion. (See pg. S-7, http://www.metrovancouver.org/Search/Pages/results.aspx?k=LFV%20AQ%20monitoring)

7.1.2 Kinder Morgan's Air Quality Assessment

KM has conducted a preliminary air quality assessment for their Trans Mountain Pipeline application. The air quality assessment addresses emissions of air contaminants and green house gases (GHGs) from pipelines, pump stations, and storage terminals both during construction and operations. It is found in Volume 5C, Section 4 entitled 'Air Quality and Greenhouse Gas Technical Report for Kinder Morgan Project'.

TM completed predictive dispersion modelling for three scenarios: existing conditions, project conditions, and cumulative conditions. Emissions from tankers at berth including both fugitive and combustion related emissions were included in the modelling. Volume 5C, Section 4.2 sets out the results of estimated VOC emissions for each phase of construction by area and operation. Butadiene is not considered. TM considers benzene, toluene, ethylene, and xylene (BTEX). The other air contaminants considered are NO_x, CO, SO_2, VOC, PM10, and PM2.5.

7.1.3 Worst Case Weather Conditions: High Temperature and Low Wind

In 2009, Metro Vancouver experienced a minor heat wave consisting of three or more consecutive days of 30 degrees or higher temperatures. On July 28, 29 and 30th of 2009, the temperatures ranged from 31–34C, with low winds of 6-10 km/h (The Weather Channel, 2014). During the summer of 2014, Metro Vancouver and surrounding areas experienced an even greater heat wave with temperatures reaching an average of 40.5C across several communities (Lytton, Pemberton, Lillooet, and Kamloops) (The Weather Channel, 2014).

These scenarios are prime examples of worst-case weather conditions that need to be considered in the modelling for the TMEP application. High temperatures and low wind slow the evaporation time of environmental pollutants (such as VOCs and BTEX) and enables them to remain in the atmosphere for a longer duration, thus increasing exposure time of contaminants to the surrounding communities and therefore risk of the cancers described here.

8 Conclusions

8.1 Limitations

As noted previously, no human health risk assessment has been made describing potential risks in the most heavily populated areas affected by the pipeline, namely Metro Vancouver. This fact, along with alternate third party human health risk assessment, makes it impossible to accurately estimate the risk posed by TMEP. We can say with certainty however that the location and weather conditions picked for the TM HHRA spill scenarios are closer to 'best-case'

than 'worst-case' scenarios. Considering the non-threshold nature of the risk of leukemia from benzene and 1,3 butadiene and the exquisitely sensitive populations living alongside the facility on the shores of Burrard inlet, this increased risk is unacceptable.

8.2 Recommendations

A full human health risk assessment under realistic but unfavorable conditions, in the location where people live, is required to adequately evaluate the human health risk of the proposed expansion. These conditions should include a period of hot weather (greater than 30C for 3 or more days) in a stagnant air weather pattern. The weather conditions that produce more volatilization of the chemicals of concern and can trap them in the Burrard Inlet for extended periods are predicted to increase under local climate change modeling (Pacific Climate Impacts Consortium: Plan2Adapt. http://www.pacificclimate.org/analysis-tools/plan2adapt, accessed 22May15).

The US EPA has developed a series of recommendations for undertaking risk assessment for early life exposure to compounds that are mutagenic in nature. These calculations specifically address the particular vulnerability of young children by incorporating age-dependant adjustment factors (ADAFs) to better estimate the impacts of cumulative (whole life) exposures. Much of this work has been collated in the EPA's Child-Specific Exposure Factors handbook (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=199243) and (http://www.epa.gov/oswer/riskassessment/sghandbook/riskcalcs.htm). Such calculations and adjustments should be included when constructing a HHRA for the TM exposure scenario.

It is important to recognize the impact this project will have on climate change and understanding the project risks in terms of future climate projections. The authors of this report acknowledge climate change as a serious public health concern and consider this a paramount factor that is completely ignored in the TMEP application. As noted above, temperature increases have been observed in recent years and are expected to continue. By continuing to build large fossil energy infrastructure such as the expansion proposed here, TM will contribute to temperature increases that may be catastrophic for human health and the environment (IPCC 5th Assessment –Synthesis for Policy Makers. http://www.ipcc.ch/, accessed 22May15). Increased temperatures will worsen the impact of the pipeline, accompanying infrastructure and increased marine transport by increasing risk of fires and air pollution from additional off-gassing of volatile carcinogenic compounds like benzene and 1,3 butadiene.

The humanity of a society is judged by how well it protects and cares for its most vulnerable individuals. Benzene and 1,3-butadiene are known human carcinogens. In our opinion, the

potential for increased exposure to these toxic chemicals as a result of the TMEP poses an unacceptable risk to the health of Burnaby and North Shore residents.

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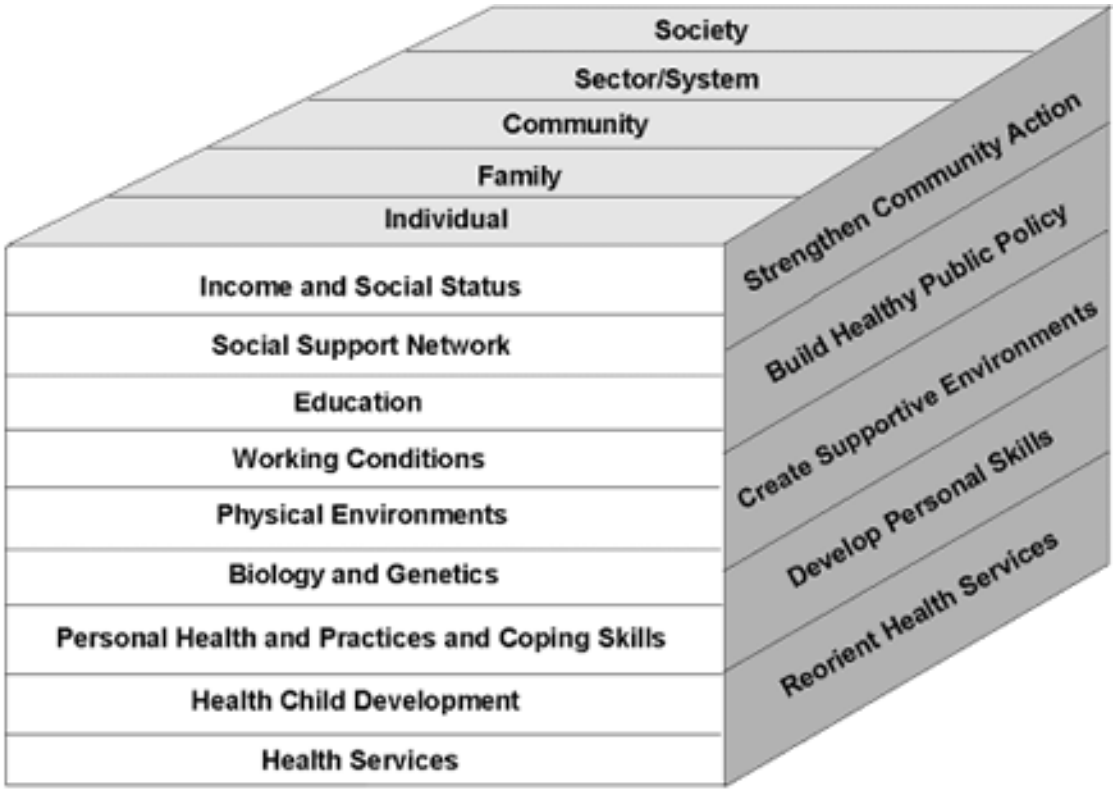
10 Appendices

10.1 Databases and Key Search Terms Used for Data Collection

Databases	Key Search terms
Kinder Morgan Trans Mountain Pipeline Expansion Application	1,3 butadiene, Benzene, human health, oil spill, Kinder Morgan, Pipeline, Dilbit, AML, Westridge terminal, benzene acceptable level in gasoline, benzene carcinogen, Kinder Morgan pipeline, Burnaby population statistics, benzene aromatic hydrocarbon, benzene metabolism, benzene metabolites, benzene pathways human body, benzene occupational exposure, benzene dermal, benzene inhalation, benzene consumption, benzene susceptibility, acute health effects, chronic health effects, benzene children, AML children, acute myeloid leukemia, leukemia, hematopoietic stem cells, acute myeloid Leukemia biology, benzene pregnant women, benzene neonates, Kalamazoo, etc. Snowball technique was used to identify other primary sources.
Google Scholar	
Web of Science	
Pub Med	
Environment Canada	
Agency for Toxic Substances and Disease Registry reports	
Statistics Canada	
Other Primary Data Sources	

10.2 Population Health Framework



(Public Health Agency of Canada, 2001)

10.3 Graphic Imaging from Kinder Morgan Proposed Westridge Marine Expansion Designs

Available at: http://www.transmountain.com/marine-westridge-terminal

10.4 Detailed Health Effects Information on Benzene and 1,3-Butadiene

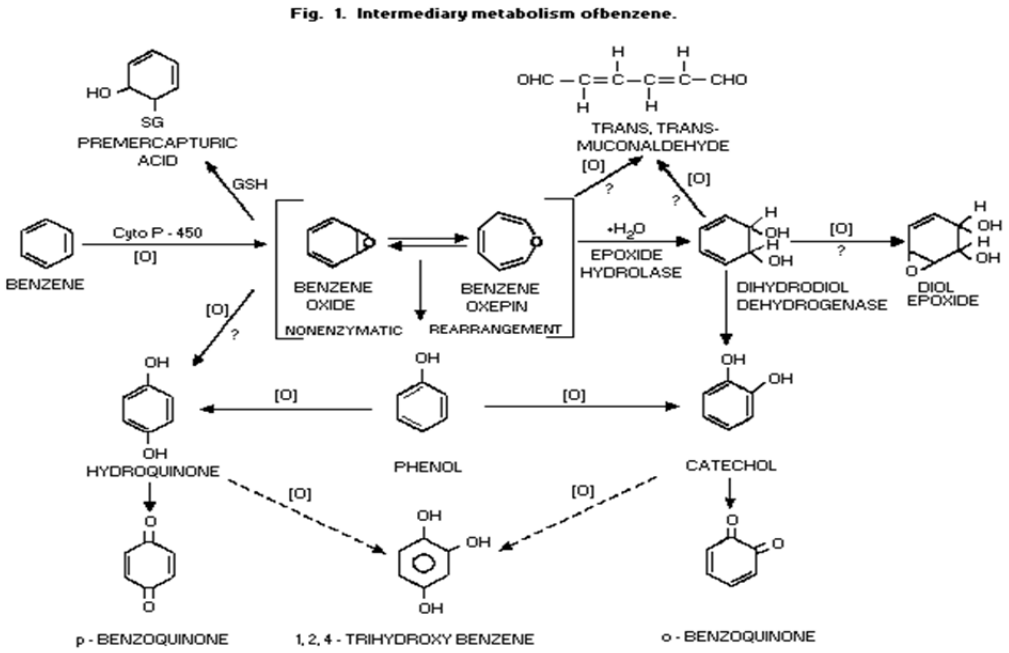
10.4.1 Metabolism of Benzene

Benzene is primarily metabolized in the liver by the cytochrome P-450 system. This system, which is responsible for the detoxification in the body, metabolizes benzene primarily into benzene oxide. Benzene oxide can hinder the normal functioning of the cytochrome system (Golding et al, 2010). In vivo studies reveal that benzene oxide-oxepin blocks one-electron oxidation by cytochrome P-450 resulting in the formation of (E, Z)-muconaldehyde (Golding et al, 2010). Oxepin is an oxygen-containing heterocyclic compound, which exists in equilibrium with benzene oxide (Vogel and Günther, 1967).

Oxide-Oxepin is known for its DNA disturbance activity, which leads to genotoxicity and carcinogenicity (Golding and Watson, 1999). A report by Ross *et al*, concluded that "susceptibility to the toxic effects of benzene has been suggested to occur partly because of polymorphisms in enzymes involved in benzene metabolism, which include cytochrome P450

2E1" (Ross and Zhou 2010). The figure below illustrates the intermediary metabolism of benzene.

Intermediary Metabolism of Benzene



(International Programme on Chemical Safety (IPCS), 1993)

Zahid *et al* (2010) found that benzene is metabolized in the liver and in the brain by (cytochrome p450) CYP 2E1 to become catechol benzene (CAT). CAT can then be oxidized to benzoquinones and form DNA damaging adducts, leading to errors in DNA repair, cancer or neurodegenerative disease. The lack of reductase enzymes in bone marrow makes these tissues particularly susceptible to benzene quinones.

10.4.2 Biology of Benzene

The type of genetic mutation determines the stage at which stem cells would be disrupted, leading to cancerous cells. These abnormalities include proliferation of the stem cells in the absence of normal growth signals, indefinite self renewal of the cells, inhibited programmed cell death, differentiation, uncontrolled cell cycle checkpoint, genomic instability, and multi organ dissemination of leukemic cells. It is proposed that the interaction of multiple anomalies stated above could lead to AML (Licht & Sternberg, 2005). Bone marrow pluripotent stem cells carry the CD34+ antigen, and are more susceptible to disruption by metabolites of benzene, particularly 1,4 benzoquinone. Low levels of 1,4 benzoquinone cause chromosomal aberrations, including single or triple copy numbers of chromosome 7 and 8 (Smith *et al*, 2000). This genetic

instability is seen in the early stages of acute myeloid leukemia (AML), and is seen in workers exposed to low levels of benzene (Zhang *et al*, 2012).

Cell proliferation in the absence of normal growth signal could result from mutations in the ABL-BCR tyrosine kinase where this pathway is activated even without the binding of growth factor to its receptor, which normally stimulates this pathway. Another FLT3 tyrosine kinase is almost always expressed in patients with AML. Once these kinases are activated, these lead to the expression of the genes, which lead to cell proliferation. The differentiation of stem cells to specific blood cell is disrupted by the chromosomal translocations and point mutations in genes responsible for differentiation, which include the retinoic acid receptor (RAR) gene, the MLL gene, HOX genes and the core binding complex factor (Licht & Sternberg, 2005).

The ability to escape cell death (aka apoptosis) is crucial in cancer development. The p53 gene regulates the apoptotic pathways. Mutation in this gene can cause the cell to escape cell death and proliferate even though it is non-desirable in the body. Additionally, the tyrosine kinase activates the PI 3-Kinase signaling pathway, which activates serine threonine kinase to phosphorylate BAD and cause the release of BCL-3 pro-survival molecules. These are survival proteins that determine the survival rate in case of cancerous cells. Mutation in this gene can also lead to malignancy (Licht & Sternberg, 2005). Genetic and epigenetic changes such as these that can lead to an increased risk of leukemia, meaning that some individuals are at higher risk of developing leukemia with a given exposure to benzene (or other leukemogen) than others.

10.4.3 Benzene Susceptibility

Benzene susceptibility can be linked to different factors including genetic makeup, age, epigenetics, ethnicity, certain blood disorders, and family history, gender. Genomic studies have concluded that genetic susceptibility is traced back to the different polymorphisms at the DNA level. One such polymorphism occurs at NQO1 gene, which codes for NAD(P)H:quinone oxidoreductase. NAD(P)H:quinone oxidoreductase is an enzyme which catalyzes the two or four electron reduction reactions of various quinones. A quinone is derived by modifying an aromatic substance, such as benzene, by converting its -CH= group to a -C(=O)- group with the arrangement of double bond. The role of NQO1 is to protect cells from oxidative reactions. Its ability to reduce quinones, which precludes generation of reactive oxygen radicals, suggests that NQO1 is a chemoprotective enzyme. Hence, NQO1 gene is responsible for the elimination of benzene in the human body. This enzyme has also been thought to stabilize the tumour suppressor gene p53 and has been demonstrated to interact with p53 in a protein-protein interaction. Lab studies have been carried out to determine that the inhibition of NQO1's metabolic functions can lead to increased risk of cancer or toxicity (Nebert et al, 2002).

Two prominent polymorphisms related to NQO1 gene have been characterized. The NQO1*2 allele has been linked to the C609T mutation in the cDNA coding for a Pro187Ser change in the enzyme. The NQO1*3 allele links to C465T change in the cDNA coding for an Arg139Trp mutation in the protein. The frequency of NQO1*2 alleles ranges between 0.22 (Caucasian) and 0.45 (Asian) in various ethnic populations. The frequency of NQO1*2 allele is higher than that of NQO1*3 and has a significant effect of the phenotype (for example, reduced catalytic function and more rapid degradation of the enzyme by ubiquitination). As a result, there is no detectable NQO1 activity in individuals with homozygous NQO1*2/NQO1*2 genotype.

An epidemiological study of a benzene exposed population reveals that NQO1*2 homozygotes exhibit as much as a seven fold greater risk of developing bone marrow toxicity, leading to such diseases such aplastic anemia and leukemia. There are other genes involved in metabolism of benzene such as the P450 2E1 (CYP2E1), myeloperoxidase (MPO), glutathione-S-transferase (GSTM1, GSTT1), microsomal epoxide hydrolase (EPHX1). The polymorphic nature of these genes could result in the mutant phenotypes of these proteins resulting in failure to eliminate the benzene from the body (Nebert et al, 2002).

Genome-wide analysis of blood mRNA from workers exposed to high (6.68 ± 2.28 mg/m^3), low (1.82 ± 1.16 mg/m^3), and no (0.06 ± 0.01 mg/m^3) levels of benzene demonstrated that chronic benzene exposure is associated with dysreguation of pathways of interest in cancer. Pathways affected include cell adhesion, MAPK signaling, apoptosis, p53 signaling and several others associated with immune response (such as B cell receptor signaling). Exposure was also associated with dysregulation of natural killer cell mediated cytotoxicity, T cell receptor signaling pathway, chronic myeloid leukemia, and hematopoietic cell lineage – all of which are identified as important in acute myeloid leukemia (Gao *et a*l, 2014). Furthermore, the same laboratory showed benzene caused dysregulation of 13 key miRNAs (small pieces of RNA which regulate RNA transcription). These miRNAs positively or negatively regulated pathways of nervous system development (discussed above), as well as RNA polymerase II promotor, and actin cytoskeleton rearrangement. Target genes for the miRNAs were SMAD4, PLCB1, NFAT5, GNAI2, PTEN, VEGFA, BCL2, CTNNB1 and CCND1. They have been implicated in key cancer pathways, including, TGF-beta signaling and Wnt signaling pathways.

More than simply up or down regulating, or mutating critical genes, occupational exposure to benzene can cause epigenetic changes to DNA in humans. These are changes in the methylation status of genes, essentially deciding if they get turned on or off. In AML and other cancers, irregular genome-wide DNA hypomethylation (decrease in methylation), gene-specific hypermethylation (increase in methylation), and loss of imprinting (loss of silencing of a gene from one parent) are often seen. Sha et al (2014) found that very low benzene exposures cause

genome-wide hypomethylation. In a study of 260 Chinese workers, benzene down regulated the mRNA poly(ADP-ribosyl)ation polymerases (PARP)s, which control the methylation state of DNA, making it less stable and more susceptible to mutations. The exact mechanism is not known. However, it is suggested this may be through changes in the methylation state of the promoter of PARP itself. PARP is also a key enzyme responsible for regulating apoptosis in regular and cancerous cells. Furthermore, even very low levels of benzene exposure (2-66ug/m3) showed changes in methylations patterns of genes associated with AML. Bollati *et al* (2007) saw decreases in methylation of long interspersed nuclear element 1 (LINE1) and *Alu1* repeat sequences; however, they discovered twice the amount of methylation (suppression) of *p15* tumour suppressor gene. Hypomethylation of MAGE-1, which is seen in many cancer types, including leukemia, was also observed.

A key danger of chronic environmental exposure to benzene is there may be a long window between the starting point of exposure and disease manifestations, making it difficult to ascertain causality. However, a recent small (33 subjects) but significant study by Li *et al* (2014) looked at workers exposed to very low levels of benzene (1-10ppm). Analysis of subjects who had even one year of exposure to these levels of benzene had upregulation of genes NOTCH1 and CD147, which as associated with development and occurrence of leukemia. NOTCH1 is a key gene in T-cell differentiation and proliferation. The increase in NOTCH1 mRNA expression was significant after one year and increased in workers exposed for longer. Upregulation has been recorded for 10% of chronic lymphocyte leukemia and is associated with poor prognosis. Mutation occurs in 50% of acute T-lymphoblastic leukemia cases. NOTCH1 controls expression c-myc, a quintessential stem oncogene which dysregulates cell-cycles progression and neoplastic transformation (Mitra et al 2007). CD147 mRNA is upregulated in many cancers and promotes invasion and metastasis and has been seen to be upregulated in patients exposed for 7 years (Li *et al*, 2014).

10.4.4 Proximity to Gas Stations & Benzene-Induced Childhood Leukemia

A recent study reported that living in close proximity to a gas station (a prime source of benzene) may be associated with childhood leukemia (Brosselin *et al*, 2009).
A total of 765 cases of acute leukemia were included in this study. Of these, 647 were cases of acute lymphocytic leukemia (ALL; 544 common B-cell ALL, 27 mature B-cell ALL, 67 T-cell ALL and 8 unspecified ALL), 118 were cases of acute nonlymphocytic leukemia (ANLL; 100 acute myeloblastic leukaemia (AML) and 18 were cases of unclassified leukaemia).

Table 3 (reprinted from Brosselin *et al*) indicates a strong association between living in the vicinity of automobile repair garage or petrol station and acute childhood leukemia (odds ratio

of 1.6, 95% CI 1.2 to 2.2). The association was strongest for “petrol stations only” (odds ratio of 2.1, 95% CI 1.1 to 4.0).

Table 3
Acute childhood leukaemia and residence next to businesses

Residence in the vicinity of businesses	Cases (n= 765)		Controls (n=1681)		OR* (95 CI)
	No	%	No	%	
Automotive repair garage					
Never	708	92.6	1590	94.6	1.0 (ref)
Ever	57	7.4	85	5.1	1.4 (1.0 to 2.0)
Petrol station					
Never	730	95.4	1633	97.2	1.0 (ref)
Ever	35	4.6	42	2.5	1.9 (1.2 to 3.0)
Garage or petrol station					
Never	689	90.0	1568	93.3	1.0 (ref)
Ever	76	10.0	107	6.4	1.6 (1.2 to 2.2)
Never	689	90.0	1568	93.3	1.0 (ref)
Garage only	41	5.4	65	3.8	1.4 (0.9 to 2.1)
Petrol station only	19	2.5	21	1.2	2.1 (1.1 to 4.0)
Both	16	2.1	21	1.2	1.7 (0.9 to 3.4)
Other businesses					
Car bodywork repair shop (yes vs no)	19	2.5	27	1.6	1.6 (0.9 to 3.0)
Factory (yes vs no)	39	5.1	92	5.5	0.9 (0.6 to 1.4)
Printing plant (yes vs no)	10	1.3	20	1.2	1.0 (0.5 to 2.2)
Missing		0	6	0.3	

*OR estimated by unconditional logistic regression models including the stratification variables, age and gender, and the number of children <15 years of age living in the household.

(Source: Brosselin *et al*, 2009)

As shown in Table 4 (also reprinted from Brosselin *et al*), Similar trends were observed for acute lymphoid leukemia (ALL) and acute myeloblastic leukemia (AML). In this analysis, the association was strongest between ALL and AML and living near the petrol station when the exposure was intrauterine only (odds ratio of 6.0 (95% CI 1.8 to 19.5) for ALL and 8.3 (95% 1.4 to 49.5) for AML).

Table 4

Acute childhood leukaemia and residence adjoining a garage or petrol station by leukaemia subtype: acute lymphoid leukaemia (ALL) and acute myeloblastic leukaemia (AML)

	ALL					AML				
	Cases (n =647)		Controls (n =1681)		OR* (95 CI)	Cases (n =100)		Controls (n =1681)		OR* (95 CI)
	No	%	No	%		No	%	No	%	
Any period of exposure										
Garage or petrol station	67	10.3	107	6.4	1.6 (1.9 to 2.3)	7	7.0	107	6.4	1.1 (0.5 to 2.5)
Garage only	37	5.7	65	3.8	1.5 (0.9 to 2.3)	3	3.0	65	3.8	0.8 (0.2 to 2.5)
Petrol station only	16	2.4	21	1.2	2.0 (1.0 to 4.0)	3	3.0	21	1.2	2.5 (0.7 to 8.8)
Garage and petrol station	14	2.1	21	1.2	1.8 (0.9 to 3.5)	1	1.0	21	1.2	0.8 (0.1 to 6.2)
Missing	0	0.0	6	0.3		0	0.0	6	0.3	
Garage or petrol station by period										
Intrauterine only	9	1.4	4	0.2	6.0 (1.8 to 19.5)	2	2.0	4	0.2	8.3 (1.4 to 49.4)
Childhood only	16	2.5	19	1.1	2.3 (1.2 to 4.5)	2	2.0	19	1.1	2.2 (0.5 to 9.8)
Both	38	5.9	77	4.6	1.3 (0.9 to 1.9)	2	2.0	77	4.6	0.4 (0.1 to 1.7)
Missing	5	0.8	13	0.8		1	1.0	13	0.8	

*OR estimated by unconditional logistic regression models including the stratification variables, age and gender, and number of children <15 years of age living in the household.

(Source: Brosselin *et al*, 2009)

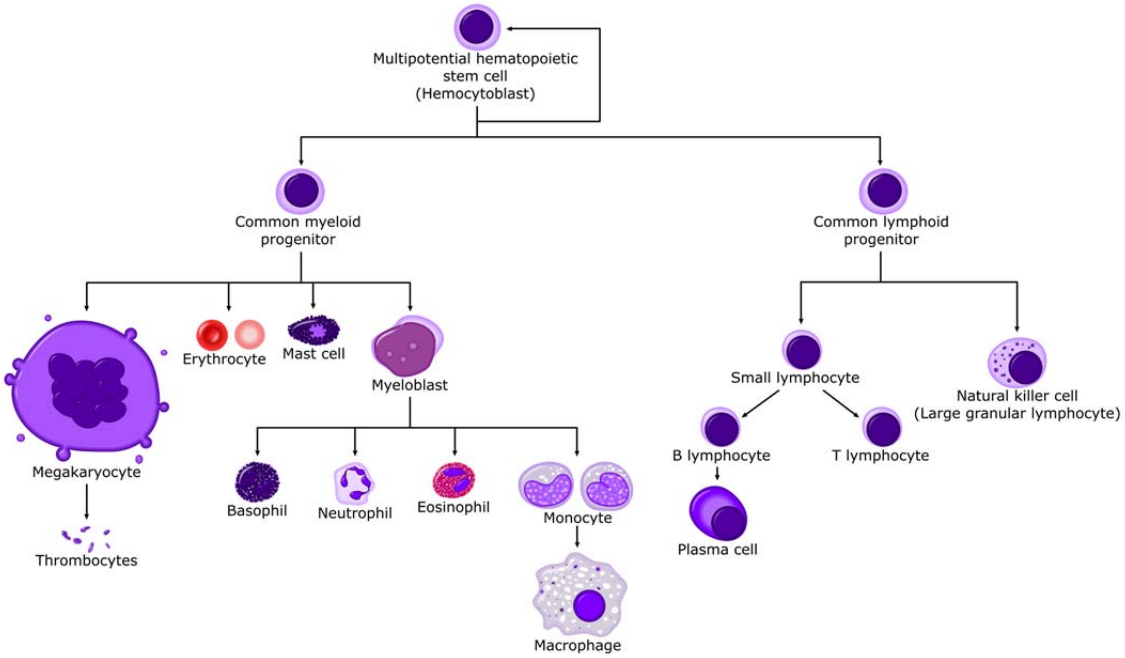
10.4.5 Biology of Butadiene

The biological mechanism of butadiene has been extensively studied. The figure below shows the metabolic pathways of butadiene based on *in vitro* and *in vivo* studies. This metabolic pathway sheds light on the mechanism of butadiene-induced carcinogenicity from the perspectives of toxicokinetics, metabolism, biomarkers, genotoxicity, and molecular biology (IARC, 2012).

Butadiene is converted by several different enzymes into monoepoxide, diepoxide, and butenediol. It is subsequently conjugated with glutathione into mercapturic acids and ultimately eliminated in the urine. Butadiene-induced carcinogenicity is due to activity by its metabolites formed during bio-transformation of butadiene in the body. Butadiene metabolism can form reactive epoxides, and their interaction with DNA results in mutagenicity, which is a likely cause of butadiene's carcinogenicity. The mutagenic response of epoxide intermediates depends on factors including their ability to concentrate in tissues, their reactivity with DNA and repair of DNA adducts (IARC, 2012). There is inter-individual variation in the mutagenic response of epoxides, for example, due to different levels of key enzyme expression. A study shows that mice deficient in microsomal epoxide hydrolase (mEH), an enzyme that transforms and reduces the concentration of epoxides in the body, are more susceptible to the mutagenic effects of butadiene (Wickliffe *et al*, 2003).

Some reports link butadiene exposure to heritable genetic mutations. Using hypoxanthine phosphoribosyltransferase (HPRT) as an embryonic stem (ES) cell marker in humans, exposure to low levels of butadiene (3.5 ppm) was associated with increased heritable mutation, although another report on slightly lower exposure differed (Pacchierotti *et al*, 2008). Mouse studies showed twice as many DNA-adducts of butadiene metabolites on germ cells as lung cells in the same mice, suggesting germ cells metabolize butadiene differently. Studies in mice show a dose-pendent increase in fetal death and heritable mutations in offspring of males exposed to low levels of butadiene (Adler *et al*, 1998). Subchronic exposure to 12.5 and 1250 ppm butadiene resulted in a significant increase in early fetal deaths and nervous system abnormalities, even at low levels. Male mice exposed to low levels of exposure was found to have increased skeletal abnormalities, especially of the skull, which is correlated to neural abnormalities (Anderson *et al*, 1996). One study that sought to extrapolate human exposure with laboratory mice data postulates that workers with low levels of benzene exposure (5–6 weeks exposure to 20–25 ppm butadiene) are more likely to have children with chromosomal abnormalities compared to background.

10.5 Acute Myeloid Leukemia Process



Available at: http://allthingsstemcell.com/category/hematopoietic-stem-cells/

10.6 Population Tables for the City of Burnaby and the District of North Vancouver

Table 3: Burnaby Population by 5-Year Age Groups

Age groups	Both sexes	Males	Females
Total - Age groups	223,220	109,250	113,970
0 to 4 years	10,805	5,615	5,190
5 to 9 years	9,900	5,055	4,850
10 to 14 years	10,415	5,335	5,080
15 to 19 years	13,130	6,805	6,320
20 to 24 years	17,625	9,075	8,550
25 to 29 years	18,100	9,140	8,965
30 to 34 years	16,410	8,150	8,265
35 to 39 years	15,915	7,640	8,275
40 to 44 years	17,240	8,145	9,095
45 to 49 years	18,270	8,785	9,485
50 to 54 years	16,970	8,155	8,820
55 to 59 years	15,005	7,235	7,775
60 to 64 years	12,535	6,055	6,480
65 to 69 years	8,710	4,250	4,460
70 to 74 years	7,330	3,370	3,960
75 to 79 years	6,050	2,850	3,205
80 to 84 years	4,530	2,010	2,520
85 years and over	4,280	1,595	2,690
Median age	39.8	38.6	40.8

Table 6 Burnaby, CY – Population by five-year age groups and sex, 2011 Census

Table 4: North Shore Population by 5-Year Age Groups

Age groups	Both sexes	Males	Females
Total - Age groups	84,410	40,900	43,510
0 to 4 years	3,975	2,035	1,935
5 to 9 years	4,880	2,450	2,435
10 to 14 years	5,610	2,935	2,680
15 to 19 years	6,395	3,305	3,095
20 to 24 years	5,180	2,660	2,520

25 to 29 years	3,465	1,765	1,705
30 to 34 years	3,405	1,595	1,815
35 to 39 years	4,865	2,175	2,690
40 to 44 years	6,545	3,045	3,500
45 to 49 years	7,490	3,430	4,060
50 to 54 years	7,840	3,795	4,045
55 to 59 years	6,335	3,160	3,175
60 to 64 years	5,280	2,575	2,705
65 to 69 years	4,110	2,030	2,080
70 to 74 years	2,965	1,380	1,590
75 to 79 years	2,435	1,110	1,325
80 to 84 years	1,850	815	1,035
85 years and over	1,770	635	1,135
Median age	43.4	42.5	44.2

10.7 Air Quality Monitoring Stations in Metro Vancouver

Stations		Air Quality Monitors													Meteorology					
		Continuous											Non-Continuous							
		Gases							Particulate Matter											
ID	Name	SO_2	TRS	NO_2	CO	O_3	THC	NH_3	PM_{10}	$PM_{2.5}$	CARB	NEPH	VOC	SP	D	Wind	T_{air}	SR	RH	BP	Precip
T1	* Vancouver-Downtown	√		√	√	√															
T2	Vancouver-Kitsilano	√		√	√	√				√						√	√		√		√
T4	Burnaby-Kensington Park	√	√	√	√	√			√	√						√	√				
T6	N. Vancouver-2nd Narrows	√		√	√	√				√	√					√					
T9	Port Moody	√	√	√	√	√			√	√	√		√		√	√	√	√	√		√
T12	Chilliwack	√		√	√	√		√	√	√	√	√	√			√	√	√	√	√	√
T13	North Delta			√		√				√						√	√				√
T14	Burnaby Mountain			√		√										√	√		√		√
T15	Surrey East			√	√	√				√						√	√				√
T17	Richmond South	√		√	√	√				√						√	√				√
T18	Burnaby South	√		√	√	√			√	√	√	√	√	√	√	√	√		√	√	√
T20	Pitt Meadows	√		√	√	√			√	√		√				√	√		√	√	√
T22	Burnaby-Burmount		√				√						√			√	√				
T23	Burnaby-Capitol Hill	√	√													√	√				
T24	Burnaby North	√	√				√		√				√			√	√				√
T26	N. Vancouver-Mahon Park	√		√	√	√			√	√						√	√	√		√	√
T27	Langley	√		√	√	√			√	√						√	√		√	√	√
T29	Hope			√	√	√			√	√						√	√		√		√
T30	Maple Ridge			√	√	√										√	√				√
T31	Richmond-Airport	√		√	√	√			√	√	√	√	√			√	√	√	√	√	√
T32	Coquitlam			√	√	√										√	√	√	√	√	√
T33	Abbotsford-Mill Lake	√		√	√	√		√	√	√						√	√				√
T35	Horseshoe Bay				√					√						√	√		√		√
T37	Alex Fraser Bridge															√	√		√		
T38	Annacis Island															√	√		√		√
T39	Tsawwassen	√		√	√	√				√						√	√		√	√	√
T45	Abbotsford Airport	This station was relocated during 2011 (station ID changed from T34 to T45)																			
20	White Rock														√						
Total Monitoring Units		16	5	21	19	20	2	2	11	17	5	4	6	1	3	25	24	5	14	8	20

SO_2 = sulphur dioxide; TRS = total reduced sulphur; NO_2 = nitrogen dioxide; CO = carbon monoxide; O_3 = ozone; THC = total hydrocarbon; NH_3 = ammonia; PM_{10} = inhalable particulate matter; $PM_{2.5}$ = fine particulate matter; NEPH = particulate light scattering; VOC = volatile organic compounds; SP = particulate speciation; D = dichotomous particulate; CARB = Carbon.

Wind = wind speed and wind direction; T_{air} = air temperature; SR = solar radiation; RH = relative humidity; BP = barometric pressure; Precip = precipitation.

√ = monitored at this location.

* = station did not operate during the entire year and data completeness is less than 75% complete.

(Source: Metro Vancouver, 2011)