Chapter 4

Air Quality, Climate Change and Health

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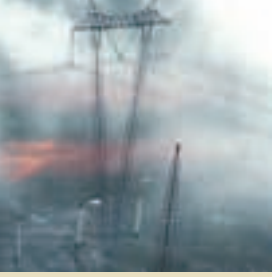

Chapter 4

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4.1 INTRODUCTION

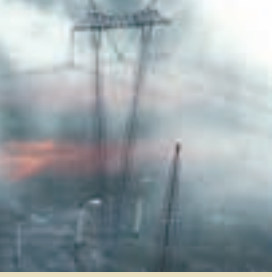
This chapter focuses on the potential impact of climate change on air quality and related health impacts in Canada. The chapter begins by providing a summary of the potential adverse health impacts of air pollution with a focus on ground-level ozone (O_3) and particulate matter (PM), and of extreme heat, including heat waves. An examination of potential additive or synergistic effects between extreme heat and air pollution was also conducted because heat may modify the nature of potential air pollution-related health impacts. Studies examining seasonal air pollution effects, air pollution effects among cities with different climates and different temperatures, as well as studies of population responses to extreme heat events are reviewed. (See Annex 1 for more information on the literature search methodology.) The literature review covered sources to 2006 except for the citing of a few authoritative sources that were published subsequent to that (e.g. the 2007 IPCC Assessment).

The chapter also examines how an increase in mean temperature (of 4°C) due to climate change might impact summer time levels of O_3 and PM in Canada. Two scenarios were considered: one examining the independent effect of increased temperature on air quality and the second examining the effect of both increased temperature and the potential contribution of changing natural emissions of volatile organic compounds (VOCs) to the formation of these pollutants. The results of these two modelling scenarios were then compared with a reference year (2002). Other potential effects of a warmer climate on anthropogenic emissions, climatology (e.g. humidity, wind) and natural constituents of the atmosphere affecting air quality, such as pollen and spores, were not considered.

Lastly, the potential impacts of modelled changes in O_3 and PM concentrations on human health were investigated. Changes in air pollution-related morbidity and mortality were estimated according to each scenario and their associated costs were calculated. The chapter reviews some measures used to manage air pollution-related risks in Canada and concludes by offering recommendations for future research efforts.

4.2 HEALTH IMPACTS OF AIR POLLUTION AND EXTREME HEAT

Air pollution exposure, both acute and chronic, is associated with a number of adverse health impacts which have been evaluated in many formal risk assessments (e.g Working Group on Air Quality Objectives and Guidelines (WGAQOG), 1999a, 1999b; World Health Organization (WHO), 2003; U.S. Environmental Protection Agency (U.S. EPA), 2005). The research available to the scientific community which describes these effects is voluminous and addresses worldwide issues as well as specific Canadian situations and mortality associations (Burnett et al., 2000; Goldberg et al., 2000; Krewski et al., 2000; Goldberg et al., 2001a, 2001b, 2001c, 2001d; Pope et al., 2002; Burnett and Goldberg, 2003; Vedal et al., 2003; Villeneuve et al., 2003; Finkelstein et al., 2004; Jerrett et al., 2004; Pope et al., 2004). Extreme heat and heat waves have also been implicated as a growing health concern. For example, large numbers of heat wave-related illnesses and deaths occurred in the U.S. and Europe in the 1990s (Ballester et al., 1997; Dematte et al., 1998; Semenza et al., 1999; Keatinge et al., 2000; McGeehin and Mirabelli, 2001; Curriero et al., 2002; Diaz et al., 2002; Hajat et al., 2002; Naughton et al., 2002; Koutsavlis and Kosatsky, 2003). In August 2003, the extreme heat wave that struck Europe was implicated in many thousands of deaths and contributed to public concern about the potential health impacts related to climate change (Ledrans and Isnard, 2003; Diaz et al., 2004; Koppe et al., 2004; Kovats et al., 2004; Johnson et al., 2005; Carcaillon et al., 2006).



The Intergovernmental Panel on Climate Change (IPCC) projects a continued rise of atmospheric greenhouse gases (GHGs) during the next century, resulting in further warming of the climate (IPCC, 2007a). Climate change has been observed through recent increases in global average air and ocean temperatures, widespread melting of snow and ice, and the rising global average sea level (IPCC, 2007a). It is very likely that such warming will result in more frequent heat waves. It may affect levels of air pollution exposure by changing local and regional weather conditions and by affecting both natural and anthropogenic sources of air pollutant emissions (Watson et al., 1998; IPCC, 2007b; U.K. Department for Environment, Food and Rural Affairs (DEFRA), 2005). Climate change may also result in changes in patterns of activity and associated air pollution exposures. The independent effects of heat, ground-level O_3 and particulate matter on human health are described below. This is followed by a discussion of the potential synergistic health effects of extreme heat and air pollution.

4.2.1 Health Impacts of Extreme Heat

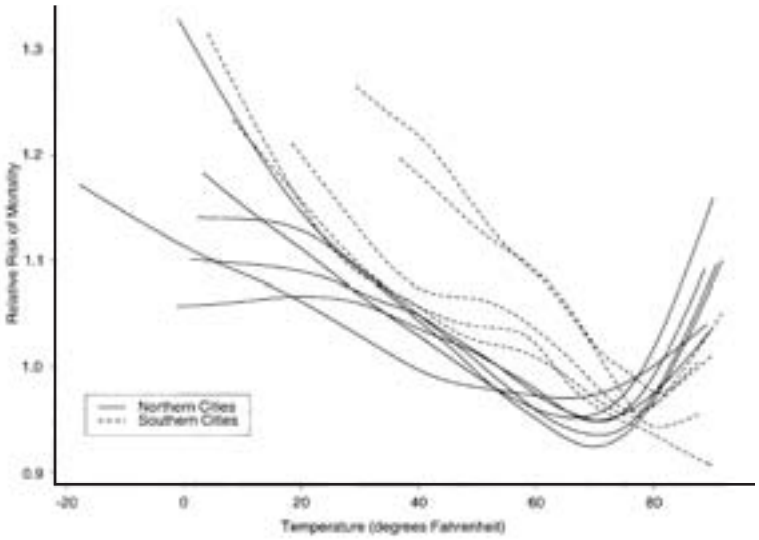
Beyond a narrow comfortable zone ("thermoneutral zone") in which body heat loss and heat gain are equal, exposure to the heat or cold can cause illness (morbidity) and eventually death (mortality). The body regulates environmental heat exchange by controlling the metabolic rate in the internal organs, blood flow through the skin, and shivering or perspiration (Rowell, 1983; Dinarello and Gefland, 2001). Normal adaptation to heat stress involves increased cardiac output and a relaxation of the blood vessels in the skin to increase blood flow from the core to the surface of the body. The production of sweat cools the body through evaporation (Bouchama and Knochel, 2002), although it is less efficient under humid conditions.

Certain people may be unable to increase cardiac and sweat output sufficiently which may lead to increases in body temperature and potential illness or death. This includes people such as seniors, post-menopausal women, or those taking certain medications including anticholinergics, diuretics, beta-blockers, estrogen replacement drugs and some antipsychotic drugs (Lee-Chiong Jr. and Stitt, 1995; Freedman and Krell, 1999; Brooks-Asplund et al., 2000; Speizer, 2001; Gauthier et al., 2005). Infants and young children are also particularly susceptible to heat-related illnesses due to an immature thermoregulatory system (Yeo, 2004). Excessive exertion among healthy adults can also result in dehydration, heat exhaustion, kidney failure, liver damage, heat stroke or death (Hart et al., 1980; Hughson et al., 1980; Barrow and Clark, 1998; Dematte et al., 1998; Bouchama and Knochel, 2002).

Over the previous two decades, it has been estimated in the U.S. that extreme heat episodes contributed to the death of several hundred people (Confalonieri et al., 2007). Many such deaths occurred in susceptible subpopulations such as seniors with pre-existing cardiovascular, cerebrovascular or respiratory conditions. The precise number of persons with heat-related illnesses seeking medical treatment in a given year in Canada or the U.S. is unknown because no reliable statistics are available. Only a small number of deaths are certified as due to heat stroke in Canada (Koutsavlis and Kosatsky, 2003).

Over a span of several weeks, people can gradually become acclimatized to heat stress by reducing their basal metabolic rate, increasing their capacity to perspire and increasing skin blood flow (Koppe et al., 2004). Epidemiological studies have revealed an approximate U- or J-shaped relationship between temperature and mortality (Ballester et al., 1997; Keatinge et al., 2000; Koppe et al., 2004). For example, the upward slope of the "J" in Figure 4.1 begins at lower temperatures and is steeper for northerly cities in the U.S. and Europe than for cities farther south (Keatinge et al., 2000; Curriero et al., 2002). In populations with cooler climates, or during heat waves occurring early in the season, people are particularly vulnerable as they are unacclimatized to heat stress. Under such conditions, the upward slope of the mortality response curve begins at lower temperatures and is steeper (Keatinge et al., 2000; Curriero et al., 2002). Adaptive measures such as heat health warning systems, air conditioned living and work spaces, or access to cooling centres by people at risk of heat stress have been shown to reduce the extent of related health impacts (Sheridan and Kalkstein, 2004; U.K. Met Office, 2006; Vittiglio, 2006; Confalonieri et al., 2007).

Figure 4.1 Dose-response curve for mortality and temperature in 11 cities of the Eastern United States[1]



Note: -20°F, 0°F, 20°F, 40°F, 60°F and 80°F are -28°C, -18°C, -6°C, 4°C, 16°C and 26°C, respectively.

Source: Curriero et al., 2002.

4.2.2 Health Impacts of Ground-Level Ozone

Ground-level ozone is a pollutant which is formed in the atmosphere primarily from nitrogen oxides and volatile organic compounds. While there are natural sources of both these ozone "precursors", human activities produce huge quantities of both–especially those activities related to the combustion of fossil fuels. Exposure to O_3 may result in various pulmonary and cardiovascular effects in healthy individuals but is especially problematic for those with existing cardiovascular and pulmonary disease (WGAQOG, 1999a, 1999b; U.S. EPA, 2006a). Considering that some 45% of all deaths in Canada result from cardiopulmonary disease, it would appear that the potentially susceptible group is very large. The main health impacts of O_3 include acute and chronic damage to the respiratory system, with increased airway reactivity, airway permeability, airway inflammation, reduction in lung function and increased respiratory symptoms. The acute reactions are of particular concern in asthmatics, including children, and others with chronic airway disease. These effects appear to be worsened as the duration of exposure to O_3 increases (Hyde et al., 1992; Krzyzanowski et al., 1992; Künzli et al., 1997; Lippmann, 2000b). Short-term exposure to O_3 has also been associated in some studies with various cardiovascular effects including acute myocardial infarction (Ruidavets et al., 2005), arrhythmias (Dockery et al., 2005) and heart rate variability (Park et al., 2005). These results provide evidence concerning possible mechanisms behind the association detected by epidemiological studies between O_3 and premature mortality (U.S. EPA, 2006a).

4.2.3 Health Impacts of Particulate Matter

Airborne PM varies in composition and concentration and is composed of both organic and inorganic constituents. Particles are usually categorized as coarse ($PM_{10-2.5}$), fine ($PM_{2.5}$) and most recently, ultrafine ($PM_{0.1}$), with aerodynamic diameters of 10 µm (micrometres) to 2.5 µm, <2.5 µm and <0.1 µm, respectively. Although regional differences are significant,

1 The J-shaped curves depicted in Figure 4.1 were obtained using a software program to handle a statistical method known as a "generalized additive model". Following publication of the figure in Curriero et al., 2002, errors in the software came to light; re-analysis of the data in question accounting for these errors led to slightly altered but still J-shaped curves, with a slightly greater range of temperatures at which the relative risk of mortality remained flat (Curriero et al., 2003).

in general, about half of $PM_{2.5}$ is directly emitted into the atmosphere whereas the other half is secondarily formed when precursor gases (sulphur dioxide [SO_2], nitrogen oxides [NOx], VOCs and ammonia [NH_3]) react or condense to form particles (Environment Canada, 2003a). Most $PM_{10-2.5}$ is directly emitted into the atmosphere from the soil, wildfires, combustion of fossil fuels, construction activities, and road and sea salt (Environment Canada, 2003a). While $PM_{10-2.5}$ can deposit widely within the lung, it is more likely to deposit in the upper portions, while both $PM_{2.5}$ and $PM_{0.1}$ penetrate deep into the lungs and elicit a range of physiological responses and even enter into the bloodstream (Delfino et al., 2005; Penn et al., 2005; Urch et al., 2005; Lipsett et al., 2006).

Epidemiological, field, controlled human exposure and toxicology studies, which have been extensively reviewed in national risk assessments (Working Group on Air Quality Objectives and Guidelines, 1999a, 1999b; U.S. EPA, 2004) have contributed to the evidence of PM-associated health impacts. PM has been associated with hospitalizations and increased respiratory and cardiovascular mortality (Burnett et al., 1999; Burnett and Goldberg, 2003; Goldberg et al., 2006). It has also been associated with asthma exacerbation, decreased lung function, inflammation and changes in heart rate variability (McConnell et al., 1999; Gong et al., 2003c; Ebelt et al., 2005). These effects have been observed at the range of concentrations found in Canadian urban entres. Particular subpopulations, including children, seniors and people with pre-existing medical conditions are more vulnerable to the health impacts of PM. Both the short-term (from days to a few weeks) and long-term (multi-year) health impacts of exposure to PM depend on the composition of the particles (which may vary with season) and the exposed population (Goldberg et al., 2000, 2001b; Gordon, 2003; Mueller-Anneling et al., 2004; Becker et al., 2005; Crighton et al., 2005; Delfino et al., 2005; Li et al., 2005; Goldberg et al., 2006; Huang and Ghio, 2006; Kreyling et al., 2006; Ostro et al., 2006; Ren et al., 2006), though this has not been consistently found.

4.2.4 Interactions of Heat and Air Pollution

Most of the underlying mechanisms that have been investigated to explain the biological effects of heat or air pollutants on health appear to belong to distinct biological pathways. However, there is clearly an overlap in the body's physiological response to the activation of these pathways; this suggests that synergistic effects are quite plausible. For example, reduced pulmonary function due to acute O_3 exposure, or reduced heart rate variability due to PM exposure, will likely impair an individual's capacity to maintain adequate tissue oxygenation under the increased cardiovascular load associated with heat stress.

Generally, any limitation in respiratory function due to pollution could interfere with cardiovascular thermoregulatory mechanisms and reduce the maximum heat load that an individual could sustain before adverse effects appeared. As another example, both heat stress and PM can lead to blood coagulation anomalies. These could precipitate an event such as a myocardial infarction in a susceptible individual who is in a state of increased cardiovascular work due to the heat. Gordon (2003) has also suggested that the physiological stress caused by marked changes in ambient temperature can alter the physiological response to toxic agents.

Despite a theoretical basis for synergistic effects of heat and air pollution, the specific potential individual and population health impacts still need to be clarified; few studies have explicitly investigated this question. There are also other potential mechanisms by which heat could influence the effect of air pollution on health. For example, higher average temperatures are in fact associated with greater exposure to air pollution because population activity patterns change; people spend more time outside and tend to leave their windows open more often. However, it remains difficult to draw any firm conclusions based solely on this evidence. Further studies with more refined air pollution exposure measurements and assessments of other modulating factors are required.

4.2.4.1 Seasonal variations in air pollution

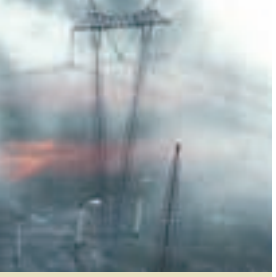
Differences in the health impacts of air pollution have been observed in studies examining the association between air pollution and health during different seasons. During the summer, intense sunlight and elevated temperatures often contribute to increased formation of ground-level O_3 (a component of photochemical smog), resulting in combined exposures to heat, and high levels of O_3 as well as PM. Differences in the response to PM_{10}—which does not follow predictable variations throughout the year—are more suggestive of an interaction with heat, provided that the effect of O_3 has been controlled for. O_3 levels are positively correlated with PM_{10} levels during the summer months (Ito et al., 2005) and negatively correlated during the winter months. As with PM_{10}, the observed season- and temperature-dependent effects of nitrogen dioxide (NO_2), sulphur dioxide (SO_2) and carbon monoxide (CO) on mortality, physician consultations and hospital admissions (Michelozzi et al., 1998; Hajat et al., 1999; Chang et al., 2005), suggest the existence of underlying interactions.

Using a case crossover design to look at hospital admissions for cardiovascular diseases in Taipei between 1997 and 2001, Chang et al. (2005) performed separate analyses within two temperature strata: cool days (mean temperature <20°C) and warm days (>20°C). They found that NO_2, CO and O_3 were significantly associated with increases in hospitalizations on warm days (after adjusting within the two strata for the effects of potential confounding factors such as temperature, humidity and other pollutants). On cool days, only PM_{10} had a statistically significant effect after adjusting for the effects of other air pollutants.

Goldberg et al. (2001d) found positive correlations between O_3 and non-accidental deaths in Montreal during the summer, whereas during the winter, the correlations were negative. In a recent review of 10 time-series studies examining the association between O_3 and mortality (Ito et al., 2005), nine studies found higher estimates of mortality risk with increasing O_3 levels during the warm months when O_3 levels were higher (winter risk due to O_3 exposure was only higher in one city in Australia). Adjusting for the effects of PM_{10} did not markedly affect the results.

In a meta-analysis of studies examining the short-term effects of air pollution in eight Italian cities, Biggeri et al. (2005) found a significant difference in the effect of PM_{10} on all-cause mortality by season, with a 0.54% increase in mortality observed in the cold season and a 2.53% increase observed in the warm season (both increases are for a 10 µg/L increase in PM_{10} concentration). The variability of the size of the effect among cities was also greater during the warm season. O_3 and other pollutants were not considered here.

In a time-series analysis of the effects of major air pollutants, Michelozzi et al. (1998) found that total mortality in Rome from 1992 to 1995 was positively associated with PM_{10} (as well as NO_2), with a 0.4% increase in mortality observed with each increase of 10 µg/m^3 PM, increasing to 1.0% in the summer months. Sunyer et al. (1996) conducted a similar study in Barcelona and found that daily variations in mortality were related to daily variations in air pollutants for the period 1985 to 1991. Adjusted for temperature and other factors, black



smoke (black particles with a diameter of less than 4.5 µm) and SO_2 were positively related to total mortality, seniors' mortality, and cardiovascular mortality. The association between SO_2 and respiratory mortality in seniors, however, was significant only during the summer months. NO_2 and O_3 were positively related with mortality in seniors and cardiovascular mortality during the summer, but again not during the winter months.

In a study that examined physician consultations for asthma in London (Hajat et al., 1999), the effect of various air pollutants was compared between seasons for different age groups. Statistically significant seasonal differences were observed for NO_2 in children, and for black smoke and PM_{10} in seniors, with larger effects observed during the summer months. Among adults, increasing O_3 levels during the summer were associated with an increase in medical consultations.

Anderson et al. (1996) found a year-round increase in respiratory mortality due to O_3 in London (U.K.), whereas all-cause and cardiovascular mortality were affected only during the warm season. This suggests that persons suffering from cardiovascular diseases may be more susceptible to a combination of O_3 and heat compared with those suffering from respiratory diseases, who appear to be equally susceptible to O_3 year-round. Black smoke was also positively associated with all-cause mortality during both seasons, but more so during the summer months. The results were similar when adjusting for the effects of PM_{10}.

4.2.4.2 Cross-city comparisons

Studies examining the effects of air pollution on health between warmer and cooler cities have also tended to suggest greater impacts on health in warmer climates. However, the results of such studies must be viewed cautiously because of the existence of potential confounding effects of other factors.

Barnett et al. (2005) examined the relationship between air pollution and child hospital admissions for respiratory diseases in five cities in Australia and New Zealand. They found that increases in respiratory disease admissions associated with $PM_{2.5}$ and PM_{10} in the 1- to 4-year-old age group occurred mostly during the warm season, whereas an association with NO_2 in the older children (aged 5 to 14 years), although greater during the warm season, was present during both warm and cool seasons. Cities with higher average temperatures were also found to have greater increases in hospital respiratory disease admissions in the 1- to 4-year-old age group associated with 1-hour NO_2 concentrations.

Diaz et al. (2004) investigated the combined effects of heat and air pollution on child mortality in Madrid, Spain, between 1986 and 1997. High temperature alone was not found to be associated with higher mortality. However, air pollutants such as total suspended particles and NOx had a strong seasonal effect on mortality. During the summer months, the relative risk of daily mortality from elevated total suspended particles was 1.53 in infants aged 1 to 5 years compared with 1.25 during the winter months. NOx had a significant effect only in the summer months in the 0- to 1-year-old age group, with a relative risk of 1.07 reported.

Aga et al. (2003) also found that temperature partly explained differences observed in air pollution-related mortality among seniors in 28 cities examined in the *Air Pollution and Health: A European Approach 2* (APHEA2) study. In cooler cities with a 9°C daily average temperature (25th percentile), a 10 µg/m^3 increase in PM_{10} caused an increase in mortality in seniors of only 0.44%. In contrast, a 0.91% increase was found in warmer cities with a 15°C daily average temperature (75th percentile). For black smoke, the mortality increase varied from 0.39% to 0.75% among cooler and warmer cities, respectively.

As part of the APHEA2 study, Katsouyanni et al. (2001) examined the short-term levels of air pollutants, ratios of PM_{10} to NO_2 and black smoke, mean temperature and humidity, as well as population characteristics in relation to overall mortality in several cities. The daily mortality increase for PM_{10} was 0.29% at the 25th percentile of average temperature, which increased to 0.82% at the 75th percentile of average temperature. The effects of black smoke depended partly on temperature. The mortality increase varied from 0.23% to 0.70% among cities at the 25th percentile of average temperature compared with those at the 75th percentile. This study may have been flawed, however, because of the use of inappropriate regression methods.

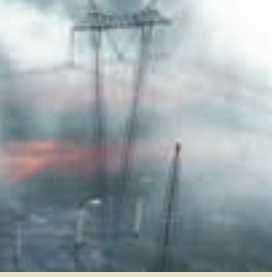
Choi et al. (1997), in one of few long-term studies examining the influence of temperature on the effects of air pollution, assessed lung cancer mortality in 47 Japanese prefectures as a function of long-term NO_2 and SO_2 levels, expenditures on tobacco, car density, temperature and geographic gradient (north to south). For the southern and warmest areas, the NO_2 effect was strongest where average temperatures were highest, possibly suggesting an additive or synergistic effect of long-term exposure to air pollution and higher temperature on risk of lung cancer mortality.

4.2.4.3 Studies of extreme heat events and their effects on air pollution-related population health risk

Heat waves provide opportunities to study the influence of temperature on the health impacts of air pollution. This is because they often combine high and/or sustained levels of both heat and air pollutants and the impacts of their interactions become more evident. Some studies have tended to suggest that interactions between heat and air pollution do occur but only become measurable above a certain temperature threshold (Sartor et al., 1997).

In a study of the effects of the 2003 heat wave in France, a daily time-series analysis for nine cities was performed (Institut de veille sanitaire (InVS), 2004). Daily O_3 concentrations, and minimum and maximum temperatures were inputted for the 8-year period from 1996 to 2003. Daily deaths were regressed on same-day and earlier temperatures and on O_3 concentrations the day of and the day before the deaths occurred. The effect of O_3 alone, temperature alone, and O_3 acting with temperature were estimated for August 3 to 17, the period of the heat wave. It was found that O_3 and temperature effects differed among some of the nine cities. Although overall the temperature effect was preponderant, O_3 itself accounted for most of the deaths in two cities, and no interaction was observed between O_3 and temperature.

Fischer et al. (2004) in Holland and Stedman (2004) in the U.K. used a risk assessment approach to estimate air pollution-related deaths during the August 2003 heat wave. Deaths were estimated on the basis of country-specific dose–response relationships for O_3 and $PM_{2.5}$ and were subtracted from the overall number of excess deaths during the heat wave. For the U.K., Stedman (2004) estimated that 21% to 38% of the total excess deaths were associated with elevated concentrations of O_3 and/or particles. Fischer et al. (2004) estimated that in the Netherlands, approximately 40% of the 1,000 to 1,400 excess deaths were related to air pollution.



Sartor et al. (1995) studied the association among daily deaths, temperature and air pollutant concentrations during a prolonged period of above normal temperatures in Belgium in 1994. Expected mortality was based on the summers of 1985 to 1993. In 1994, a net excess of 1,226 deaths occurred during a period of hot weather with above average O_3 levels. For persons more than 65 years of age, the statistical interaction between the logarithm of O_3 and temperature, both measured the day before, contributed to 40% of the logarithm of daily deaths. Additional analyses for this age group were based on tertiles of mean daily temperature (Sartor et al., 1997). At the lowest temperatures, temperature and O_3 were not correlated, and only O_3 was associated with mortality. In the middle tertile, temperature and O_3 were highly correlated, and daily deaths also varied with the concentration of O_3. In the tertile where mean temperature was highest (21–27°C), temperature was the stronger predictor of daily deaths, with a positive interaction between temperature and O_3 observed.

Katsouyanni et al. (1993) studied excess mortality across several urban areas in Greece during a heat wave in July 1987. Athens (a "high-pollution" city) was compared with a group of 14 other smaller ("low-pollution") cities. The excess of mortality in Athens was statistically different than that in the other cities after controlling for temperature, suggesting that air pollution had an effect on mortality independent from air temperature. This result is similar to the cross-city effects observed in the APHEA studies, although their designs and methodologies are different. Study limitations include the fact that mean air temperature was averaged over the entire 1-month period and there was a lack of real data for pollutants and other confounders.

4.2.4.4 Synoptic air mass studies

Studies of synoptic air masses examining the effects of air pollutants on population health outcomes (Pope and Kalkstein, 1996; Samet et al., 1998; Smoyer et al., 2000) did not produce any evidence that temperature influenced the effects of air pollutants. The synoptic air mass approach (Kalkstein, 1991) has been used to study the impact of multiple meteorological variables on health. Synopses are meteorological descriptors that take into account a number of weather parameters (e.g. temperature, humidity, cloud cover, wind direction and speed), describing these grouped variables as air masses, which are generally named according to their geographic origin. They can also be classified according to a similarity to a classic weather pattern, or by automatic classification methods that cluster coincident weather variables into groups having preset characteristics.

Using synoptic classification of air masses in four major Canadian cities (Montreal, Ottawa, Toronto and Windsor), Cheng et al. (2005) quantified an increase in heat-related mortality by the 2050s and 2080s as a result of climate change. Although the influence of temperature on air pollution effects was not examined specifically, an increase in air pollution-related mortality, largely driven by O_3, was observed for one of the scenarios where emissions were not modified.

4.2.4.5 Laboratory and field studies

Laboratory and field studies, although limited, have provided some evidence of interactions between air pollutants and temperature. Some data from laboratory studies suggest that high ambient temperatures increase the toxicity of CO (Yang et al., 1988). The temporal association between peak expiratory flow rates and ambient O_3 was studied in a group of 287 children and 523 non-smoking adults in Tucson, Arizona (Krzyzanowski et al., 1992). In children, peak expiratory flow rates were reduced on days when there was a higher O_3 concentration. In adults, peak expiratory flow rates were reduced in asthmatics who spent more time outdoors on days when O_3 levels were higher. After adjusting for other co-variates, significant interactions among O_3, PM_{10} and temperature were found; the impairment of the respiratory response due to low-level ambient O_3 increased with temperature and PM_{10}.

4.2.5 Vulnerable Populations

As noted earlier, it is clear that many factors can affect an individual's health risks resulting from exposure to environmental stresses such as ambient air pollution and/or heat.

Population groups most vulnerable to the effects of natural and anthropogenic air pollution emissions and/or extreme heat are relatively well identified (Neas et al., 1996; Lippman, 2000a; Ledrans and Isnard, 2003; Jerrett et al., 2004; Newhouse and Levetin, 2004; Carcaillon et al., 2006):

- seniors and those in institutions, such as residential care homes;
- young children and asthmatics;
- people with chronic diseases, particularly cardiovascular and respiratory illnesses, renal disease, diabetes and obesity, as well as those taking certain medications; and
- people of lower socio-economic status and those living in densely populated urban neighbourhoods.

The latter group is at greater risk of the adverse health impacts of air pollution mainly because of living conditions that lead to higher exposures to ambient PM, gaseous pollutants and traffic emissions. In addition, people of low socio-economic status have a greater incidence of illness (Finkelstein et al., 2005).

Under conditions of combined heat and pollution, vulnerable groups may experience greater risks than the general population. However, it is difficult to evaluate who might exhibit more sensitivity to potential synergistic effects; such questions have not yet been formally addressed in the literature. Evidence pointing to specific groups that might be particularly sensitive is very limited.

4.2.5.1 Persons with cardiovascular disease

As already noted, Anderson et al. (1996) found that the association between O_3 and related respiratory mortality in London (U.K.) was significant throughout the year, whereas association with cardiovascular mortality was significant only during the warm season. Similar findings were reached by Sunyer et al. (1996). This could suggest that synergistic effects are more prevalent in people with cardiovascular diseases. In addition, such people appear to be affected by air pollution only when temperatures are warmer.

4.2.5.2 Seniors

Roberts (2004) observed interactions between air pollutants and ambient temperatures while studying mortality in people aged >65 years, and Aga et al. (2003) found suggestive evidence for this in a study of this age group. In another APHEA study of all-age mortality (Katsouyanni et al., 2001), the effect modification by temperature on air pollution-related mortality was comparable to that found by Aga et al. (2003) in seniors. Mortality due to air pollution in an urban study was slightly higher in seniors than in the general population; there was also a slightly higher effect on mortality for the general population in warmer cities. However, the effect modification by temperature of air pollution-related mortality was approximately the same in seniors and the general population. This study therefore suggests that although the adverse health impacts of air pollution are greater in seniors, the impacts of combined exposure to pollution and variations in temperature may be similar in seniors and in the general population. Most of the victims of heat waves in Europe and North America have been seniors whose health is already fragile (Bouchama and Knochel, 2002; Curriero et al., 2002; Diaz et al., 2002; Hémon and Jougla, 2003; Ledrans and Isnard, 2003; Fischer et al., 2004; Kovats et al., 2004; Toulemon and Barbieri, 2004). Future research should investigate the possible synergistic role of poor air quality in contributing to morbidity and mortality during heat waves.

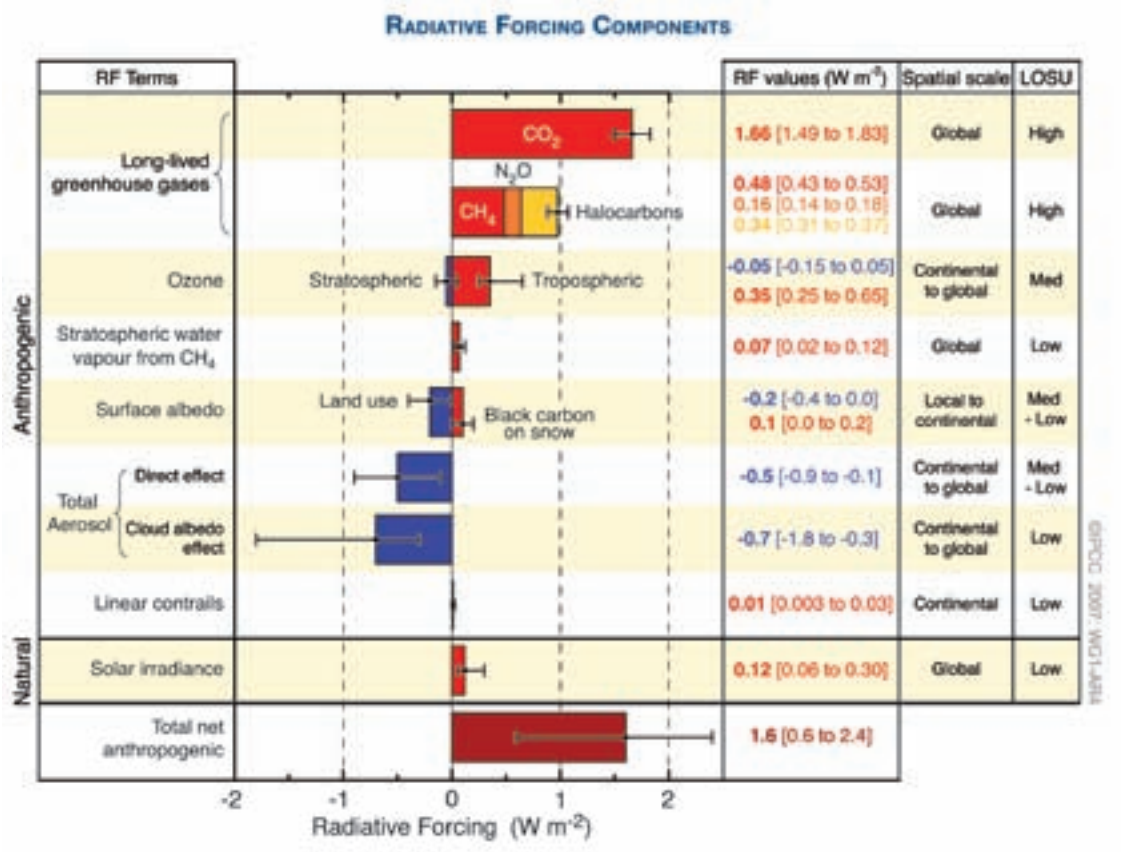
4.2.5.3 Children

Hajat et al. (1999) observed seasonal variations in the effect of pollution on medical consultations for asthma in children as well as in seniors. Similarly, Barnett et al. (2005) found that in children aged 1 to 15 years, the association between air pollution and hospital admissions was greater during the warm season. Diaz et al. (2004) also found that air pollutants had a greater impact on child mortality during the warm season.

4.3 CLIMATE CHANGE AND AIR POLLUTION

Carbon dioxide (CO_2) and other carbon-based GHGs contribute to climate change, whereas airborne PM (other than black carbon) can have a cooling effect by reflecting incoming solar radiation back into space (IPCC, 2007a). Figure 4.2 illustrates some of the major physical and chemical factors that influence the warming and cooling of the Earth's atmosphere (IPCC, 2007a).

Figure 4.2 Major factors influencing atmospheric warming and cooling



Global average radiative forcing (RF) (watts per square metre) estimates and ranges in 2005 for anthropogenic carbon dioxide (CO_2), methane (CH_4), nitrous oxide (N_2O) and other important agents and mechanisms, together with the typical geographical extent (spatial scale) of the forcing and the assessed level of scientific understanding (LOSU). The net anthropogenic radiative forcing and its range are also shown.

Source: IPCC, 2007a. *Climate Change 2007: The Physical Science Basis, Summary for Policymakers* (Figure SPM.2, page 4)

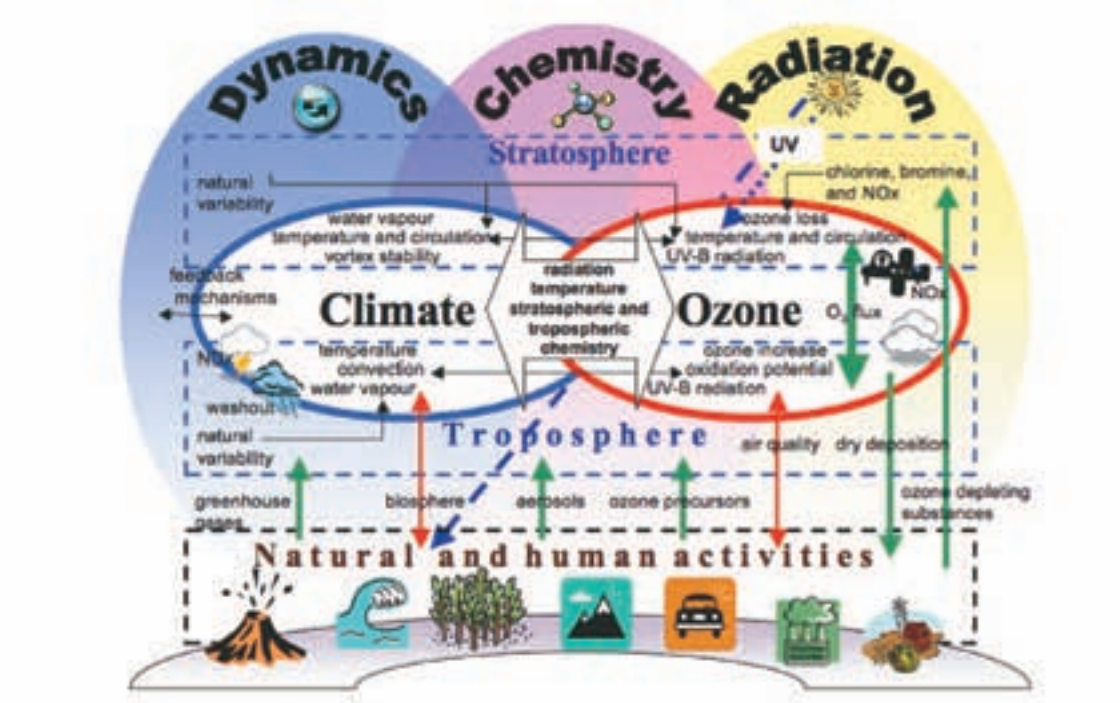
The atmosphere and the earth's surface receive heat energy from the sun in the form of visible and invisible radiation. Global atmospheric warming occurs because incoming solar radiation energy is retained; this is mediated in part by GHGs and other atmospheric components such as halocarbons, nitrous oxide (N_2O), methane (CH_4), CO_2, tropospheric O_3, dusts, and black carbon from fossil fuel and biomass burning. Warming is counterbalanced by the cooling effects of stratospheric O_3, sulphate aerosols, organic carbon from fossil fuel and biomass burning, and by an increased reflectivity of the land surface due to deforestation in snow-covered forests (IPCC, 2007a). Under the present atmospheric conditions, with rising levels of CO_2 and other GHGs, the overall warming effect is of greater magnitude than the overall cooling effect. The reduction of secondary aerosol precursors, NOx, NH_3, SO_2 and VOCs, through efforts to improve air quality, is expected to lead to an increase in temperature. The cooling effect of sulphate aerosols may have partially masked the extent of global warming (U.K. DEFRA, 2005).

4.3.1 Effect of Climate Change on Particulate Matter and Ozone

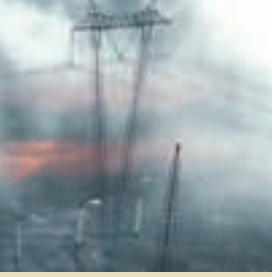
Climate change could alter air quality by modifying the complex mechanisms affecting the formation and behaviour of PM and O_3. As outlined by Bernard et al. (2001), Lloyd (2001), IPCC (2007b) and others, climate change could influence levels of ambient pollutants by modifying (1) weather and consequently local and regional air pollution levels; (2) anthropogenic emissions; and (3) natural emissions, which are strongly controlled by temperature.

Figure 4.3 illustrates the complexity of the atmosphere, and the physical and chemical interactions that affect the behaviour and concentration of air pollutants. Changes in the height of atmospheric layers that determine the vertical distribution of pollutants can alter their concentration (U.S. EPA, 1989; Hogrefe et al., 2004; Laurila et al., 2004b). Global warming may also accelerate the transfer of O_3 from the stratosphere to the troposphere where it would add to the formation of smog (Collins et al., 2003).

Figure 4.3 Interactions among climate, atmospheric composition, chemical and physical processes driven by solar radiation energy and natural and human activities



Source: Adapted from Integrated Global Observing Strategy (IGOS), 2004.



Mickley et al. (2004) conducted an analysis of the effect of projected future climate conditions (1950–2052) on pollutant levels in the U.S. Their general circulation model simulations included black carbon and carbon monoxide (CO) as tracers, and indicated that the concentration of both pollutants increased by 5 to 10% during pollution episodes, although actual emission levels for both tracers were kept constant over the simulation period. This suggests that the magnitude and duration of air pollution episodes could increase as a result of a warmer climate. Changes in the frequency and magnitude of simulated air pollution episodes were also associated with reduced cyclonic weather patterns, which are usually indicative of air pollution. Increases in anticyclonic conditions would cause more frequent inversions that would result in severe air pollution episodes (Hulme and Jenkins, 1998).

Leung and Gustafson Jr. (2005) modelled regional climate change scenarios (based on the IPCC A1B scenario for the years 2045 to 2055) to estimate the potential effects of climate change on U.S. air quality compared to the 1995–2005 period. They found that during autumn in the western U.S., increases in air temperature of up to 4°C increased solar radiation, reduced rainfall frequency, increased air stagnation associated with large high-pressure systems and resulted in deteriorated air quality.

Prather et al. (2003) summarized results from 14 independent three-dimensional global tropospheric chemistry models. Using six varying global emissions estimates, they arrived at global average increases in tropospheric O_3 for the year 2030 of between 5 ppb and more than 20 ppb, in the case of two of the more extreme emissions scenarios. Using different assumptions, Anderson et al. (2001), Tuovinen et al. (2001), Knowlton et al. (2004), Laurila et al. (2004a) and Langner et al. (2005) found similar results.

Photo Credits: Environzone, Environnement Canada (http://www.eg.gc.ca/envirozine)

Ottawa under smog

Hogrefe et al. (2004) conducted an extensive assessment of the implications of climate change for O_3 formation in the U.S. for the 1990s, 2020s, 2050s and 2080s. A global climate model using the IPCC Special Report on Emission Scenarios A2 scenario (one of the most pessimistic scenarios) was coupled with a regional model to obtain current and future regional climate fields. Projections of future biogenic emissions and anthropogenic emissions remained the same. Results for five consecutive summers during the 2020s, 2050s and 2080s suggested that daily maximum 8-hour O_3 levels would be increased by 2.7 ppb, 4.2 ppb and 5.0 ppb, respectively, in the central and eastern parts of the U.S.. Larger increases were projected in certain urban corridors, whereas some decreases were observed in other specific locations. An increase in the number of days with exceedances of the current U.S. 8-hour O_3 standard and an increase in the length of the O_3 episodes were also observed.

4.4 AIR QUALITY MODELLING IN CANADA

A study was undertaken for the Assessment to investigate how a global climate 4°C warmer might affect air quality in Canada. Using A Unified Regional Air-quality Modelling System (AURAMS), a model developed at the Meteorological Service of Canada, levels of O_3 and PM were projected. This section describes the study methodology used and the modelling results.

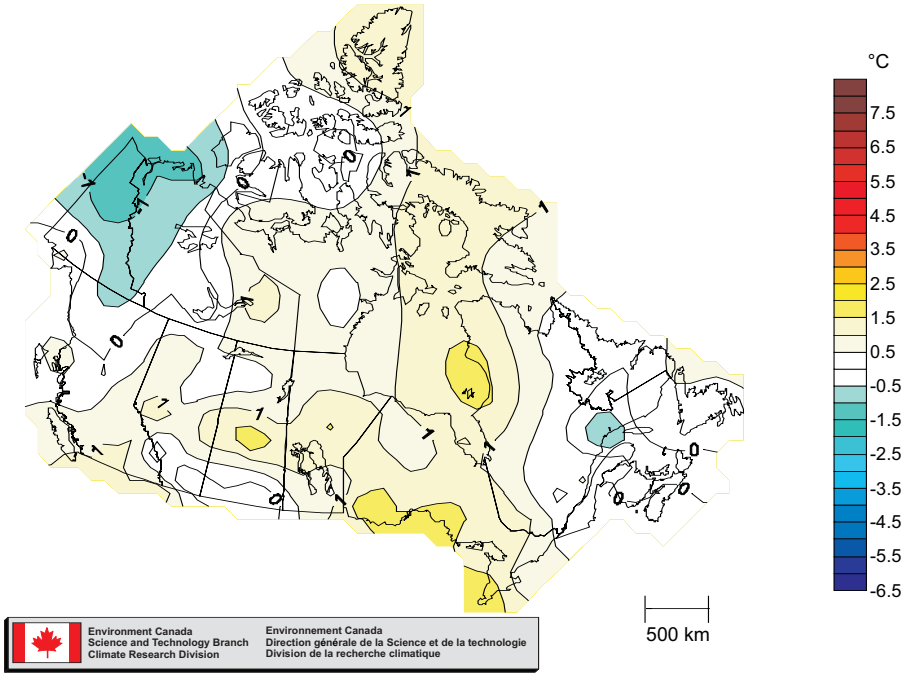
4.4.1 AURAMS: Scenarios and Assumptions

Two scenarios of increasing complexity were used to isolate (1) the individual effects of an increase in temperature on air quality (scenario CC4) and (2) the effects of changes in biogenic emissions of air pollutant precursors along with increases in temperature (scenario CC4b). Because air pollutant levels and temperatures tend to be highest during the summer months, the air pollutant concentrations were modelled for June, July and August. A detailed description of the model and its components is given in Annex 2.

A single temperature increase was used to represent projected climate warming. The Third IPCC Assessment Report (2001b) stated that globally averaged surface temperatures are expected to increase by 1.4 to 5.8°C over the period 1990–2100, based on a range of climate model simulations. Although this may not be representative of specific climate changes at the regional level, it provides a reasonable temperature bracket within which to work. For comparability with previous studies, a 4°C increase in average surface temperature was selected for the current study. This temperature increase is identical to that used by Morris et al. (1995) but is somewhat more conservative than the 5.8°C simulated by Hogrefe et al. (2004). The Fourth IPCC Assessment Report projects that the global average temperature in the decade from 2090–2099 is "likely" to be 1.1 to 6.4°C higher than that of the 1980–1999 period, depending on the emissions outlook used (IPCC, 2007a). The modelled temperature increase in the current exercise therefore represents one plausible future scenario within this range.

The year 2002 was used as a reference year for the simulations. Although no single summer can truly represent average climate conditions, the only alternative to performing simulations with multiple years is to choose a base year that is as representative as possible of the average. Figure 4.4 illustrates how the meteorological conditions experienced during summer 2002 compared with the 30-year normal, based on the Climate Trends and Variations Bulletin for 2002 (Environment Canada, 2002a).

Figure 4.4 Year 2002 regional summer time deviations in temperature from "average"



Temperatures across most of Canada were 0.5°C above normal for the summer (June, July and August) of 2002, and as a whole, Canada experienced its 19th warmest summer above normal since nationwide records began in 1948. Figure 4.4 does illustrate, however, that the warmth was neither extreme nor uniform throughout the country. From southern British Columbia through to the western edge of Quebec, and up into Nunavut, temperatures ranged anywhere from 0.5°C to 1.5°C above normal. Yukon, northwestern Northwest Territories and a small area around the mouth of the St. Lawrence River were the only areas that experienced a cooler than normal summer.

The analysis focussed on contaminant measurements that reflect current risk management goals in Canada: namely, the 8-hour daily O_3 maximum and the 24-hour $PM_{2.5}$ mean concentration, which are the metrics used for the Canada-wide Standards (CWS) for these pollutants. For the purpose of the present analysis, only the numerical targets were used, and an exceedance is assumed once the average 8-hour concentration of O_3 or the 24-hour average of $PM_{2.5}$ exceeds the CWS of 65 ppb and 30 µg/m^3, respectively, throughout the course of a day. For simplicity, the CWS levels are used as reference values throughout the analysis, although they do differ from the values of the American National Air Quality Standards. As described later (section 4.6.1), the CWS are based on feasibility of attainment and thus population health impacts may occur even when pollutant levels are within the CWS. Resultant impacts on health are presented later in the report (Section 4.5) based on all population exposures, regardless of the attainment status of the CWS for PM and ozone.

Compared with actual measurements, model simulations may present some discrepancies that can be attributed to various factors. Model results are nonetheless valuable for estimating the directions in which the future atmospheric composition may evolve. It is generally recognized that the differences between two simulations present less uncertainty than those in the reference simulation when compared to actual observations. Therefore, the present analysis will emphasize differences among the two modelling scenarios and the base case.

It is important to understand that the scenarios used in the current study were not developed with the objective of predicting how climate change would precisely impact air quality. Rather, the current study evaluated only two of the potential factors associated with climate change, namely increases in temperature and higher biogenic emissions (VOCs from plants and nitric

oxide [NO] from soil). As mentioned previously, climate change would also interfere with present climatic conditions and likely modify anthropogenic emissions. All of these factors could in turn affect ambient levels of O_3 and $PM_{2.5}$. Therefore, no definitive conclusions, only indications, can be drawn from the results of the current study regarding the effects of a future warmer climate on the levels of O_3 or PM in the air.

4.4.2 Modelling Results

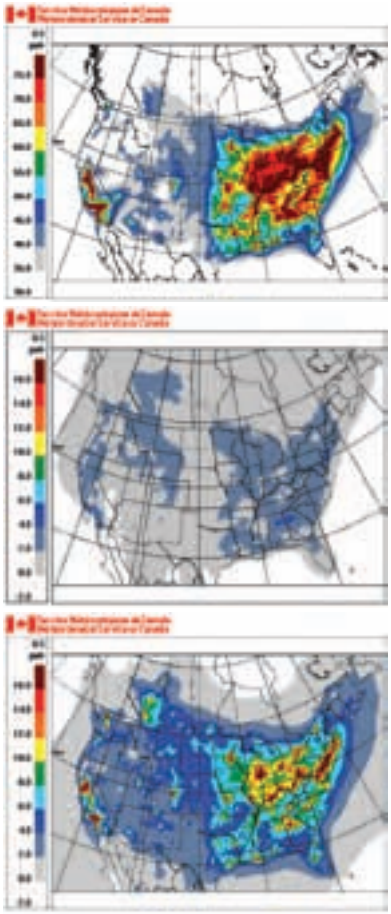
The following section presents the results of the scenario simulations for O_3 and $PM_{2.5}$. Overall, results for O_3 support the hypothesis that in a 4°C warmer climate, increased emissions of biogenic VOCs and soil NO may result in higher ambient O_3 concentrations. In contrast, reductions in the average $PM_{2.5}$ concentration were observed from baseline. Such reductions in $PM_{2.5}$ may be explained by changes in the sulphate–nitrate–ammonium–water system that controls a large fraction of the PM mass. However, given the relative uncertainty attached to PM atmospheric processes and modelling, further research is needed to better elucidate the effect of climate change on PM formation.

4.4.2.1 Ozone

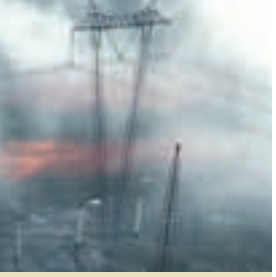
Changes in average daily 8-hour maximum

Figure 4.5 presents the average daily 8-hour O_3 maximum over the three summer months for the base case (BC) simulation (top panel); the relative change in the CC4 simulation, representing a temperature increase of 4°C, compared with the BC simulation (middle panel); and the relative change in the CC4b simulation, representing the same 4°C temperature increase and taking into account a change in biogenic emissions of air pollutant precursors, compared with the BC simulation (bottom panel).

Figure 4.5 Summer time average daily 8-hour O_3 maximum (ppb)



Note: Top panel: Base case simulation. Middle panel: Relative change in the CC4 simulation compared with the base case simulation. Bottom panel: Relative change in the CC4b simulation compared with the base case simulation.



In the BC simulation, the average summer time daily 8-hour O_3 maximum value was 33.5 ppb, with values ranging from 5.4 ppb to 142.3 ppb. The highest concentrations (above 75 ppb) were simulated in the most densely populated areas of North America. In Canada, the highest average daily 8-hour O_3 maximum values are projected for southern Ontario, Quebec and in the Vancouver area, where they reach values of approximately 60 to 70 ppb.

In the CC4 simulation, an overall increase in the average daily 8-hour O_3 maximum values was observed, induced by the increase in air temperature. The average daily 8-hour O_3 maximum value was 34.5 ppb, an overall 0.9 ppb increase from the BC simulation. As in previous studies (Morris et al., 1995; Aw and Kleeman, 2003; Hogrefe et al., 2004), an increase in temperature accelerated chemical reaction rates and therefore increased the rate at which O_3 and other oxidants were produced. The increase in O_3 in the CC4 simulation was not higher than 6.0 ppb in any of the modelled locations.

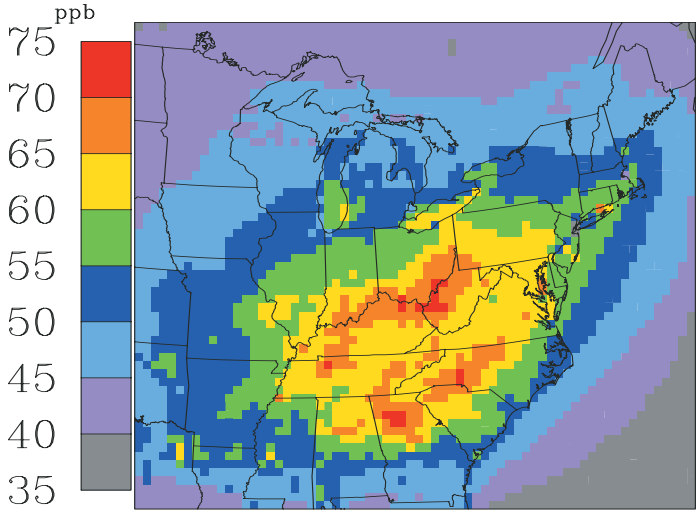
In the CC4b simulation, where biogenic emissions are also stimulated due to increased temperature, the overall average daily 8-hour O_3 maximum was 36.9 ppb, a 3.4 ppb increase from the BC simulation. The increase in the average daily 8-hour O_3 maximum also exceeded 10 ppb in places. The highest Canadian increases in O_3 concentrations (10 to 18 ppb) from the BC simulation were projected for Montreal, Toronto, Vancouver, Calgary, Edmonton, and Winnipeg. A large increase of up to 18 ppb can also be noted in Alberta, mainly in the vicinity of oil sands developments near Fort McMurray. The increased soil NO emissions over the Prairie regions (e.g. in Alberta, Saskatchewan and midwestern U.S.) also contribute to the increase in O_3 concentrations modelled for those areas.

In the CC4b simulation, biogenic emissions of VOC species, such as isoprene and monoterpenes, increased by 25 to 50%, and in some areas by as much as 100%, as a result of the 4°C increase in temperature. The biogenic monoterpene emissions increased most prominently over the northwestern and southeastern U.S., with the exception of the Illinois and Ohio area. Isoprene emissions increased over the boreal forest regions of Canada. The large increases in O_3 concentrations observed in the Illinois and Ohio regions and extending to large cities in the adjoining states are attributable to the increase in biogenic emissions of VOCs in the surrounding regions and to locally increased emissions of NO from soil (up by 15%).

In a similar study, Hogrefe et al. (2004) obtained future year climate conditions by coupling the MM5 and GISS models.[2] Results for the 2080s, where the temperature increased by 4.3°C for the GISS and by 5.8°C for the MM5, are comparable to those obtained from the CC4b scenario here. Figure 4.6 depicts the spatial distribution of the average summer time daily 8-hour O_3 maximum projected by Hogrefe et al. (2004) for the 1990s. Figure 4.7 represents the change in the 2080s scenario from 1990. Despite many differences between the current study and that of Hogrefe et al. (2004), the magnitude of the projected change in both the CC4b simulation here and the corresponding scenario from Hogrefe et al. (2004) is similar.

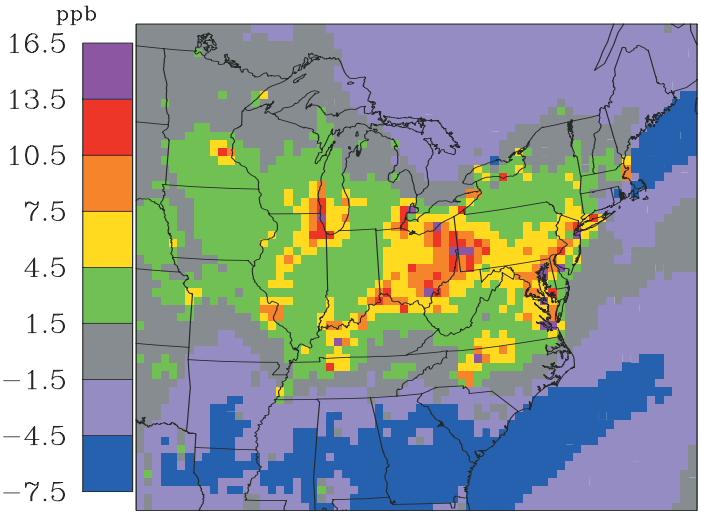
2 MM5 is the Pennsylvania State University/National Centre for Atmospheric Research mesoscale regional climate model. More information about this model is available in Grell et al. (1994). GISS AOM is the Goddard Institute for Space Studies coupled Atmosphere-Ocean Model. More information about this model is available in Russell et al. (1995).

Figure 4.6 Summer time projected average daily maximum 8-hour O_3 maximum concentration (ppb) for the 1990s



Source: Hogrefe et al., 2004.

Figure 4.7 Changes in summer time average daily maximum 8-hour O_3 concentration (ppb) projected under the 2080s climate change scenario simulation relative to that for the 1990s

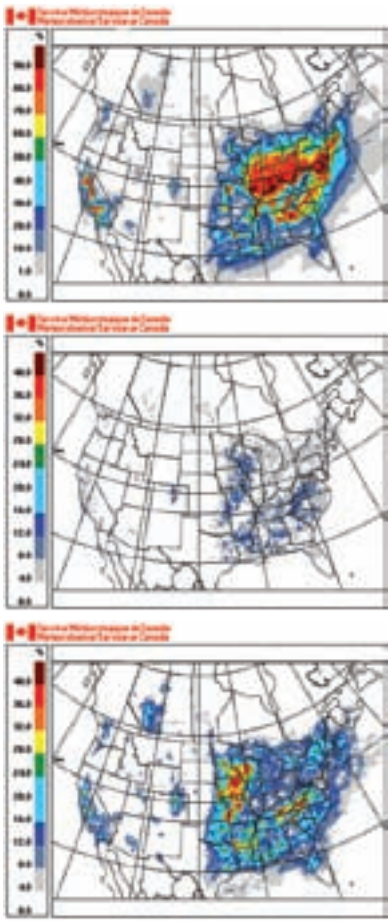


Source: Hogrefe et al., 2004.

Changes in the frequency of exceedances

The top panel of Figure 4.8 presents the number of exceedances of the CWS for O_3 in summer in the BC simulation. Multiple exceedances during the day are counted only as a single exceedance. In Canada, the highest number of exceedances ranges from about 40 to 80% along the Quebec–Windsor corridor. Areas near Vancouver and in Alberta also present a significant number of exceedances of the CWS for O_3.

Figure 4.8 Percentage of days with average 8-hour O_3 concentration exceeding the Canada-wide Standard of 65 ppb



Note: Top panel: Base case simulation. Middle panel: Difference in number of exceedances between the CC4 simulation and the base case simulation. Bottom panel: Difference in number of exceedances between CC4b simulation and the base case simulation.

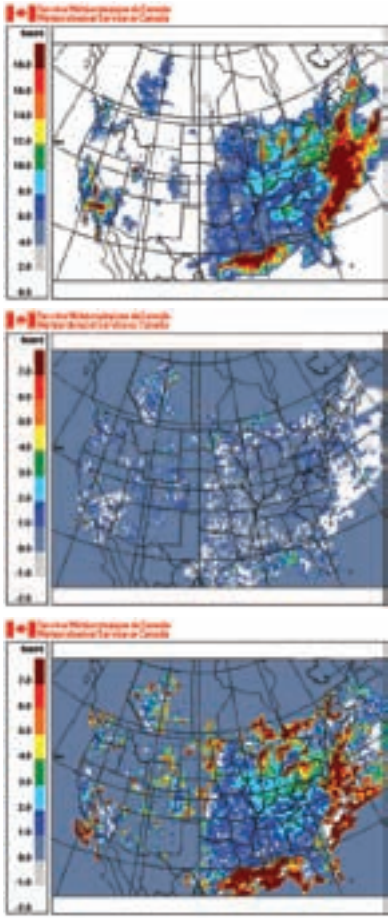
The middle panel of Figure 4.8 presents the relative increase in the number of summer time O_3 exceedances between the CC4 and the BC simulations. The average number of exceedances rose slightly from 10.0% in the BC simulation to 11.2%. In Canada, changes in the number of exceedances are less intense, and are generally below 10%. Changes at the local level varied from -5.4 to 22.8%.

The bottom panel of Figure 4.8 presents the relative increase in the number of summer time O_3 exceedances between the CC4b and the BC simulations. The CC4b scenario projected greater changes in the number of exceedances than the CC4 simulation, with some regions of the U.S. exhibiting an increase of more than 40%. Overall, the average number of exceedances in the CC4b simulation was 14.8%, with local variations ranging from 0 to 51.1%. In Canada, the largest increases are again observed in the Quebec–Windsor corridor, and range from 10 to 25% in Alberta and Vancouver.

Changes in the duration of exceedances

The top panel of Figure 4.9 presents the simulated average duration (in hours) of summer time O_3 exceedances in the BC simulation. The average duration of exceedances varied between 0 and 52 hours, but tended to be shorter above the continental landmass, where episodes generally lasted between 0 and 20 hours.

Figure 4.9 Duration (hours) of summer time episodes with O_3 concentration greater than 65 ppb



Note: Top panel: Base case simulation. Middle panel: Difference in average duration of exceedance between the CC4 simulation and the base case simulation. Bottom panel: Difference in average duration of exceedance between CC4b simulation and the base case simulation.

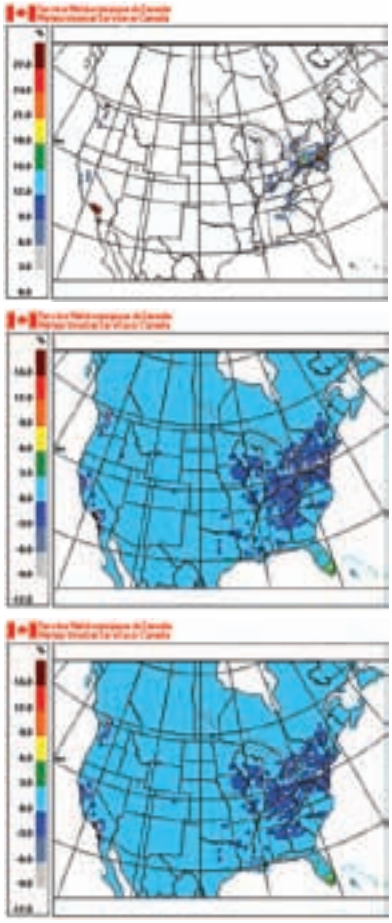
To highlight changes in the persistence of extreme O_3 events, the middle panel of Figure 4.9 presents the changes in CC4 simulation relative to the BC simulation. Changes above the continental landmass generally ranged from -4 to +7 hours with an average change of 0.1 hour.

The bottom panel of Figure 4.9 presents the changes in the CC4b simulation relative to the BC simulation, and indicates that several parts of Canada would experience average durations of summer time O_3 exceedance of more than 7 hours. The duration of O_3 episodes in some locations in Canada, however, would decrease slightly. The average length of O_3 exceedance increased by 1.5 hours from the BC simulation. In the CC4 and CC4b simulations, the average duration of O_3 exceedances would increase by up to 30 hours in some regions.

4.4.2.2 Fine particulate matter

Figure 4.10 presents the percentage of days with average 24-hour $PM_{2.5}$ concentrations exceeding the CWS of 30 µg/m^3 for the BC, CC4 and CC4b simulations. Simulated summer time $PM_{2.5}$ concentrations seem to be less influenced than O_3 concentrations by the imposed temperature increase. In the BC simulation, the number of days with an exceedance was 4.5% overall, though in some regions including Los Angeles, Vancouver, New Orleans, Toronto and the Ohio River Valley the increase was over 20%. Although not presented graphically, mean daily maximum 24-hour $PM_{2.5}$ concentrations were 8.9 µg/m^3 in the BC simulation.

Figure 4.10 Percentage of days with an average 24-hour $PM_{2.5}$ concentration exceeding the Canada-wide Standard of 30 µg/m³



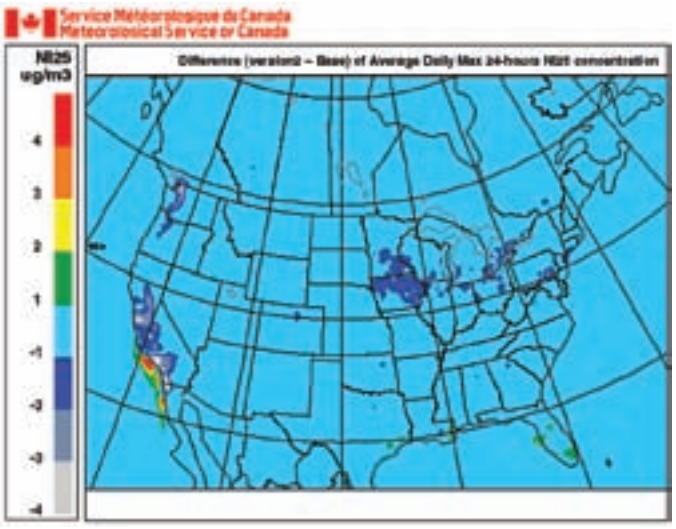
Notes: Top panel: Base case simulation. Middle panel: Difference in number of exceedances between the CC4 simulation and the base case simulation. Bottom panel: Difference in number of exceedances between the CC4b simulation and the base case simulation.

In the CC4 and CC4b simulations, little change was observed overall from the BC simulation (approximately +0.5%). Mean daily maximum 24-hour $PM_{2.5}$ concentrations increased by 1 and 2% (to 9.0 and 9.1 µg/m³) in the CC4 and CC4b simulations, respectively. The duration of the exceedances exhibited a slightly sharper change than mass concentrations, increasing by 6% from the BC simulation to the CC4 simulation, and almost 7% for CC4b simulation. This increase is larger than the increase between the BC and CC4 simulations for O_3 (3.5%), but much smaller than the increase between the BC and the CC4b results (43%).

It is striking that in the CC4 and CC4b simulations, some industrialized and/or urban areas in Canada (particularly the Quebec-Windsor corridor, Winnipeg and southern B.C.) would actually experience a decrease in the percentage of days with average 24-hour $PM_{2.5}$ concentrations exceeding the CWS by 0 to 10%. The small difference between the levels simulated in the CC4 and CC4b simulations suggests that $PM_{2.5}$ levels show very little response to a change in biogenic emissions. Based on the current limited understanding of how biogenic VOCs produce $PM_{2.5}$ (Pun et al., 2002; Aw and Kleeman, 2003) and the current results, $PM_{2.5}$ levels appear not to be strongly influenced by such factors. Changes observed in $PM_{2.5}$ levels between the BC simulation and the CC4 and CC4b simulations may relate to a change in the chemistry of the sulphate–nitrate–ammonium–water system that controls a large fraction of PM mass. Figure 4.11

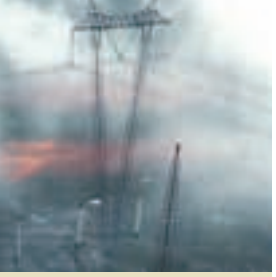
presents the difference in particle nitrate (NO_3^-) concentration during the summer between the BC and CC4b simulations. Certain regions would experience a decrease in particulate NO_3^- concentration whereas others would see an increase. Patterns of particulate NO_3^- change match those of $PM_{2.5}$.

Figure 4.11 Difference between the base case and CC4b simulations of the NO_3^- fraction of $PM_{2.5}$ during the summer



Decreasing particulate NO_3^- levels with increasing temperature have been observed in other studies. Aw and Kleeman (2003) conducted a similar analysis of the effect of temperature variability on levels of O_3 and $PM_{2.5}$. They found that for some locations, in particular regions that had similar concentrations of gas-phase NH_3 and nitric acid (HNO_3), and especially those with relatively high initial temperatures, particulate NO_3^- concentrations decreased with increasing temperature.

Increasing temperature produces a net effect of two opposing processes: (1) increasing the rate of reaction by which particles are formed (thereby increasing particle concentration) and (2) increasing the volatility of the semi-volatile components of $PM_{2.5}$ (thereby decreasing particle formation). Aw and Kleeman (2003) found that, among regions with high concentrations of gas-phase NH_3 and relatively low initial temperatures, particulate NO_3^- concentrations were less sensitive to an increase in temperature, and minor reductions or sometimes small increases in particulate ammonium nitrate (NH_4NO_3) concentration were observed. However, the work of Aw and Kleeman (2003) was limited geographically to the South Coast Basin surrounding Los Angeles, California. In this area, particles of ammonium nitrate dominate the $PM_{2.5}$ mass fraction, and changes in the concentration of secondary sulphate aerosols in this region had a negligible effect on the observed change in $PM_{2.5}$ mass. In regions where SO_2 emissions are larger, such as eastern Canada and the eastern U.S., Aw and Kleeman (2003) suggested that increased temperatures would lead to higher particulate sulphate concentrations and a reduction in particulate NO_3^-. However, even for the short period analyzed in this study it seems that the decreases in particulate NO_3^- levels are large enough to drive the observed changes in $PM_{2.5}$ mass.



4.5 ESTIMATION OF HUMAN HEALTH IMPACTS ASSOCIATED WITH CHANGES IN AMBIENT AIR QUALITY DUE TO CLIMATE CHANGE

Modelling was undertaken to explore the potential health impacts and associated costs that could result from changes in the atmospheric concentration of O_3 and $PM_{2.5}$ in a warmer climate. Estimates were obtained by comparing modelled air quality during three summer months of the year 2002 (base year) with modelled O_3 and $PM_{2.5}$ conditions that could prevail in North America if the average ambient temperature was 4°C higher. Changes in ambient concentrations associated with increased biogenic emissions of VOCs were also obtained. This exercise made use of Health Canada's Air Quality Benefit Assessment Tool (AQBAT) for characterizing human health risks. A detailed description of AQBAT, including its inputs and outputs is provided in Annex 3. The AQBAT analysis here is based on air pollution concentrations overall, not solely on locations that exceeded the CWS.

Valuation of health impacts of air quality changes (positive or negative) is usually completed to complement cost estimates of air pollution mitigation measures. Such a cost-benefit analysis provides a value estimate of the health benefits compared to the cost of specific pollution management measures. The effects estimated in the current analysis are not complemented with an estimation of the mitigation cost of climate change or GHGs, which is beyond the scope of this Assessment. However, it provides an indication of the impacts Canadian society would face if temperatures rose by 4°C, with anthropogenic emissions of air contaminants held constant at 2002 levels.

The precise nature of the potential health impacts is difficult to assess because such quantification requires the integration of multiple variables, including human behaviour, changes in emissions and other climate variables. The estimation of the effect of climate change on both ozone and $PM_{2.5}$ is complex and depends on multiple variables. While the relationships with ozone are relatively well understood, those with PM are still the subject of much investigation, and these results should stimulate additional research. Overall, climate change, according to the CC4b (temperature and biogenic increases) scenario, was predicted to result in a 4.6% increase in cost to Canadian society.

4.5.1 Estimated Health Benefits: Incremental Climate Change Scenario

The estimated human health impacts of changes from baseline levels of O_3 and $PM_{2.5}$ concentrations to levels projected under scenario CC4b (+4°C and increased biogenic emissions) are presented in Annex 4, Tables 4.6 to 4.8. The CC4 temperature-only results are presented in Tables 4.3 to 4.5.

The national average 8-hour O_3 concentration was increased in the CC4b scenario by 14.7%, whereas the 24-hour $PM_{2.5}$ concentration was reduced by 10.5%. The estimated increase in O_3-related mortality in the CC4b scenario is 658 deaths over the modelled 3-month period, with an associated cost of over $3 billion (Table 4.6). The increase in the number of Acute Respiratory Symptom Days (ARSD) corresponds to 2,940,278 cases and a cost of over $42 million. Overall, the total cost of increasing O_3 levels under this scenario is $3.167 billion. Given the results of the modelling runs, this is primarily associated with temperature-induced biogenic increases rather than a direct temperature effect on ozone formation.

The modelled reduction in $PM_{2.5}$ is associated with decreases in all health endpoints, including a reduction of 346 premature deaths and 810,934 ARSD under the CC4b scenario for the 3-month period in question (Table 4.7). The overall benefit of $PM_{2.5}$ reduction corresponds to reduced costs of $1.8 billion. Based on the modelling runs, the PM results are relatively insensitive to the biogenic changes, and are more directly tied to the temperature increase than was the case with O_3. Given the considerable uncertainty attached to this aspect of the modelling, more research is required to provide greater confidence in both the direction and magnitude of the change in PM levels.

The overall pollutant effect in scenario CC4b is endpoint-dependent because there are increases in the incidence of some endpoints and decreases in others (Table 4.8). Overall, premature mortality would be increased by 312 deaths and significant increased morbidity of several types would be observed, whereas the number of adult chronic bronchitis cases would be reduced by 450. Other health benefits include 20 fewer cardiac emergency room visits, 54 fewer cardiac hospital admissions, and 3,479 fewer child acute bronchitis episodes. The overall cost to society would be approximately $1.4 billion over the modelled 3-month period.

4.5.2 Climate Associated Air Quality Impacts in Perspective

An estimation of the amount of illness and death that could be prevented in the complete absence of ambient levels of O_3 and $PM_{2.5}$ was also calculated. This calculation was performed to put into perspective the increase of illness and death associated with the climate change scenario. This estimate was produced by setting O_3 and $PM_{2.5}$ concentrations to zero and then estimating the differences to the baseline and climate change scenarios.

The average baseline morbidity and mortality estimates associated with O_3 and $PM_{2.5}$, based on comparisons with the zero-pollutant scenario, are provided in Annex 4, Tables 4.9 and 4.10, respectively. The AQBAT simulations indicated an increase from baseline in the incidence of O_3-related morbidity and mortality that ranged from 4.4 to 5.3% for the CC4 scenario. The range for the CC4b scenario was 19.5 to 23.3%. In other words, the inclusion of biogenic emissions resulted in considerably higher incidences of health impacts.

Inclusion of the biogenic component had the opposite effect on PM. Temperature increase alone significantly reduced PM impacts (15.3 to 19.1%) though this result was tempered by inclusion of associated biogenic processes (11.9 to 15.3%).

These results indicate that while these pollutants appear to operate in opposite directions under the influence of climate change, the increase in O_3 formation would result in negative net health impacts overall. This result is most influenced by biogenic aspects of the air quality modelling, and based on this estimate would result in an approximate 4.6% increase in the air pollutant-related health burden to Canadian society, subject to a 4°C temperature increase with anthropogenic emissions held at 2002 levels. Recall that future changes in anthropogenic emissions due to economic growth, technological change or regulation would also significantly affect air quality, but were not considered in this modelling exercise.

4.6 RISK MANAGEMENT AND ADAPTATION

4.6.1 Norms and Trends

In June, 2000, the Canadian federal, provincial and territorial governments, with the exception of Quebec, agreed on the Canada-wide Standards (CWS) for $PM_{2.5}$ and O_3. The CWS are long-term air quality management goals that seek to minimize the risks these substances pose to human health and the environment. Although the health impacts attributed to PM and O_3 have been observed at very low concentrations, the CWS attempt to balance reducing risks from $PM_{2.5}$ and O_3 exposure with technologically feasible and cost-effective measures to reduce ambient levels of air pollutants (CCME, 2006). Specifically, the CWS numerical targets and timeframes are (Canadian Council of Ministers of the Environment (CCME), 2000):

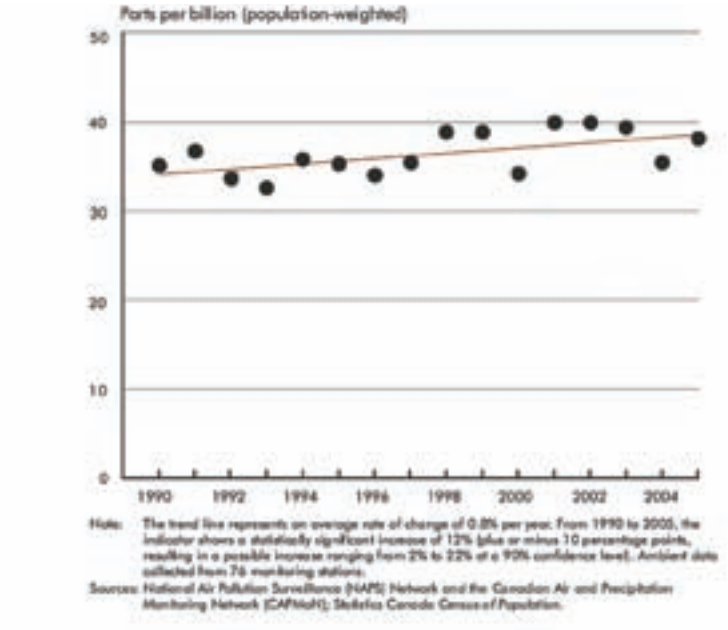
- $PM_{2.5}$: 30 µg/m^3 based on a 24-hour averaging time. Achievement is based on the 98th percentile measurement annually, averaged over 3 consecutive years, by 2010; and
- O_3: 65 ppb based on an 8-hour averaging time. Achievement is based on the fourth highest measurement annually, averaged over 3 consecutive years, by 2010.

In addition to the numerical standards, the CWS also contain provisions that commit jurisdictions to the principles of "Continuous Improvement" and "Keeping Clean Areas Clean." Although the processes governing these two principles have taken some time to elucidate, they are likely to become increasingly important under a warming climate. As larger geographic areas come under the influence of larger and warmer air masses containing air contaminants, those areas currently in marginal compliance with the numerical standards may begin to approach or exceed the CWS.

4.6.1.1 Ground-level ozone

In Canada, population-weighted concentrations of ground-level O_3 have shown a slight increase in recent years (Government of Canada, 2007). Between 1990 and 2005, population-weighted ozone concentrations increased by 12%, with a margin of error of plus or minus ten percentage points (Figure 4.12). O_3 is highest in the Quebec–Windsor corridor, the southern Atlantic region, and to a lesser degree the Lower Fraser Valley of British Columbia.

Figure 4.12 Historical levels of ozone in Canada



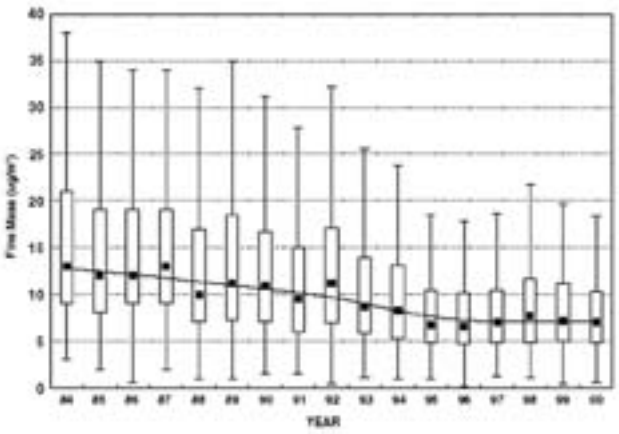
Source: Government of Canada, 2007.

4.6.1.2 Fine particulate matter

Trends in ambient concentrations of $PM_{2.5}$ in Canada are difficult to assess because of changes in monitoring methods over time (Environment Canada, 2003b). Annual averages of $PM_{2.5}$ concentrations for 11 urban sites across Canada are shown in Figure 4.13 (Environment Canada, 2003a). Although there is a slight decreasing trend in mean $PM_{2.5}$ concentration over time, mean levels since the mid-1990s are relatively stable. A large reduction in the value of the 98th percentile is also observed. Such reductions in $PM_{2.5}$ concentrations from the 1980s and early 1990s are likely due to decreases in SO_2 emissions resulting from acid rain prevention programs (Environment Canada, 2004).

More recent reports show that population-weighted levels showed no statistically significant increase or decrease between 2000–2005 (Government of Canada, 2007).

Figure 4.13 Trend in annual mean $PM_{2.5}$ (µg/m³), 1984–2000 (10th percentile, 98th percentile and mean)



Source: Environment Canada, 2003a.

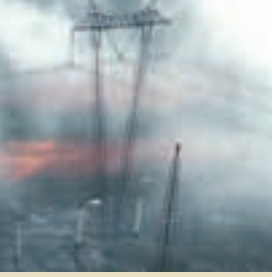
4.6.1.3 Canadian sources and transboundary pollution

Several North American airsheds extend beyond national boundaries, resulting in transboundary pollution and air quality problems whereas other areas are dominated by local sources and conditions. Two regions in Canada with considerable transboundary air pollution are the Great Lakes Basin airshed and the Georgia Basin–Puget Sound airshed (Environment Canada, 2004). A recent report, *Transboundary Air Pollution in Ontario* (Yap et al., 2005) documents that, during smog episodes, transboundary air pollution in Ontario reaches or exceeds the levels resulting from local sources. Canada and the United States currently jointly manage aspects of air quality through the Canada-United States Air Quality Agreement (AQA) (Environment Canada, 2006).

4.6.2 Air Quality Index Forecasts and Adaptation

4.6.2.1 The Air Quality Health Index

Air quality forecasts and the publication of current air quality conditions provide the public with opportunities to undertake short-term adaptive measures that reduce their exposure to air pollutants. With air quality indices designed around ambient air quality objectives for individual pollutants, attention is often focussed on the air quality advisories that are issued when these objectives are exceeded. However current scientific evidence shows that health risks increase more or less linearly as air quality deteriorates, and that several pollutants simultaneously contribute to the health risks.



Health Canada and Environment Canada, in conjunction with provinces, municipalities and a variety of stakeholders from the health and environmental communities, have developed a new index that better reflects the immediate health risks associated with the smog mixture. This new Air Quality Health Index (AQHI) was designed to be a personal health protection tool to be used by the public on a daily basis. The AQHI scale is accompanied by health advice targeted to vulnerable groups—children, seniors, and people with cardiovascular and respiratory disease—as well as to the general population, to enable individuals to make informed decisions about reducing their exposure to air pollution and their associated health risks. Several communities have piloted the new index and over the next three years it will be implemented throughout Canada as part of a renewed air quality forecast program. For example, information on the City of Toronto's AQHI can be obtained at www.toronto.ca/health/aqhi/.

4.6.2.2 Outreach to the public, at-risk populations and health professionals

Significant social marketing efforts will be undertaken to make the Air Quality Health Index (AQHI) as effective as the Ultraviolet (UV) Index. For example, the federal government will work in collaboration with the communications industry, including The Weather Network and local media providers, to identify the ideal means of communicating the AQHI to the public through different media (i.e. television, print, radio, automated telephone, Internet). Lessons learned from the 2006 pilots utilizing websites, radio and automated telephone for communicating the AQHI will be taken into consideration. Government On-Line officials will also be engaged to investigate the feasibility of creating a nationally branded federal portal for the index, providing one-stop access to air quality measurements and forecasts via the AQHI in various jurisdictions.

To facilitate education and endorsement of the AQHI by health professionals, Health Canada will develop a toolkit for health professionals based on recent research. The toolkit will detail the health impacts of air pollution, information on how to interpret and explain the AQHI to their patients, and appropriate actions to reduce exposure to air pollution while maintaining a balance of healthy lifestyle factors. Materials will also be generated for health professionals to distribute to their clientele.

Outreach materials will be developed for the general public to provide consistent health and air quality messaging. Additional efforts will concentrate on messaging targeting susceptible populations and their caregivers (children, seniors, and people with cardio-vascular and respiratory disease). It is envisioned that these resources will be developed in concert with and distributed by non-governmental organizations as well as health professionals.

4.6.2.3 Links to other adaptation programs

Several communities across Canada have already implemented different types of heat alert systems (see Chapter 8, Vulnerabilities, Adaptation and Adaptive Capacity in Canada) and many more are investigating their applicability in their community. The Government of Canada committed in 2007 to working with stakeholders on developing best practices for the implementation of Heat Alert Systems and Infectious Disease Alert Systems. Lessons learned from the AQHI could provide valuable information to Health Canada's pilot heat alert and response initiative. For example, as is the case with air quality, the most heat-vulnerable groups include seniors, children, and people with pre-existing conditions. The AQHI's communication and outreach methods could prove valuable in designing health messaging around the effects of heat on vulnerable populations.

4.7 DISCUSSION AND CONCLUSIONS

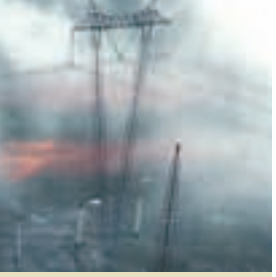
Overall, results from epidemiological studies have provided some evidence, although sparse, that temperature may influence the impact of air pollution on health. This interaction may be due to true synergistic effects, but other plausible explanations must also be considered. Higher average temperatures are in fact associated with greater exposure to air pollution because population activity patterns change (e.g. people spend more time outside and tend to leave their windows open, though air conditioning may temper this).

Results from atmospheric modelling conducted for this Assessment focussed only on changes in temperature and biogenic emissions, ignoring possible changes in anthropogenic emissions. Nevertheless, modelling has produced a useful basis for future work because the results have suggested that climate change could change the ambient tropospheric levels of O_3 and $PM_{2.5}$. Results have also suggested that increased temperature may lead to increases in O_3 concentrations but decreases in $PM_{2.5}$ concentrations. However, modelling that accounts for all potentially related factors, such as precipitation and anthropogenic emissions is required to adequately assess the direction for $PM_{2.5}$ under warmer climatic conditions.

Modelled changes in air pollutants certainly imply that climate change could lead to increases in illnesses and premature death in Canada. Valuation estimates clearly indicate that climate change would result in increased financial burden over the 3-month summer period, largely due to O_3. A similar assessment for a 12-month period would theoretically result in a larger number, but not necessarily four times larger because the highest air pollution levels are primarily experienced in summer. The results of quantifying the human health impacts with AQBAT clearly suggest that climate change in Canada may result in significant health costs to society.

The scenarios used here are only a first step in improving understanding of the effects of climate change on air quality. Only two factors, temperature and biogenic emissions, of the multitude of the factors that may be affected by climate change were considered. More detailed Global Climate Model, Regional Climate Model, AURAMS and AQBAT assessments might provide more accurate estimates of how climate change might affect air pollution in Canada and its related health impacts.

Future assessments of the potential health impacts related to climate change should integrate the broader chain of events that would likely be involved. More accurate estimations of the potential health effects which take into account population growth and change, the climate for various future time periods (e.g. around the years 2020, 2050 and 2080), anthropogenic and natural emissions, atmospheric modelling, changes in human exposure to pollutants, environmental changes associated with climate change and GHG mitigation measures should be conducted. For instance, changes in weather patterns could modify the energy demands associated with the cooling and heating of buildings, which in turn could result in changes in pollutant emissions. These assessments should also consider the existing disparities in the social, economic and health status of the residents of different provinces and territories. However, they would still leave many uncertainties.



4.8 ANNEXES

Annex 1: Literature Search Methodology

Five bibliographical databases were searched, using several different terms combined in a logical expression, to identify all relevant articles in the published literature (Figure 4.14).

Figure 4.14 Bibliographic databases and search terms and logical expressions used to identify relevant studies in the literature

Databases
Ovid MEDLINE(R) 1966 to June Week 4 2005 Ovid MEDLINE(R) In-Process & Other Non-Indexed Citations July 05, 2005 Biological Abstracts/RRM 1992 to 2002 Biological Abstracts 2002 to June 2005 Current Contents/All Editions 1993 Week 27 to 2005 Week 28
Search terms and logical expression (? and $ denote wildcards)
heat-wave? or heatwave? or (heat wave?) OR heat or hot$ or warm$ or (high$ temperature?) AND temperature? or weather or meteorolog$ or climat$ or season$ AND (air pollut$) or (atmospheric pollut$) or “air quality” or ozone OR (particles OR particulate) and pollut$ AND health or morbidity or mortality or death? or admission? or consult$ or disease? or disorder?

This search resulted in over 600 references published by 2006. These were manually selected, based on their title and abstract, resulting in approximately 50 core articles. Several other key articles cited as references to these papers or found elsewhere were added to the list. All articles were then reviewed, and if deemed relevant, were classified and retained for analysis.

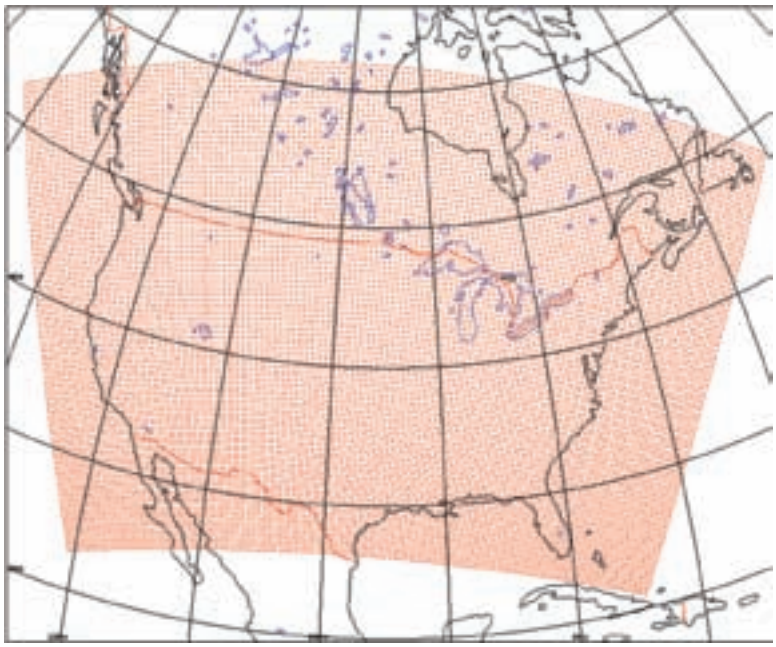
Annex 2: AURAMS Model

1. Model Description

A Unified Regional Air-quality Modelling System (AURAMS) is a unified regional air quality modelling system developed by Environment Canada for research and policy applications. Designed as a “one-atmosphere” system, AURAMS allows the study of interactions between NOx, VOCs, NH_3, O_3, and primary and secondary airborne PM smaller than 2.5 µm ($PM_{2.5}$). It can therefore be used to address a variety of interconnected tropospheric air pollution problems ranging from surface O_3 to acid rain to PM over the entire North American continent.

AURAMS has been exercised over domains covering the whole or parts of the North American continent at spatial resolutions of 20 to 42 km (Figure 4.15). Although initial applications of AURAMS were limited to episodic events, it is now being used for seasonal and annual simulations, as well as in experimental mode for next-day forecasting of air quality. Evaluations of the performance of AURAMS can be found in the peer-reviewed literature (Bouchet et al., 2003; Gong et al., 2003b; Makar et al., 2004; McKeen et al., 2005; Gong et al., 2006) and in recent joint U.S.and Canada assessments (Canada-U.S. Subcommittee on Scientific Co-operation, 2004). For determination of O_3 and PM concentrations for the present study, AURAMS was run at 42-km resolution on a continental domain.

Figure 4.15 AURAMS modelling domains in North America



The three major components of the system, namely the meteorological driver, the emission processor and the chemical transport model, are described below. Additional details on the various processes represented in the chemical transport model are also given.

2. Meteorological Component

AURAMS is driven "off-line" by the Canadian operational forecast model, Global Environmental Multiscale model (GEM). GEM is a non-hydrostatic, two-time-level implicit semi-Lagrangian model (Côté et al., 1998a, 1998b). For air quality applications, meteorological fields from a high-resolution regional window positioned over the air quality modelling domain are stored at the frequency required by the AURAMS (i.e. 900 seconds), then interpolated spatially to match the grid used by the air quality model.

3. Emission Components

Anthropogenic emission component

Hourly point-, area- and mobile-source emissions files are prepared by the Spare-Matrix Operating Kernel Emissions processor (SMOKE), from the year 2000 Canadian and the year 2001 U.S. national criteria-air-contaminant emission inventories for the AURAMS domain. The total Canadian anthropogenic emissions for the year 2000 are presented in Table 4.1. Similar information for the year 2001, which is stored in the U.S. national emission inventory, can be readily accessed online. AURAMS-ready emission fields include 17 gas-phase species, as well as primary $PM_{2.5}$ and PM_{10} emissions. Within the AURAMS chemical transport component, PM emissions are disaggregated according to size and chemical species as a function of the source stream. A plume-rise calculation is also applied to major point sources. Two other types of emissions are represented online in AURAMS: biogenic emissions using the Biogenics Emissions Inventory System, Version 3.09 (BEISv3.09) algorithm and sea salt emissions from wave-breaking (Gong et al., 2003a).

Table 4.1 Total Canadian anthropogenic emissions (in tons) from area, mobile and non-road sources for the year 2000

	Area*	Mobile†	Non-Road‡
TPM	17,490,531	21,242	69,771
PM_{10}	105,311,545	21,162	68,787
$PM_{2.5}$	842,433	19,415	60,102
SO_2	202,559	28,005	63,323
NO_x	435,225	936,794	774,694
VOC	1,938,958	446,438	354,872
CO	1,870,862	6,313,751	2,915,483
NH_3	591,966	19,695	998

* In this table, includes industrial sources and small, non-mobile sources that are inventoried as a group (e.g. wood stoves and incinerators).

† Cars, trucks and other on-road mobile sources.

‡ Off-road mobile sources like tractors and backhoes.

Biogenic emission component

Plants emit VOCs that can serve as precursors of O_3 and of $PM_{2.5}$ by reacting in the atmosphere with other chemicals such as NOx, or with solid or semi-solid particles. These VOCs include isoprene and methyl butanol, whose highest concentrations occur in the air above large forests and above extensive agricultural crops. The biogenic emissions inventory module BEISv3.09, developed in the U.S. by a U.S. Environmental Protection Agency–National Oceanic and Atmospheric Administration partnership (U.S. EPA 2006b, 2006c), was used within AURAMS to estimate biogenic VOC emissions from vegetation and NO emissions from soil.

AURAMS chemical transport model

The AURAMS chemical transport component includes a representation of all the processes that influence the formation, release and fate of O_3 and PM. Up to eight chemical components are considered to contribute to PM composition: sulphate, nitrate, ammonium, black carbon, primary organic carbon, secondary organic carbon, crustal material and sea salt. These PM chemical components are assumed to be internally mixed in each of the 12 size bins of the PM sectional size distribution, spanning diameters between 0.01 µm and 40.96 µm. No data assimilation is performed in AURAMS.

AURAMS uses a non-oscillatory semi-Lagrangian advection scheme (Pudykiewicz et al., 1997; Sirois et al., 1999) to describe the transport of up to 145 individual chemical tracers. The gas-phase mechanism is a modified version of the ADOM-II mechanism (Stockwell and Lurmann, 1989) and incorporates 42 gas-phase species and 114 reactions based on Lurmann et al. (1986). It has been extended to include secondary organic aerosol formation based on the approach of Jiang (2003), inorganic heterogeneous chemistry (i.e. gas-particle partitioning of the sulphate–nitrate–ammonium–water system) (Makar et al., 2003), and aqueous-phase chemistry based on a modified ADOM mechanism coupled with explicit aerosol components. Aerosols are represented by a size-segregated multi-component algorithm (the Canadian Aerosol Module (CAM)) (Gong et al., 2003a). CAM relies on a sectional approach, and includes major aerosol processes in the atmosphere: generation, hygroscopic growth, coagulation, nucleation, condensation, dry deposition and sedimentation, below-cloud scavenging as well as aerosol activation. Finally, dry deposition of gases and size-dependent aerosols is based on the resistance approach (Zhang et al., 2001, 2002), and wet deposition processes to gases and aerosol include cloud-to-rain conversion and below-cloud scavenging as well as evaporation (Gong et al., 2003b).

Annex 3: AQBAT Model

1. Model Description

The Air Quality Benefits Assessment Tool (AQBAT) is a computer simulation tool designed to estimate the relative human health and welfare benefits or risks associated with changes in Canada's ambient air quality. AQBAT allows users to define a wide range of specific scenarios by combining and linking various air pollutants, health endpoints, geographic areas and scenario years. AQBAT can provide economic valuation estimates based on changes in the incidence of health impacts associated with changes in air quality. AQBAT is the successor to the Air Quality Valuation Model (AQVM). It consists of one Excel file containing Excel user forms and toolbars with numerous controls, and Visual Basic programming to help the user define, run, examine and save a specific scenario. An expert panel of the Royal Society of Canada reviewed socio-economic analysis tools and provided specific comments on AQVM, the predecessor of AQBAT. The expert panel supported AQVM and the inputs used in the model (Royal Society of Canada, 2001).

AQBAT contains files of historical and projected population data, and can access preset data files of historical and hypothetical pollutant concentrations, as well as baseline health endpoint rates. The model utilizes and controls the @Risk (trademark) add-in software to perform Monte Carlo simulations, which entail sampling the input distributions, tracking the calculated sample outputs, and providing descriptive statistics on the distributions of these outputs.

2. AQBAT Approach

The net risk or benefit associated with changes in the ambient air pollutant concentration is estimated using concentration response functions (CRF) that are assigned to specific pollutant concentrations and to exposed populations. The AQBAT user defines a CRF for a pollutant-health endpoint combination and assigns it to one or more geographic areas. Different geographic areas may be assigned either the same quantified impact of a pollutant on a health endpoint, or different ones (i.e. there can be multiple CRFs for the same pollutant-health endpoint combination). Each CRF applies to every year of the scenario.

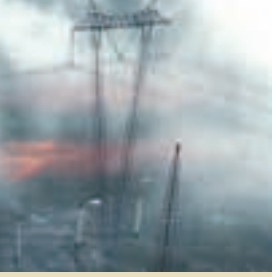
The count estimate formulation, in its simplified form, consists of multiplying the following factors:

- CRF expressed as % excess adverse endpoints per unit concentration increase of pollutant;
- pollutant concentration change as the difference between the status quo and forecast concentrations;
- baseline rate of incidence of the health endpoint in the target population; and
- target population count.

These counts are derived for individual scenario years.

3. Concentration Response Function

The CRF is a quantification of the impact of an air pollutant on a health endpoint. It is a statistically derived estimate of the percent excess health endpoint associated with a unit increase in the pollutant concentration. It is generally derived from a statistical model, or from the pooling of estimates from several models or studies. CRFs therefore have an



uncertainty in their quantification, reflected in the selection of a distribution form (normal, triangular or 3-point discrete), with corresponding parameter inputs in AQBAT. During a scenario model simulation run, the @Risk tool samples these distribution functions, from which output sample values are calculated and tracked; these outputs therefore have a distribution themselves. There are 13 different health endpoints in this model:

Acute Exposure Mortality

Acute Respiratory Symptom Days

Adult Chronic Bronchitis Cases

Asthma Symptom Days

Cardiac Emergency Room Visits

Cardiac Hospital Admissions

Child Acute Bronchitis Episodes

Chronic Exposure Mortality

Elderly Cardiac Hospital Admissions

Minor Restricted Activity Days

Emergency Room Visits Due to Respiratory Illnesses

Hospital Admissions Due to Respiratory Illnesses

Days of Restricted Activity

Each of these represents a health endpoint resulting either from an acute or short-term exposure, or from a long-term or chronic exposure (see Table 4.13 for all CRFs). As well, each of these endpoints corresponds to a certain proportion of a specific population age group. These characteristics of the health endpoints are pre-defined in AQBAT; the user cannot change them.

4. Economic Valuation Estimates

Economic valuation estimates for health outcomes consider potential economic and social consequences associated with the adverse health impacts that result from air pollution, including medical costs, work loss, out-of-pocket expenses, and pain and suffering. Although the economic valuation estimates used to assess potential benefits may include medical costs, they must not be interpreted in their totality as savings to the health care system. The valuation estimates must be considered merely as an indication of the relative value that society places on health benefits represented by decreases in the risk of death or disease (see Table 4.12 for AQBAT valuation information).

One economic measure of value that represents the reasons why people desire reductions in health risks is called “willingness to pay.” Willingness to pay is a measure of the monetary tradeoffs that people are willing to make in exchange for reductions in risks of mortality or morbidity. Unlike many goods and services that Canadians can purchase and enjoy, the prevention of health risks cannot be directly purchased in the marketplace. Therefore, there are no price and quantity data from which the economic values can be easily estimated.

Over the last five decades, economists have developed and refined a number of techniques to estimate the economic value of avoiding adverse health impacts. These techniques estimate values by examining how people would trade money for reductions in health risks. There are a number of empirical methods for valuation; these essentially fall into two categories—those that rely on observed market behaviour (“revealed preference methods”) and those that do not (“stated preference methods”).

5. Netting

Netting is the subtraction of one health endpoint count from another health endpoint count (see under section 8, Outputs, later in Annex 3 for a discussion of counts), because the former endpoint may overlap with the latter. The result of not carrying out the netting may be a double counting of the latter endpoint. One common example of netting is that Respiratory Hospital Admissions (RHAs) are subtracted from Respiratory Emergency Room Visits (RERVs), under the assumption that some RHAs started out as RERVs. In this example, not carrying out the subtraction would result in some RHAs being counted as both health endpoints. The netting results in conservative estimates. The user can decide whether a CRF is already netted, or alternatively, whether AQBAT is to apply the preset netting adjustments to the health endpoint counts that result in applying the scenario model CRFs.

6. Grouping

Grouping is the accumulation of similar health endpoints. Acute Exposure Mortality counts and Chronic Exposure Mortality counts are usually added up to obtain total mortality counts. This could be done outside of AQBAT after the simulation has been run, but grouping during the simulation reduces the workload for the user. As well, grouping during the simulation produces exact percentile values for the distributions of grouped endpoint outputs, whereas these can be approximated only outside the simulation. However, grouping during the simulation requires tracking more outputs, and therefore, a longer run time for the simulation.

7. Geographic Areas

AQBAT was developed for Canada's geography. There are 442 geographic areas in the current version of AQBAT, based on the 2001 Census Geography as determined by Statistics Canada. Each of the 442 geographic areas is one or two of the following five types by level (acronym and/or count):

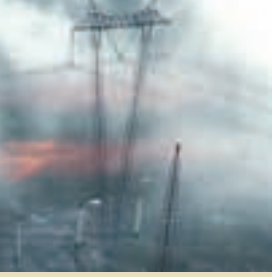
- National level: Canada as a whole
- Provincial level: provinces or territories (13)
- Specific (lowest) level: Census Agglomeration Area (CA, 113), Census Metropolitan Area (CMA, 27), and Census Division (CD, 288)

Census Agglomeration Areas (CAs) are towns with a total population between 10,000 and 100,000 and Census Metropolitan Areas (CMAs) are cities with a total population of 100,000 or more.

8. Outputs

AQBAT provides estimates of excess or reduced health endpoints in a geographic area that is associated with an inputted pollutant concentration change. The CRF has an uncertainty and is input into AQBAT as a distribution function. The pollutant concentration change, the baseline rates of health endpoints and the population count are deterministic, based on specific choices or inputs to define the scenario model in AQBAT. The resulting output count therefore also has an uncertainty, expressed as a distribution. There are two additional scenario model output types: percent of baseline and economic valuations. Both of these use the counts as a factor in their calculation.

AQBAT allows for the generation of up to 20,000 outputs, tracked by @Risk during the simulation; one iteration results in a sampled value for each tracked output. Percentile values, means, standard deviations and other statistics are determined from the simulation for each set of output sample values. For each output, the user can examine the distribution of values on a chart. There are tools available in AQBAT to filter or "home in" on a smaller subset of outputs, when necessary. All inputs defining the specific scenario model, along with the output distributions can also be stored in an external Excel workbook for investigation and analysis outside of AQBAT.



9. Definition of the AQBAT Estimates

The atmospheric modelling for O_3 and $PM_{2.5}$ was carried out by scientists at Environment Canada to estimate average air pollutant concentrations in 2,446 Census Consolidated Subdivisions (CCSs) (of the census year 2001) during the months of June, July and August 2002. These concentration levels were then averaged over all CCSs in a Census Division (CD), to determine a concentration level for each of the 288 CDs under the corresponding hypothesis. AQBAT estimates were based on a modelled baseline and a scenario where ambient temperature was increased by 4°C without changes in biogenic emissions of air pollutant precursors (scenario CC4) and a scenario where ambient temperature was increased by 4°C with associated changes in biogenic emissions (scenario CC4b). These estimates were used as an indication of the potential health impacts of these two climate change scenarios (CC4 and CCC4b). AQBAT estimates of the differences between the CC4 and CC4b scenarios were also used to show the effect of biogenic emissions.

AQBAT estimates of the differences between a scenario in the absence (anthropogenic and biogenic) of air pollutants (Zero) and baseline, CC4 and CC4b scenarios were completed. These estimates were obtained to put into perspective the various estimates against the overall mortality and morbidity associated with air pollutants. AQBAT estimates for the environmental states identified in Table 4.2 were obtained.

Table 4.2 The six estimates computed as part of this project

AQBAT Estimates	Baseline State	Compared to
1	Baseline	CC4
2	Baseline	CC4b
3	CC4	CC4b
4	Zero	Baseline
5	Zero	CC4
6	Zero	CC4b

It should be noted that the AQBAT estimates for the current project are for a 3-month summer period, as the atmospheric modelling for this project covered only the months of June, July and August 2002. Adjustment to the baseline mortality or morbidity rates was completed to report the results for the 3-month period only. Using 2002 as a base year for the simulations means that the meteorological inputs are generated for summer 2002. Although no single summer can really represent the climate average, the only alternative to performing years of simulation is to choose a base year that is as representative as possible of that average (see section 4.4.1 for additional details).

10. AQBAT Results

AQBAT estimates were generated nationally and for each of the provinces and territories as well as for each of the CDs with a population of more than 200,000 people. Twenty thousand @Risk iterations were completed for each analysis. Results are provided individually for each of the two pollutants, and the overall effect is also provided separately. Absolute estimates and the valuation associated with each of the health endpoints are provided in each table of results in Annex 4. The list of endpoints for each of the pollutants differs somewhat, as the health impacts of O_3 and $PM_{2.5}$ are not identical.

It is important to remember that AQBAT estimates cover only the months of June, July and August. These estimates would likely be higher if computed for a 12-month period, but would not necessarily be four times higher as this project covers the predominant period for smog events.

Some may argue that chronic health impacts should not be part of the Assessment as only a 3-month period was modelled. However, the analysis is used as an indication of the long-term trend in the potential shift in air pollution as indicated by one segment (June, July and August) and is therefore representative of the potential impacts on chronic health impacts that would be observed over the long term.

Some values are truncated at Zero because the developers of AQBAT concluded that pollution associated with O_3 and PM could not result in positive effects to human health.

Annex 4: Detailed AQBAT Results, Valuation Information and Concentration Response Functions

Table 4.3 O_3: AQBAT estimates from baseline to scenario CC4, national data

Endpoint		Absolute Changes from Baseline Mean (95% CI*)
Acute Exposure Mortality	Count Valuation	-156 (-207, -105) -$726,056,312 (-$1,653,546,752, -$311,456,512)
Acute Respiratory Symptom Days	Count Valuation	-696,586 (-1,607,940, 0) -$10,153,797 (-$37,319,304, $0)
Asthma Symptom Days	Count Valuation	-92,291 (-150,091, -34,619) -$5,148,005 (-$12,521,288, -$1,129,731)
Minor Restricted Activity Days	Count Valuation	-201,800 (-87,599, 0) -$7,921,113 (-$40,626,480, $0)
Respiratory Emergency Room Visits	Count Valuation	-374 (-783, 0) -$147,277 (-$328,433, $0)
Respiratory Hospital Admissions	Count Valuation	-92 (-172, -11) -$421,657 (-$831,809, -$49,016)
Acute Exposure Mortality + Chronic Exposure Mortality	Count Valuation	-156 (-207, -105) -$726,056,312 (-$1,653,546,752, -$311,456,512)
Cardiac Hospital Admissions + Respiratory Hospital Admissions	Count Valuation	-92 (-172, -11) -$421,657 (-$831,809, -$49,016)
Cardiac Emergency Room Visits + Respiratory Emergency Room Visits	Count Valuation	-374 (-783, 0) -$147,277 (-$328,443, $0)
All Endpoints	Count Valuation	Not applicable -$749,848,162 (-$1,678,030,080, -$333,939,776)

Note: A negative value represents a negative effect to society and should therefore be considered as an increase in cost or health impacts. A positive value represents a positive effect to society and should therefore be considered as a reduction in cost or health impacts.

* CI, Confidence Interval

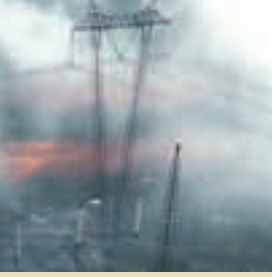


Table 4.4 $PM_{2.5}$: AQBAT estimates from baseline to scenario CC4, national data

Endpoint		Absolute Changes from Baseline Mean (95% CI)
Acute Respiratory Symptom Days	Count Valuation	1,010,696 (0, 2,602,081) $14,691,284 ($0, $57,391,520)
Adult Chronic Bronchitis Cases	Count Valuation	559 (0, 1106) $184,819,544 ($0, $487,192,096)
Asthma Symptom Days	Count Valuation	40,878 (9827, 71,808) $2,279,833 ($332,484, $5,871,109)
Cardiac Emergency Room Visits	Count Valuation	24 (0, 65) $9,646 ($0, $26,454)
Cardiac Hospital Admissions	Count Valuation	68 (36, 99) $383,991 ($182,677, $625,964)
Child Acute Bronchitis Episodes	Count Valuation	4,298 (0, 9,159) $1,470,337 ($0, $4,073,960)
Chronic Exposure Mortality	Count Valuation	428 (227, 629) $1,995,154,492 ($718,112,448, $4,843,215,872)
Respiratory Emergency Room Visits	Count Valuation	176 (100, 253) $69,460 ($34,899, $110,597)
Respiratory Hospital Admissions	Count Valuation	44 (29, 58) $200,064 ($182,240, $296,629)
Restricted Activity Days	Count Valuation	620,980 (366,139, 874,903) $32,435,121 ($0, $71,797,136)
Acute Exposure Mortality + Chronic Exposure Mortality	Count Valuation	428 (227, 629) $1,995,154,492 ($718,112,448, $4,843,215,872)
Cardiac Hospital Admissions + Respiratory Hospital Admissions	Count Valuation	111 (76, 146) $584,055 ($365,122, $840,583)
Cardiac Emergency Room Visits + Respiratory Emergency Room Visits	Count Valuation	201 (115, 291) $79,107 ($40,695, $123,901)
All Endpoints	Count Valuation	Not applicable $2,231,513,773 ($929,518,016, $5,080,671,744)

Note: A negative value represents a negative effect to society and should therefore be considered as an increase in cost or health impacts. A positive value represents a positive effect to society and should therefore be considered as a reduction in cost or health impacts.

Table 4.5 Both pollutants: AQBAT estimates from baseline to scenario CC4, national data

Endpoint		Absolute Changes from Baseline Mean (95% CI)
Acute Exposure Mortality	Count Valuation	-156 (-207, -105) -$726,056,312 (-$1,653,546,752, -$311,456,512)
Acute Respiratory Symptom Days	Count Valuation	314,110 (-1,233,611, 2,119,159) $4,537,487 (-$24,191,136, $43,016,324)
Adult Chronic Bronchitis Cases	Count Valuation	559 (0, 1106) $184,819,544 ($0, $487,192,096)
Asthma Symptom Days	Count Valuation	-51,413 (-116,874, 14,486) -$2,868,172 (-$8,925,413, $702,022)
Cardiac Emergency Room Visits	Count Valuation	24 (0, 74) $9,646 ($0, $30,420)
Cardiac Hospital Admissions	Count Valuation	68 (36, 99) $383,991 ($182,677, $625,964)
Child Acute Bronchitis Episodes	Count Valuation	4,298 (0, 9159) $1,470,337 ($0, $4,073,960)

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Endpoint		Absolute Changes from Baseline Mean (95% CI)
Chronic Exposure Mortality	Count Valuation	428 (227, 629) $1,995,154,492 ($718,112,448, $4,843,215,872)
Minor Restricted Activity Days	Count Valuation	-201,800 (-873,599, 0) -$7,921,113 (-$40,626,480, $0)
Respiratory Emergency Room Visits	Count Valuation	-198 (-616, 184) -$77,817 (-$252,547, $73,374)
Respiratory Hospital Admissions	Count Valuation	-48 (-130, 33) -$221,593 (-$617,189, $153,411)
Restricted Activity Days	Count Valuation	620,980 (336,139, 874,903) $32,435,121 ($0, $71,797,136)
Acute Exposure Mortality + Chronic Exposure Mortality	Count Valuation	272 (64, 481) $1,269,098,181 (-$402,231,328, $4,193,872,384)
Cardiac Hospital Admissions + Respiratory Hospital Admissions	Count Valuation	19 (-69, 105) $162,398 (-$289,217, $594,437)
Cardiac Emergency Room Visits + Respiratory Emergency Room Visits	Count Valuation	-173 (-591, 210) -$68,171 (-$243,020, $85,423)
All Endpoints	Count Valuation	Not applicable $1,481,665,612 (-$198,432,752, $4,416,672,768)

Note: A negative value represents a negative effect to society and should therefore be considered as an increase in cost or health impacts. A positive value represents a positive effect to society and should therefore be considered as a reduction in cost or health impacts.

Table 4.6 O_3: AQBAT estimates from baseline to scenario CC4b, national data

Endpoint		Absolute Changes from Baseline Mean (95% CI)
Acute Exposure Mortality	Count Valuation	-658 (-876, -442) -$3,065,838,265 (-$6,989,489,664, -$1,324,193,408)
Acute Respiratory Symptom Days	Count Valuation	-2,940,278 (-6820468, 0) -$42,578,915 (-$155,551,872, $0)
Asthma Symptom Days	Count Valuation	-394,438 (-644,376, -147,054) -$22,064,012 (-$54,112,592, -$4,533,992)
Minor Restricted Activity Days	Count Valuation	-871327 (-3,816,480, 0) -$34,602,731 (-$179,627,168, $0)
Respiratory Emergency Room Visits	Count Valuation	-1,558 (-3,282, 0) -$613,496 (-$1,381,587, $0)
Respiratory Hospital Admissions	Count Valuation	-382 (-719, -46) -$1,756,359 (-$3,479,363, -$206,747)
Acute Exposure Mortality + Chronic Exposure Mortality	Count Valuation	-658 (-876, -442) -$3,065,838,265 (-$6,989,489,664, -$1,324,193,408)
Cardiac Hospital Admissions + Respiratory Hospital Admissions	Count Valuation	-382 (-719, -46) -$1,756,359 (-$3,479,363, -$206,747)
Cardiac Emergency Room Visits + Respiratory Emergency Room Visits	Count Valuation	-1,558 (-3,282, 0) -$613,496 (-$1,381,587, -$0)
All Endpoints	Count Valuation	Not applicable -$3,167,453,777 (-$7,102,102,016, -$1,420,562,304)

Note: A negative value represents a negative effect to society and should therefore be considered as an increase in cost or health impacts. A positive value represents a positive effect to society and should therefore be considered as a reduction in cost or health impacts.

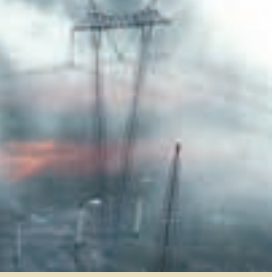

Table 4.7 $PM_{2.5}$: AQBAT estimates from baseline to scenario CC4b, national data

Endpoint		Absolute Changes from Baseline Mean (95% CI)
Acute Respiratory Symptom Days	Count Valuation	810,934 (-1, 208,7633) $11,786,653 ($0, $46,570,748)
Adult Chronic Bronchitis Cases	Count Valuation	450 (0, 892) $149,476,645 ($0, $396,918,784)
Asthma Symptom Days	Count Valuation	32,817 (7,890, 57,660) $1,835,379 ($274,370, $4,700,622)
Cardiac Emergency Room Visits	Count Valuation	162 (92, 233) $63,595 ($32,510, $99,810)
Cardiac Hospital Admissions	Count Valuation	54 (29, 80) $309,809 ($145,737, $504,069)
Child Acute Bronchitis Episodes	Count Valuation	3,479 (0, 7,450) $1,186,541 ($0, $3,302,251)
Chronic Exposure Mortality	Count Valuation	346 (183, 509) $1,610,567,933 ($589,880,640, $3,874,233,856)
Respiratory Emergency Room Visits	Count Valuation	142 (80, 204) $55,795 ($27,981, $88,450)
Respiratory Hospital Admissions	Count Valuation	35 (23, 47) $160,722 ($95,211, $237,069)
Restricted Activity Days	Count Valuation	498,629 (293,856, 702,787) $26,020,581 ($0, $57,630,600)
Acute Exposure Mortality + Chronic Exposure Mortality	Count Valuation	346 (183, 509) $1,610,567,933 ($589,880,640, $3,874,233,856)
Cardiac Hospital Admissions + Respiratory Hospital Admissions	Count Valuation	89 (61, 118) $470,531 ($289,620, $675,508)
Cardiac Emergency Room Visits + Respiratory Emergency Room Visits	Count Valuation	162 (92, 233) $63,595 ($32,510, $99,810)
All Endpoints	Count Valuation	Not applicable $1,801,407,857 ($751,893,632, $4,075,342,080)

Note: A negative value represents a negative effect to society and should therefore be considered as an increase in cost or health impacts. A positive value represents a positive effect to society and should therefore be considered as a reduction in cost or health impacts.

Table 4.8 Both pollutants: AQBAT estimates from baseline to scenario CC4b, national data

Endpoint		Absolute Changes from Baseline Mean (95% CI)
Acute Exposure Mortality	Count Valuation	-658 (-876, -442) -$3,065,838,265 (-$6,989,489,664, -$1,324,193,408)
Acute Respiratory Symptom Days	Count Valuation	-2,129,344 (-6,178,484, 1,332,933) -$30,792,263 (-$136,676,912, $21,992,590)
Adult Chronic Bronchitis Cases	Count Valuation	450 (0, 892) $149,476,645 ($0, $396,918,784)
Asthma Symptom Days	Count Valuation	-361,620 (-612,340, -112,404) -$20,228,634 (-$50,705,608, -$3,634,177)
Cardiac Emergency Room Visits	Count Valuation	20 (0, 60) $7,800 ($0, $24,460)

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Endpoint		Absolute Changes from Baseline Mean (95% CI)
Cardiac Hospital Admissions	Count Valuation	54 (29,80) $309,809 ($145,737, $504,853)
Child Acute Bronchitis Episodes	Count Valuation	3,479 (0, 7,450) $1,186,541 ($0, $3,320,251)
Chronic Exposure Mortality	Count Valuation	346 (183, 509) $1,610,567,933 ($589,880,640, $3,874,233,856)
Minor Restricted Activity Days	Count Valuation	-871,327 (-3,816,480, 0) -$34,602,731 (-$179,627,168, $0)
Respiratory Emergency Room Visits	Count Valuation	-1,416 (-3,146,134) -$557,701 (-$1,315,710, $52,460)
Respiratory Hospital Admissions	Count Valuation	-347 (-684, -11) -$1,595,637 (-$3,295,326, -$46,996)
Restricted Activity Days	Count Valuation	498,629 (293,856, 702,787) $26,020,581 ($0, $57,630,600)
Acute Exposure Mortality + Chronic Exposure Mortality	Count Valuation	-312 (-584, -44) -$1,455,270,331 (-$5,706,124,288, $1,622,349,824)
Cardiac Hospital Admissions + Respiratory Hospital Admissions	Count Valuation	-293 (-630, 43) -$1,285,828 (-$2,997,135, $273,121)
Cardiac Emergency Room Visits + Respiratory Emergency Room Visits	Count Valuation	-1,397 (-3,124, 153) -$549,901 (-$1,309,949, $59,708)
All Endpoints	Count Valuation	Not applicable -$1,366,045,919 (-$5,622,451,200, $1,765,228,032)

Note: A negative value represents a negative effect to the society and should therefore be considered as an increase in cost or health impacts. A positive value represents a positive effect to the society and should therefore be considered as a reduction in cost or health impacts.

Table 4.9 O_3: AQBAT estimates from absence of air pollutants to baseline, CC4 and CC4b scenarios

	Baseline*	CC4*	CC4b*	Baseline to CC4 (% change)	Baseline to CC4b (% change)
Acute Exposure Mortality	-3,449	-3,616	-4,158	4.84	20.56
Acute Respiratory Symptom Days	-14,381,861	-15,019,056	-17,186,586	4.43	19.50
Asthma Symptom Days	-2,041,466	-2,143,840	-2,481,305	5.01	21.55
Minor Restricted Activity Days	-4,720,256	-4,971,734	-5,818,014	5.33	23.26
Respiratory Emergency Room Visits	-8,046	-8,441	-9,691	4.91	20.44
Acute Exposure Mortality + Chronic Exposure Mortality	-3,449	-3,616	-4,158	4.84	20.56
Cardiac Hospital Admissions + Respiratory Hospital Admissions	-1,973	-2,070	-2,376	4.92	20.43
Cardiac Emergency Room Visits + Respiratory Emergency Room Visits	-8,046	-8,441	-9,691	4.91	20.44

* Only average values are presented for simplicity.

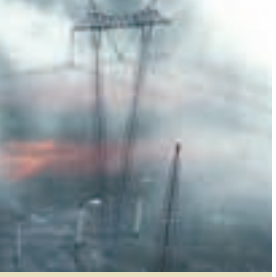

Table 4.10 $PM_{2.5}$: AQBAT estimates from absence of air pollutants to baseline, CC4 and CC4b scenarios

	Baseline*	CC4*	CC4b*	Baseline to CC4 (% change)	Baseline to CC4b (% change)
Acute Respiratory Symptom Days	-6,320,702	-5,343,659	-5,533,850	-15.46	-12.45
Adult Chronic Bronchitis Cases	-4,002	-3,321	-3,456	-17.02	-13.64
Asthma Symptom Days	-263,555	-221,890	-230,118	-15.81	-12.69
Cardiac Emergency Room Visits	-160	-135	-141	-15.63	-11.88
Cardiac Hospital Admissions	-441	-373	-386	-15.42	-12.47
Child Acute Bronchitis Episodes	-35,429	-28,669	-30,000	-19.08	-15.32
Chronic Exposure Mortality	-2,916	-2,451	-2,541	-15.95	-12.86
Respiratory Emergency Room Visits	-1,146	-970	-1,005	-15.36	-12.30
Restricted Activity Days	-4,102,799	-3,444,088	-3,574,568	-16.06	-12.87
Acute Exposure Mortality + Chronic Exposure Mortality	-2,916	-2,451	-2,541	-15.95	-12.86
Cardiac Hospital Admissions + Respiratory Hospital Admissions	-724	-613	-634	-15.33	-12.43
Cardiac Emergency Room Visits + Respiratory Emergency Room Visits	-1,306	-1,105	-1,145	-15.39	-12.33

* Only average values are presented for simplicity.

Table 4.11 Both pollutants: AQBAT estimates from absence of air pollutants to baseline, CC4 and CC4b scenarios

	Baseline*	CC4*	CC4b*	Baseline to CC4 (% change)	Baseline to CC4b (% change)
Acute Exposure Mortality	-3,449	-3,616	-4,158	4.84	20.56
Acute Respiratory Symptom Days	-20,702,562	-20,362,715	-22,720,437	-1.64	9.75
Adult Chronic Bronchitis Cases	-4,002	-3,321	-3,456	-17.02	-13.64
Asthma Symptom Days	-2,305,021	-2,365,730	-2,711,422	2.63	17.63
Cardiac Emergency Room Visits	-160	-135	-141	-15.63	-11.88
Cardiac Hospital Admissions	-441	-373	-386	-15.42	-12.47
Child Acute Bronchitis Episodes	-35,429	-28,669	-30,000	-19.08	-15.32
Chronic Exposure Mortality	-2,916	-2,451	-2,541	-15.95	-12.86
Minor Restricted Activity Days	-4,720,256	-4,971,734	-5,818,014	5.33	23.26
Respiratory Emergency Room Visits	-9,192	-9,410	-10,696	2.37	16.36

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	Baseline*	CC4*	CC4b*	Baseline to CC4 (% change)	Baseline to CC4b (% change)
Restricted Activity Days	-4,102,799	-3,444,088	-3,574,568	-16.06	-12.87
Acute Exposure Mortality + Chronic Exposure Mortality	-6,366	-6,068	-6,699	-4.68	5.23
Cardiac Hospital Admissions + Respiratory Hospital Admissions	-2,697	-2,682	-3,010	-0.56	11.61
Cardiac Emergency Room Visits + Respiratory Emergency Room Visits	-9,353	-9,546	-10,836	2.06	15.86

* Only average values are presented for simplicity.

Table 4.12 AQBAT valuation information

Endpoint	Sources of derivation	Form	Parameter 1	Parameter 2	Parameter 3
Acute Exposure Mortality	Jones-Lee et al., 1985; Cropper and Freeman, 1991; Rowe et al., 1995	Discrete	Low valuation= $2,637,960 (probability=33%)	Central valuation= $4,506,516 (probability=50%)	High valuation= $9,013,031 (probability=17%)
Acute Respiratory Symptom Days	Stieb et al., 2002	Normal	Mean valuation= $14.06	SD of valuation= $10.82	
Adult Chronic Bronchitis Cases	Viscusi et al., 1991; Krupnick and Cropper, 1992	Discrete	Low valuation= $192,351 (probability=33%)	Central valuation= $292,374 (probability=34%)	High valuation= $511,105 (probability=33%)
Asthma Symptom Days	Stieb et al., 2002	Triangular	Minimum valuation=$7.57	Most likely valuation=$30.29	Maximum valuation=$129.81
Cardiac Emergency Room Visits	Stieb et al., 2002	Normal	Mean valuation= $393.85	SD of valuation= $65.64	
Cardiac Hospital Admissions	Stieb et al., 2002	Normal	Mean valuation= $5,689	SD of valuation= $984.64	
Child Acute Bronchitis Episodes	Krupnick and Cropper, 1989	Discrete	Low valuation= $166.43 (probability=33%)	Central valuation= $343.96 (probability=34%)	High valuation= $510.39 (probability=33%)
Chronic Exposure Mortality	Jones-Lee et al., 1985; Cropper and Freeman, 1991; Rowe et al., 1995	Discrete	Low valuation= $2,637,960 (probability=33%)	Central valuation= $4,506,516 (probability=50%)	High valuation= $9,013,031 (probability=17%)
Minor Restricted Activity Days	Stieb et al., 2002	Discrete	Low valuation= $21.64 (probability=33%)	Central valuation= $35.70 (probability=34%)	High valuation= $61.66 (probability=33%)
Respiratory Emergency Room Visits	Stieb et al., 2002	Normal	Mean valuation= $393.85	SD of valuation= $65.64	
Respiratory Hospital Admissions	Stieb et al., 2002	Normal	Mean valuation= $4,595	SD of valuation= $656.42	
Restricted Activity Days	Stieb et al., 2002	Normal	Mean valuation= $51.93	SD of valuation= $27.04	

Note: SD, standard deviation.

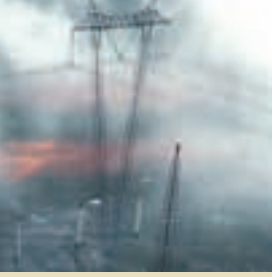

Table 4.13 AQBAT concentration response function

Source for derivation of CRF	Pollutant	Averaging period	Health endpoint	Specified population	Regression type	Baseline rate (events per quarter per million specified population)	Excess for a unit concentration increase	
							Mean	Standard error
Internal AHED sub-analysis based on R. Burnett's 1981–2000 data and methods	O_3	1 hour	Acute Exposure Mortality	Total population	Poisson	CD specific	0.127%	0.0212%
Krupnick, 1990	O_3	1 hour	Acute Respiratory Symptom Days	94% (non-asthmatics) of the total population	Linear	16,000,000	0.0786%	0.0386%
Stock et al., 1988; Whittemore and Korn, 1980	O_3	1 hour	Asthma Symptom Days	6% (asthmatics) of the total population	Logistic	12,000,000	0.173%	0.0552%
Ostro and Rothschild, 1989	O_3	1 hour	Minor Restricted Activity Days	94% (non-asthmatics) of the total population	Poisson	2,000,000	0.0530%	0.291%
Burnett et al., 1997; Stieb et al., 2000	O_3	1 hour	Respiratory Emergency Room Visits	Total population	Poisson	CD specific	0.0791%	0.0355%
Burnett et al., 1997	O_3	1 hour	Respiratory Hospital Admissions	Total population	Poisson	CD specific	0.0791%	0.0355%
Krupnick, 1990	$PM_{2.5}$	24 hours	Acute Respiratory Symptom Days	94% (non-asthmatics) of the total population	Linear	16,000,000	0.266%	0.139%
Abbey et al., 1995	$PM_{2.5}$	24 hours	Adult Chronic Bronchitis Cases	Population 25 years old and over	Logistic	1,600	1.33%	0.689%
Whittemore and Korn, 1980	$PM_{2.5}$	24 hours	Asthma Symptom Days	6% (asthmatics) of the total population	Logistic	12,000,000	0.144%	0.0559%
Burnett et al., 1995; Stieb et al., 2000	$PM_{2.5}$	24 hours	Cardiac Emergency Room Visits	Total population	Linear	CD specific	0.0711%	0.0170%
Burnett et al., 1995	$PM_{2.5}$	24 hours	Cardiac Hospital Admissions	Total population	Linear	CD specific	0.0711%	0.0170%
Dockery et al., 1996	$PM_{2.5}$	24 hours	Child Acute Bronchitis Episodes	Population under 20 years old	Logistic	16,000	2.76%	1.73%
Krewski et al., 2000	$PM_{2.5}$	24 hours	Chronic Exposure Mortality	Total population	Poisson	CD specific	0.678%	0.164%

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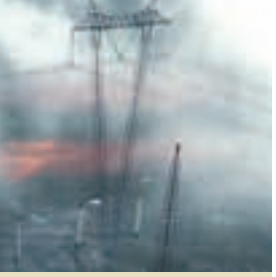
Source for derivation of CRF	Pollutant	Averaging period	Health endpoint	Specified population	Regression type	Baseline rate (events per quarter per million specified population)	Excess for a unit concentration increase	
							Mean	Standard error
Burnett et al., 1995	$PM_{2.5}$	24 hours	Respiratory Emergency Room Visits	Total population	Linear	CD specific	0.0754%	0.0132%
Burnett et al., 1995	$PM_{2.5}$	24 hours	Respiratory Hospital Admissions	Total population	Linear	CD specific	0.0754%	0.0132%
Ostro, 1987	$PM_{2.5}$	24 hours	Restricted Activity Days	94% (non-asthmatics) of the population 20 years old and over	Poisson	4,700,000	0.482%	0.101%

Note: CD, census division



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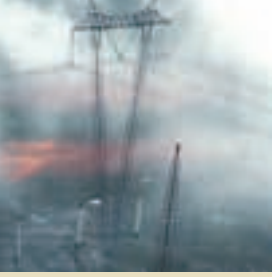

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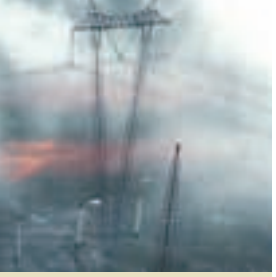

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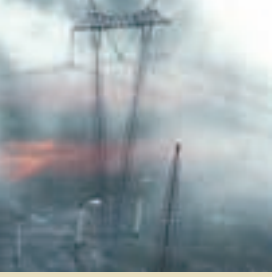

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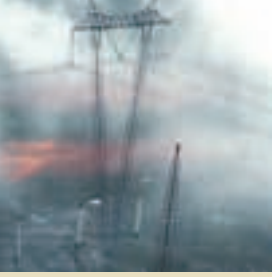

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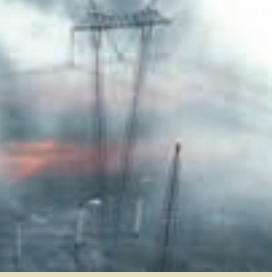

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