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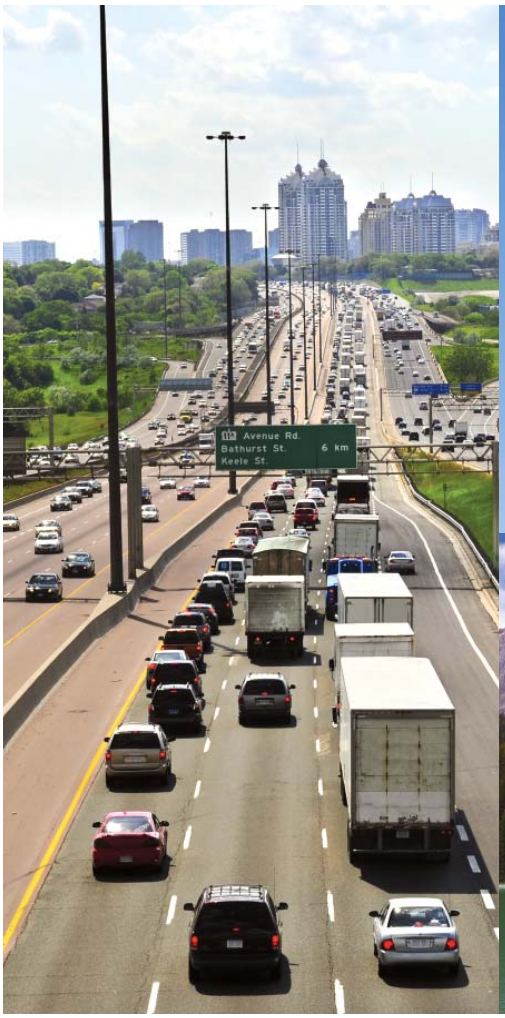
Health Risks and Benefits Associated with the Use of 10% Ethanol-blended Gasoline in Canada



Canada

Health Risks and Benefits Associated with the Use of 10% Ethanol-blended Gasoline in Canada

Prepared by:

Water, Air and Climate Change Bureau
Healthy Environments and
Consumer Safety Branch

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Executive summary

Transportation activities have a significant impact on urban air pollution and consequently, human health. This risk assessment addressed the human health implications that might arise from changes in air quality due to increased use in Canada of E10 fuel, a formulation of gasoline containing 10% ethanol per volume. The health impacts of E10 were compared to the baseline, conventional gasoline.

Studies of ethanol-blended fuels have indicated that ethanol addition to gasoline may modify the vehicle emission and secondary formation of several chemicals including: ethanol, acetaldehyde, benzene, 1,3-butadiene, formaldehyde, carbon monoxide (CO), nitrogen dioxide (NO_2), ozone (O_3), particulate matter (PM), peroxyacetyl nitrate (PAN), and sulphur dioxide (SO_2). Though some of these substances have multiple routes of exposure, for the purposes of evaluating the widespread use of E10 fuel compared to gasoline, inhalation exposure was expected to dominate and was the focus of the risk assessment.

Ambient concentrations of hazardous air pollutants were determined using a combination of: modelling of vehicle emissions; quantification of changes in the emission inventory based on changes in vehicle emissions; and, atmospheric modelling. To assess the impact of E10 fuel, two geographical regions of Canada were considered:

- West domain: an area extending from the western shore of Vancouver Island to the B.C./Alberta border, and 500 km on either side of the Canada/U.S. border; and
- East domain: an area covering the most densely-populated areas of southeastern Canada in Ontario and Quebec, and most of the northeastern U.S.

Atmospheric modelling of both the West and East domains indicated that widespread use of E10 had almost no impact on concentrations of several criteria air contaminants (NO_2, SO_2, O_3, and $PM_{2.5}$) and formaldehyde, and some impact on benzene, 1,3-butadiene, acetaldehyde, and CO, compared to conventional gasoline. Overall, atmospheric modelling results indicated that a change to E10 fuel in Canada would not substantially change the levels of pollutants observed in ambient air.

For the criteria air contaminants (CO, NO_2, O_3, $PM_{2.5}$, and SO_2), human health risks and benefits for widespread use of E10 fuel were estimated using Health Canada's Air Quality Benefit Assessment Tool (AQBAT). Changes in cancer risk due to a change in fuel were estimated for exposure to benzene, 1,3-butadiene, formaldehyde and acetaldehyde. Overall, there were no substantial differences in the predicted health effects for widespread use of E10 fuel compared to conventional gasoline.

The techniques used in this assessment are considered conservative and are based on the current scientific understanding of vehicle emissions, atmospheric chemistry, human exposure and human health effects of environmental pollutants.

Preface

Air pollution and climate change concerns have prompted governments to introduce measures to reduce the use of fossil fuels. One recent fossil fuel reduction initiative that has been widely implemented is the "greening of fuel" by the addition of ethanol to gasoline. Health Canada has completed a risk assessment evaluating the potential risks and benefits to the health of Canadians from the use of E10, a formulation of gasoline containing 10% ethanol per volume. E10 is the most common proportion for ethanol-blended fuel and is considered usable in gasoline engines without the need for mechanical modifications.

The risk assessment of E10 fuel focused on the human health implications due to changes in air quality that might arise from its widespread use in Canada. The risk assessment compared the health impacts of E10 blended fuel to those of conventional gasoline. Risk characterization was performed for: ozone (O_3), nitrogen dioxide (NO_2), particulate matter of 2.5 µm or less in diameter ($PM_{2.5}$), sulphur dioxide (SO_2), carbon monoxide (CO), acetaldehyde, benzene, 1,3-butadiene, formaldehyde, ethanol, and peroxyacetyl nitrate (PAN).

This risk assessment is important due to the emergence of Canada's Renewable Fuels Strategy, announced in 2006, which requires increased use of biofuels, including ethanol. The strategy will require a 5% renewable content for gasoline by 2010, and has the potential to reduce greenhouse gas (GHG) emissions by about 4 megatonnes per year, the equivalent, in terms of GHG emissions, of taking almost one million vehicles from the road.

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1.0 Introduction

The Canadian government introduced an amendment to the *Canadian Environmental Protection Act* (CEPA) aimed in part at reducing the amount of greenhouse gas emissions from the transportation sector. The amendment, which came into force on September 28, 2009, provides changes to CEPA that allow for the development of regulations requiring minimum levels of biofuel content in gasoline, diesel fuel and heating oil. Regulations enabled by this amendment will require the use of a prescribed amount of renewable fuel that will lead to a reduction in greenhouse gas emissions by approximately four megatonnes per year—about the equivalent of taking one million vehicles off the road. This could be achieved through implementing federal regulations by 2010 requiring an average of 5% renewable content in the national gasoline pool. Also, a requirement for 2% renewable content in diesel fuel and heating oil is intended to be implemented by 2011 or earlier, subject to technical feasibility.

An example of a renewable fuel is E10, a formulation of gasoline containing 10% ethanol per volume. This is the most common form of ethanol-blended fuel, as E10 can be used interchangeably with gasoline in most vehicles without any engine modifications. Currently, most ethanol-blended fuel in Canada is at the E10 level or lower and is available at over 1,000 gas stations across the country.

Initially (in the 1970s and 1980s), ethanol-blended gasoline was introduced in order to reduce exhaust emissions of carbon monoxide (CO); however, advances in emission control technologies have reduced this relative advantage of ethanol blends. Presently, the promotion of ethanol-blended gasoline focuses on the renewable aspect of the ethanol content and the associated reductions in greenhouse gas emissions. Corn and wheat, the primary feedstocks used in fuel-grade ethanol production, capture carbon dioxide (CO_2) as they grow, leading to reduced life-cycle greenhouse gas emissions compared to conventional gasoline. As a result of support from Government of Canada programs, including the Ethanol Expansion Program and the Renewable Fuels Strategy, production of ethanol for fuel has increased steadily from approximately 200 million litres (ML) in 2004 to over 1,300 ML in 2009, with further increases expected. Additionally, research into second-generation technologies to produce ethanol from non-food renewable resources (e.g., straw, crop waste, forestry residues) is continuing.

Motor vehicle emissions are a major source of urban air pollutants in Canada. Their components include carbon monoxide (CO), particulate matter (PM), ozone (O_3), nitrogen dioxide (NO_2), benzene, 1,3-butadiene, and aldehydes. These air pollutants are known to be associated with multiple human health effects including respiratory difficulties, cardiovascular health issues, cancer, and mortality. Changes in fuel composition, such as the addition of ethanol, can impact the quality and quantity of vehicle emissions. As such, it is important to examine the potential changes in human exposure that could result from a major change in fuel composition such as the blending of ethanol with gasoline.

1.1 Scope of assessment

The transportation sector is a major contributor to urban air pollution. Given the ubiquitous exposure of individuals in urban centres to air pollution, and the significant health effects associated with air pollutants, this risk assessment studied the human health implications due to changes in air quality that might arise from widespread use of E10 fuel in Canada.

The health impacts of vehicle emissions associated with E10 fuel were evaluated relative to those of conventional gasoline. Other possible emissions from the production, transportation, and distribution of E10 fuel, and the potential effects of leaked E10 fuel on aquifer contamination were beyond the scope of this analysis. Potential health benefits from reduced greenhouse gases due to the use of renewable fuel (ethanol) and its economic impacts were also beyond the scope of this assessment.

The use of motor vehicle fuels, including conventional gasoline or E10 fuel, results in the release of pollutants to the atmosphere. In addition, these pollutants may undergo subsequent atmospheric reactions, leading to the formation of secondary air pollutants. For example, peroxyacetyl nitrate (PAN) is a product of the photo-oxidation of acetaldehyde in polluted air. Studies of ethanol-blended fuels have indicated that ethanol addition to fuel may modify human exposure to several chemicals including: ethanol, acetaldehyde, benzene, 1,3-butadiene, carbon monoxide (CO), nitrogen dioxide (NO_2), ozone (O_3), particulate matter (PM), peroxyacetyl nitrate (PAN), and sulphur dioxide (SO_2). Though some of these substances have multiple routes of exposure, for the purposes of evaluating the widespread use of E10 fuel compared to gasoline, inhalation exposure was expected to dominate and was the focus for this assessment.

2.0 Atmospheric modelling

A necessary step in evaluating human inhalation exposure to pollutants is determining the ambient concentrations of the various pollutants. For this risk assessment, ambient concentrations of hazardous air pollutants were determined using a combination of: modelling of vehicle emissions; quantification of changes in the emission inventory based on changes in vehicle emissions; and, atmospheric modelling. Scenarios were considered for a West domain (extending from the western shore of Vancouver Island to the B.C./Alberta border, and 500 km on each side of the Canada/U.S. border) and an East domain (covering the most densely populated areas of southeastern Canada in Ontario and Quebec, and most of the northeastern U.S.), including the major cities located in the two domains. For the West domain, the years 2000 and 2010 were modelled, in which all on-road, gasoline-powered vehicles (except motorcycles) were considered to be using either conventional fuel or E10 fuel. For the East domain, only the year 2000 was modelled, in which all on-road, gasoline-powered vehicles (except motorcycles) were considered to be using either conventional fuel or E10. As emission scenarios for the years 2000 and 2010 had been previously designed, these years were chosen for the basis of this assessment. For each modelled domain and year, conventional gasoline was the baseline scenario against which the E10 fuel scenario was compared.

2.1 Vehicle emissions modelling

Quantification of the effects of E10 fuel on vehicle emissions was performed using the Canadian version of the MOBILE6.2 model (MOBILE6.2C). Developed by the U.S. Environmental Protection Agency, MOBILE is the most common and recognized vehicle emissions model used in Canada and the United States. Emissions for different vehicle classes, temperatures, and speeds were modelled for CO, nitrogen oxides (NO_x), $PM_{2.5}$, benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and other volatile organic compounds (VOCs). Results from the modelling indicated that for all scenarios, use of E10 fuel reduced emissions of CO, VOCs, benzene, and 1,3-butadiene compared to the baseline (conventional gasoline). Considering all three scenarios (West 2000, West 2010, and East 2000), the modelled decreases in emission rates between E10 fuel and conventional gasoline were: for CO, 8.9 to 19.4%; for 1,3-butadiene, 20.0 to 22.9%; for benzene including tailpipe and evaporative emissions, 20.8 to 23.4%; and, for other VOCs including tailpipe and evaporative emissions, 6.0 to 6.9%.

These reductions in emissions were attributed to improved fuel combustion due to the 10% blend of ethanol. Additionally, the reductions in CO emissions for the West 2010 scenario (8.9%) were less than for the West 2000 scenario (19.4%). This reduced efficacy of ethanol to lower CO emissions for the 2010 scenario was attributed to the replacement of older vehicles by newer vehicles with improved emissions control technology. This supports the observation from recent vehicle emission studies that the efficiency of ethanol in reducing vehicle emissions has less impact in more recent and advanced vehicles.

Increases in acetaldehyde emissions for E10 fuel of 118.2 to 136.8% were observed for all three scenarios and attributed to the oxidation of ethanol to acetaldehyde during combustion. No changes in emission quantities were observed for E10 fuel relative to conventional gasoline for NO_x, $PM_{2.5}$, NH_3 ammonia, and SO_2. Variable results were obtained for formaldehyde. The change in emission rates for E10 fuel ranged from an increase of 4.5% (West 2000 scenario) to a decrease of 5.2% (West 2010 scenario). This variability reflects the change in vehicle inventories between 2000 and 2010, and the impact of emission regulations introduced in 2004 for formaldehyde.

2.2 Modelling ambient air pollutant concentrations

Modelling was judged to be the most reliable way of obtaining source information as no Canadian monitoring data existed that was representative of atmospheric concentrations of vehicle-related pollutants in locations using E10 fuel for the entire fleet. Baseline (conventional gasoline) emission inventories were developed for the East domain (2000) and West domain (2000 and 2010). The baseline emission inventories were adjusted using MOBILE6.2C emissions results for scenarios in which all gasoline-powered vehicles (except motorcycles) were operated using E10 fuel. Atmospheric modelling of the various scenarios was conducted using the Community Multiscale Air Quality model (CMAQ) version 4.3. A modified version of the Statewide Air Pollution Research Center (SAPRC-*99*) chemical mechanism was employed within CMAQ to model the formation of secondary pollutants (e.g., O_3) from primary pollutants (e.g., VOCs) in the emissions inventories.

Atmospheric modelling estimates pollutant concentrations at different heights above the ground surface. Pollutant concentrations for the lowest model layer, representing a height of approximately 20 m above ground level, were extracted from the CMAQ model output. Model results consisted of multiple grid cells covering the geographic areas of the East (36 x 36 km) and West (12 x 12 km) domains. The cell mapping of the domains was not a perfect overlay of Canadian census division (CD) mapping. Since the human health risk characterization utilized in this risk assessment was based on CD mapping, the modelled concentrations were weighted in order to allocate a concentration to each CD in the East and West domains. The modelled concentration allocated to a specific CD was weighted based on the proportion of a cell covering the CD and the concentration of pollutants modelled in that cell, for each of the cells overlapping with the CD. These weighted concentrations were also scaled using the National Air Pollution Surveillance Network (NAPS) summer averages. Scaling is a technique by which ratios of modelled results from different scenarios are applied to actual monitored concentrations in order to obtain more representative and accurate atmospheric concentrations for modelled scenarios. Summer NAPS monitoring results from a five-year period (1998–2002) were used as a multi-year average and were considered to be more representative of typical concentrations, and less susceptible to year-to-year variations that result from climatic variability and human activities.

Modelled summer atmospheric pollutant concentrations for the West domain are provided in Table 1 (2000) and Table 2 (2010). For the West domain, widespread use of E10 fuel reduced summer atmospheric concentrations of benzene, 1,3-butadiene, and CO for the 2000 and 2010 scenarios, and increased atmospheric concentrations of acetaldehyde for both years. The differences in atmospheric concentrations between E10 and conventional gasoline for the West domain were:

- Year 2000: benzene, -9.0%; 1,3-butadiene, -7.4%; CO, -8.2%; and, acetaldehyde, +2.6%;
- Year 2010: benzene, -7.4%; 1,3-butadiene, -6.0%; CO, -3.5%; and, acetaldehyde, +1.2%.

Decreases in atmospheric concentrations of the air pollutants were attributed to improved fuel combustion due to the ethanol content in the E10 fuel. In comparison, the increase in acetaldehyde was attributed to increases in exhaust emissions and increased photochemical oxidation of ethanol in the atmosphere. The magnitude of these changes was found to be less important between the E10 and conventional fuel scenarios for 2010 than the two scenarios for 2000. As with the exhaust emissions modelling, this difference was attributed to the replacement of older vehicles in the fleet by newer vehicles with more advanced emissions control technologies. Additionally, for the West domain, no noticeable changes in the atmospheric concentrations of formaldehyde, NO_2, O_3, SO_2, $PM_{2.5}$, and PAN were observed between the E10 and gasoline fuel scenarios. Summer atmospheric concentrations for Vancouver were extracted from the West domain results for the gasoline baseline and E10 fuel scenarios for year 2000 and 2010. The trends for Vancouver were similar to those for the West domain described previously.

Table 1. Year 2000 summer atmospheric concentrations for the West domain

April–September (mean)				
Chemical/averaging time	**B 2000[1]**	**E10 2000**	**Delta**	**%**
Benzene (µg/m³)/24h	1.39	1.26	-0.13	-9.0
1,3-Butadiene (µg/m³)/24h	0.21	0.19	-0.02	-7.4
Acetaldehyde (µg/m³)/24h	2.29	2.35	0.06	2.6
Formaldehyde (µg/m³)/24h	2.25	2.25	0.00	0.0
CO (ppm)/24h	0.52	0.47	-0.04	-8.2
NO_2 (ppb)/24h	10.72	10.72	0.00	0.0
O_3 (ppb)/1h	37.48	37.43	-0.05	-0.1
O_3 (ppb)/8h	33.49	33.45	-0.04	-0.1
O_3 (ppb)/24h	21.74	21.72	-0.02	-0.1
SO_2 (ppb)/24h	1.53	1.53	0.00	0.0
$PM_{2.5}$ (µg/m³)/1h	13.16	13.15	0.00	0.0
$PM_{2.5}$ (µg/m³)/24h	5.19	5.19	0.00	0.0
PAN[2] (ppb)/24h	0.34	0.34	0.00	-0.2

Notes:
1. Baseline for 2000 is 1998–2002 NAPS average.
2. PAN results are unscaled.

Abbreviations: B = baseline (gasoline); Delta = change from baseline; % = percentage change from baseline.

Table 2. Year 2010 summer atmospheric concentrations for the West domain

April–September (mean)				
Chemical/averaging time	**B 2010**	**E10 2010**	**Delta**	**%**
Benzene (µg/m³)/24h	1.15	1.06	-0.09	-7.4
1,3-Butadiene (µg/m³)/24h	0.13	0.13	-0.01	-6.0
Acetaldehyde (µg/m³)/24h	2.17	2.19	0.03	1.2
Formaldehyde (µg/m³)/24h	2.08	2.08	0.00	-0.2
CO (ppm)/24h	0.51	0.49	-0.02	-3.5
NO_2 (ppb)/24h	8.72	8.72	0.00	0.0
O_3 (ppb)/1h	36.11	36.09	-0.02	-0.1
O_3 (ppb)/8h	32.25	32.23	-0.02	-0.1
O_3 (ppb)/24h	21.09	21.09	-0.01	0.0
SO_2 (ppb)/24h	1.64	1.64	0.00	0.0
$PM_{2.5}$ (µg/m³)/1h	13.54	13.54	0.00	0.0
$PM_{2.5}$ (µg/m³)/24h	5.35	5.35	0.00	0.0
PAN[1] (ppb)/24h	0.33	0.33	0.00	-0.1

Notes:
1. PAN results are unscaled.

Abbreviations: B = baseline (gasoline); Delta = change from baseline; % = percentage change from baseline.

For the East domain (Table 3), atmospheric modelling indicated that widespread use of E10 fuel had almost no effect on the summer atmospheric concentrations of all pollutants of interest. The differences in atmospheric concentrations between the use of E10 fuel and conventional gasoline for 2000 were: -1.3% for benzene; -0.9% for 1,3-butadiene; -0.7% for CO; and +0.3% for acetaldehyde. The magnitude of the modelled changes in the East domain was less than the modelled changes for the West domain. It was inferred that emissions associated with the transportation sector over the entire inventory are less important in the East domain, causing the alteration in vehicle emissions to have less impact on atmospheric concentrations.

Table 3. Year 2000 summer atmospheric concentrations for the East domain

April–September (mean)				
Chemical/averaging time	**B 2000[1]**	**E10 2000**	**Delta**	**%**
Benzene (µg/m³)/24h	0.83	0.82	-0.01	-1.3
1,3-Butadiene (µg/m³)/24h	0.09	0.09	0.00	-0.9
Acetaldehyde (µg/m³)/24h	1.56	1.56	0.00	0.3
Formaldehyde (µg/m³)/24h	2.55	2.55	0.00	0.0
CO (ppm)/24h	0.43	0.43	0.00	-0.7
NO_2 (ppb)/24h	12.58	12.58	0.00	0.0
O_3 (ppb)/1h	44.79	44.79	-0.01	0.0
O_3 (ppb)/8h	41.36	41.36	-0.01	0.0
O_3 (ppb)/24h	29.31	29.31	0.00	0.0
SO_2 (ppb)/24h	5.43	5.43	0.00	0.0
$PM_{2.5}$ (µg/m³)/1h	20.14	20.14	0.00	0.0
$PM_{2.5}$ (µg/m³)/24h	8.84	8.84	0.00	0.0
PAN[2] (ppb)/24h	0.32	0.32	0.00	0.0

Notes:
1. Baseline (gasoline) for 2000 is 1998–2002 NAPS average.
2. PAN results are unscaled.

Abbreviations: B = baseline (gasoline); Delta = changes from baseline; % = percentage changes from baseline.

Summer atmospheric concentrations for Toronto and Montreal were extracted from the East domain results for the baseline and E10 fuel scenarios for the year 2000. Results obtained from modelling the Toronto 2000 summer atmospheric concentrations were similar to those for the East domain. Only minor changes in pollutant concentrations due to the use of E10 fuel were observed. The largest difference between fuel scenarios was a decrease in benzene concentration by 1.0% for E10 fuel. For the Montreal summer 2000 atmospheric modelling, decreases of 2.6% for benzene, 1.6% for CO, and 1.0% for 1,3-butadiene were observed for E10 fuel compared to conventional gasoline. Additionally, there was an increase of 1.7% for the 8-hour O_3 concentration in Montreal for the E10 fuel, while no difference in O_3 concentration was observed for Toronto. Similar to the differences between the East and West domain results, the modelling of widespread use of E10 fuel had less impact on the atmospheric pollutant concentrations in Toronto and Montreal compared to Vancouver.

Overall, atmospheric modelling results indicated that simulated fleet-wide use of E10 fuel had almost no impact on several criteria air contaminant (NO_2, SO_2, O_3, and $PM_{2.5}$) concentrations compared to conventional gasoline, regardless of location or year. Compared to the East domain, the transportation sector in the West domain is a greater proportion of overall pollution. For this reason, simulated use of E10 fuel for the West domain had some impact on pollutants which are primarily associated with traffic, including benzene, 1,3-butadiene, acetaldehyde, and CO. The largest decrease in pollutant concentration was 9.0% for benzene and the largest increase was 2.6% for acetaldehyde, and both were observed in the 2000 West domain results. Additionally, a change in fuel emissions had less effect on the atmospheric pollutant concentrations in the East domain. Overall, the modelled results indicated that a change to E10 fuel in Canada would reduce some pollutants of interest and slightly increase acetaldehyde.

3.0 Personal exposure modelling

In order to determine the overall daily human exposure to various pollutants for Toronto and Vancouver residents, the chemical concentration data for different microenvironments (MEs) were established for the baseline conventional gasoline based on the scientific literature. MEs considered for this risk assessment included: indoor-homes; indoor-homes with attached garages; indoor-public buildings; indoor-other (offices); indoor-parking garage; in-vehicle; outdoor-parking garage; outdoor-roadside; outdoor-gas station at a pump island–no refuelling in progress; and outdoor-gas station-refuelling in progress.

For the E10 fuel, the ME concentrations were estimated from the baseline information using a scaling method for Toronto and Vancouver (year 2000 for both cities). Scaling equations specific for each ME were developed to quantify the hypothetical ME concentrations that would result from changes in ambient concentration and emission changes from the widespread use of E10 fuel. The magnitude of the changes in scaled pollutant concentrations for E10 fuel was dependent on the ME and pollutant. ME concentrations of ethanol, acetaldehyde, and formaldehyde increased compared to the baseline, while benzene and 1,3-butadiene decreased. Detailed ME concentrations for $PM_{2.5}$, O_3, NO_2, PAN, and CO were not required, as a different risk characterization approach based on ambient pollutant concentrations was used to quantify human health risks from these pollutants (Section 4).

The changes in ME concentration between the gasoline baseline and the E10 fuel were influenced by the changes in vehicle emissions, resultant changes in ambient atmospheric concentrations, and the proportion of the ME concentration attributable to vehicle emissions. The MEs in which the pollutant concentrations were largely due to vehicle emissions would be the most impacted by changes in emissions. As expected, the MEs with the largest changes were: indoor-parking garage; outdoor-parking garage; outdoor-roadside; outdoor-gas station at a pump island; and outdoor-gas station-refuelling in progress. Larger relative changes in ME concentration were predicted for Vancouver as greater changes in atmospheric concentration were modelled for Vancouver for the E10 fuel compared to conventional gasoline.

3.1 Exposure assessment

To evaluate the inhalation exposure of Canadians to various vehicle-emission air pollutants, the Canadian Exposure Model (CEM) was employed. CEM is a stochastic model designed to quantify inhalation exposure to pollutants in various MEs. Exposure estimates were generated for six age groups: 0 to 0.5 years; 0.5 to 4 years; 5 to 11 years; 12 to 19 years; 20 to 59 years; and, 60+ years. This model uses specific chemical concentration data, in combination with age-specific time-activity distribution data, and applies appropriate inhalation rates and body weights to develop distributions of potential daily exposures. Time-activity data was derived from population survey data collected by Health Canada under the Canadian Human Activity Pattern Survey (CHAPS), which provided information regarding the amount of time respondents spent in each microenvironment (ME).

Inhalation exposure to ethanol, benzene, 1,3-butadiene, acetaldehyde, and formaldehyde was determined for the six age groups for Toronto (Table 4) and Vancouver (Table 5). The level of exposure was found to be age-dependent, with toddlers (0.5 to 4 years) having the greatest exposure to all compounds. This age group has the highest inhalation rate to body weight ratio, which resulted in the highest intake on a per weight basis. The 60+ years age group was the least exposed to ethanol, benzene, 1,3-butadiene, and formaldehyde; the 20 to 59 age group had the lowest exposure to acetaldehyde.

Widespread use of E10 fuel decreased exposure to benzene and 1,3-butadiene, increased exposure to ethanol and acetaldehyde, and had a negligible effect on formaldehyde exposure. For Toronto, the greatest percent decreases in exposure, compared to the gasoline baseline, were observed in the 20 to 59 age group, 7.2% for benzene and 4.9% for 1,3-butadiene. The greatest percent increases in exposure for Toronto,

when compared to the gasoline baseline were in the 5 to 11 age group for ethanol (0.6%), and in the 20 to 59 age group for acetaldehyde (0.8%). For Vancouver, the magnitude of change in exposure for E10 fuel use was slightly greater, which was as expected due to the larger relative changes in ME concentrations quantified for this city. The greatest decreases were also observed in the 20 to 59 age group for Vancouver, 9.6% and 9.0% for benzene and 1,3-butadiene, respectively. The greatest increase in ethanol exposure was 0.6% for the 5 to 11 age group, and in acetaldehyde exposure, 1.1% for the 20 to 59 age group. The observed differences in the modelled exposures between the age groups are a result of their differing time-activity patterns, inhalation rates, the relative concentration of the pollutants in the different MEs, and the magnitude of the effect of vehicle-related emissions to these MEs.

Table 4. Baseline and E10 scenario exposure results by age group for Toronto

Group	Mean baseline exposure (µg/kg-bw/d)[1]	Mean E10 exposure (µg/kg-bw/d)	Changes from baseline to E10	
			Absolute difference	Percentage difference
0–0.5 years				
Total ethanol	175.02	175.84	0.82	0.47
Total benzene	0.49	0.46	-0.02	-5.06
Total 1,3-butadiene	0.06	0.06	0.00	-2.51
Total acetaldehyde	6.92	6.94	0.02	0.31
Total formaldehyde	6.30	6.30	0.00	0.02
0.5–4 years				
Total ethanol	376.55	378.33	1.79	0.47
Total benzene	1.15	1.09	-0.05	-4.56
Total 1,3-butadiene	0.14	0.13	0.00	-2.39
Total acetaldehyde	13.79	13.83	0.04	0.31
Total formaldehyde	13.19	13.19	0.00	0.02
5–11 years				
Total ethanol	248.01	249.43	1.42	0.57
Total benzene	1.01	0.95	-0.06	-5.77
Total 1,3-butadiene	0.11	0.10	0.00	-3.50
Total acetaldehyde	8.24	8.29	0.05	0.55
Total formaldehyde	8.56	8.56	0.00	0.03
12–19 years				
Total ethanol	145.26	146.02	0.76	0.52
Total benzene	0.54	0.52	-0.03	-4.66
Total 1,3-butadiene	0.06	0.06	0.00	-2.74
Total acetaldehyde	4.64	4.66	0.02	0.43
Total formaldehyde	4.89	4.89	0.00	0.02
20–59 years				
Total ethanol	130.39	131.05	0.66	0.51
Total benzene	0.58	0.54	-0.04	-7.21
Total 1,3-butadiene	0.06	0.05	0.00	-4.92
Total acetaldehyde	4.01	4.04	0.03	0.79
Total formaldehyde	4.33	4.33	0.00	0.05
60+ years				
Total ethanol	118.38	118.97	0.59	0.50
Total benzene	0.40	0.38	-0.02	-5.61
Total 1,3-butadiene	0.05	0.04	0.00	-3.18
Total acetaldehyde	4.26	4.28	0.02	0.42
Total formaldehyde	4.17	4.17	0.00	0.02

Note:
1. µg/kg-bw/d = microgram per kilogram body weight per day.

Table 5. Baseline and E10 scenario exposure results by age group for Vancouver

Group	Mean baseline exposure (µg/kg-bw/d)[1]	Mean E10 exposure (µg/kg-bw/d)	Changes from baseline to E10	
			Absolute difference	Percentage difference
0–0.5 years				
Total ethanol	174.64	175.46	0.81	0.47
Total benzene	0.48	0.44	-0.04	-7.57
Total 1,3-butadiene	0.06	0.06	0.00	-7.05
Total acetaldehyde	6.91	6.95	0.04	0.62
Total formaldehyde	6.27	6.27	0.00	0.02
0.5–4 years				
Total ethanol	375.47	377.25	1.79	0.48
Total benzene	1.14	1.06	-0.08	-7.38
Total 1,3-butadiene	0.14	0.13	-0.01	-7.13
Total acetaldehyde	13.80	13.89	0.09	0.65
Total formaldehyde	13.13	13.14	0.00	0.02
5–11 years				
Total ethanol	247.86	249.28	1.42	0.57
Total benzene	1.01	0.92	-0.09	-8.68
Total 1,3-butadiene	0.11	0.10	-0.01	-8.26
Total acetaldehyde	8.25	8.33	0.08	0.92
Total formaldehyde	8.48	8.48	0.00	0.03
12–19 years				
Total ethanol	145.58	146.35	0.77	0.53
Total benzene	0.55	0.51	-0.04	-7.50
Total 1,3-butadiene	0.06	0.05	0.00	-7.52
Total acetaldehyde	4.64	4.68	0.04	0.78
Total formaldehyde	4.84	4.85	0.00	0.02
20–59 years				
Total ethanol	130.51	131.18	0.66	0.51
Total benzene	0.59	0.53	-0.06	-9.57
Total 1,3-butadiene	0.06	0.05	-0.01	-8.97
Total acetaldehyde	4.01	4.06	0.05	1.14
Total formaldehyde	4.32	4.32	0.00	0.05
60+ years				
Total ethanol	119.24	119.83	0.60	0.50
Total benzene	0.41	0.37	-0.03	-8.25
Total 1,3-butadiene	0.05	0.04	0.00	-7.74
Total acetaldehyde	4.28	4.31	0.03	0.76
Total formaldehyde	4.13	4.13	0.00	0.02

Note:
1. µg/kg-bw/d = microgram per kilogram body weight per day.

In addition to assessing exposure for the six age groups, the CEM was used to assess the exposures of three predefined receptors. Receptor 1 was an adult (20 to 59 age group), who commuted by car every day to work in an office, lived in a house with an attached garage, and for the day of analysis, visited a gas station to refuel a car. Receptor 2 was a pre-school child (0.5 to 4 age group) who spent the day indoors or outdoors at a house without an attached garage. Receptor 3 was an adult (20 to 59 age group) who commuted by car every day to work at a construction site, lived in a house without an attached garage, and for the day of analysis, visited a gas station to refuel a car.

Modelled exposures for the predefined receptors were similar for Toronto and Vancouver (Table 6). Receptor 2 had the greatest exposure to all pollutants except benzene, for which Receptor 3 had the greatest exposure. Comparing exposures between the E10 fuel and conventional gasoline scenarios, Receptor 3 was the most impacted by changes in vehicle emissions, as a significant proportion of time was spent in the outdoor-roadside ME. Changes in exposure for Receptor 3 included:

- Toronto: benzene, -13.2%; and acetaldehyde, +4.6%;
- Vancouver: benzene, -15.2%; and acetaldehyde, +5.1%.

3.2 Estimated time-weighted exposures

The CEM was also utilized to estimate the time-weighted exposures (TWEs), which are an expression of the daily levels of a pollutant to which an individual would be exposed. TWEs were used to quantify the risk of benzene, 1,3-butadiene, acetaldehyde, and formaldehyde associated with the two fuel scenarios. Estimates were computed for the six age groups in Toronto (Table 7) and Vancouver (Table 8). For the simulated shift to widespread E10 use, the TWE was decreased for benzene and 1,3-butadiene, increased for ethanol and acetaldehyde, and showed a negligible effect for formaldehyde—the same pattern as for general inhalation exposure estimates. The differences in TWE estimates for the two fuel scenarios were slightly greater in Vancouver compared to Toronto. The greatest percent changes in TWEs with ethanol-blended fuel occurred in the 20 to 59 age group and were:

- Toronto: benzene, -7.4%; and acetaldehyde, +0.7%;
- Vancouver: benzene, -9.6%; and acetaldehyde, +1.0%.

Table 6. Baseline and E10 scenario exposure inhalation intake (µg/kg-bw/d)[1] results for predefined receptors in Toronto and Vancouver

	Ethanol			Benzene			1,3-Butadiene			Acetaldehyde			Formaldehyde		
	B[2]	E10	%	B	E10	%	B	E10	%	B	E10	%	B	E10	%
Toronto															
Receptor 1	180.34	181.59	0.69	1.14	1.04	-8.81	0.07	0.06	-8.18	4.30	4.45	3.48	3.69	3.69	0.13
Receptor 2	325.81	327.81	0.61	0.83	0.82	-0.73	0.12	0.12	0.02	13.40	13.42	0.11	12.10	12.10	0.03
Receptor 3	77.34	78.38	1.35	1.22	1.06	-13.16	0.11	0.10	-10.10	3.44	3.60	4.57	3.66	3.68	0.49
Vancouver															
Receptor 1	180.34	181.59	0.69	1.14	1.02	-10.66	0.07	0.06	-11.39	4.30	4.47	3.84	3.69	3.69	0.13
Receptor 2	325.81	327.79	0.61	0.82	0.78	-4.88	0.12	0.12	-5.51	13.46	13.52	0.47	11.96	11.96	0.02
Receptor 3	77.33	78.39	1.36	1.22	1.03	-15.15	0.11	0.09	-13.95	3.44	3.61	5.09	3.66	3.68	0.50

Notes:
1. µg/kg-bw/d = microgram per kilogram body weight per day.
2. B = conventional gasoline baseline.
Receptor 1 = adult office worker; Receptor 2 = child at home; Receptor 3 = adult construction worker.

Table 7. Time-weighted exposures for the baseline and E10 scenarios for Toronto

Group	Mean baseline TWE[1] (µg/m³)	Mean E10 TWE (µg/m³)	Changes from baseline to E10	
			Absolute difference	Percentage difference
0–0.5 years				
Ethanol	655.12	658.01	2.89	0.44
Benzene	1.64	1.58	-0.07	-3.96
1,3-Butadiene	0.22	0.21	0.00	-1.81
Acetaldehyde	25.81	25.87	0.06	0.23
Formaldehyde	23.43	23.43	0.00	0.01
0.5–4 years				
Ethanol	650.93	653.83	2.90	0.45
Benzene	1.86	1.77	-0.09	-4.83
1,3-Butadiene	0.23	0.22	-0.01	-2.50
Acetaldehyde	24.48	24.56	0.08	0.31
Formaldehyde	22.93	22.94	0.00	0.02
5–11 years				
Ethanol	612.33	615.31	2.98	0.49
Benzene	2.14	2.02	-0.12	-5.64
1,3-Butadiene	0.24	0.23	-0.01	-3.31
Acetaldehyde	21.19	21.28	0.10	0.46
Formaldehyde	21.07	21.07	0.01	0.03
12–19 years				
Ethanol	634.03	636.96	2.94	0.46
Benzene	2.04	1.94	-0.09	-4.65
1,3-Butadiene	0.23	0.22	-0.01	-2.60
Acetaldehyde	21.82	21.89	0.08	0.35
Formaldehyde	21.64	21.64	0.00	0.02
20–59 years				
Ethanol	625.34	628.23	2.89	0.46
Benzene	2.51	2.33	-0.18	-7.35
1,3-Butadiene	0.26	0.25	-0.01	-4.78
Acetaldehyde	20.85	20.99	0.14	0.67
Formaldehyde	21.23	21.24	0.01	0.04
60+ years				
Ethanol	638.07	641.00	2.93	0.46
Benzene	1.96	1.85	-0.11	-5.58
1,3-Butadiene	0.23	0.23	-0.01	-3.05
Acetaldehyde	23.79	23.88	0.09	0.38
Formaldehyde	22.45	22.46	0.00	0.02

Note:
1. TWE = time weighted exposure.

Table 8. Time-weighted exposures for the baseline and E10 scenarios for Vancouver

Group	Mean baseline TWE[1] (µg/m³)	Mean E10 TWE (µg/m³)	Changes from baseline to E10	
			Absolute difference	Percentage difference
0–0.5 years				
Ethanol	655.12	658.01	2.89	0.44
Benzene	1.64	1.53	-0.11	-6.75
1,3-Butadiene	0.22	0.20	-0.01	-6.56
Acetaldehyde	25.82	25.97	0.14	0.56
Formaldehyde	23.39	23.39	0.00	0.01
0.5–4 years				
Ethanol	650.93	653.83	2.90	0.45
Benzene	1.86	1.72	-0.14	-7.45
1,3-Butadiene	0.23	0.21	-0.02	-7.05
Acetaldehyde	24.50	24.65	0.16	0.64
Formaldehyde	22.90	22.90	0.00	0.02
5–11 years				
Ethanol	612.33	615.31	2.98	0.49
Benzene	2.14	1.96	-0.18	-8.27
1,3-Butadiene	0.24	0.22	-0.02	-7.79
Acetaldehyde	21.22	21.39	0.17	0.80
Formaldehyde	20.99	20.99	0.01	0.03
12–19 years				
Ethanol	634.03	636.96	2.94	0.46
Benzene	2.04	1.89	-0.15	-7.31
1,3-Butadiene	0.23	0.21	-0.02	-7.16
Acetaldehyde	21.84	21.99	0.15	0.69
Formaldehyde	21.58	21.58	0.00	0.02
20–59 years				
Ethanol	625.34	628.23	2.89	0.46
Benzene	2.51	2.27	-0.24	-9.60
1,3-Butadiene	0.26	0.24	-0.02	-8.74
Acetaldehyde	20.86	21.07	0.21	1.01
Formaldehyde	21.19	21.20	0.01	0.04
60+ years				
Ethanol	638.07	641.00	2.93	0.46
Benzene	1.96	1.80	-0.16	-8.15
1,3-Butadiene	0.23	0.22	-0.02	-7.50
Acetaldehyde	23.81	23.98	0.17	0.71
Formaldehyde	22.41	22.41	0.00	0.02

Note:
1. TWE = time weighted exposure.

TWE estimates were also determined for the predefined receptors in Toronto and Vancouver (Table 9). A similar pattern of results was observed for the predefined receptors as for the six age groups in both cities. The greatest impacts of simulated E10 fuel use on TWEs of the predefined receptors were: decreases in benzene exposure for Receptor 3 of 12.3% in Toronto and 13.9% in Vancouver; and, increases in acetaldehyde exposure for Receptor 1 of 3.2% in Toronto and 3.6% in Vancouver.

Table 9. Time-weighted exposures (µg/m^3) for predefined receptors in Toronto and Vancouver

	Ethanol			Benzene			1,3-Butadiene			Acetaldehyde			Formaldehyde		
	B[1]	E10	%	B	E10	%	B	E10	%	B	E10	%	B	E10	%
Toronto															
Receptor 1	966.06	972.60	0.68	5.36	4.93	-8.13	0.31	0.29	-7.50	23.53	24.29	3.23	19.04	19.06	0.10
Receptor 2	626.20	629.15	0.47	1.34	1.33	-0.62	0.20	0.20	0.00	25.46	25.49	0.08	22.73	22.73	0.00
Receptor 3	433.95	437.28	0.77	4.07	3.57	-12.26	0.34	0.31	-8.97	17.68	18.08	2.27	17.31	17.35	0.23
Vancouver															
Receptor 1	966.06	972.60	0.68	5.36	4.82	-10.06	0.31	0.28	-10.76	23.53	24.37	3.57	19.04	19.06	0.10
Receptor 2	626.20	629.15	0.47	1.34	1.28	-4.07	0.20	0.19	-4.79	25.50	25.61	0.42	22.63	22.63	0.00
Receptor 3	433.95	437.28	0.77	4.07	3.51	-13.93	0.34	0.30	-12.47	17.68	18.15	2.67	17.31	17.35	0.23

Notes:
1. B = conventional gasoline baseline.
Receptor 1 = adult office worker; Receptor 2 = child at home; Receptor 3 = adult construction worker.

4.0 Human health risk characterization

Risks were estimated for the criteria air contaminants using a risk characterization model developed by Health Canada, the Air Quality Benefits Assessment Tool (AQBAT). The health risks associated with exposure to benzene, 1,3-butadiene, and acetaldehyde were characterized using the U.S. EPA unit risks. For inhalation exposures, unit risks are the excess lifetime cancer risks associated with continuous exposure to a chemical at a concentration of 1 $\mu g/m^3$ in the air. Cancer risk associated with formaldehyde exposure was estimated using a model developed by the Chemical Industry Institute of Toxicology (CIIT). Quantitative risk characterizations were not completed for ethanol or peroxyacetyl nitrate (PAN) as no reliable inhalation risk estimates exist for these two compounds. Instead, their risks were estimated by comparing their reference concentrations with estimated TWEs and atmospheric levels, respectively.

4.1 Criteria air contaminants (CO, NO_2, O_3, $PM_{2.5}$, and SO_2)

Risks were estimated for the criteria air contaminants (CO, NO_2, O_3, $PM_{2.5}$, and SO_2) using the Air Quality Benefit Assessment Tool (AQBAT). This model combines changes in ambient pollutant concentrations with concentration-response functions derived from epidemiological literature. The differences in pollutant concentrations between the gasoline baseline and the E10 fuel scenarios were used to calculate the changes in various health outcomes across various populations.

Risk characterizations for the pollutants addressed by AQBAT do not require receptor-specific exposure assessment data because the dose-response relationships are based on ambient monitoring data. Ambient pollutant levels are integrated with model dose-response values to estimate the number of resultant or avoided health outcomes across the population. In addition, AQBAT can provide economic valuation estimates based on changes in the incidence of health effects associated with changes in air quality. AQBAT risk estimates are generated in relation to census divisions (CD). For the West domain, results were limited to the Greater Vancouver Regional District (GVRD) representing one CD. For the East domain, AQBAT results included 10 CDs covering parts of Ontario, Quebec, and New Brunswick.

Risk/benefit estimates from the simulation of wide-spread use of E10 for GVRD (West domain) were generated by AQBAT for 2000 and 2010 (Tables 10 and 11, respectively) and covered an estimated population of about 2 million people. For both years, the overall simulated effect of E10 use resulted in a decrease in premature mortality, hospital admissions, emergency room visits, acute respiratory symptom days, restricted activity days, and bronchitis.

Widespread use of E10 fuel resulted in overall health benefits, though very small in magnitude. Reduced effects were associated with changes in CO, NO_2, O_3, and $PM_{2.5}$ in both years, while a minor increased effect was associated with SO_2. For 2000, the health benefit for all endpoints was valued at $9.3 million, largely due to an estimated reduction of 1.8 premature deaths valued at approximately $8.9 million (Table 10). For 2010, the health benefit estimated for E10 fuel use compared to the gasoline baseline was valued at $6.5 million for all endpoints, largely due to a reduction of 1.3 premature deaths valued at $6.2 million (Table 11). These estimates should be regarded as negligible as they are well within the margin of error for this type of analysis.

AQBAT risk/benefit estimates for the 2000 East domain (Table 12), representing a population of about 8 million people, were less than the estimates derived for the 2000 West domain. Health benefits for the E10 fuel scenario were derived for CO, O_3, and SO_2, while increased risks were derived for NO_2 and $PM_{2.5}$. For all pollutants, benefits were realized for premature mortality (reduction of less than 1 premature death), emergency room visits, acute respiratory symptom days, and asthma days, with a valuation of $4.0 million. Similarly, as stated above, these estimates should be regarded as negligible.

Table 10. Changes in pollutant-specific valuation (count) for Greater Vancouver Regional District (West domain) from baseline to E10 scenarios, year 2000

Pollutant	Premature mortality $	Hospital admissions $	Emergency room visits $	Acute respiratory symptom days $	Asthma symptom days $	Restricted activity days $	Bronchitis $	All $
O_3	4.4 million (<1)[A]	0 (<1)[R]	0.007 million (2.6)[R]	0.1 million (5,339.0)	0.05 million (901.4)	0.04 million (1,536.7)[MR]		4.6 million
NO_2	0.3 million (<1)[A]							0.3 million
$PM_{2,5}$	0.8 million (<1)[C]	0 (<1)[R] 0 (<1)[C]	<1,000 (<1)[R] <1,000 (<1)[C] <1,000 (<1)[T]	0.008 million (319.9)	<1000 (9.3)	0.01 million (207.9)[R]	<1,000 (1.4)[CA] 0.07 million (<1)[AC]	0.9 million
SO_2	**-0.005 million** (<1)[A]							**-0.005 million**
CO	3.5 million (<1)[A]	0.06 million (9.1)[EC]						3.6 million
All-pollutant model	8.1 million (1.6)[A] 0.8 million (<1)[C] 8.9 million (1.8)[T]	0 (<1)[R] 0 (<1)[C] 0.05 million (9.1)[EC]	0.008 million (2.7)[R] <1,000 (<1)[C] 0.008 million (2.7)[T]	0.1 million (5,658.9)	0.05 million (910.6)	0.04 million (1,536.7)[MR] 0,01 million (207.9)[R]	<1,000 (1.4)[CA] 0.07 million (<1)[AC]	9.3 million
Abbreviations:	A: Acute exposure C: Chronic exposure T: Acute + Chronic	R: Respiratory C: Cardiac EC: Elderly cardiac T: Resp + Card + EC	R: Respiratory C: Cardiac T: Card + Resp			R: Restricted MR: Minor restricted	CA: Child acute episodes AC: Adult chronic cases	

Note: Negative values represent negative effects to society and should be considered an increase in cost or health effects. Positive values represent positive effects to society and should be considered a reduction in cost or health effects.

Table 11. Changes in pollutant-specic valuation (count) for Greater Vancouver Regional District (West domain) from baseline to E10 scenarios, year 2010

Pollutant	Premature mortality $	Hospital admissions $	Emergency room visits $	Acute respiratory symptom days $	Asthma symptom days $	Restricted activity days $	Bronchitis $	All $
O_3	3.5 million (<1)[A]	0 (<1)[R]	0.005 million (1.8)[R]	0.06 million (3,363.0)	0.03 million (567.9)	0.02 million (968.1)[MR]		3.6 million
NO_2	0.1 million (<1)[A]							0.1 million
$PM_{2,5}$	0.6 million (<1)[C]	0 (<1)[R] 0 (<1)[C]	<1,000 (<1)[R] <1,000 (<1)[C] <1,000 (<1)[T]	0.004 million (178.8)	<1,000 (5.3)	0.007 million (122.4)[R]	<1,000 (<1)[CA] 0.04 million (<1)[AC]	0.6 million
SO_2	**-0.002 million** (<1)[A]							**-0.002 million**
CO	2.1 million (<1)[A]	0.03 million (5.0)[EC]						2.1 million
Modèle de tous les polluants	5.7 million (1.1)[A] 0.6 million (<1)[C] 6.2 million (1.3)[T]	0 (<1)[R] 0 (<1)[C] 0.03 million (5.0)[EC]	0.005 million (1.8)[R] <1,000 (<1)[C] 0.005 million (1.8)[T]	0.07 million (3,541.8)	0.03 million (573.1)	0.02 million (968.1)[MR] 0.007 million (122.4)[R]	<1,000 (<1)[CA] 0.04 million (<1)[AC]	6.5 million
Abbreviations:	A: Acute exposure C: Chronic exposure T: Acute + Chronic	R: Respiratory C: Cardiac EC: Elderly cardiac T: Resp + Card + EC	R: Respiratory C: Cardiac T: Card + Resp			R: Restricted MR: Minor restricted	CA: Child acute episodes AC: Adult chronic cases	

Note: Negative values represent negative effects to society and should be considered an increase in cost or health effects. Positive values represent positive effects to society and should be considered a reduction in cost or health effects.

Table 12. Changes in pollutant-specific valuation (count) for East domain (10 CDs) from baseline to E10 scenarios, year 2000

Pollutant	Premature mortality $	Hospital admissions $	Emergency room visits $	Acute respiratory symptom days $	Asthma symptom days $	Restricted activity days $	Bronchitis $	All $
O_3	2.1 million (<1)[A]	0 (<1)[R]	0.004 million (1.3)[R] 0.004 million (1.3)[T]	0.05 million (2,479.2)	0.03 million (418.2)	0.02 million (713.0)[MR]		2.1 million
NO_2	**<-1,000 (<-1)[A]**							**<-1,000**
$PM_{2,5}$	**-0.3 million (<-1)[C]**	0 (<1)[R] 0 (<1)[C]	**<-1,000 (<-1)[R]** **<-1,000 (<-1)[C]** **<-1,000 (<-1)[T]**	**-0.003 million (-111.6)**	**<-1,000 (-3.2)**	**-0.004 million (-72.0)[R]**	**<-1,000 (<-1)[CA]** **-0.02 million (<-1)[AC]**	**-0.3 million**
SO_2	0.005 million (<1)[A]							0.005 million
CO	2.1 million (<1)[A]	0.03 million (5.4)[EC]						2.1 million
Modèle de tous les polluants	4.2 million (<1)[A] **-0.3 million (<-1)[C]** 3.9 million (<1)[T]	0 (<1)[R] 0 (<1)[C] 0.03 million (5.4)[EC]	0.004 million (1.2)[R] **<-1,000 (<-1)[C]** 0.004 million (1.2)[T]	0.04 million (2,367.5)	0.02 million (415.0)	0.02 million (713.0)[MR] **-0.004 million (-72.0)[R]**	**<-1,000 (<-1)[CA]** **-0.02 million (<-1)[AC]**	4.0 million
Abbreviations:	A: Acute exposure C: Chronic exposure T: Acute + Chronic	R: Respiratory C: Cardiac EC: Elderly cardiac T: Resp + Card + EC	R: Respiratory C: Cardiac T: Card + Resp			R: Restricted MR: Minor restricted	CA: Child acute episodes AC: Adult chronic cases	

Note: Negative values represent negative effects to society and should be considered an increase in cost or health effects. Positive values represent positive effects to society and should be considered a reduction in cost or health effects.

4.2 Benzene, 1,3-butadiene, formaldehyde, and acetaldehyde

Carcinogenic risks were characterized for benzene, 1,3-butadiene, formaldehyde, and acetaldehyde. Lifetime average exposures were determined from the TWE estimates (Section 3.2). These lifetime exposure estimates were evaluated using cancer unit risks from the U.S. EPA Integrated Risk Information System for benzene, 1,3-butadiene, and acetaldehyde. The unit risk for benzene is a range and for this assessment, cancer risks were estimated using the high and low ends of the range. Cancer risk due to formaldehyde exposure was estimated using the model developed by CIIT and used by Health Canada for the Priority Substance List assessment. In the case of formaldehyde, use of the biologically based mechanistic model was determined to be the most appropriate for this risk assessment.

Changes in cancer risk estimates are provided in Table 13. Compared to the conventional gasoline scenario, the simulated widespread use of E10 fuel resulted in decreased cancer risk estimates for benzene and 1,3-butadiene, while increased cancer risks were estimated for acetaldehyde. The magnitude of change in cancer risk was slightly greater for Vancouver than Toronto, as expected, based on the observed relative differences in exposure between the cities. The greatest decrease in cancer risk for the E10 fuel scenario was associated with benzene-low, with decreases of 1.2 and 1.6 per million people observed for Toronto and Vancouver, respectively. The greatest increase in cancer risk for the E10 fuel scenario was associated with acetaldehyde, with increases of 0.3 and 0.4 per million people observed for Toronto and Vancouver, respectively. No change in cancer risk was observed for formaldehyde in either city, reflecting the high degree of similarity in formaldehyde exposure between the fuel scenarios.

Additionally, the overall cancer risk for the estimated lifetime exposure to benzene, 1,3-butadiene, acetaldehyde, and formaldehyde was less for the E10 fuel compared to conventional gasoline. The decrease in overall cancer risk per million people ranged from 0.4 to 1.3, for Toronto, and was slightly greater at 0.8 to 1.9, for Vancouver (Table 13). As high and low unit risk values were used to estimate the risk associated with benzene, a range in overall cancer was obtained for each city. Of note, for the purpose of determining an overall cancer risk, estimated risks of the individual pollutants were presumed to be additive, while recognizing the potential limitation of this approach if the pollutants act synergistically. The changes in lifetime cancer risk should be considered as negligible, in consideration of the margin of error for this type of analysis.

Table 13. Changes in lifetime cancer risk estimated per million people for Toronto and Vancouver from baseline fuel to E10 scenario, year 2000

Pollutant	Change in cancer risk (per million people)	
	Toronto	Vancouver
Benzene – high[a]	-0.33	-0.45
Benzene – low[a]	-1.17	-1.60
1,3-Butadiene[a]	-0.35	-0.72
Acetaldehyde[a]	+0.26	+0.42
Formaldehyde[b]	0.00	0.00
Overall	-0.42 to -1.26	-0.75 to -1.90

Notes:
Risks were estimated using [a] = 70 year lifetime exposure and [b] = 80 year lifetime exposure. Positive numbers represent an increase in cancer risk and negative figures a decrease in cancer risk.

4.3 Ethanol and PAN

Inhaled ethanol is not known to be carcinogenic. There is evidence that ethanol is an eye and respiratory system irritant in humans at high concentrations. The California EPA has developed a draft health protective concentration of 100 mg/m^3 (53 ppm) for ethanol based on the lowest observed adverse effect level (LOAEL) for sensory irritation in human volunteers exposed to ethanol vapour. This concentration is many times higher than the maximum ethanol concentration used in the exposure assessment for this analysis.

Similar to ethanol, the health effects literature on peroxyacetyl (PAN) is limited and the carcinogenicity of PAN cannot be evaluated. The California EPA has developed a draft health protective concentration of 3.2 µg/m^3 (0.6 ppb) for chronic exposures based on the no observed adverse effect level (NOAEL) from studies with laboratory rats. This concentration is almost double the maximum estimated 24-hour average concentration of PAN for any of the locations or years considered in this risk assessment.

5.0 Considerations

This risk assessment relied on several models, and refinement and/or validation of these models would improve the certainty of the results. For example, higher resolution emissions and atmospheric modelling would allow for increased detection of changes in atmospheric pollutant concentrations due to a change in vehicle emissions.

Increased precision and refinement of input data for the Canadian Exposure Model (CEM) would increase the accuracy of the personal exposure estimates. This would be achieved with a greater number of microenvironment (ME) monitoring studies conducted in Canada, especially for the MEs with limited information (e.g., in-vehicle, public buildings, gas stations, and parking garages). Furthermore, the derivation methods used to determine the impact of vehicle-related emissions to ME concentrations have not been validated to ensure accuracy of this novel approach. Additionally, the present Canadian time-activity data (i.e., CHAPS) was collected in 1994–95 and has limited information for some age groups, especially infants. As time-activity information is critical for conducting accurate and reliable human health risks assessments, it would be valuable to obtain more recent and comprehensive data that would reflect changes in social activity patterns that have occurred over the last 15 years. Validation of the CEM would also increase the certainty of the results.

6.0 Conclusions

This risk assessment indicates that increasing the use of E10 fuel in Canada would result in a possibly negligible decrease in the number of adverse health effect incidents. This decline would result from a reduction in ambient air concentrations of select pollutants resulting from increased E10 fuel use. In general, there were no substantial differences in predicted health effects between the conventional gasoline baseline and E10 fuel scenarios. While the health effects of most pollutants associated with the combustion of E10 fuel could be quantified, current knowledge of the health effects associated with the inhalation of ethanol itself limited the assessment of this pollutant to a qualitative discussion. Data were also limited with respect to the characterization of all possible microenvironments (MEs) and the estimation of baseline exposure.

The modelling of ME concentrations utilized in this document is innovative and requires further work, including field validation, in order to determine the accuracy of the various approaches. The techniques used in this assessment are considered conservative and include the current scientific understanding of vehicle emissions, atmospheric chemistry, human exposure, and human health effects of environmental pollutants.

GLOSSARY

AQBAT	Air Quality Benefit Assessment Tool
B.C.	British Columbia
CD	census division
CEM	Canadian Exposure Model
CEPA	*Canadian Environmental Protection Act*
CHAPS	Canadian Human Activity Pattern Survey
CIIT	Chemical Industry Institute of Toxicology
CMAQ	Community Multiscale Air Quality
CO	carbon monoxide
CO_2	carbon dioxide
E10	10% ethanol (per volume) blended gasoline
GVRD	Greater Vancouver Regional District
LOAEL	lowest observed adverse effect level
ME	microenvironment
µg/kg-bw/d	microgram per kilogram body weight per day
$\mu g/m^3$	microgram per cubic metre
mg/m^3	milligram per cubic metre
ML	million litres
NAPS	National Air Pollution Surveillance Network
NH_3	ammonia
NOAEL	no observed adverse effect level
NO_2	nitrogen dioxide
NO_x	nitrogen oxides
O_3	ozone
PAN	peroxyacetyl nitrate
PM	particulate matter
$PM_{2.5}$	particulate matter 2.5 µm or less in diameter
ppb	parts per billion
ppm	parts per million
SAPRC	Statewide Air Pollution Research Center
SO_2	sulphur dioxide
TWE	time-weighted exposure
U.S. EPA	United States Environmental Protection Agency
VOC	volatile organic compound