Final Report:

HEALTH IMPACTS EXPOSURE TO OUTDOOR AIR POLLUTION IN HAMILTON, ONTARIO

Prepared For:

Hamilton Public Health Services
Clean Air Hamilton

Prepared By:

SENES Consultants Limited

February 2012

CleanAir HAMILTON

SENES Consultants Limited

Specialists in Energy, Nuclear and Environmental Sciences

FINAL

HEALTH IMPACTS OF EXPOSURE TO OUTDOOR AIR POLLUTION IN HAMILTON, ONTARIO

Prepared for:

Hamilton Public Health Services
Clean Air Hamilton

Prepared by:

SENES Consultants Limited
121 Granton Drive, Unit 12
Richmond Hill, Ontario
L4B 3N4

February 2012

EXECUTIVE SUMMARY

Air quality in Hamilton is a significant concern for public health. Clean Air Hamilton (CAH) has been working with the City of Hamilton to improve air quality and the health of Hamilton's citizens for over twelve years with significant strides being made in reducing air emissions, while also integrating innovative methods of monitoring and reporting local air quality data.

A health assessment of air quality was included in the 1997 Hamilton Air Quality Initiative (HAQI) Report. The last Air Quality Health Assessment study was published in 2003 with annual air pollutant data included up to 1999 (Sahsuvaroglu and Jerrett, 2003). Since then, there have been substantial improvements in air quality within Hamilton, creating a need to update the earlier work. Although different and more detailed analyses could be conducted with the data with more sophisticated methodologies, this study was meant to be an update and comparison to the 2003 study and hence the methodology of the Sahsuvaroglu and Jerrett (2003) study was followed. The current study does however, considers more recent air pollutant concentration levels, as well as updated health data obtained from the literature.

The health outcomes in this report were calculated using measured air quality data and observed base mortality and morbidity rates from Hamilton as well as excess relative risks for various air quality pollutants obtained from the literature. Revised relative health risks were identified and used to determine health outcomes from year to year. Consequently, temporal variations in health outcomes are due to the combined changes in air quality data, excess relative risks for each air pollutants and changes to observed base event rates for Hamilton.

The air pollutants that were evaluated in this study were fine particulate matter (PM_{10} and $PM_{2.5}$), nitrogen dioxide, sulphur dioxide, ozone and carbon monoxide. While NO is also a pollutant associated with vehicular and industrial emissions, it was evaluated as NO_2 since it is rapidly converted in the atmosphere to NO_2 and also since epidemiology and health studies focus on NO_2. The air quality data used in the assessment were considered representative of typical exposures in the community; however it is generally recognized that residents living in close proximity to major roadways may be exposed to higher levels of some pollutants than those who live further from roadways.

Overall, the average measured air quality for the Hamilton region has improved during the study period with the exception of ozone concentrations, which have increased. Ozone concentrations may have increased due to trans-boundary pollution. In addition, the health data indicate that the total non-traumatic mortality and total respiratory hospital admissions have remained relatively constant over the study period while total cardiovascular hospital admissions have decreased since approximately 2001.

Based on these general trends, the results of this study demonstrate that the health outcomes associated with airborne pollutants have improved or remained constant over the study period with the exception of those associated with ozone levels. The calculated numbers of events for non-traumatic acute mortality has changed from 229 in the 2003 study to 186 in this study. Hospital admissions associated with respiratory health effects have remained largely unchanged (407 in 2003 to 395 in 2008). The largest calculated decrease was seen in hospital admissions due to cardiovascular effects where the numbers decreased from 1239 to 322. From these results it can be determined that there are clear health benefits associated with the declining air pollutant concentrations in Hamilton.

Calculations were also carried out with the Ontario Medical Association's Illness Cost of Air Pollution (ICAP) model (available in CAH Annual Reports), as well as with the Health Canada's Air Quality Benefits Assessment Tool (AQBAT) model. These two models are commonly used to determine the burden of illness associated with air pollution and are discussed in more detail in the report. It should be noted that different inputs are used with the two models, making it difficult to make meaningful comparisons between the outputs of the models.

Cost benefits associated with the improvements in air quality in Hamilton were calculated using both the ICAP and AQBAT models. The ICAP model was restricted to costs associated with ozone; whereas the AQBAT model calculated the cost benefits associated with the decreasing concentrations of fine particulate matter, nitrogen dioxide, sulphur dioxide and carbon monoxide. These cost benefits outweighed the costs associated with the increased concentrations of ozone.

When interpreting the results of the study, it should be noted that the study was an extension to the Sahsuvaroglu and Jerrett (2003) work and thus the focus was on relative risks of acute exposures and thus health outcomes associated with chronic exposure to air pollutants were not evaluated. There may be the possibility that some health outcomes are double-counted by using separate relative risk values for each pollutant; however efforts were made to ensure as many studies as possible were based on multi-pollutant models to minimize these influences. Finally, while the results of this report indicate that air quality improvements can result in lower health outcomes, the equation used to assess this relationship is very linear and does not take spatial variation into account such as distance to major roadways or industries. There are studies that indicate increased adverse effects associated with people living closer to major roadways and highways. Thus, it is important for policy and decision makers to consider some of issues when looking at outcomes associated with air pollutants.

Overall, decreasing levels of air pollutants in Hamilton have resulted in a decreased health burden on the general population. In the future, further reductions in pollutant levels will lead to further decreased health burdens on the citizens of Hamilton.

TABLE OF CONTENTS

LIST OF TABLES

Page No.

LIST OF FIGURES

Page No.

LIST OF FIGURES (Cont'd)

Page No.

GLOSSARY

Acute effects	An adverse health effect that is caused suddenly, rapidly or within a short timeframe after exposure.
Asthma	A common disorder in which chronic inflammation of the bronchial tubes (bronchi) makes them swell, narrowing the airways.
Atherosclerosis	A stage of arteriosclerosis involving fatty deposits (atheromas) inside the arterial walls, thus narrowing the arteries.
Bronchii	The large air tubes leading from the trachea to the lungs that convey air to and from the lungs.
Cancer	An abnormal growth of cells which tend to grow in an uncontrolled way and, in some cases, to metastasize (spread).
Cardiac arrest	The stopping of the heartbeat.
Cardiopulmonary	Of, relating to, or involving the heart and the lungs.
Cardiovascular	Of, relating to, or involving the heart and the blood vessels.
Carotid intima-media thickness	A measure of the thickness of artery walls. It is often used a surrogate endpoint for evaluating the progression of atherosclerotic cardiovascular disease.
Chronic effects	An adverse health effect that is caused after prolonged or repeated instances of exposure over a longer timeframe.
Epidemiology	Branch of medicine that deals with the study of the causes, distribution, and control of disease in populations.
Exposure	Contact between environmental releases or contaminants and local populations.
Health endpoints	Disease symptoms or deaths - generally used to describe a health effect (or a probability of that health effect) resulting from exposure to a particular source.
Interquartile range (IQR)	The difference between the value of a variable below which lie 25 per cent of the population, and that below which lie 75 per cent: a measure of the spread of the distribution.
Ischemic heart disease	Disease characterized by ischaemia (reduced blood supply) of the heart muscle.
Ischemic stroke	Stroke caused by an interruption in the flow of blood to the brain.
Morbidity	A measure of illnesses within a geographic area (can be a numerical count or a calculated rate).
Mortality	A measure of deaths within a geographic area (can be a numerical count or a calculated rate).
Myocardial infarction	Heart attack, results from the interruption of blood supply to a part of the heart, causing heart cells to die.
Non-traumatic mortality	Death not causing, caused by, or associated with trauma and especially traumatic injury.

Pulmonary	Having to do with the lungs
Relative coefficient/risk	In this context, a risk coefficient is a relative risk factor (in units of excess relative risk per unit change in air quality) which is applied to baseline risks.
Respiratory	Of, relating to, used in, or affecting respiration

1.0 INTRODUCTION

Air quality in Hamilton is of significant concern to public health. To this end, Clean Air Hamilton has been working with the City of Hamilton to improve air quality and the health of Hamilton's citizens for over twelve years with significant strides being made in reducing air emissions, while also integrating innovative methods of monitoring and reporting local air quality data.

A health assessment of air quality was included in the 1997 Hamilton Air Quality Initiative (HAQI) Report for Clean Air Hamilton. The last Air Quality Health Assessment study was published in 2003 with annual air pollutant data included up to 1999 (Sahsuvaroglu and Jerrett, 2003). Since then, there have been substantial improvements in air quality within Hamilton, creating a need to update the earlier work. The current report updates the public health impact of exposure using the more recent air pollutant concentration levels, as well as updated health data obtained from the literature. This study was funded by Hamilton Public Health Services (HPHS), and managed through Clean Air Hamilton (CAH) with the City of Hamilton.

This report updates the public health impact of exposure using the methodology from Sahsuvaroglu and Jerrett (2003) with recent air pollutant concentration levels, as well as updated health data. A monetary value of these air quality improvements was also assigned using models which relate air quality and the associated costs of health impacts. This report provides an assessment of changes in health effects due to air pollution exposure over time. It is anticipated that quantification of the health and cost implications of reducing air pollution in the City will provide support for improving Hamilton's profile in terms of air quality in a positive way.

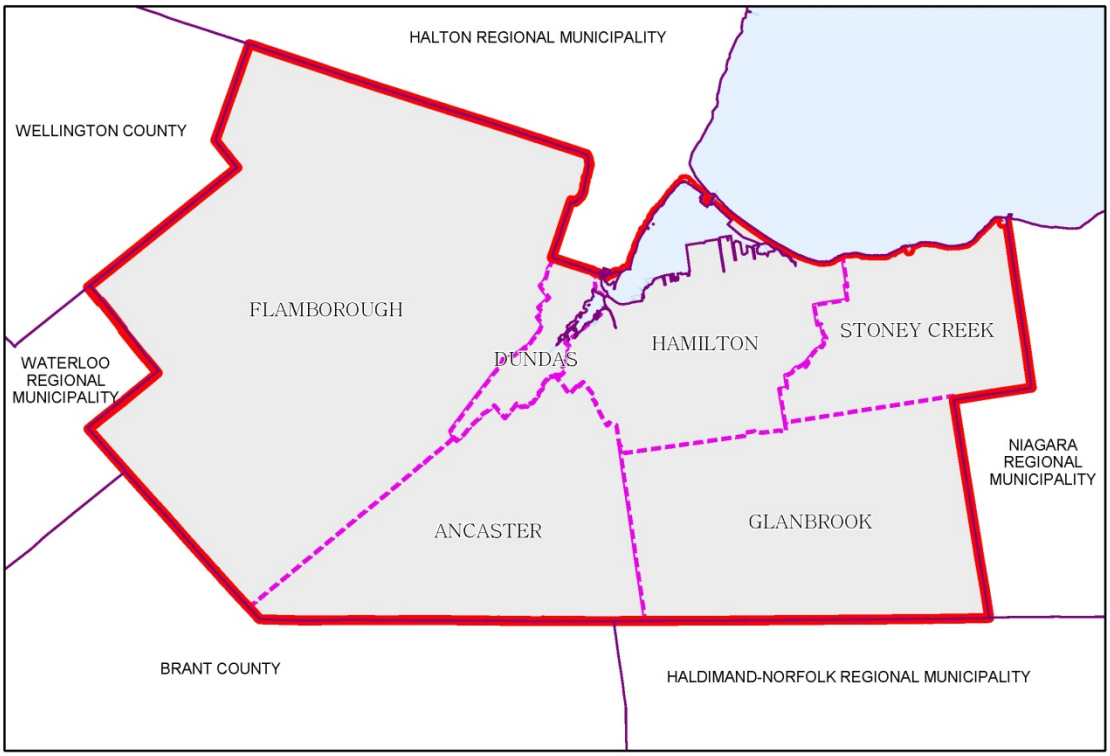
To conduct this analysis, a literature review was conducted to update the health information provided in Sahsuvaroglu and Jerrett (2003); as part of this task, the health risk coefficients[1] to be utilized in the current analysis were identified (Section 2). Second, additional data was required for the assessment of health impacts following the approach of Sahsuvaroglu and Jerrett (2003); baseline risks of mortality in exposed populations were identified, assessed and prepared for analysis. The analysis described in this report involves the calculation of the air quality related-health effects based on the available health care and air pollution data from Hamilton. These updated estimates of potential health impacts were compared to information provided in Sahsuvaroglu and Jerrett (2003).

Health data were made available from Hamilton Public Health Services including mortality data for 1996 to 2005 and morbidity data from 1997 to 2008. Air quality data was obtained from the Ontario Ministry of Environment (MOE) and encompassed 1997 to 2009. The amount of data

[1] In this context, a risk coefficient is actually a relative risk factor (in units of excess relative risk per unit change in air quality) which is applied to baseline risks.

available was dependant on the pollutant measured and is discussed in more detail in Section 3. The geographic boundary of the City of Hamilton for both sets of data is outlined in the red heavy line in Figure 1-1.

Figure 1-1 City of Hamilton Boundaries



Source: City of Hamilton, 2011

As a sensitivity analysis, as well as to assign ("crude") monetary value to the estimated health impacts, Health Canada's Air Quality Benefits Assessment Tool (AQBAT) model and Canadian Medical Association's Illness Costs of Air Pollution (ICAP) model were utilized.

The results provided in this report are intended for wide dissemination, including a presentation to Clean Air Hamilton, and the 2012 Upwind/Downwind Conference.

2.0 LITERATURE REVIEW

There were three areas of focus for the literature review. The first area involved the investigation of research conducted on air quality improvements and consequent potential health impacts, in order to provide a review of current methodologies to assess these changes. Where applicable, research was directed first on Canadian studies, then North American, European and finally other international locations.

The second area of focus was to provide key updates on the relative risks identified in the previously published study (Sahsuvaroglu and Jerrett 2003). An extensive literature review of the topic of air quality and health impacts was not conducted, as other reviews have reported on these matters in significant detail (U.S.EPA, 2009, WHO, 2005, etc.). Professional judgement was used to extract the most relevant and applicable information from published literature review work, specifically for use in the assessment of mortality and morbidity attributable to air pollution exposure in Hamilton, Ontario. It is important to note that the identification of potentially significant relative risks inherently causes a selection bias in the results, which could result in an overestimation of the true risk associated with air pollution. Within this section, a detailed review is provided for particulate matter, as this has been recognized as a key research area and an area of concern.

The third focus of the literature review was to provide a summary of information on some specific issues of concern for Hamilton: health impacts of exposures to polycyclic aromatic hydrocarbons and dioxins/furans, mobile monitoring and proximity to roadways.

2.1 AIR QUALITY IMPROVEMENTS AND HEALTH

Epidemiological research studies on air pollution and health impacts have largely focused on the adverse health outcomes that arise from exposure to increasing concentrations of air pollutants. In many countries, air pollution levels have been declining due to advances in pollution control technologies and adherence to air pollution guidelines, policies and regulations identified and enforced by government. Consequently there is a growing body of research that now seeks to identify the potential consequent improvements in health outcomes that may result from decreases in air pollution concentrations, despite the difficulty in directly identifying these effects (Samet and Krewski, 2007).

Several cohort studies that followed up study participants have given researchers an opportunity to assess health impacts with the improvements of air quality levels over extended periods of time. In the Harvard Six Cities Study (Dockery *et al.*, 1993), reductions in air pollution concentrations were observed between the 1970s and the late 1980s. In the follow up analysis conducted by Laden *et al.*, (2006) with an extended cohort, decreased mortality was associated

with lower $PM_{2.5}$ concentrations. Similarly, in the American Cancer Society's (ACS) cohort that was also followed up over time, researchers found evidence suggesting a decrease in the relative risk of mortality associated with a reduction in $PM_{2.5}$ (Pope *et al.* 2002).

Other studies have assessed whether improvements in air quality can impact life expectancy. In fact, life expectancy was shown to increase by 0.61 (+/- 0.20 SE) years for a decrease in 10 $\mu g/m^3$ of fine particulate matter in the United States (Pope *et al.* 2009). The study used data from 51 U.S metropolitan areas from the late 1970s to early 2000's. The researchers also estimated that reductions in air pollution concentrations may have accounted for as much as 15% of life expectancy increases in the study area, indicating that measurable and significant improvements in life expectancy are possible with reductions in ambient fine particulate air pollution.

There are additional types of studies that have looked into assessing the effectiveness of air quality interventions that result in air quality improvements. These studies primarily address specific situations, interventions or "natural experiments" where measurable improvements in air quality were observed and health effects were assessed. For example, during the Atlanta Olympic Games (Friedman *et al.* 2001) traffic reduction measures were implemented resulting in lower air quality concentrations (e.g., peak ozone was reduced by 28%). Researchers found that emergency visits for asthma events in children decreased 42% during that time period while emergency visits rates for other causes did not change.

In another "natural experiment," as a result of the ban on the use of coal for domestic heating in Dublin, Ireland, concentrations of black smoke and sulphur dioxide had decreased by 35.6% and 11.3% respectively, 6 years following the ban. The researchers calculated a decrease in age-standardized non-traumatic deaths of 5.7%, resulting in 287 fewer deaths in Dublin (Clancy *et al.* 2002). Cardiovascular death rates contributed approximately 45% of the non-trauma deaths, and respiratory deaths accounted for 15% (Clancy *et al.* 2002). Also, as a result of a 13 month strike at the Utah Valley steel mill, PM_{10} concentrations in the Valley dropped approximately 15 $\mu g/m^3$, and the total death rates over that time were calculated to be reduced by 3.2% (Pope, 1989).

In Hong Kong, the sulphur content of fuel oil for power generation and road transport was reduced to 5% or less by weight in 1990 over a very distinct timeline (Hedley *et al.* 2002). Substantial reductions in SO_2 concentrations were observed, as much as 80% in some areas, while airborne sulphate concentrations fell by 38%. It was estimated that as a result, average life expectancy per year increased by 20 days for women, and 41 days for men (Hedley *et al.* 2002).

Mindell and Joffee (2004) assessed the health impacts of achieving local air quality objectives in the UK. The researchers estimated that if the 2004 24 h PM_{10} (38.5 $\mu g/m^3$) objective at the local

roadside monitoring data was attained, then there would be approximately 1-21 fewer annual deaths, depending on which study the exposure-response coefficients were drawn from. Alternatively, the researchers estimated that attaining the 2009 PM_{10} annual mean objective of 15.4 $\mu g/m^3$ would result in a decrease of between 8-20 deaths (Mindell and Joffe 2004). With respect to morbidity, attaining the 2009 annual objective would result in a reduction of approximately 20 respiratory and 14-20 circulatory hospital admissions, and lead to a 5% reduction in emergency visits for asthma (Mindell and Joffe 2004).

Researchers have also conducted biological tests which confirm health effects resulting from exposure to air pollution. Neuberger and colleagues examined standard spirometry test results of over 3,000 Austrian school children over a period of 5 years, in two distinct areas: which either experienced unchanged NO_2 or decreasing NO_2 levels (Neuberger *et al.* 2002). Researchers found that airway dysfunction indicators such as MEF25 (maximum expiratory flow rate at 25% vital capacity) had the most marked improvements in areas with declining NO_2 levels. This finding is important as it highlights that health effects can be reversed if air quality is improved. In a study of ambient air pollution levels and respiratory health of Swiss children by Bayer-Oglesby *et al.* (2005), an association was found between the decline in air pollution levels between 1993 and 2000 and an improvement in respiratory health in children. Between that period, the average decrease of PM_{10} was 9.8 $\mu g/m^3$ (12.7 $\mu g/m^3$ in urban areas and 4.0 $\mu g/m^3$ in rural areas); and between 1992-1993 and 1998-2001 nonallergic related health issues including chronic cough, bronchitis, common cold, nocturnal dry cough, and conjunctivitis symptoms decreased 4.5-8.9% on average (Bayer-Oglesby *et al*. 2005).

Another approach to assessing the impacts of air quality improvements have been conducted through cost benefit studies, where monetary benefits of reducing adverse health effects have been calculated. For example, Wong *et al*. (2003) predicted that, due to the forecasted reduction in criteria air pollutants in the United States from 1990 to 2010, the following reductions associated with child health would occur:

- 10,000 fewer asthma hospital admissions in children 1-16 years old, with estimated benefits ranging from $20 million to $46 million (1990 U.S.$);
- 40,000 fewer emergency department visits in children 1-16, with estimated benefits ranging from $1.3 to $5.8 million;
- 20 million school absences avoided by children 6-11 years old, with estimated benefits of $0.7-1.8 billion; and
- 10,000 fewer infants of low birth weight, with estimated benefits of $230 million.

In another study, an analysis of the effects of the U.S Clean Air Act (from 1970-1990) showed that monetary benefits ($22,171 billion) of the Act outweighed the monetary costs ($523 billion) associated with the implementation of the Act (Samet and Krewski 2007). Over 4/5 of these benefits would be derived from avoided mortality (@ $4.6 million per life).

Thus, there are multiple methods that researchers have employed that identify health benefits as a result of improved air quality: the extended cohort analyses, life expectancy calculations, biological assessments, specific interventions or natural experiments resulting in improved air quality and cost-benefit analysis.

2.2 IDENTIFICATION OF RISK COEFFICIENTS

To identify the health risk coefficients in the literature (i.e., dose-response relationships as a relative risk), a specific literature search was conducted. The literature review was initially conducted using the MEDLINE and PubMed search engines. The search began using the key word combinations of "air pollution" with health effects, mortality, morbidity, health effects for all publications since 2002. Using similar criteria to the 2003 study for consistency, articles were reviewed and subsequently selected based on relevance and suitability of exposure and outcome measurements.

To allow for identification of risk coefficients in the literature, articles were excluded if:

- Study health outcomes were not related specifically to non-traumatic mortality or hospital admissions (morbidity);
- Studies were focused on indoor air pollutants and/or tobacco smoke;
- Studies specifically considered children or elderly or susceptible populations;
- Studies did not provide numerical estimates for the effects of air pollution (e.g., a correlation coefficient should have been identified for the association between air pollution and health); and
- Studies were based on advancing methodological techniques and used either simulated or existing data.

Articles were categorized by study type (acute effects and chronic effects) and results were summarized by pollutant. Multi-pollutant models that provide maximum control for co-pollutants were also highlighted. Additional articles were identified either through published reviews or reference lists included in key articles to provide a more complete summary.

All identified studies that provided usable regression estimates were summarized, with a number of items identified, such as location, year, regression coefficients, standard errors and other study details. In order to make the regression estimates comparable, all results were converted to a standard metric: percentage change of health outcome associated with an increase of 10 $\mu g/m^3$ or 10 ppb of each pollutant (1ppm increase in the case of CO).

Because of the general health outcome categories that have been used historically for this study, many articles could not be used, primarily because the health outcomes were too specific (e.g.,

particular ICD-9/10 codes), and in some cases the age groups were not generally applicable (e.g., significant only for >65 years old). It is important to note that only the more significant results are included in the summary tables quantifying the impacts; inherently, selection bias is introduced to this analysis. Recommendations for future updates of this study could include the identification of specific illnesses of most importance to a specific community. Appendix A contains tables summarizing the results of the literature review.

Particulate Matter (PM)

The U.S. EPA (2009) conducted detailed reviews of the literature associated with exposure to air particulate matter. Including available evidence from atmospheric chemistry and exposure assessment studies enabled them to also develop causal determinations for a variety of health outcome categories. Table 2-1 summarizes the U.S. EPA (2009) causality determinations for the health effects that can result from inhalation exposure to PM_{10} and $PM_{2.5}$.

The U.S. EPA (2009) indicated that epidemiological, controlled human exposure and animal toxicological studies provide only suggestive evidence for relationships between short-term exposure to PM_{10} and cardiovascular effects, respiratory effects, and increased mortality. However, they observed that short-term epidemiologic studies do consistently report positive associations between short-term exposure to PM_{10} and cardiovascular outcomes that are similar in magnitude to those observed in $PM_{2.5}$ studies. With respect to respiratory health effects due to short-term exposure to PM_{10}, there is limited evidence of a relationship.

Another key observation is the considerable uncertainty that surrounds PM_{10} concentrations. One reason for this may be that less robust monitoring data are often available, relative to $PM_{2.5}$, and greater potential for errors in estimating ambient exposures to PM_{10}, relative to $PM_{2.5}$. In addition, there is high variability in the chemical and biological composition of PM_{10}.

Table 2-1 U.S. EPA (2009) Causality Determinations for Health Effects Associated with Inhalation Exposure to PM_{10} and $PM_{2.5}$

Health Outcome	Causality Determination*
PM_{10}	
Effects of Short-Term Exposure	
Cardiovascular Effects	Suggestive
Respiratory Effects	Suggestive
Mortality	Suggestive
$PM_{2.5}$	
Effects of Short-Term Exposure	
Cardiovascular Effects	Causal
Respiratory Effects	Likely to be causal
Mortality	Causal
Effects of Long-term Exposure	
Cardiovascular Effects	Causal
Respiratory Effects	Likely to be causal
Mortality	Causal
Reproductive and Developmental Effects	Suggestive
Cancer, Mutagenicity, and Genotoxicity	Suggestive

Note:
CAUSAL RELATIONSHIP
Evidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures. That is, the pollutant has been shown to result in health effects in studies in which chance, bias, and confounding could be ruled out with reasonable confidence.

LIKELY TO BE A CAUSAL RELATIONSHIP
Evidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures, but important uncertainties remain. That is, the pollutant has been shown to result in health effects in studies in which chance and bias can be ruled out with reasonable confidence but potential issues remain.

SUGGESTIVE OF A CAUSAL RELATIONSHIP
Evidence is suggestive of a causal relationship with relevant pollutant exposures, but is limited because chance, bias and confounding cannot be ruled out.

As seen from the above table, causal effects related to fine particulate matter relate to primarily to $PM_{2.5}$ and not PM_{10} exposure and the focus on health effects in the last few years has been on $PM_{2.5}$.

The following articles provided relative risks (RR) that were used in the current study.

PM_{10}

Bell *et al*, (2004) reviewed the epidemiological literature on time series analyses and summarized the findings from several key studies:

- The National Morbidity Mortality Air Pollution Study (NMMAPS) was designed specifically to address the effects of biases due to city selection and publication as well as to determine the influence of co-pollutants. An average RR of 0.21% increase in non-traumatic mortality for an increase in 10 $\mu g/m^3$ in PM_{10} was identified.

- The 'Air Pollution and Health: A European Approach' or APHEA was a large-scale research project in Europe. Higher RRs were identified in Central Eastern Europe (0.8%) than in Western Europe (0.4%). An extension of the original APHEA study, termed APHEA-2, investigated up to 29 cities and determined that PM was significantly associated with daily mortality counts (Katsouyanni *et al*., 2003, Analitis *et al*., 2006). The RR was calculated at 0.6% for an increase in 10 $\mu g/m^3$ in PM_{10}.

Dominici *et al*. (2005) reanalyzed air quality and health data for 90 cities utilizing the NMMAPS database and reported associations between PM_{10} and mortality to account for the calculation limitation identified in the generalized additive model (GAM) used for the initial evaluations. Results for the original study resulted in a RR estimate of 0.41% for a 10 $\mu g/m^3$ in PM_{10}, with reanalysis adjusting the value further to 0.27% and 0.21%.

Samoli *et al.* (2008) report results from the APHENA study (Air Pollution and Health: A Combined European and North American Approach). This study combined the data from the European APHEA (Air Pollution on Health: a European Approach) [22 cities] and the U.S. NMMAPS (National Morbidity, Mortality and Air Pollution Study) [90 cities] with data from 12 Canadian cities) for a total of 124 cities. This approach allowed use of common protocols in multicity studies to address possible heterogeneity and consistency issues. Across all cities, an increase of 10 $\mu g/m^3$ of PM_{10} resulted in a 0.2-0.6% increase in all-cause mortality, across all ages. Risk estimates for Canadian cities (0.84%) were more than twice as high as those for Europe (0.33%) and the United States (0.29%). The reasoning for higher estimates in the Canadian data was suggested to be either due to more accurate exposure and outcome data, or PM_{10} acting as a surrogate of true causal pollutants, or that that the association between toxic components and PM_{10} in Canada truly differs. The observed differences could not be attributed to methodological differences, as consistent analysis was used. In addition, the differences could also not be assigned to any specific source mix differences as these were not observed.

Zanobetti *et al.* (2002) investigated mortality displacement in air pollution and health effects. Using data from 10 cities within the APHEA-2 study network, the researchers assessed dependency of daily deaths to PM_{10} on that day and up to the previous 40 days. The meta-analysis for all cities averaged results from the same day and the previous day impacts (time lags of 0 and 1days) and found a 0.7% increase in mortality for a 10 $\mu g/m^3$ in PM_{10}. The paper also found that although exposure in the first week (mostly the first two days) before the mortality event had a strong impact, the exposure in the preceding month increased the estimate of the overall effect substantially (approximately two-fold). This is consistent with results from cohort studies that generally have higher risk estimates than time series studies. Zanobetti *et al.* (2002) suggest while short-term time series studies capture very early deaths, there are still public health effects that occur over a longer period of time. In terms of a public health impact, it suggests

that short-term associations likely *underestimate* the mortality impacts and that cohort studies are capable of a more accurate assessment.

Recent analyses done by Larrieu *et al.* (2007) in 8 French cities reported significant increases in cardiovascular hospital admissions associated with increases in PM_{10} exposure (0.2% for 10 µg/m^3 increase).

The following box provides a summary of the studies that were considered in deriving updated relative risks for non-traumatic mortality and cardiovascular hospital admissions associated with PM_{10} that were used in this study.

Identified Relative Risks for PM_{10}

Non-traumatic mortality	
0.21% (95%CI: 0.04-0.33) for 10µg/m^3	Bell *et al*., (2004)
0.41% (95%CI: 0.2-0.6) for 10µg/m^3	Bell *et al*., (2004)
0.80% (95%CI: 0.06-1. 8) for 10µg/m^3	Bell *et al*., (2004)
0.6% (95%CI: 0.4 -0.8) for 10µg/m^3	Bell *et al*., (2004)
0.2% - 0.6% for 10µg/m^3	Brook *et al*., (2010)
0.41% (95%CI: 0.35-0. 47) for 10µg/m^3	Dominici *et al*., (2005)
0.27% (95%CI: 0.21-0.33) for 10µg/m^3	Dominici *et al*., (2005)
0.21% (95%CI: 0.15-0.27) for 10µg/m^3	Dominici *et al*., (2005)
0.29% (95%CI: 0.18-0.4) for 10µg/m^3	Samoli *et al*., (2008)
0.84% (95%CI: 0.3-1.04) for 10µg/m^3	Samoli *et al*., (2008)
0.33% (95%CI: 0.22-0.44) for 10µg/m^3	Samoli *et al*., (2008)
0.7% (95% CI: 0.43-0.97) for 10µg/m^3	Zanobetti *et al*., (2002)
Cardiovascular hospital admissions	
0.2% (95%CI: 0.1-1.2) for 10 µg/m^3	Larrieu *et al*., (2007)

$PM_{2.5}$

As seen from Table 2-1, the U.S. EPA (2009) concluded that short-term exposure to $PM_{2.5}$ is positively associated with a broad range of respiratory and cardiovascular effects, as well as mortality. For cardiovascular observations, experimental findings of health effects were identified by the U.S.EPA, such as increased emergency department visits/hospital admissions and atherosclerosis, among others. For respiratory effects, while some findings are consistent (e.g., increased emergency department visits and hospital admissions, alterations in lung function and other respiratory symptoms in asthmatic children) there is still some uncertainty for most of these endpoints. Finally, short and long-term epidemiologic studies have both reported evidence for increased hospital admissions for respiratory infections in response to $PM_{2.5}$ exposures,

which may result from exposure making the individuals more susceptible to respiratory infections. However, no risk values have been identified.

The American Heart Association has recently updated its assessment for risks of cardiovascular health impacts associated with short-term exposure to $PM_{2.5}$ (Brook *et al.,* 2010). Time-series studies estimate that a 10 $\mu g/m^3$ increase in mean 24 hour $PM_{2.5}$ concentration increases the relative risk (RR) for daily non-traumatic mortality by approximately 0.2% to 0.6%. Brook *et al.,* (2010) also note that a 10 $\mu g/m^3$ increase during the preceding day contributes, on average, to the premature death of approximately one susceptible person per day in a region of 5 million people (based on annual U.S. death rates in 2005).

The Harvard Six Cities Study (Dockery *et al*., 1993) and the ACS study (Pope *et al*., 1995) were pioneering long-term studies which demonstrated that there was evidence linking $PM_{2.5}$ exposure to adverse cardiovascular events. Extended analysis of the original Harvard Six Cities Study (Leden *et al*., 2006) found PM to be strongly associated with cardiovascular mortality. Schwartz *et al.* (2008), in a more recent analysis of the Harvard Six Cities Study noted that health effects due to changes in exposure were observable within 2 years.

Similarly, extended analyses of the original ACS study (Pope *et al.,* 2002; and Pope *et al*., 2004) reported $PM_{2.5}$ exposure to be strongly associated with a host of adverse cardiovascular events, including ischemic heart disease, dysrhythmia, cardiac arrest and heart failure. Socioeconomic and/or demographic factors were also shown to correlate with variations to PM exposure across and within cities, in an extended analysis of the ACS study (Jerrett *et al*., 2003; Jerrett *et al*., 2004). Jerrett *et al*. (2005) also analyzed a subcohort of the ACS sample from the metropolitan Los Angeles area and reported strong $PM_{2.5}$-mortality effects, specifically due to ischemic heart disease.

Schwartz (2001) analyzed data from the Third National Health and Nutrition Examination survey and observed PM_{10} exposures to be associated with cardiovascular risk indicators, specifically elevated levels of fibrinogen, platelets and white blood cells. Souza *et al.* (1998) analyzed lung tissue samples of corpses from Sao Paolo, Brazil and report that individuals who lived in polluted areas had evidence of chronic inflammatory lung injury when compared to individuals who lived in cleaner areas. Kunzli *et al.* (2005) examined data from 798 patients from the metropolitan Los Angeles area and report strong links between $PM_{2.5}$ exposure and increase in carotid intima-media thickness, an indicator of subclinical atherosclerosis.

Short-term $PM_{2.5}$ exposure has also been associated with ischemic heart disease and elevated risk of myocardial infarction (MI). Peters *et al.* (2001) examined 772 patients (with MI) in the Boston area and reported increased risk of MI as a result of short-term $PM_{2.5}$ exposure. A similar study of 691 patients with MI in Southern Germany also observed adverse $PM_{2.5}$-MI effects

(Peters *et al.*, 2004). A large-scale study across 21 cities in the U.S. examined over 300,000 MI events and reported that short-term PM_{10} exposure increased the risk of MI (Zanobetti *et al.*, 2005; von Klot *et al*., 2005). D'Ippoliti *et al*. (2003) also found adverse $PM_{2.5}$-MI effects for a sample from Rome, Italy, but a few studies also showed inconsistent PM-MI associations, including a study from the state of Washington (Sullivan *et al.*, 2005).

Many of these studies, though important to the literature and status of science, could not be included in identifying the RRs for this study because they were either specific to age groups or to health outcomes. The studies included are identified below.

Bell *et al.* (2004) summarized results from time-series studies that estimate health effects resulting from short-term exposures to particulate matter. The key findings summarized the large multicity studies, namely the American Cancer Society cohort and the Harvard Six Cities studies: 0.4% increase and 0.1% increase, respectively for an increase of 10 $\mu g/m^3$ in fine particulate matter. In the AHA's update, Brook *et al* (2010) reported world-wide averages of 0.4 to 1% increase in daily mortality for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ within 5 days of exposure. Specifically for fine particulates and all-cause non-traumatic mortality, Brunekreef *et al* (2005) reported a 0.3-2.9% increase in mortality per 10 $\mu g/m^3$ increase in fine particulates. Two other studies were also included. Ostro *et al*. (2006) and Zanobetti and Schwartz (2009) who reported 0.1% and 0.98% increase in mortality (respectively) for a 10 $\mu g/m^3$ increase in fine particulates.

Bell and colleagues (2008) and Peng and colleagues (2008) in the U.S.EPA MCAPS study evaluated the association between $PM_{2.5}$ and the risk of cardiovascular hospital admissions between 1999 and 2005 in 202 U.S. countries and determined a 0.8% (CI 0.6% to 1%) increase in risk for a 10 $\mu g/m^3$ increase in $PM_{2.5}$.

The following box provides a summary of the studies that were considered in deriving updated relative risks for non-traumatic mortality and cardiovascular hospital admissions associated with $PM_{2.5}$ that were used in this study.

Identified Relative Risks for $PM_{2.5}$

Non-traumatic mortality	
0.1% - 0.4% for 10$\mu g/m^3$	Bell *et al*., (2004)
0.4% - 1.0% for 10$\mu g/m^3$	Brook *et al*., (2010)
0.3% -2.9% for 10$\mu g/m^3$	Dominici *et al*., (2005)
0.1% (95%CI: 0.2-1.0) for 10$\mu g/m^3$	Ostro *et al*., (2006)
0.98% (95% CI: 0.75-1.22) for 10$\mu g/m^3$	Zanobetti and Schwartz (2009)
Cardiovascular hospitalization	
0.1% (95%CI: 0.2-1.0) for 10$\mu g/m^3$	Bell *et al*., (2008); Peng *et al* (2008)

Nitrogen Oxides

The two most prevalent oxides of nitrogen are nitrogen dioxide (NO_2) and nitric oxide (NO). However, NO is rapidly oxidized by ozone to NO_2 and therefore the epidemiological studies have focussed on the health effects of NO_2 as opposed to NO.

The U.S. EPA (2008) summarized the extensive literature regarding the health effects resulting from exposure to nitrogen oxides (primarily NO_2). Epidemiological studies in both short and long term exposure assessments have investigated mortality (Villeneuve *et al*., 2003 and Beelen *et al*., 2008 respectively), hospital admissions (Larrieu *et al*, 2007) and emergency department visits (e.g., Szyszkowicz, 2009). Recent research has also shown associations with susceptible or vulnerable populations such as children (Jerrett *et al*., 2008).

Villeneuve *et al*. (2003) assessed non-traumatic mortality in Vancouver, Canada and found a 3.5% increase associated with a 17.5 ppb increase in NO_2. In the meta-analysis conducted by Stieb *et al*. (2002), an increase of 24 ppb in NO_2 was significantly associated with a 2.8% increase in non-traumatic mortality rates. Long term exposure assessment projects also found a significant association (e.g., Beelen *et al*., 2008 and Filleul *et al*., 2005), indicating that chronic impacts were also important to consider. Other mortality estimates were specific to particular health outcomes, such as ischemic stroke (Hong *et al*., 2002), fatal case of myocardial infarction (Rosenlund *et al*., 2009) and lung cancer (Beelen *et al*., 2008).

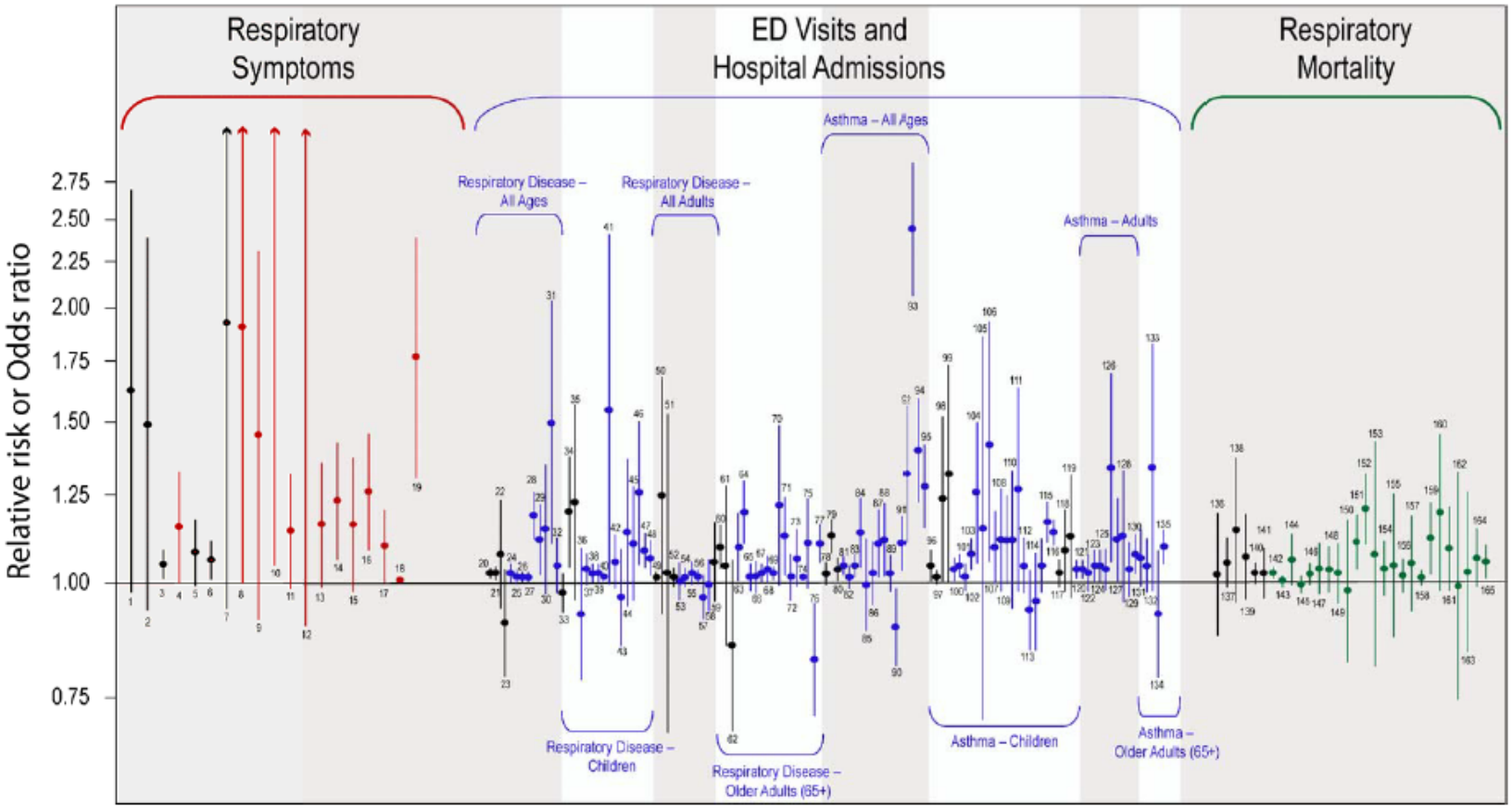
Exposure to NO_2 and morbidity were assessed for ischemic heart disease admissions in Montreal and Edmonton by Szyszkowicz (2007 and 2008). Both health endpoints were significantly correlated to increases in NO_2 concentrations. Admissions to ERs for chest pain were also related to increases in NO_2 concentrations in 6 Canadian cities (Szyszkowicz, 2009). Larrieu *et al*. (2007) assessed overall cardiovascular hospital admissions with NO_2 exposure, identifying a 0.53% increase in admissions for an increase of 5.3 ppb in NO_2. Figure 2-1 provides results of studies summarized specifically for respiratory outcomes. Generally, a significant association is observed for specific health endpoints across these studies.

For this study, the following box indicates the relative risks utilized. The Villeneuve (2003) study was chosen as Canadian content, and the Stieb (2002) study was chosen as it provided a meta-analysis of a significant component of literature. Larrieu (2007) was identified as one of the only studies providing associations to cardiovascular admissions that could be compared to the previous study.

Identified Relative Risks for NO_2

Non-traumatic mortality	
0.2% (95%CI: 0.45-3.6) for 10 ppb	Villeneuve *et al*., (2003)
1.16% (95% CI: 0.88-1.46) for 10 ppb	Steib *et al*., (2002)
Cardiovascular admissions	
0.94% (95% CI: 0.19-1.89) for 10 ppb	Larrieu *et al*., (2007)

Figure 2-1 Summary of Epidemiological Studies Examining Short-term Exposure to Ambient NO_2 and Respiratory Outcomes



Notes: Effect estimates for studies conducted in Canada or the U.S. are in black. Other colours represent other countries. Circles represent effect estimates. Lines represent 95% CI.

Source: U.S. EPA (2008).

Ozone

In 2009, the U.S. EPA published a review of literature describing health impacts associated with exposure to ozone (O_3) (USEPA, 2009). The following were identified as key summaries about the strength of the association:

- Positive association between increasing ambient O_3 concentrations and excess risk for non-accidental and cardiopulmonary-related daily mortality, with some seasonal variation existing during summer months.
- Clear evidence of causality for the associations observed during acute ($\leq$ 24 h) O_3 exposure.
- Limited body of evidence in the literature suggesting that O3 directly and/or indirectly contributes to cardiovascular-related morbidity.

One recent study that examined long-term exposure to O_3 reported a positive association between ambient O_3 concentration and respiratory causes of death, and this association remained after controlling for $PM_{2.5}$ using co-pollutant models (Jerrett *et al.,* 2009). In another recent study Krewski *et al*., (2009) observed a positive association between summer O_3 exposure and all cause non-traumatic as well as cardiopulmonary disease mortality. This association was robust to control for ecologic variables. No association with mortality was found when examining year-round O_3 exposure.

Two Canadian studies assessed morbidity impacts due to O_3 exposure. Cakmak *et al.* (2006) examined the relationship between O_3 exposure and hospital admissions for cardiac disease in 10 large Canadian cities. Overall the authors found a positive association, although the association varied between cities, with some cities showing no association. Seasonal variation in the association was not assessed. Szyszkowicz (2008) found a positive association between emergency department visits for acute ischemic stroke and one day lagged O_3 concentration among men aged 20-64 years during the warm season, in Edmonton, Canada. A similar association was not seen for men or women aged 65 and older. Also, no association was seen with same-day O_3 levels.

Several studies were averaged with respect to O_3 exposure and mortality to identify the relative risks utilized in this paper. Janke *et al*. (2009) identified impacts in a multipollutant study within the UK. Bell et al (2004) reviewed data from 95 US urban communities, and found that an increase of 10 ppb resulted in an increase of 0.52% in non-traumatic mortality. In a later meta-analysis study of 39 different time-series studies, Bell *et al*. (2005) identified a 0.87% increase over the same pollutant concentrations. Ito *et al*. (2005) identified a range of associations, depending on assumptions taken. Finally, Stieb *et al.* (2002) conducted a meta-analysis and identified a 0.5% increase in non-traumatic mortality as a result of a 10 ppb increase in O_3. For

cardiovascular morbidity, Larrieu *et al*. (2007) identified a 0.2% increase associated to a 10 ppb increase in O_3 within the general population.

Identified Relative Risks for ozone

Non-traumatic mortality	
1.57% (95%CI: 1.0-2.14) for 10ppb	Janke *et al*., (2009)
0.52% (95%CI: 0.27-0.77) for 10ppb	Bell *et al*., (2004)
0.87% (95%CI: 0.55-1.18) for 10ppb	Bell *et al*., (2005)
0.39% (95%CI: 0.26-0.51) for 10ppb	Ito *et al*., (2005)
0.37% (95%CI: 0.2-0.54) for 10ppb	Ito *et al*., (2005)
0.8% (95%CI: 0.55-1.0) for 10ppb	Ito *et al*., (2005)
0.5% (95% CI: 0.32-0.38) for 10ppb	Steib *et al.,* (2002)
Cardiovascular admissions	
0.2% for 10ppb	Larrieu *et al*., (2007)

Sulphur dioxide

Ballester *et al.* (2002) investigated the relationship between particulates and SO_2 in 13 Spanish cities as part of the EMECAM project studying the effects of short term effects of air pollution on mortality. For an increase in 10µg/m^3 for SO_2 in a two pollutant model, a 0.2 % in non-traumatic mortality was observed (95% CI: 0.09-0.7%) using a 24 hour mean value of SO_2. Stieb *et al*. (2002) conducted a meta-analysis of time-series analysis on air pollutants and health. An increase in 9.4 ppb SO_2 resulted in a 0.9% increase in all-cause mortality.

With respect to cardiovascular morbidity, Fung *et al.* (2005) assessed air quality impacts on Windsor residents in Ontario, Canada. An increase of 2.6% was observed for cardiac hospital admission rates for adults over the age of 65 (95% CI: 0.5-6.4), calculated for an interquartile range (IQR) increase of 19.3 ppb. Across seven Canadian cities, Stieb *et al.* (2009) identified an increase of 2.1% (95% CI: 0.2-4.0) in emergency department admissions for angina and myocardial infarctions for an increase of 5.1 ppb of SO_2. In Europe, Sunyer *et al.* (2003) assessed the relationship between daily SO_2 levels and cardiovascular hospital admissions in several European studies. Significant associations were observed for admissions specifically for ischemic heart disease in adults over 65 years of age: 1.2% increase for 10µg/m^3 (95% CI: 0.8-1.6).

Associations were also observed for respiratory morbidity indicators resulting from exposure to SO_2. For examples, Wilson *et al.* (2005) found that an interquartile (IQR) increase of 6.3 ppb in SO_2 was associated with a 5% (95% CI: 2–7%) increase in all respiratory admissions and a 6%

(95% CI: 1–12%) increase in asthma visits. Some studies do suggest a temporal association where higher impacts are observed over warmer seasons (Villeneuve *et al.*, 2003).

Identified Relative Risks for SO_2

Non-traumatic mortality	
0.36% (95% CI: 0.28-0.48) for 10ppb	Stieb *et al.* 2002
Respiratory admissions	
3% (95% CI: 1–5%) for 10ppb	Wilson *et al*. 2005

Carbon monoxide

Carbon monoxide was shown to have a significant association with non-traumatic mortality in Stieb *et al.* (2002) meta-analysis assessment. An increase in 1.1 ppm CO led to a 1.7% increase non-traumatic mortality (95% CI: 1.2-2.2). In Korea, Hong *et al.* (2002) identified a 6% increase per 0.7 ppm increase in CO (95% CI: 2-9%) for deaths resulting from ischemic stroke. Long-term studies, such as Rosenlund *et al.* (2009), also found associations between CO and cardiac-related deaths, specifically that an IQR increase resulted in an increase of 1.14% of fatal myocardial infarction rates.

Morbidity assessments were conducted in various cities in Canada found associations between exposure to CO and ER admissions for ischemic heart disease (IHD) (Szyszkowicz, 2008), angina/MC infarctions and heart failure (Stieb *et al.*, 2009) and ER admissions for chest pains (Szyszkowicz, 2009). Villeneuve *et al.* (2006) identified a seasonal association between exposure to CO and both acute ischemic stroke and hemorrhagic stroke, with impacts observed significantly over the summer season. Associations between general respiratory morbidity impacts and exposure to CO were not identified.

Identified Relative Risks for CO

Non-traumatic mortality	
1.7% (95% CI: 1.08-1.98) for an increase in 10ppb	Stieb *et al.* 2002

2.3 BENZO(A)PYRENE AND DIOXINS AND FURANS

The following paragraphs provide some pertinent information on polycyclic aromatic hydrocarbons such as benzo(a)pyrene and dioxins and furans based on material primarily that reported by the U.S. Agency for Toxic Substances and Disease Registry (ATSDR 1995).

Benzo(a)pyrene and other Carcinogenic PAHs

Polycyclic aromatic hydrocarbons (PAHs) are a large group of chemicals that are formed during the incomplete burning of carbonaceous substances like coal, oil and gas (e.g., automobile exhaust) or tobacco. PAHs are usually found as a mixture containing two or more of these compounds. One of the most common and more carcinogenic PAHs is benzo(a)pyrene.

Background levels of PAHs in the air are reported to be in the 0.15 to 19.3 ng/m^3 range in North American urban areas while levels of PAHs in urban air may be 10 times greater than those found in rural areas. PAH levels in Europe and Asia are much greater than in North America. PAHs are found in tobacco smoke, smoke from wood burning stoves and fireplaces, and in some foods such as barbequed foods.

The International Agency for Research on Cancer (IARC 2010) has determined that some PAHs are probably carcinogenic to humans. Occupational health studies have shown that people who have breathed mixtures of PAHs for long periods of time have developed lung cancer.

While there is sufficient information available in the scientific literature to relate exposure to a potential effect, so that a risk assessment could be conducted; risk assessments only provide estimates of theoretical increases in incidence of cancer from exposure to contaminants which cannot be directly translated into health outcomes and burdens on the hospitals in the municipalities. Therefore, no quantitative health outcomes are presented for exposures to PAHs.

Dioxins and Furans

Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDF), commonly known as dioxins and furans, are a group of similar structured compounds that are toxic, persistent and bioaccumulative. One of these compounds, 2,3,7,8-tetrachlorodibenzo-p-dioxin or 2,3,7,8-TCDD, is one of the most toxic dioxins and has received the most attention. Other compounds with similar toxic properties are called “dioxin-like” compounds.

Currently, dioxins and furans are primarily released to the environment during combustion of fossil fuels (coal, oil, and natural gas) and wood, and during incineration processes (municipal, medical waste and hazardous waste incineration). Smoking can also contribute to an individual’s

exposure. Typical levels of dioxins in outdoor air in urban areas averaged about 2.3 pg/m³ which is about 1000 times lower than measured PAH levels. It is noted that breathing dioxins and furans in air is a minor route of exposure for most people with food intake being the primary route of exposure.

The International Agency for Research on Cancer (IARC 1997) has determined that 2,3,7,8-TCDD can cause cancer in people. After breathing dioxins and furans, the contaminants can enter your body through your lungs and pass into the blood stream. Exposure to dioxins and furans over a long time has been shown to lead to cancer of the liver and thyroid, and other types of cancer.

While there is sufficient information available in the scientific literature to relate exposure to a potential effect so that a risk assessment could be conducted; risk assessments only provide estimates of theoretical increases in incidence of cancer from exposure to contaminants which cannot be directly translated into health outcomes and burdens on the hospitals in the municipalities. Therefore, no quantitative health outcomes are presented for exposures to dioxins and furans.

2.4 MOBILE AIR QUALITY MONITORING

Vehicles and industry are a major source of emissions of air pollutants and there have been studies that have looked at the effect of these sources on air pollutant concentrations. The studies focussed on fine particles, nitrogen oxides and sulphur dioxide. As indicated in Section 2.2, nitrogen dioxide is the form of nitrogen dioxides that is associated with health effects due to the fact that NO reacts very quickly in the atmosphere to form NO_2.

Wallace *et al.,* (2009) reported the results of oxides of nitrogen (NO_x) representing traffic sources, and sulfur dioxide (SO_2) representing industry sources, from mobile air quality surveys conducted in Hamilton since 2005. Very high levels of NO_x (>600ppb) were measured near major highways. Similar observations were made for SO_2 levels near industrial sources.

Close proximity to roads has shown to be a significant factor in health impacts. Therefore, assessing the pollution levels in these areas is important. While personal monitoring can provide the most direct and accurate measure of human exposures, they are often cumbersome and exorbitantly expensive. Thus, mobile monitoring has started to afford a more accurate, real-time measurement option in air pollution level exposure assessment.

The Hamilton Mobile Monitoring Surveys identified concentrations of NO_x that pose significant concerns for individuals spending considerable time in traffic, especially due to congestion. Residential areas have relatively low levels of ambient pollutant concentrations, but NO_x levels

rise sharply on arterial roads with highest concentrations measured on highways with frequent heavy duty truck traffic. In Hamilton the industrial sector is often thought to contribute to the majority of air pollution within the city. However, the mobile monitoring survey identified that the highest concentrations and exposures to residents are due to vehicle emissions instead (Rotek Environmental, 2011).

Additional findings from the mobile monitoring study include high levels of vehicle exhausts when idling at red lights as well as while dropping off or picking up children from school. Further, the effects of wind direction with respect to highways can create localized traffic pollution concentrations.

2.4.1 Proximity to Roadways

The key findings from the Health Effects Institute (HEI, 2010) special report on traffic-related air pollution included the observation that zones most impacted by traffic-related pollution range from between up to 300 to 500 metres from highways and major roads. In North America, this accounts for approximately 30-45% of the population. For example, Beckerman *et al*. (2008) measured ultrafine particulates (UFP) and NO_2 levels both upwind and downwind of Highway 401 in Toronto to assess the distance decay of pollutants by distance. They found that UFP and NO_2 decreased to background within 300–500m on the downwind side of highway, while on the upwind side UFP and NO_2 levels decreased to background within 100–200m of the highway.

In the Children's Health Study in southern California; a significant association was found between asthma history and distance to freeway (OR 1.89, 1.19-3.02) and model-based freeway pollution in particular NO_2 (OR 2.22, 1.36-3.63) (Gauderman *et al.*, 2005). In the Cincinnati Allergy and Air Pollution Study cohort, GIS and traffic classification was used to categorize traffic exposures based on type, traffic volume and distance from road. Significant increases in prevalence of wheeze in infants living very near (<100 m) stop-and-go bus and truck traffic were observed (Ryan *et al*, 2005). Current research indicates that exposure to pollution may start causing effects in utero. For example, Brauer *et al.,* (2006) compiled residential histories for over 70,000 mothers reporting singleton births in Vancouver, Canada between 1999 and 2002 and found that residence within 50 m of highways was associated with a 26% increase in small for gestational births [95% CI, 1.07–1.49] and an 11% (95% CI, 1.01–1.23) increase in low full-term birth weights. Adults have also shown to be affected by proximity to roadways. In U.S. male veterans in south-east Massachusetts, persistent wheeze increased in men living within 50 m of major roadway, compared with those living >400 m away (Garshick *et al.*, 2003).

2.5 SUMMARY OF RELATIVE RISK VALUES USED IN THIS ASSESSMENT

The following table summarizes the relative risk values that were used in this assessment. These values were derived from the relative risks provided in the summary boxes in the previous section. When there was one or more study identified, an average of the relative risks was used to capture the relative risks in all studies. This is an uncertainty in the derivation of the relative risk and may result in an underestimate or overestimate of health outcomes; however it is not an unreasonable nominal estimate of the relative risk values. Where there were no updates of relative risks, the relative risks from Sahsuvaroglu and Jerrett (2003) were used as they were considered to be valid. There are no data to determine relative risks for respiratory hospital admissions for $PM_{2.5}$ and CO.

Air Pollutant	NT Mortality (changes per 10 pollution units)	Respiratory Hospital Admissions (changes per 10 pollution units)	Cardiovascular Hospital Admissions (changes per 10 pollution units)
$PM_{2.5}$(µg/m^3)	0.77	-	0.8
PM_{10} (µg/m^3)	0.45	2.1*	0.7
SO_2 (ppb)	0.36	3.0	1.1*
NO_2 (ppb)	0.68	4.9*	0.94
CO (ppm)	1.7	-	1.95*
O_3 (ppb)	0.72	2.8*	0.2

Note: - no data available in the literature to determine a relative risk.

* - relative risks obtained from Sahsuvaroglu and Jerrett (2003) as no new data available.

In order to make the regression estimates comparable, all results were converted to a standard metric: percentage change of health outcome associated with an increase of 10 µg/m^3 or 10 ppb of each pollutant (1ppm increase in the case of CO).

3.0 DATA AND METHODS

3.1 HEALTH DATA

Under specific terms and conditions of a data sharing agreement with the Ontario Ministry of Health and Long-term Care, the City of Hamilton Public Health Services has access to mortality and hospital discharge data via the IntelliHEALTH system[2]. The data were provided solely for the purposes of this Air Quality and Health Impact Study, and was were in accordance with the conditions included in Appendix B in order to satisfy the requirements outlined in the agreement. The geographic boundary of the data is shown in Figure 1-1.

Mortality Data

Mortality data describe the main/primary causes of death indicated by data from death certificates from the Ontario Office of the Registrar General. Geographic information is based on place of residence from the municipality on the death certificate, not where the death occurred. City of Hamilton residents who died anywhere in Ontario are included in the data provided. The data presented are based on underlying cause of death (i.e. the disease or injury which initiated the events leading directly to death), classified by the codes of the International Statistical Classification of Diseases and Health Related Problems 9th and 10th Revisions (see ICD-9 to ICD-10 Conversion paragraph for more information). Mortality data are available for calendar years 1986-2005.

Hospital Discharge Data

Hospital discharge data describe the main causes of hospital admission indicated by acute care inpatient discharges and excluded birth-related discharges of newborn and stillborn infants. Counts do not represent individuals but counts of discharge records. Some individuals are admitted to hospital several times for the same condition and some conditions are more likely to have multiple hospital admittance compared to others. Geographic information is based on patient's place of residence from the reported municipality at the time of admission. City of Hamilton residents hospitalized in the province of Ontario are included. Data are based on most responsible diagnosis (i.e. the diagnosis with the longest duration of treatment), classified by the codes of the International Statistical Classification of Diseases and Health Related Problems 9th and 10th Revisions (see ICD-9 to ICD-10 Conversion paragraph for more information). Hospital admission data is available for calendar years 1997-2008.

Hospital Admission Code Conversion (ICD-9 to ICD-10)

ICD codes are revised periodically to reflect new medical information. The 10th Revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-10)

[2] IntelliHealth is a knowledge repository that contains clinical and administrative data collected from various sectors of the Ontario healthcare system.

represents a considerable change from the 9th revision (ICD-9). Hospital admission data for the fiscal years 1996 to 2001 are coded based on ICD-9 and data from 2002 to 2008 are coded based on ICD-10. Hospital admission data presented for the calendar year 2002 contain both ICD-9 and ICD-10 codes. Mortality data for the calendar years 1986 to 1999 are coded based on ICD-9 and data from 2000 to 2005 are coded based on ICD-10. Changes between revisions may impact the comparability of hospital admission and mortality data based on different ICD versions if significant differences in the way the data are coded were made. However, this did not cause any challenges in this study, as the general categories of health outcomes were very similar.

ICD-9 and ICD-10 Codes

Table 3-1 describes the codes used to extract the mortality and hospital admission data.

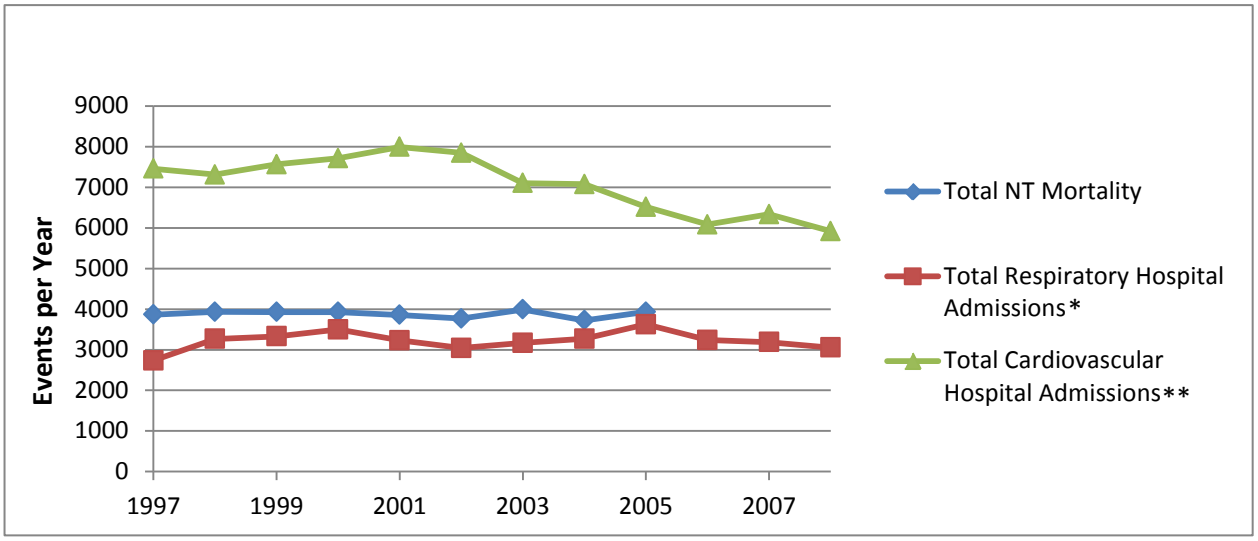
Table 3-1 ICD-9 and corresponding ICD-10 Codes

Mortality Data	**ICD-9**	**ICD-10**
All Non-Traumatic (NT) mortality	001-799	A00-R999
Disease of the Circulatory System	390-459	I00-I999
Disease of the Respiratory System	460-519	J00-J999
Hospital admission Data	**ICD-9**	**ICD-10**
All Non-Traumatic (NT) morbidity	001-799	A00-R999
Disease of the Circulatory System	390-459	I00-I999
Congestive Heart Failure	428	I500
Disease of the Respiratory System	460-519	J00-J999

Data were available by age and gender. For consistency with the previous reports, total annual values were utilized. The mortality and morbidity data are summarized in Figure 3-1 from 1997 to 2008. It should be noted that the mortality data are only available up to 2005 whereas the morbidity data is available up until 2008. Appendix C provides a summary of these data.

As seen from the figure, non-traumatic mortality (NT mortality) values are relatively constant. There appears to be a slight decrease in the total numbers of respiratory hospital admissions and a larger decrease in the number of cardiovascular hospital admissions in Hamilton.

Figure 3-1 Mortality and Morbidity Rates for Hamilton



* Approximated by Acute Care Hospital Discharges for Disease of the Respiratory System
** Approximated by Total Acute Care Hospital Discharges for Disease of the Circulatory System

3.2 AIR QUALITY DATA

The City of Hamilton provided the air quality data set for this study. Hamilton air trends were provided from the data from the MOE's local air monitoring network stations (Frank Dobroff, 2011, personal communication). The data provided were from the sites most relevant for an air pollution exposure assessment and health study. Air monitoring stations in Hamilton (i.e., stations in the Hamilton Air Monitoring Network) which are used primarily for air quality monitoring within an industrial area were not used as they do not accurately represent general population exposure. Appendix C provides a summary of these data.

Figure 3-2 provides a map of the monitoring stations where data were obtained.

The annual pollutant concentrations from this data set are summarized in the following figures (Figures 3-3 to 3-8). As seen from these figures, the measured air concentrations in Hamilton for most pollutants, while quite varied from year to year, have generally decreased over time with the exception of ozone which has increased.

Air concentrations for $PM_{2.5}$, PM_{10}, NO_2 and SO_2 are slightly higher at station 29000 – located in Hamilton Downtown than at the other stations. This difference is likely due to the proximity of this station to higher car and truck traffic, as well as heavy industry. Ozone, however, is largely formed from VOCs and NO_x compounds in the presence of sunlight, and takes some time to form and may arise from transport outside of the region, primarily from the Ohio Valley Area.

Consequently, the highest ozone measurements are observed at station 29114 – Hamilton Mountain.

Figure 3-2 Air Quality Monitoring Locations

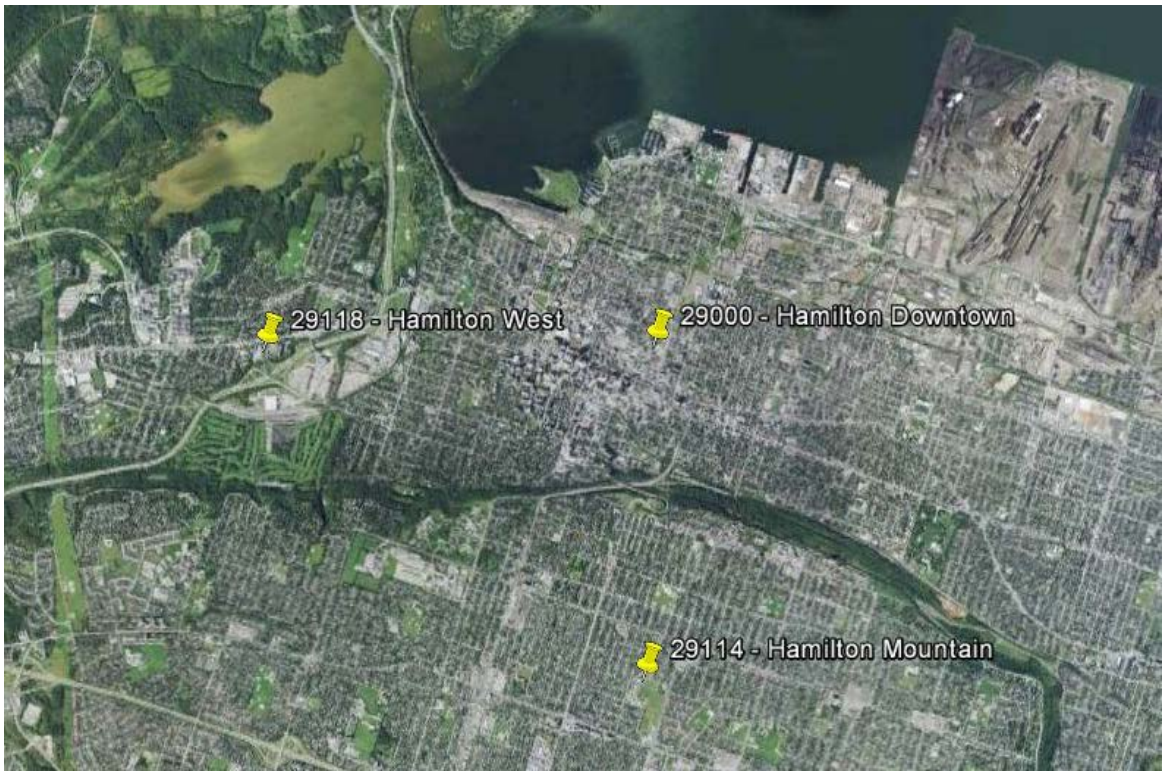


Figure 3-3 Summary of Air Quality Data for Fine Particulate Matter - $PM_{2.5}$

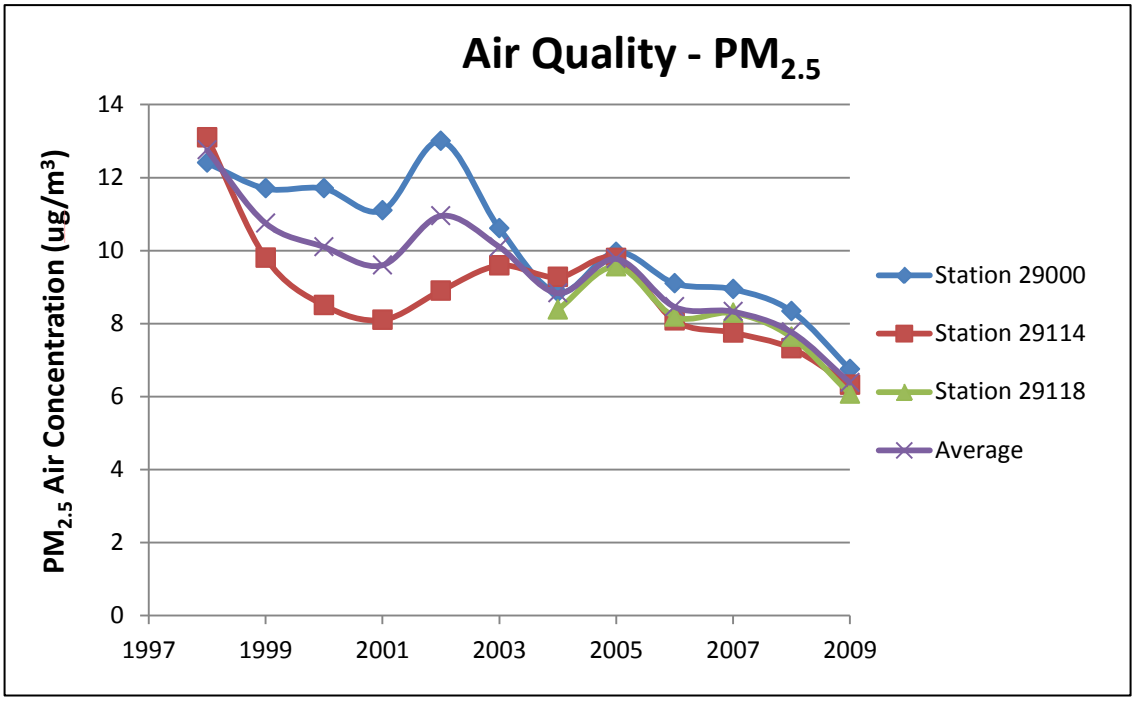


Figure 3-4 Summary of Air Quality Data for Fine Particulate Matter - PM_{10}

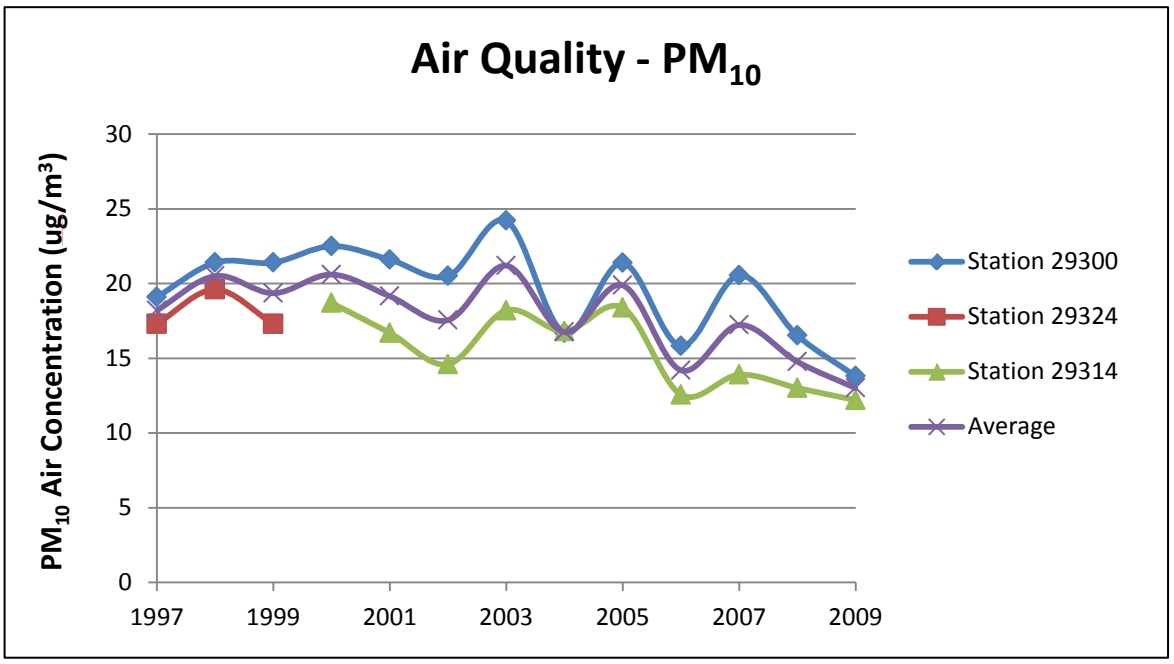


Figure 3-5 Summary of Air Quality Data for Nitrogen Dioxide - NO_2

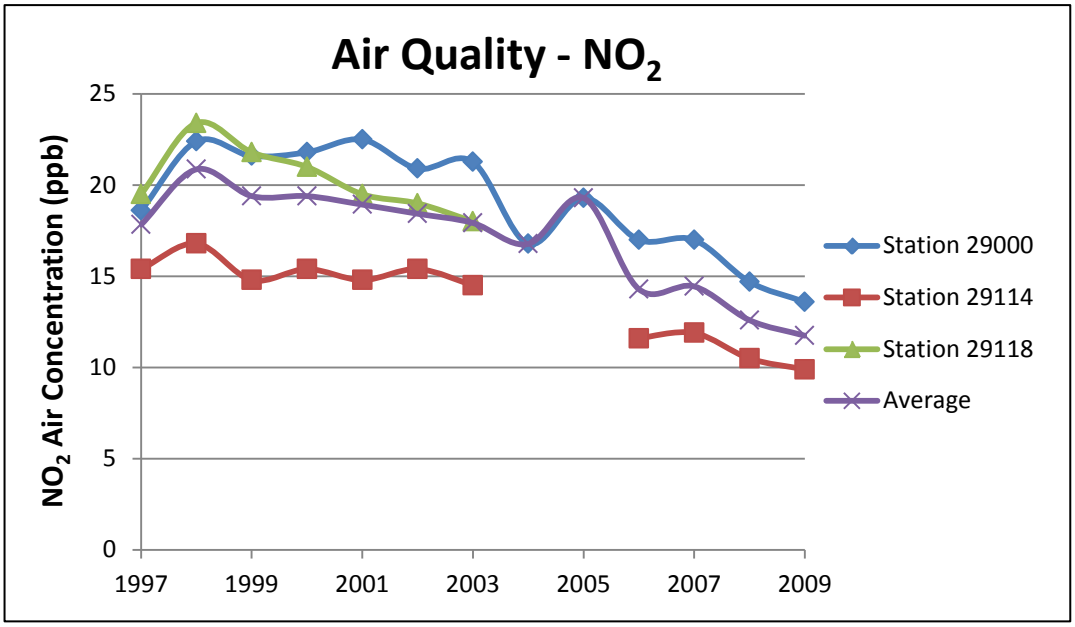


Figure 3-6 Summary of Air Quality Data for Sulphur Dioxide - SO_2

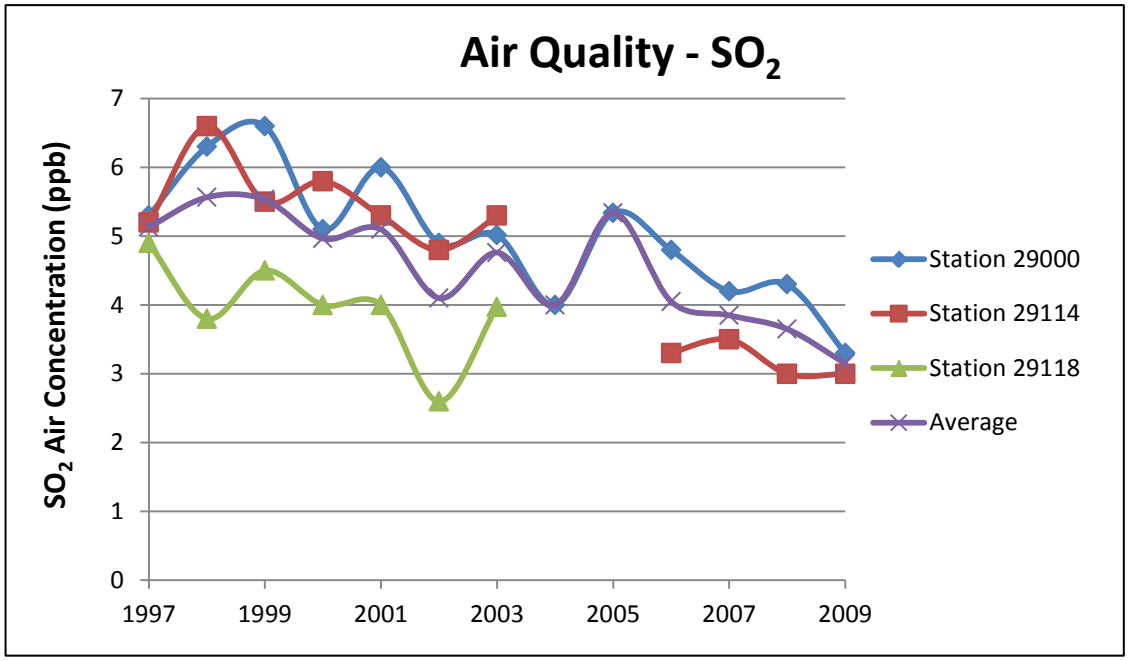


Figure 3-7 Summary of Air Quality Data for Ozone - O_3

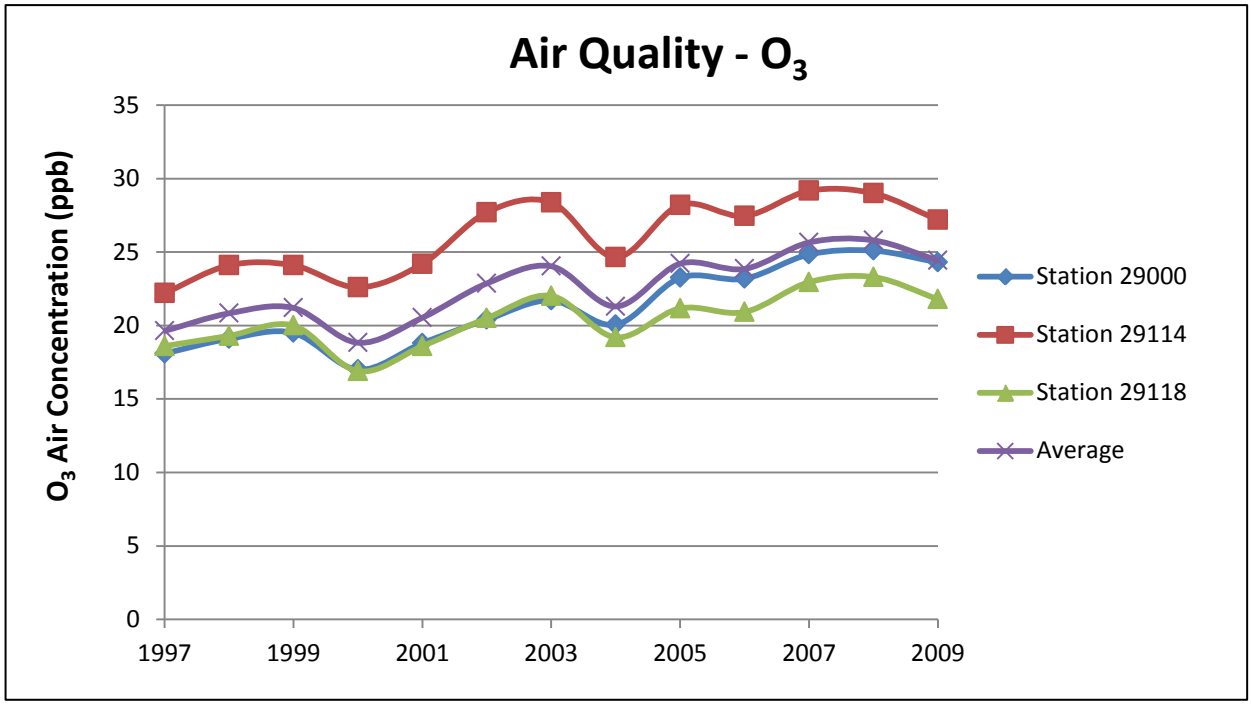
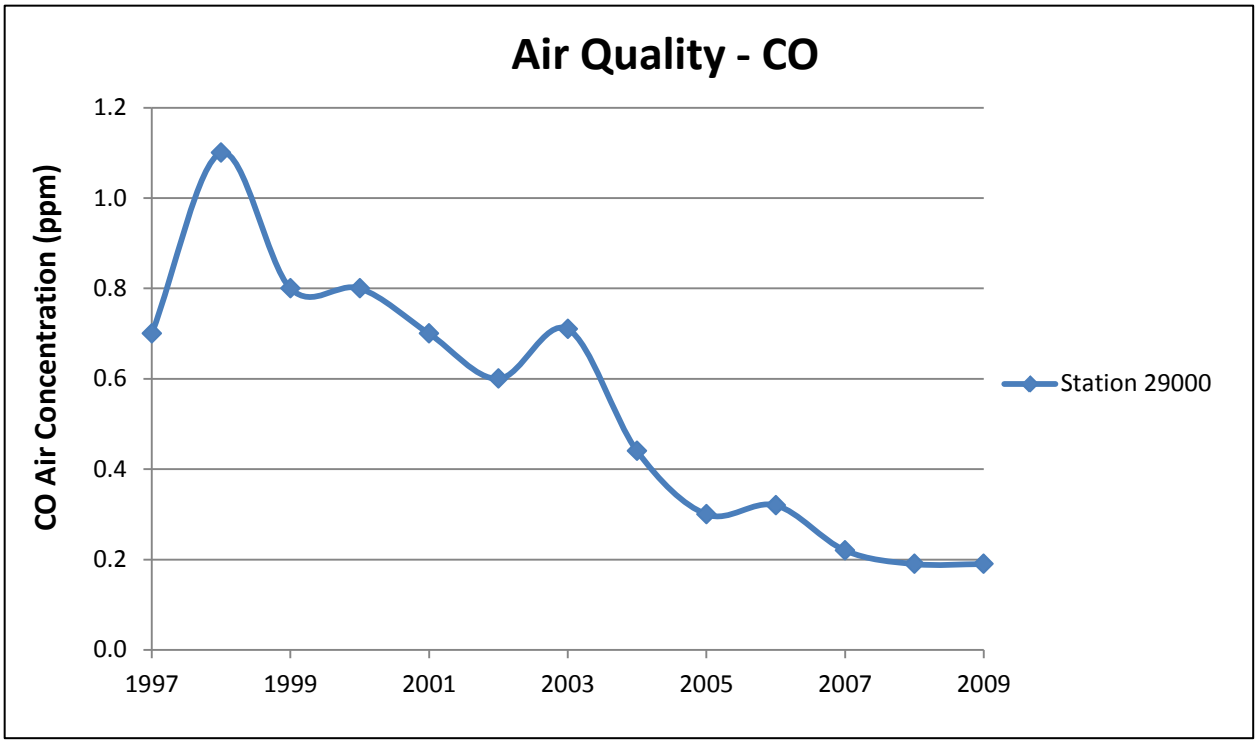


Figure 3-8 Summary of Air Quality Data for CO



3.3 METHODOLOGY FOR CALCULATING HEALTH OUTCOMES

To assess the mortality and hospital admissions associated with air pollution, the methodology established by Pengelly *et al.*'s initial 1997 HAQI report, and repeated in the Sahsuvaroglu and Jerrett (2003) update was used for consistency and comparability over time.

Generally, the following calculation was used.

$$B * \Delta H\% * P = HO$$

where,

B = base number of annual health outcomes
Δ H% = percent change in health outcome per unit increase of pollutant
P = annual pollutant average
HO = annual health outcome attributable to (current) air pollution exposure

Δ H% is calculated from the relative risks (RR) associated with pollutant increases identified in the literature review.

A sample calculation is provided below for PM_{10} and non-traumatic (NT) mortality.

Year	Base Number of Annual Health Outcomes – B (Events per year)	Relative Risk (RR)	Percent Change in Health outcome per 10 unit change of pollutant – Δ H%	Annual pollutant average – P (µg/m^3)	Health Outcome - HO (Annual Incidences per Year)
1998	3,934	0.45	0.00045	20.50	36.29
1999	3,930		0.00045	19.20	33.96
2000	3,928		0.00045	20.60	36.41
2001	3,857		0.00045	19.10	33.15
2002	3,765		0.00045	17.65	29.90
2003	3,992		0.00045	21.10	37.90
2004	3,725		0.00045	16.75	28.08
2005	3,934		0.00045	19.88	35.19
				Average	33.86

It should be noted that in Sahsuvaroglu and Jerrett (2003) update, no health outcomes were calculated for $PM_{2.5}$; however, as the focus of health effects of fine particulate matter has now shifted from PM_{10} to $PM_{2.5}$ as the causal agent; therefore health outcomes have been predicted for $PM_{2.5}$ in this report.

In addition, if relative risks were obtained from Sahsuvaroglu and Jerrett (2003), then the Health Outcomes were adjusted by 42% to account for the fact that a reanalysis of the National Morbidity, Mortality and Air Pollution Study (NMMAPS) data indicated that risk estimates were overestimated by 36 to 42% (Dominici *et al.*, 2002). The adjustments were conducted at the time when a modelling error had been discovered in the software used to run the calculations. The same adjustment as in the NMMAPS analysis was therefore used in Sahsuvaroglu and Jerrett (2003) as well as in this report.

4.0 RESULTS

As outlined previously, the estimated health outcomes discussed in this section are calculated using measured air quality data in Hamilton, observed base mortality and morbidity rates from Hamilton and relative risks obtained from the literature. In the current model, relative risks are held constant from year to year; consequently, temporal variations in health outcomes are due only to the increase or decrease of measured air pollutant concentrations and observed base event rates for Hamilton.

As discussed in Section 3 (Figure 3.1), the total non-traumatic mortality and total respiratory hospital admissions have remained relatively constant over the study period while total cardiovascular hospital admissions has been declining since approximately 2001. Similarly, average measured air pollutant concentrations for the Hamilton region (Figures 3-3 to 3-7) has generally decreased over the study period with the exception of O_3, which has increased. Based on these general trends it is observed that the health outcomes associated with airborne pollutants will have improved or remained constant over the study period with the exception of those associated with O_3 levels. It should be noted that this is conditional on the number of exposed persons remaining (approximately) constant over the study period.

Estimated Hamilton-specific year to year annual health outcomes are presented in Figures 4-1, 4-2 and 4-3 for non-traumatic acute exposure mortality, respiratory hospital admissions and cardiovascular hospital admissions respectively. Non traumatic mortality was calculated between 1997 and 2005 as no measured base mortality rates were available for years beyond 2005. Respiratory and cardiovascular hospital admission rates, however, were available up until 2008 and thus the evaluated period for these health outcomes was 1997 to 2008. Appendix D provides a summary of the results.

Figure 4-1 presents the non-traumatic mortality attributable to the studied air pollutants. It can be seen that, based on current estimates, ozone (O_3) and nitrogen dioxide (NO_2) levels measured in Hamilton are responsible for more non traumatic acute exposure deaths per year over the study period than those of any of the other air pollutants. The non-traumatic mortality due to all air pollutants are seen to either remain fairly constant or decrease over the 1997-2005 period with the exception of incidences associated with ozone, which are seen to increase.

It is estimated that there are between 40 and 70 non traumatic deaths per year attributable to both ozone and NO_2, while annual deaths due to PM_{10} and $PM_{2.5}$ are both between 25 and 40. Estimated annual non-traumatic mortality due to SO_2 and CO is much lower by comparison at below 10 deaths per year each.

Figure 4-1 Modeled Health Outcomes for Non Traumatic Mortality for Air Pollutants

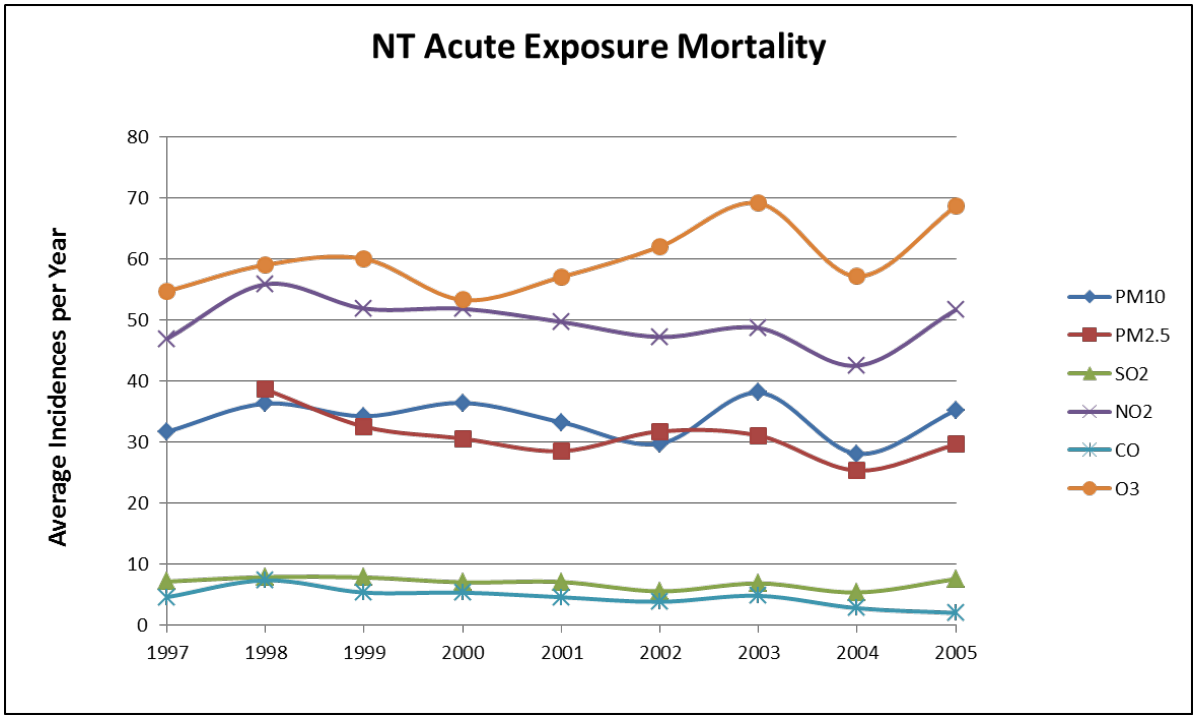
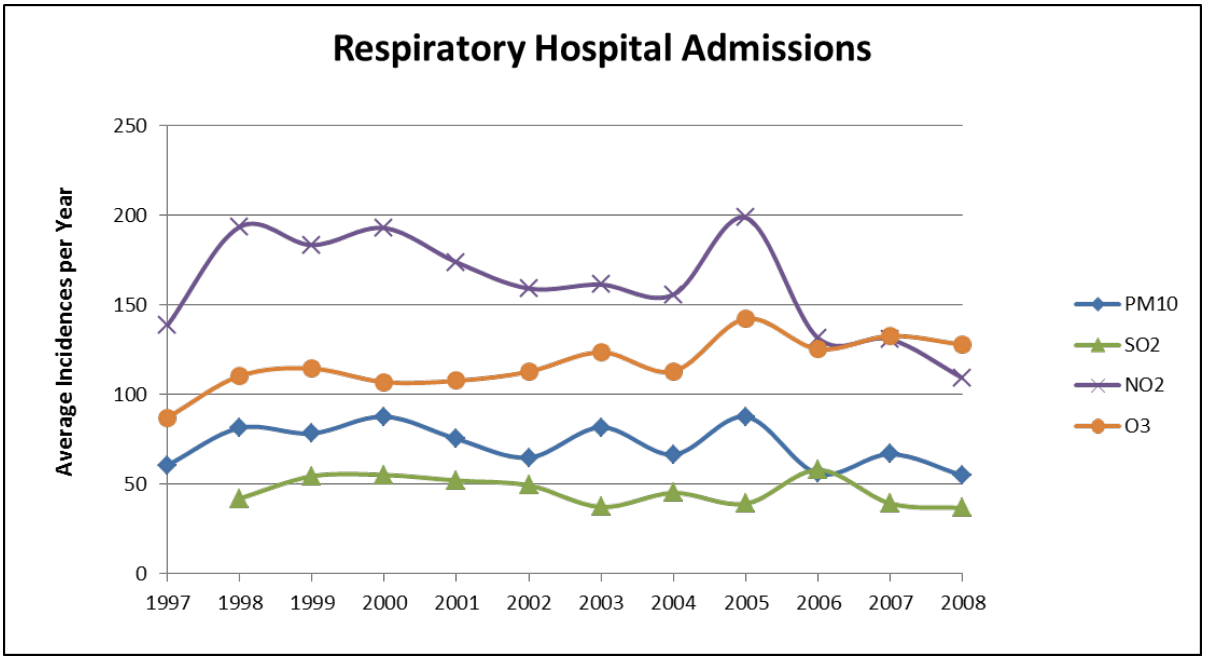


Figure 4-2 Modeled Health Outcomes for Respiratory Hospital Admissions for Air Pollutants



Notes:

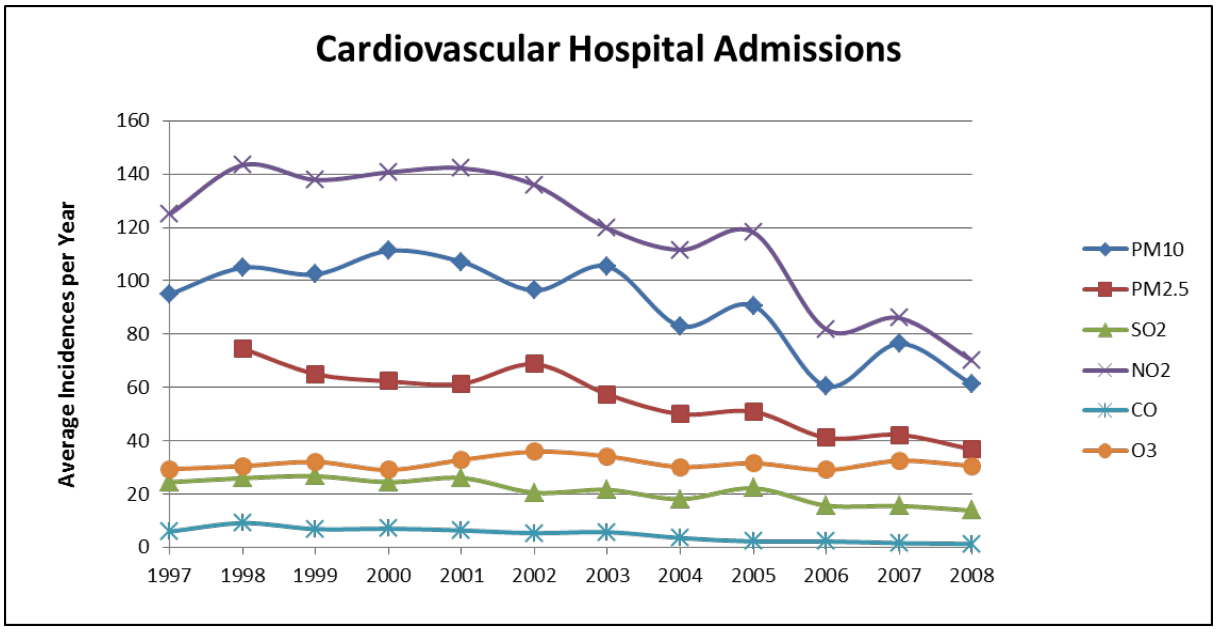
$PM_{2.5}$ and CO not assessed because no appropriate relative risk values available in literature.

PM_{10}, NO_2 and O_3 respiratory hospital admissions adjusted by 42% as per Sahsuvaroglu and Jerrett (2003).

Figure 4-2 presents the estimated year to year respiratory hospital admissions due to the studied airborne pollutants. It should be noted that for this outcome no appropriate relative risk values were available for $PM_{2.5}$ and CO and thus respiratory hospital admissions due to these pollutants could not be estimated.

Similar to non-traumatic acute exposure deaths, ozone is seen to be one of the top contributors to respiratory hospital admissions. According to current estimates, NO_2 is the leading pollutant responsible for respiratory hospital admissions for the majority of the study period with ozone becoming the leading pollutant in 2008. PM_{10} and SO_2 are the cause of fewer respiratory hospital admissions; average incidences for these pollutants are both between 25 and 100 as compared to between 75 and 200 for NO_2 and ozone. Similar to non-traumatic acute mortality, annual respiratory hospital admissions are decreasing or remaining constant with the exception of admissions due to ozone exposures.

Figure 4-3 Modeled Health Outcomes for Cardiovascular Hospital Admissions for Air Pollutants



Notes:

SO_2 and CO cardiovascular hospital admissions adjusted by 42% as per Sahsuvaroglu and Jerrett (2003).

Figure 4-3 above presents the annual cardiovascular hospital admissions attributable to studied air pollutants. It can be seen that NO_2 and PM_{10} levels are the key factors in cardiovascular hospital admissions. $PM_{2.5}$ is the next most important, followed by O_3 and SO_2. Of the pollutants studied, CO is estimated to cause the fewest cardiovascular hospital admissions over the study period. As with the non-traumatic mortality and respiratory hospital admissions, cardiovascular hospital admissions per year due to the studied pollutants are seen to be following

either decreasing or constant trends with the exception of O_3, which is seen to increase slightly over the 1997-2008 study period.

Figures 4-4 to 4-9 provide summaries of the three health outcomes for each air pollutant. As seen from the figures, respiratory hospital admissions are the key health outcome associated with NO_2, SO_2 and O_3 where as cardiovascular hospital admissions are associated with exposure to PM_{10}, $PM_{2.5}$ and CO. However, it should be noted that for $PM_{2.5}$ and CO, there were no available relative risk values for respiratory admissions and therefore, these outcomes could not be calculated.

Figure 4-4 Modeled Summary of Health Outcomes for PM_{10}

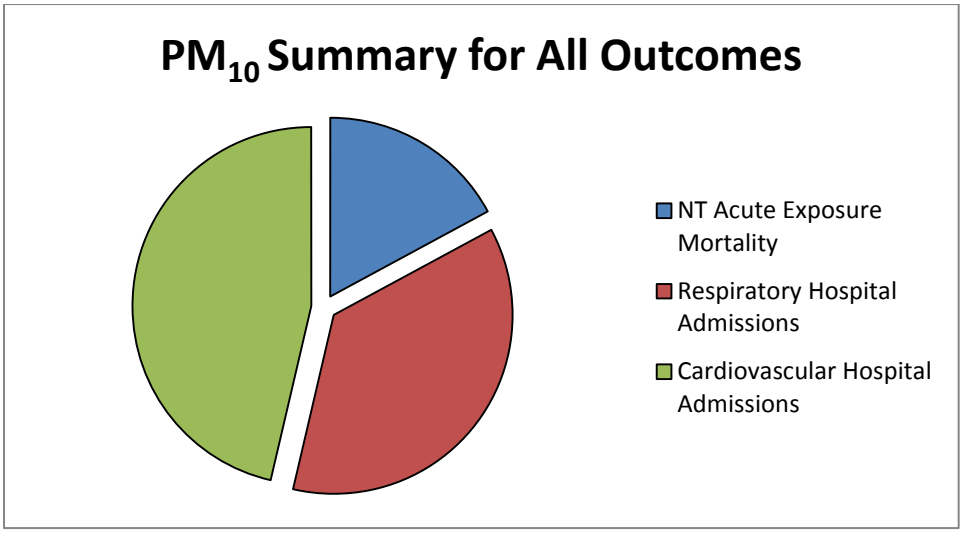
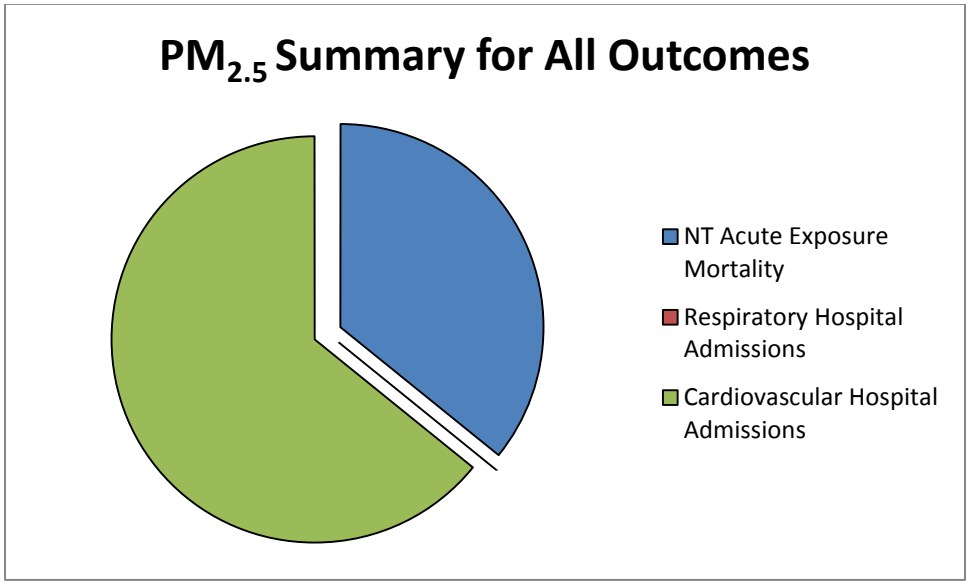


Figure 4-5 Modeled Summary of Health Outcomes for $PM_{2.5}$



Notes: Respiratory hospital admissions not assessed because no appropriate relative risk values available in literature.

Figure 4-6 Modeled Summary of Health Outcomes for NO_2

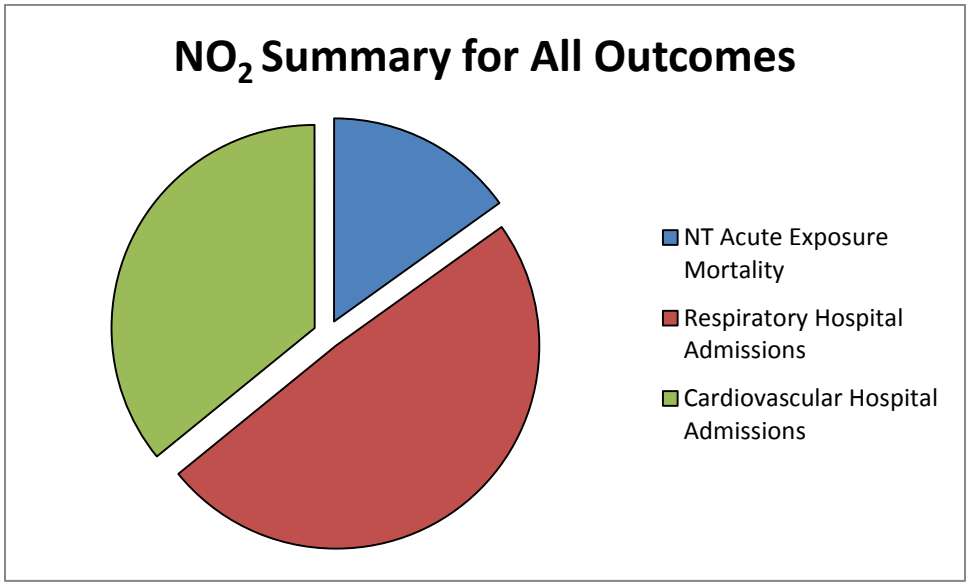


Figure 4-7 Modeled Summary of Health Outcomes for SO_2

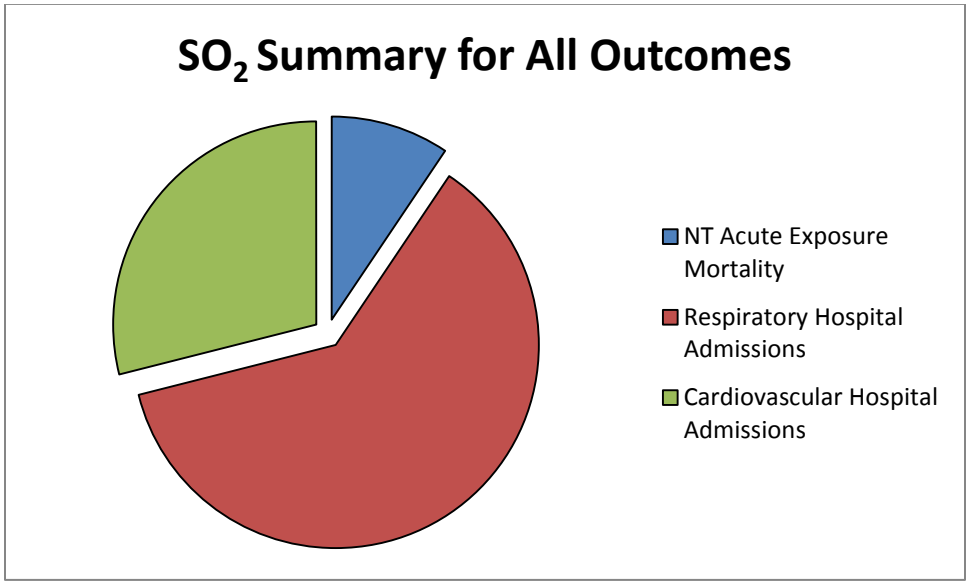


Figure 4-8 Modeled Summary of Health Outcomes for O_3

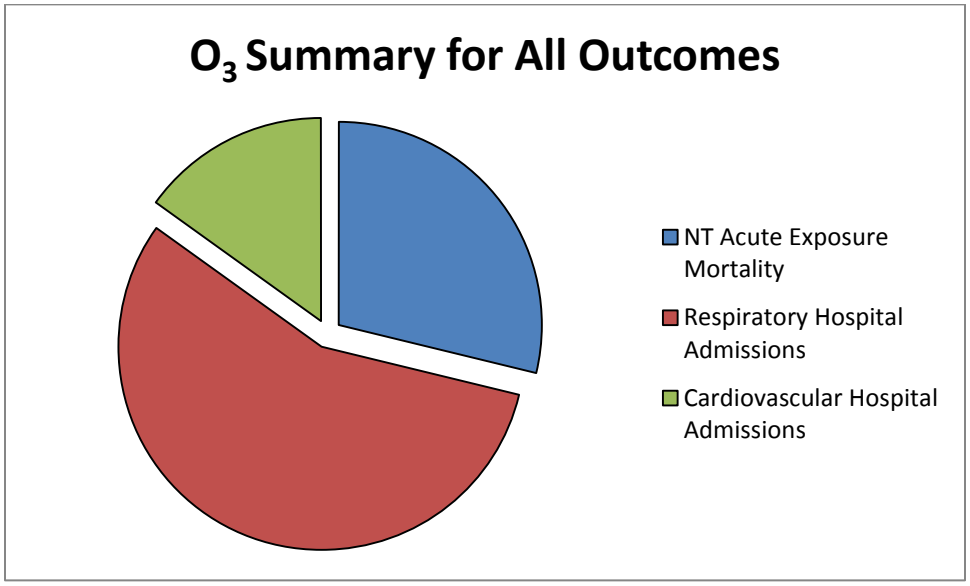
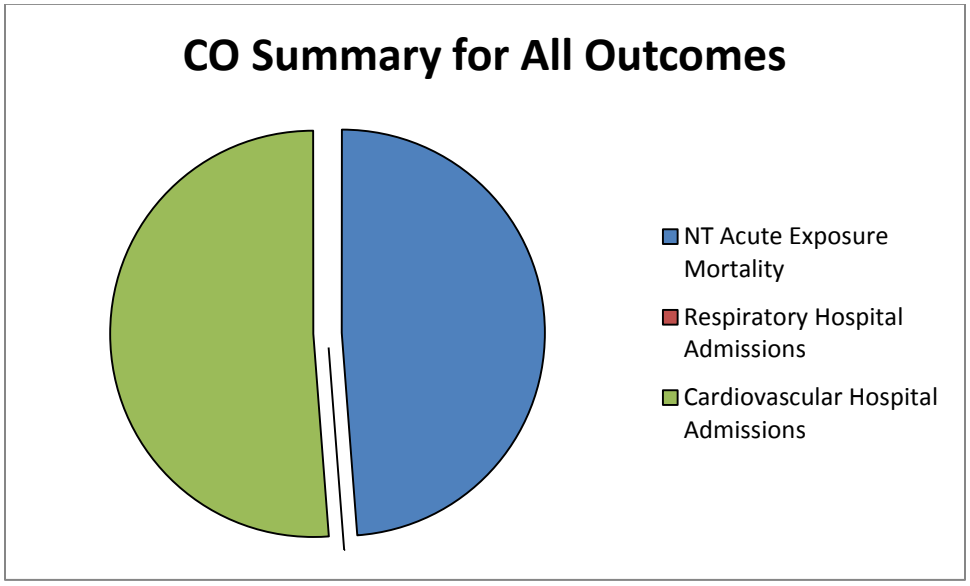


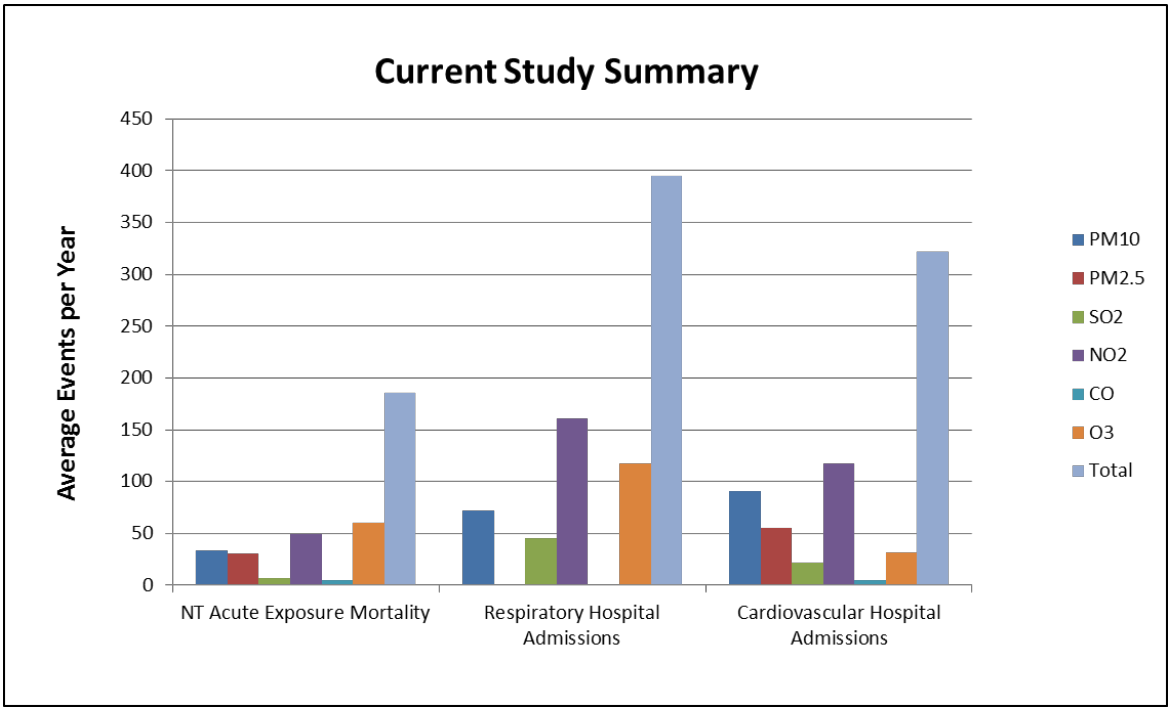
Figure 4-9 Modeled Summary of Health Outcomes for CO



Notes: Respiratory hospital admissions not assessed because no appropriate relative risk values available in literature.

Figure 4-10 provides a summary of the health outcome results averaged over the current study period. As discussed above, the top pollutants contributing to non-traumatic acute exposure mortality and respiratory hospital admissions are O_3 and NO_2 while the key factors impacting cardiovascular (CV) hospital admissions over this period are NO_2 and PM_{10}.

Figure 4-10 Modeled Summary of Health Outcomes for Air Pollutants



A comparison of relative risks employed in the current study to those used in two previous studies is presented in Table 4-1. The '1997' relative risk values were provided in Pengelly *et al.* (1997) while the '2003' relative risk values were employed by Sahsuvaroglu and Jerrett (2003). It should be noted that at the time of the 1997 study, there were less scientific reports available on the impacts of specific pollutants on human health. Since 1997, more reports have been provided in the literature. In cases where the current literature review indicated no appropriate updated relative risk values, relative risks utilized by Sahsuvaroglu and Jerrett (2003) were adopted. The health outcomes calculated using these values were adjusted to account for a maximum risk rate overestimation of 42% as was done in Sahsuvaroglu and Jerrett (2003).

All relative risks employed in the current study are lower than or equal to those used in the 2003 work while the relative risks utilized in 1997 for non-traumatic mortality associated with CO and O_3 and respiratory hospital admissions associated with all pollutants are lower. This may be due to re-analysis of the studies resulting in more appropriate values of relative risks.

These differences reflect the data available in the scientific literature. In recent years, much greater attention has been paid to the statistical methods used and the evaluation of relative risks. Thus, advances in all of these areas are reflected in the changing risk values in the three studies.

Table 4-1 Comparison of Relative Risks Between Studies

	NT Acute Exposure Mortality (changes per 10 units pollutant)			Respiratory admissions (changes per 10 units pollutant)			CV admissions (changes per 10 units pollutant)		
	1997[a]	2003	Current	1997[a]	2003[b]	Current	1997[a]	2003[b]	Current
PM_{10} ($\mu g/m^3$)	1	0.76	0.45	0.7	2.1	2.1[a]	0.6	1.4	0.7
$PM_{2.5}$ ($\mu g/m^3$)	-	2.88	0.77	-	-	-	-	-	0.8
SO_2 (ppb)	0.6	2	0.36	0.4	3.7	3	-	1.1	1.1[a]
NO_2 (ppb)	1.15	1.9	0.68	0.4	4.9	4.9[a]	-	6.55	0.94
CO (ppm)	1.1	3.68	1.7	-	-	-	5	1.95	1.95[a]
O_3 (ppb)	0.3	1.38	0.72	0.8	2.8	2.8[a]	-	4.5	0.2

Note:

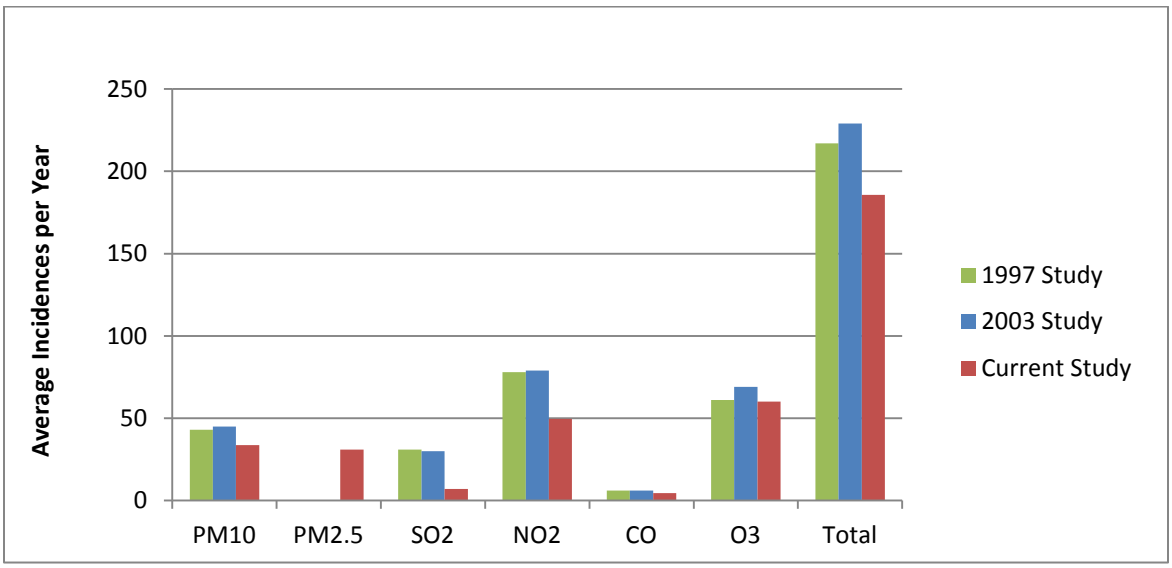
a Pengelly *et al.* (1997).

b Sahsuvaroglu and Jerrett (2003).

c no appropriate updated relative risk values were available, values utilized in Sahsuvaroglu and Jerrett (2003) were adopted.

A comparison of non-traumatic mortality estimates from studies completed using the three sets of relative risk values provided in Table 4-1 is shown in Figure 4-11. As expected, in many cases the current estimates are lower than annual mortality incidences calculated by Pengelly *et al* (1997) and Sahsuvaroglu and Jerrett (2003); this decrease is due, in part, to reductions in the relative risks employed in combination with generally decreasing air pollutant levels in the Hamilton region in recent years.

Figure 4-11 Comparison of Modeled Non Traumatic Mortality with Previous Studies for Air Pollutants



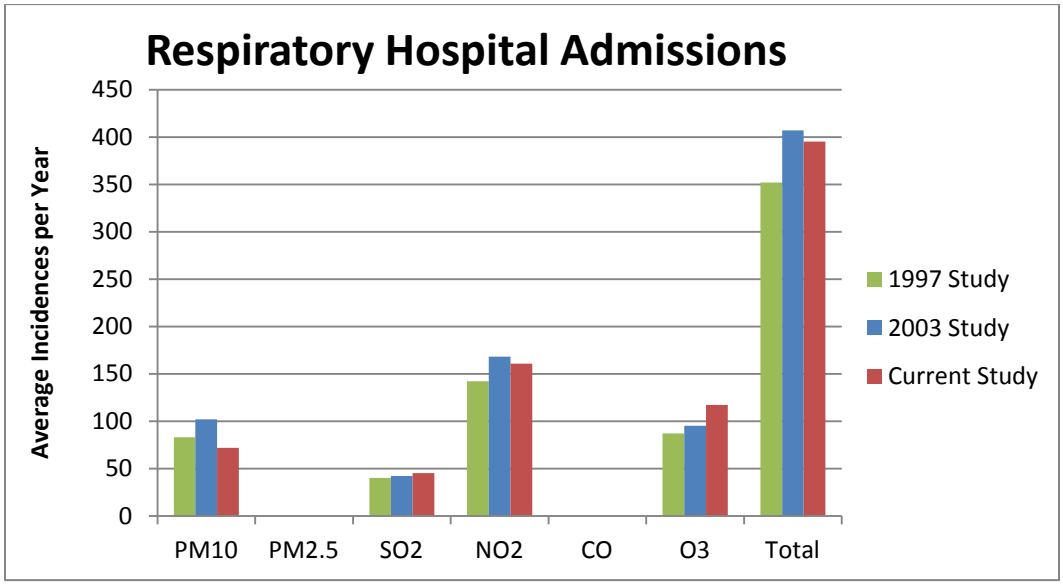
Notes: All '1997 Study' and '2003 Study' data adjusted by 42% to account for overestimate of RR values (Sahsuvaroglu and Jarrett (2003)).

'1997 Study' Pengelly *et al.* (1997).

'2003 Study' Sahsuvaroglu and Jarrett (2003).

Figure 4-12 shows a comparison of average respiratory hospital admissions as estimated by the current and previous studies. All of the outcomes are similar with the only marked increase over previous studies being incidences due to ozone levels. This result is not surprising considering the recent increase in ozone levels which would not have been captured in either of the previous estimates.

Figure 4-12 Comparison of Modeled Respiratory Hospital Admissions with Previous Studies for Air Pollutants



Notes:

All ''1997 Study' and '2003 Study' data adjusted by 42% to account for overestimate of RR values (Sahsuvaroglu and Jerrett (2003)).

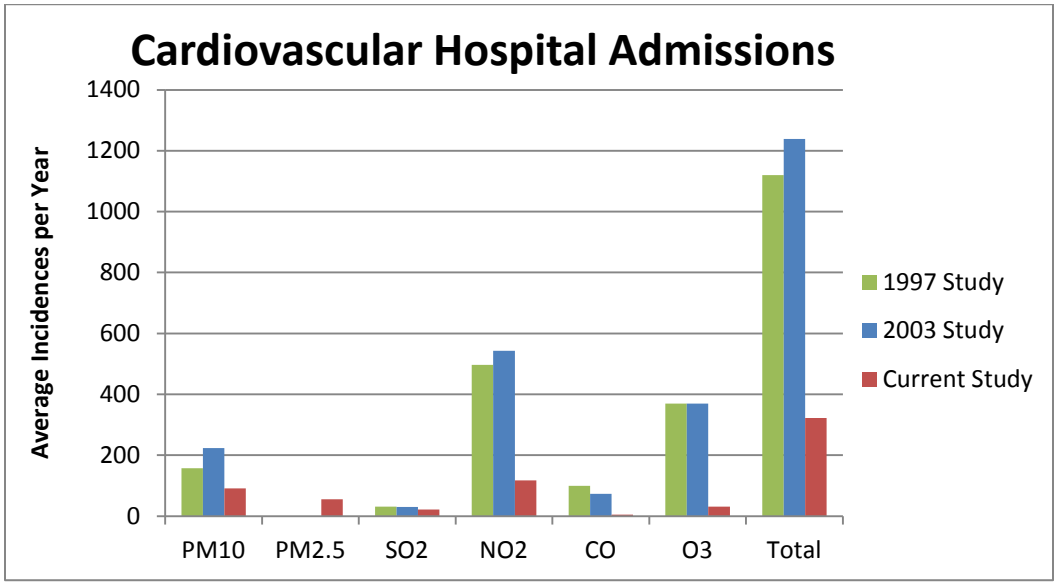
PM_{10}, NO_2 and O_3 '1997-2008' values adjusted by 42% as using 2003 study values (Sahsuvaroglu and Jerrett (2003)).

'1997 Study' Pengelly *et al.* (1997).

'2003 Study' Sahsuvaroglu and Jerrett (2003).

A comparison of average cardiovascular hospital admissions calculated in the current and previous studies is shown in Figure 4-13. In this comparison, it can be seen that there is a dramatic decrease in average incidences per year calculated in the current study as compared to previous estimates; this decrease is largely due to the considerably lower relative risk values selected for use in the current study for PM_{10}, NO_2 and ozone.

Figure 4-13 Comparison of Modeled Cardiovascular Hospital Admissions with Previous Studies for Air Pollutants



Notes:

All 1997 Study' and '2003 Study' data adjusted by 42% to account for overestimate of RR values (Sahsuvaroglu and Jerrett (2003)).

SO_2 and CO 'Current Study' values adjusted by 42% as using 2003 study values (Sahsuvaroglu and Jerrett (2003))

''1997 Study' Pengelly *et al.* (1997).

'2003 Study' Sahsuvaroglu and Jarrett (2003).

5.0 ALTERNATIVE MODEL APPROACHES

As a sensitivity analysis and to obtain a "crude" estimation of health savings, calculations were carried out with the Ontario Medical Association's Illness Cost of Air Pollution (ICAP) model using data already available in CAH Annual Reports. Calculations of health impacts were also determined using the Health Canada Air Quality Benefits Assessment Tool (AQBAT) model. These two models are generally used to determine the burden of illness associated with air pollution and are discussed in more detail in the following sections. It should be noted that the inputs associated with the three models are different, making it inherently difficult to compare the outputs of the different models and to do a sensitivity analysis

5.1 ICAP

ICAP was developed to assign health care costs to the impacts of exposure to air pollution. It has the ability to forecast health outcomes and health care costs. The model was originally prepared for Ontario on behalf of the Ontario Medical Association in 2000. In 2005, ICAP Version 2.0 included updated air pollution, health incidence rates and relative risks estimated for the different health outcomes.

The third version was prepared for the Canadian Medical Association so that it could be expanded and applied to assess effects at a national level. ICAP Version 3.0 now includes estimates and calculations for all provinces, has the most recent (data available at the time of the model version) underlying air quality and health data, and has also incorporated the most recent scientific advances in air pollution and health research.

ICAP's main intent is to quantify the health effects of exposure to air pollution to provide policy makers with a means of advocating for the improvement of air quality. It has been used by several provinces, local medical officers and educators. The latest report also suggests that community groups and private citizens have used the model to influence local policies.

Some of the aspects of the ICAP model are discussed below:

1. Spatial resolution. The level of data available is only for census divisions (as defined by Statistics Canada).

2. Data parameters:
    a. Default values available by census division include ambient air quality concentrations for seven pollutants and population broken down by age and gender (based on the latest census available). The pollutants are: ozone, PM_{10}, $PM_{2.5}$, SO_4, CO, SO_2, NO_2.

b. Provincial-level data include base incidence rates, average length of hospital stays, and healthcare unit costs for different health outcomes, as well as value of lost time.
c. Common data across all provinces, which include health risks of exposure to air pollution, value of a statistical life, and the value of the quality of life.

3. Data estimation:
    a. Population forecasting can be chosen (based on StatCan) – low growth, medium growth –medium migration, medium growth-central-west migration, and high growth.
    b. Air Quality data. These are based on NAPS data that are then kreiged to create a surface. Note that for $PM_{2.5}$, there are only 41 stations throughout Canada, compared to NO_2 which has 140 NAPS stations across the country. The model allows for us to set our own air pollution baseline, and our own forecasts.
    c. Health risks are expressed as relative risks, derived from the epidemiological literature and assessed with an expert panel. Calculations are derived from Base Incidence Rates that vary by location, age, illness type. Base Incidence Rates (expressed as incidence per 100,000 persons) - relative risks are associated with a 10-unit change in pollutant concentrations.
    d. Economic damage functions are estimated for the value of avoiding early death, reducing or avoiding pain and suffering, costs of health care treatment and the value of lost time/productivity due to illness.

In applying this model to this project, a number of limitations were encountered:

- The starting year is 2006 and therefore any information collected prior to 2006 cannot be applied in the model. This was a problem for comparison to the current project as mortality data for Hamilton were only available up until 2005.
- Another limitation of the model for this study was that the model can provide incremental health outcomes only for scenarios with increasing air quality as compared to the baseline year. As such, the focus of ICAP is on calculating damages or costs associated with incremental air concentration increases and not the calculation of economic savings due to incremental air concentration decreases. In the case of this study, where the air pollutant concentrations have decreased, the model had limited utility for assessing the effects of incremental concentration changes. Incremental health results could only be obtained for ozone as these concentrations increased with time.
- The model is quite tedious since all air pollutants cannot be evaluated in one run. Therefore numerous runs are needed to get any results. In addition, the output format is somewhat inconvenient to work with, especially in trying to do model comparisons.

- The model only considers total mortality outcomes and not acute or chronic mortality outcomes as do other models.

Table 5-1 provides a comparison between the relative risks in ICAP and those used in the current study. As seen in the table, the relative rates associated with mortality are similar; however the relative risks for respiratory admissions are different. For cardiovascular admissions, the relative risks are similar for $PM_{2.5}$ and SO_2 but quite different for the other air pollutants. Since this study was attempting to do a comparison between the results of the current study and the ICAP model, the relative risks were changed in ICAP to the ones used in the current study. As discussed above, the inputs are different, thus comparisons between results are more qualitative. than quantitative.

Table 5-1 Comparison of Relative Risks Between ICAP and the Current Study

	NT Acute Exposure Mortality (changes per 10 units pollutant) range of RR estimates		Respiratory admissions (changes per 10 units pollutant) range of RR estimates		CV admissions (changes per 10 units pollutant) range of RR estimates	
	ICAP*	Current	ICAP	Current	ICAP	Current
PM10 (µg/m³)	-	0.45	-	2.1	-	0.7
PM2.5 (µg/m³)	1.0	0.77	1.2	-	0.9	0.8
SO_2 (ppb)	0.4	0.36	7.5	3	1.9	1.1
NO_2 (ppb)	0.8	0.68	7.4	4.9	7.6	0.94
CO (ppm)	-	1.7	-	-	-	1.95
O_3 (ppb)	0.5	0.72	1.2	2.8	1.9	0.2

Note: The ICAP NT mortality is total (acute and chronic) whereas for the current study it is acute.

5.2 AQBAT

AQBAT is Health Canada's tool to estimate the human health and welfare benefits or damages associated with changes in Canada's ambient air quality. It is a computer simulation tool that allows for the definition of wide range of specific scenario models from the flexibility of combining and linking of pollutants, health endpoints, geographic areas and scenario years. AQBAT contains historical and projected population data, and accesses preset data files of historical and hypothetical pollutant concentrations along with data files of baseline health endpoint rates. The tool performs Monte Carlo simulations to provide a range of possible health effects outcomes and associated economic valuations for each scenario, ranged around a central value that provides the best estimate of the simulation.

The current pollutants in AQBAT are four gas and two particle type pollutants, including CO, NO_2, O_3, SO_2, PM_{10} and $PM_{2.5}$, with 1 hour maximum and 24 hour average metrics for the gases and 24 hour average metrics for PM_{10} and $PM_{2.5}$. There is also an 8 hour maximum metric for ozone. The summary statistics derived for the pollutants generally apply to all months of the

year. In addition, however, there are seasonal distinctions for ozone because O_3 concentrations vary according to the time of year much more than the other pollutants.

There are a wide range of health outcomes that are considered in AQBAT such as acute exposure mortality, chronic exposure mortality, cardiac hospital admissions (elderly and adult), respiratory hospital admissions, cardiac emergency room visits, respiratory emergency room visits, adult chronic bronchitis cases, child acute bronchitis episodes, asthma symptom days, acute respiratory symptom days, minor restricted activity days and restrictive activity days. For this project, the focus was on acute exposure mortality, cardiac hospital admissions and respiratory hospital admissions.

To calculate the economic impact of air pollution, AQBAT uses health endpoint valuations which assign a monetary value to a health outcome. Mortality valuation ("value of a statistical life") is based on someone's willingness to pay to reduce mortality risks or willingness to accept compensation to experience increased mortality risks (i.e. wage premiums for riskier jobs). The morbidity outcomes are valued using a variety of approaches which evaluate costs of treatment (e.g., medical costs), lost productivity, pain and suffering and averting expenditures. The model is more versatile than ICAP as it can output costs associated with benefits as well as damages.

The AQBAT model has been applied for similar assessments including the City of Toronto, for British Columbia, and for evaluating the total cost of air pollution in the transportation sector across Canada, among others.

In applying this model to this project, a number of limitations for the current study were encountered:

- It does not appear that air concentrations can be input into the model prior to 2003 so that a comparison can be made to baseline.
- The model provides incremental health outcomes due to an incremental change in air pollution and does not seem to be able to provide a total health outcome which makes it difficult to compare to the model used in this current study.

Table 5-2 provides a comparison between the relative risks in AQBAT and those used in the current study. As seen in the table, the relative rates associated with mortality are similar; however there is little information on relative risks for respiratory and cardiovascular admissions. Relative risks are only provided for $PM_{2.5}$. Since this study was attempting to do a comparison between the results of the current study and the AQBAT model, the relative risks were changed in AQBAT to the ones used in the current study. As discussed above, the inputs are different, thus comparisons between results are more qualitative than quantitative.

Table 5-2 Comparison of Relative Risks Between AQBAT and the Current Study

	NT Acute Exposure Mortality (changes per 10 units pollutant) range of RR estimates		Respiratory admissions (changes per 10 units pollutant) range of RR estimates		CV admissions (changes per 10 units pollutant) range of RR estimates	
	AQBAT	Current	AQBAT	Current	AQBAT	Current
PM10 (µg/m³)	-	0.45	-	2.1	-	0.7
PM2.5 (µg/m³)	-	0.77	0.75	-	0.71	0.8
SO_2 (ppb)	0.46	0.36	-	3	-	1.1
NO_2 (ppb)	0.75	0.68	-	4.9	-	0.94
CO (ppm)	1.9	1.7	-	-	-	1.95
O_3 (ppb)	0.84	0.72	-	2.8	-	0.2

5.3 RESULTS

As discussed in the previous section, there were a number of limitations for the use of these two models for this application that made it very difficult to compare the results from these alternative models to the results generated for this study. Thus the results presented here provide an illustration of results from the models using relative risks generated in the study and a subset of the air quality data for Hamilton due to the limitations of the model for this application. They are not intended to be compared quantitatively.

5.3.1 Results from ICAP

As discussed in Section 5.2, one of ICAP's limitations for the context of this current study is that the earliest starting point in ICAP is set to 2006, while the mortality data used in the current study is for the period prior to 2006. For this reason, comparisons were only made to incremental health outcomes predicted by AQBAT and not total incidences as predicted by the current model. However, there are limitations which affect this comparison as well, namely that ICAP can only provide results for burden of illness (i.e. damages) due to incremental increases in air pollutant concentrations and it cannot show benefits due to incremental decreases. Therefore the model could not be applied in this study, especially as concentrations of fine particulate matter ($PM_{2.5}$ and PM_{10}), sulphur dioxide, nitrogen dioxide and carbon monoxide decreased between 1997 and 2009.

Figure 5-1 provides the results for the number of increased cases attributable to an ozone increase since 1997. As seen from the figure, approximately 30 more cases were attributable to increases in ozone concentrations with respiratory hospital admissions accounting for the majority of the cases. The current model predicts approximately a 5 times higher result. This may be due to the fact that ICAP is predicting an incremental increase whereas the current result

is predicting a total. If the incremental change for respiratory hospital admissions is determined, it is in the order of 50 more cases.

Figure 5-1 Annual Health Outcomes Attributable to Increase in Ozone since 1997

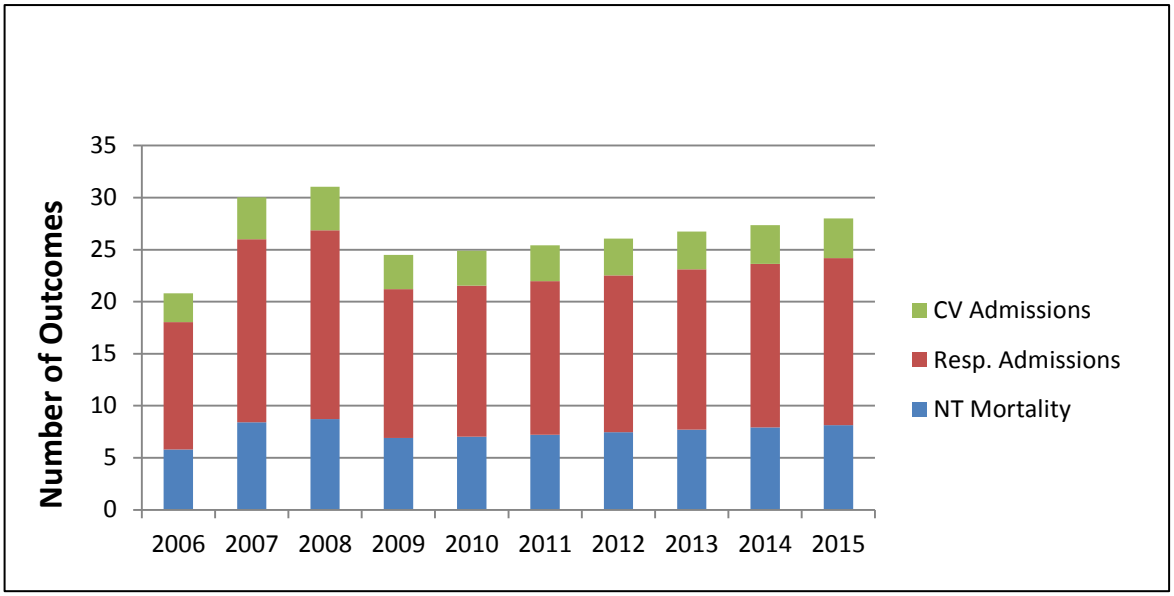
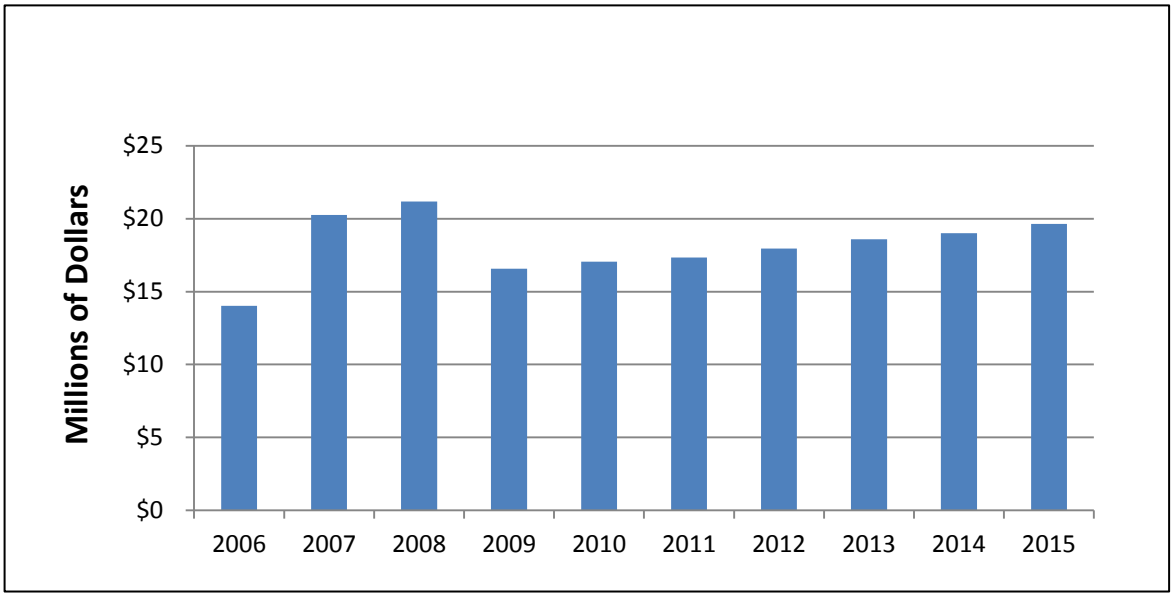


Figure 5-2 provides the cost associated with the increase in ozone concentrations. As seen from the Figure, ICAP predicts that an average increase in ozone concentration of approximately 5 ppm is associated with a cost of $ 20 million.

Figure 5-2 Economic Damages Attributable to Increase in Ozone since 1997



5.3.2 Results from AQBAT

The AQBAT model was able to calculate the benefits associated with declining air concentrations which makes it the better model for considering health outcomes and costs for Hamilton; however, there are some limitations to using the model as described in Section 5.1 that limit its utility for this application. For example, AQBAT is primarily used for calculating incremental health outcomes due to an incremental change in air quality from baseline and not for estimating total annual incidences as needed for a comparison to the current study results. No direct comparisons between the results from AQBAT and the results from the current study can be made.

Figures 5-3 to 5-5 show the results obtained from AQBAT for acute non-traumatic mortality, respiratory hospital admissions and cardiovascular hospital admissions. As seen from the figures, there are benefits associated with the decreases in air pollutant levels, with declines in concentrations of NO_2 and $PM_{2.5}$ accounting for the largest benefits in mortality and cardiovascular admission health outcomes.

Figure 5-3 Changes in Non-Traumatic Mortality Attributable to Changes in Air Quality

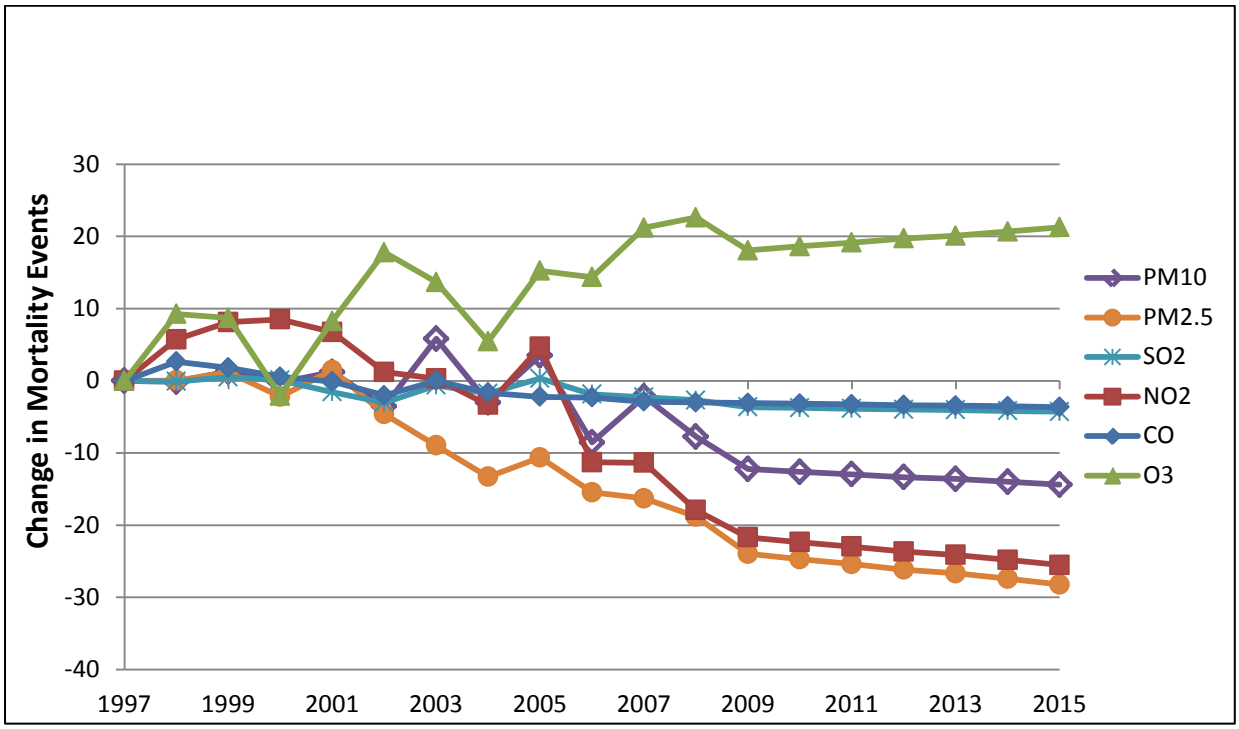


Figure 5-4 Changes in Respiratory Hospital Admissions Attributable to Changes in Air Quality

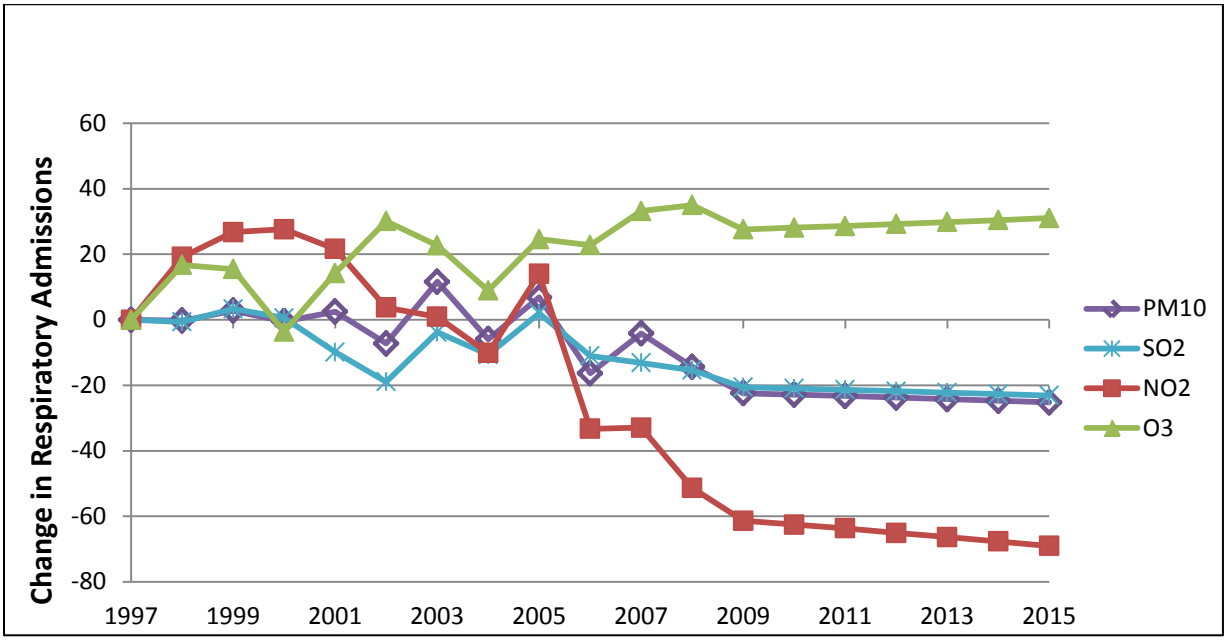


Figure 5-5 Changes in Cardiovascular Hospital Admissions Attributable to Changes in Air Quality

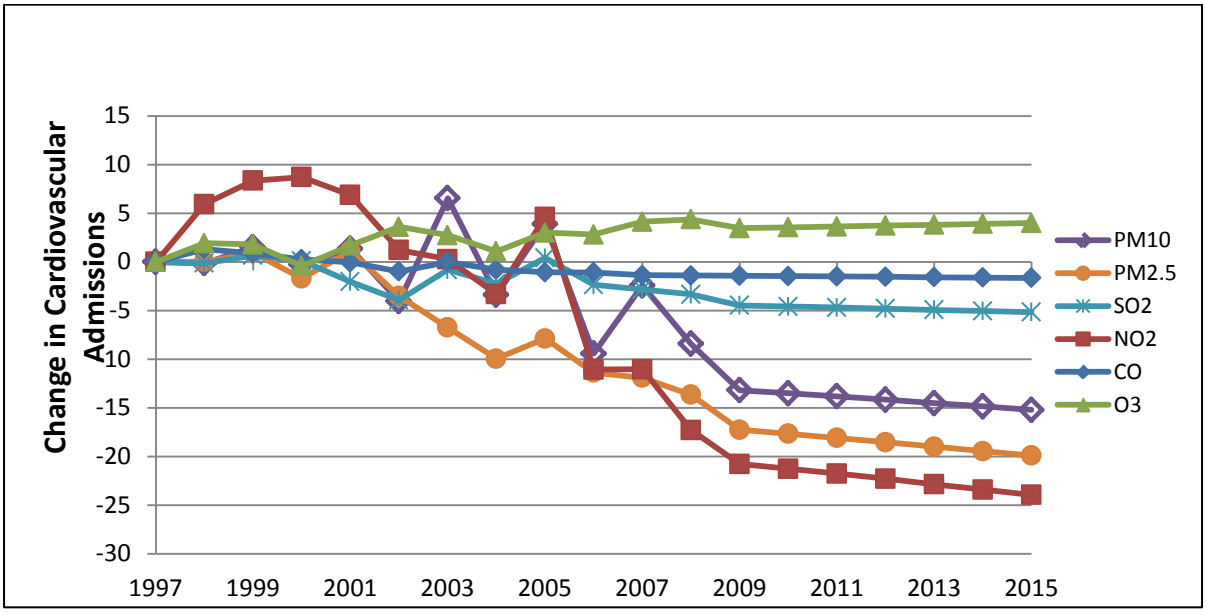
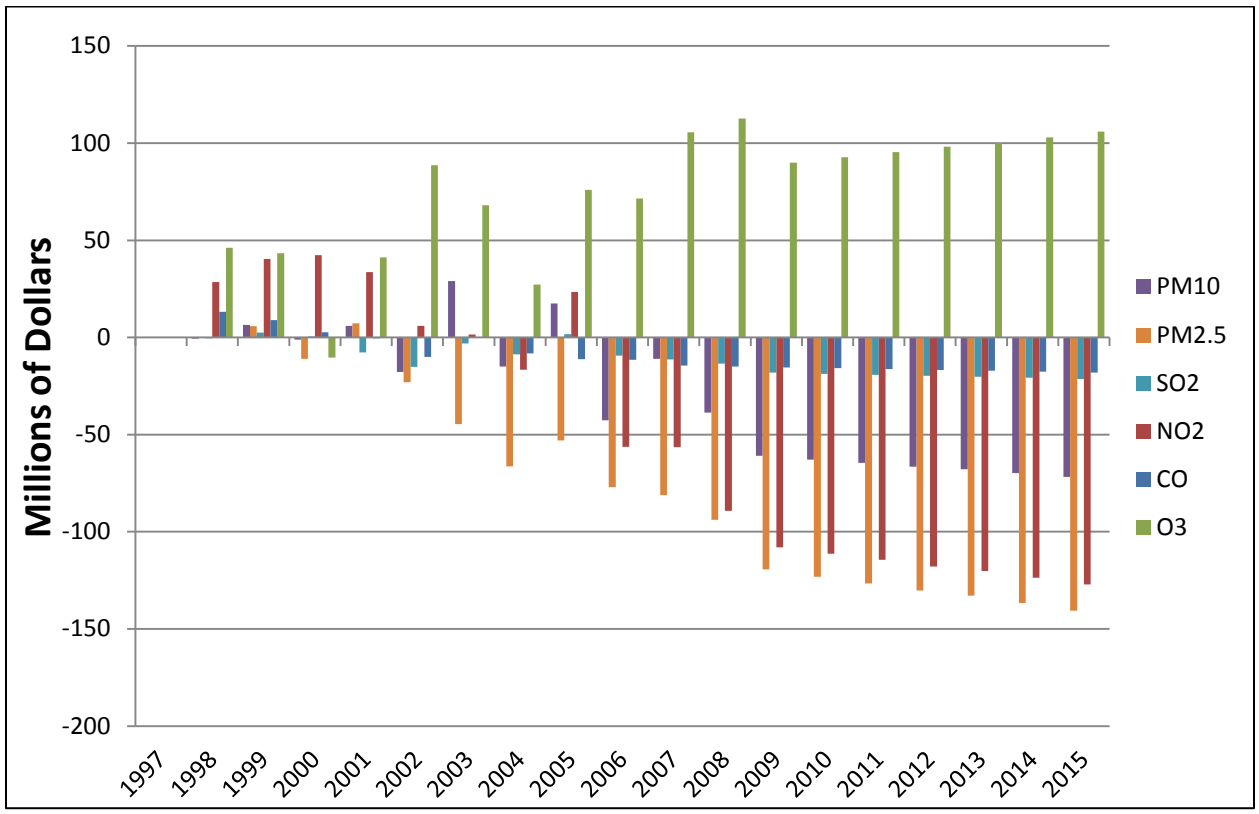


Figure 5-6 provides the economic valuation associated with the change in air quality in Hamilton. As seen from the figure, there are benefits associated with the decreasing concentrations of fine particulate matter, nitrogen dioxide, sulphur dioxide and carbon monoxide which outweigh the costs associated with the increased concentrations of ozone. It should be

noted that the costs associated with the increase in ozone concentrations determined from the AQBAT model is approximately 5 times higher than the cost obtained from the ICAP model. Thus care should be taken in the interpretation of the monetary costs/benefits associated with air pollution using these models.

Figure 5-6 Annual Economic Valuation Attributable to Changes in Air Quality



6.0 CONCLUSIONS

This report provided an updated health impacts assessment of exposure of Hamilton residents to air pollutants. The methodology used in this report was based on the 1997 HAQI report and Sahsuvaroglu and Jerrett (2003). With the improved methodologies, modeling and association capabilities, different and more detailed analyses could be conducted with the data; however, this study was meant to be an update and comparison to the Sahsuvaroglu and Jerrett (2003) study using the most recent air pollutant concentration levels and health endpoints.

The health outcomes in this report were calculated using measured air quality in Hamilton, observed base mortality and morbidity rates from Hamilton and relative risks obtained from the literature. For these revised calculations, relative risks for exposures to individual pollutants were held constant from year to year; consequently, temporal variations in health outcomes are due to the changing levels of measured air quality and observed base event rates for Hamilton.

The air pollutants that were evaluated in this study were fine particulate matter (PM_{10} and $PM_{2.5}$), nitrogen dioxide, sulphur dioxide, ozone and carbon monoxide. While NO is also a pollutant associated with vehicular and industrial emissions, it was evaluated as NO_2 since it is rapidly converted in the atmosphere to NO_2 and epidemiology and health studies focus on NO_2. The air quality data used in the assessment were considered representative of typical exposures in the community; however it is generally recognized that residents living in close proximity to major roadways may be more exposed. However, studies have shown that these pollutant concentrations drop off rapidly within 100 to 200 meters of the roadway.

The health data indicate that the total non-traumatic mortality and total respiratory hospital admissions have remained relatively constant over the study period while total cardiovascular hospital admissions has declined since 2001. Similarly, average measured air quality in the Hamilton region has improved or remained constant over the study period with the exception of ozone, which has increased. The modeling of health outcomes resulted in the number of events for non-traumatic acute mortality changing from 229 in the 2003 study to 186 in this study. It is estimated that there are between 40 and 70 non traumatic deaths per year attributable to both ozone and NO_2, while annual deaths due to PM_{10} and $PM_{2.5}$ are both between 25 and 40. Estimated annual non-traumatic mortality due to SO_2 and CO is much lower by comparison at below 10 deaths per year.

Hospital admissions associated with respiratory health effects remained largely unchanged (407 in 2003 to 395 in 2008). According to current estimates, NO_2 and ozone are the leading pollutants responsible for respiratory hospital admissions. PM_{10} and SO_2 are the cause of fewer respiratory hospital admissions with average incidences for these pollutants between 25 and 100 as compared to between 75 and 200 for NO_2 and ozone. The largest calculated decrease was

seen in hospital admissions due to cardiovascular effects where the numbers decreased from 1239 to 322.

These results of this study indicate that there are health benefits associated with the decreasing air pollutant concentrations in Hamilton.

Calculations were also carried out using the Ontario Medical Association's Illness Cost of Air Pollution (ICAP) model and Health Canada's Air Quality Benefits Assessment Tool (AQBAT). These two models are used to determine the health and financial burden of illness associated with air pollution. It should be noted that the inputs associated with the models are different and therefore, direct comparisons between the models were not made.

Costs associated with the change in air quality in Hamilton were calculated using both models. ICAP was restricted to increased costs associated with ozone. AQBAT calculated the benefits associated with the decreasing concentrations of fine particulate matter, nitrogen dioxide, sulphur dioxide and carbon monoxide; these benefits outweighed the costs associated with the increased concentrations of ozone.

When interpreting the results of the study, it should be noted that the study was an extension to the Sahsuvaroglu and Jerrett (2003) work and thus the focus was on relative risks of acute exposures and thus health outcomes associated with chronic exposure to air pollutants were not evaluated. There may be the possibility that some health outcomes are double-counted by using separate relative risk values for each pollutant; however efforts were made to ensure as many studies as possible were based on multi-pollutant models to minimize these influences. Finally, while the results of this report indicate that air quality improvements can result in lower health outcomes, the equation used to assess this relationship is very linear and does not take spatial variation into account such as distance to major roadways or industries. There are studies that indicate increased adverse effects associated with people living closer to major roadways and highways. Thus, it is important for policy and decision makers to consider some of issues when looking at outcomes associated with air pollutants.

In summary, this report demonstrated that as the air quality in Hamilton improves, the number of non-traumatic deaths, hospital admissions (for respiratory and cardiovascular disorders) and the overall cost to the economy due to air pollution decreases.

7.0 REFERENCES

Aga, E., Samoli, E., Touloumi, G., Anderson, H. R., Cadum, E., Forsberg, B., *et al*. 2003. *Short-Term Effects of Ambient Particles on Mortality in the Elderly: Results from 28 Cities in the APHEA2 Project*. Eur Respir J Suppl, 40, 28s-33s.

Agency for Toxic Substances and Disease Registry 1995. *Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs).* August.

Analitis, A., Katsouyanni, K., Dimakopoulou, K., Samoli, E., Nikoloulopoulos, A. K., Petasakis, Y., *et al.* 2006. *Short-Term Effects of Ambient Particles on Cardiovascular and Respiratory Mortality.* Epidemiology, 17(2), 230-233.

Ballester, F., Saez, M., Perez-Hoyos, S., Iniguez, C., Gandarillas, A., Tobias, A., *et al.* 2002. *The EMECAM Project: A Multicentre Study on Air Pollution and Mortality in Spain: Combined Results for Particulates and for Sulfur Dioxide.* Occup Environ Med, 59(5), 300-308.

Bayer-Oglesby, L., Grize, L., Gassner, M., Takken-Sahli, K., Sennhauser, F. H., Neu, U., *et al.* 2005. *Decline of Ambient Air Pollution Levels and Improved Respiratory Health in Swiss Children.* Environ Health Perspect, 113(11).

Beelen, R., Hoek, G., van den Brandt, P. A., Goldbohm, R. A., Fischer, P., Schouten, L. J., *et al.* 2008. *Long-Term Effects of Traffic-Related Air Pollution on Mortality in a Dutch Cohort (NLCS-AIR Study).* Environ Health Perspect, *116*(2), 196-202.

Bell, M. L., Dominici, F., and Samet, J. M. 2005. *A Meta-Analysis of Time-Series Studies of Ozone and Mortality with Comparison to the National Morbidity, Mortality, and Air Pollution Study.* Epidemiology, 16(4), 436-445.

Bell, M. L., Ebisu, K., Peng, R. D., Walker, J., Samet, J. M., Zeger, S. L., *et al.* 2008. *Seasonal and Regional Short-Term Effects of Fine Particles on Hospital Admissions in 202 US Counties, 1999-2005.* Am J Epidemiol, 168(11), 1301-1310.

Bell, M. L., McDermott, A., Zeger, S. L., Samet, J. M., and Dominici, F. 2004. *Ozone and Short-Term Mortality in 95 US Urban Communities, 1987-2000.* JAMA, 292(19), 2372-2378.

Bell, M. L., Samet, J. M., and Dominici, F. 2004. *Time-Series Studies of Particulate Matter*. Annu Rev Public Health, 25, 247-280.

Brauer, M., Lencar, C., Tamburic, L., Koehoorn, M., Demers, P., and Karr, C. 2008. *A Cohort Study of Traffic-Related Air Pollution Impacts on Birth Outcomes.* Environ Health Perspect. 116, 680-686.

Brook, R. D., Rajagopalan, S., Pope, C. A., III, Brook, J. R., Bhatnagar, A., Diez-Roux, A. V., *et al.* 2010. *Particulate Matter Air Pollution and Cardiovascular Disease: An Update to the Scientific Statement from the American Heart Association*. Circulation, 121(21), 2331-2378.

Brunekreef, B. and Forsberg, B. 2005. *Epidemiological Evidence of Effects of Coarse Airborne Particles on Health*. Eur Respir J, 26(2), 309-318.

Cakmak, S., Dales, R. E., and Judek, S. 2006. *Respiratory Health Effects of Air Pollution Gases: Modification by Education and Income.* Arch Environ Occup Health, 61(1), 5-10.

Clancy, L., Goodman, P., Sinclair, H., and Dockery, D. W. 2002. *Effect of Air-Pollution Control on Death Rates in Dublin, Ireland: An Intervention Study.* The Lancet, 360(9341), 1210-1214.

D'Ippoliti, D., Forastiere, F., Ancona, C., Agabiti, N., Fusco, D., Michelozzi, P., *et al.* 2003. *Air Pollution and Myocardial Infarction in Rome: A Case-Crossover Analysis*. Epidemiology, 14, 528-535.

Dockery, D. W., Pope, C. A., III, Xu, X., Spengler, J. D., Ware, J. H., Fay, M. E., *et al*. 1993. *An Association Between Air Pollution and Mortality in Six U.S. Cities.* N Engl J Med, 329(24), 1753-1759.

Dominici, F., McDermott, A., Daniels, M., Zeger, S. L., and Samet, J. M. 2005. *Revised Analyses of The National Morbidity, Mortality, and Air Pollution Study: Mortality Among Residents of 90 Cities.* J Toxicol Environ Health A, 68(13-14), 1071-1092.

Dominici, F., McDermott, A., Zeger, S. L., and Samet, J. M. 2002. *On the Use of Generalized Additive Models in Time-Series Studies of Air Pollution and Health*. Am J Epidemiol, 156(3), 193-203.

Dominici, F., Peng, R. D., Bell, M. L., Pham, L., McDermott, A., Zeger, S. L., *et al.* 2006. *Fine Particulate Air Pollution and Hospital Admission for Cardiovascular and Respiratory Diseases.* JAMA, 295(10), 1127-1134.

Filleul, L., Rondeau, V., Vandentorren, S., Le Moual, N., Cantagrel, A., Annesi-Maesano, I., *et al.* 2005. *Twenty Five Year Mortality and Air Pollution: Results from the French PAARC Survey*. Occup Environ Med, 62(7), 453-460.

Forbes, L. J., Patel, M. D., Rudnicka, A. R., Cook, D. G., Bush, T., Stedman, J. R., *et al.* 2009. *Chronic Exposure to Outdoor Air Pollution and Diagnosed Cardiovascular Disease: Meta-Analysis of Three Large Cross-Sectional Surveys*. Environ Health, 8, 30.

Friedman, M. S., Powell, K. E., Hutwagner, L., Graham, L. M., and Teague, W. G. 2001. *Impact of Changes in Transportation and Commuting Behaviors During the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma.* JAMA, 285(7), 897-905.

Fung, K. Y., Luginaah, I., Gorey, K. M., and Webster, G. 2005. *Air Pollution and Daily Hospital Admissions for Cardiovascular Diseases in Windsor, Ontario*. Can J Public Health, 96(1), 29-33.

Garshick, E., Laden, F., Hart, J.E., Caron, A. 2003. *Residence Near a Major Road and Respiratory Symptoms in U.S. Veterans.* Epidemiology, *14*(6), 728-736.

Gauderman WJ, A. E., Lurmann, F, Kuenzli N, Gilliland F, Peters J, and McConnell R. 2005. *Childhood Asthma and Exposure to Traffic and Nitrogen Dioxide*. Epidemiology, 16(6) 737-743.

Goldberg, M. S., Burnett, R. T., Brook, J., Bailar, J. C., III, Valois, M. F., and Vincent, R. 2001. *Associations Between Daily Cause-Specific Mortality and Concentrations of Ground-Level Ozone in Montreal, Quebec*. Am J Epidemiol, 154(9), 817-826.

Health Effects Institute (HEI) 2010. *Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposures, and Health Effects*.

Hedley, A. J., Wong, C.-M., Thach, T. Q., Ma, S., Lam, T.-H., and Anderson, H. R. 2002. *Cardiorespiratory and All-Cause Mortality After Restrictions on Sulphur Content of Fuel in Hong Kong: An Intervention Study*. The Lancet, 360(9346), 1646-1652.

Hong, Y. C., Lee, J. T., Kim, H., and Kwon, H. J. 2002. *Air Pollution: A New Risk Factor in Ischemic Stroke Mortality*. Stroke, 33(9), 2165-2169.

International Agency for Research on Cancer (IARC) 2010. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans; Some Non-heterocyclic Polycyclic Aromatic Hydrocarbons and Some Related Exposures.* Volume 92.

International Agency for Research on Cancer (IARC) 1997. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Polychlorinated Dibenzo-para-dioxins and Polychlorinated Dibenzofurans.* Volume 69.Ito, K., De Leon, S. F., and Lippmann, M. 2005. *Associations Between Ozone and Daily Mortality: Analysis and Meta-Analysis.* Epidemiology, *16*(4), 446-457.

Janke, K., Propper, C., and Henderson, J. 2009. *Do Current Levels of Air Pollution Kill? The Impact of Air Pollution on Population Mortality in England*. Health Econ, 18(9), 1031-1055.

Jerrett, M., Burnett, R. T., Brook, J., Kanaroglou, P., Giovis, C., Finkelstein, N., *et al.* 2004. *Do Socioeconomic Characteristics Modify the Short Term Association Between Air Pollution And Mortality? Evidence from a Zonal Time Series in Hamilton, Canada.* J Epidemiol Community Health, 58(1), 31-40.

Jerrett, M., Burnett, R. T., Ma, R., Pope, C. A., III, Krewski, D., Newbold, K. B., *et al.* 2005. *Spatial Analysis of Air Pollution and Mortality in Los Angeles*. Epidemiology, 16(6), 727-736.

Jerrett, M., Burnett, R. T., Pope, C. A., III, Ito, K., Thurston, G., Krewski, D., *et al.* 2009. *Long-Term Ozone Exposure and Mortality*. N Engl J Med, 360(11), 1085-1095.

Jerrett, M., Eyles, J., Dufournaud, C., and Birch, S. 2003. *Environmental Influences on Healthcare Expenditures: An Exploratory Analysis from Ontario, Canada*. J Epidemiol Community Health, *57*(5), 334-338.

Jerrett, M., Shankardass, K., Berhane, K., Gauderman, W. J., Kãnzli, N., Avol, E., *et al.* 2008. *Traffic-Related Air Pollution and Asthma Onset in Children: A Prospective Cohort Study with Individual Exposure Measurement.* Environ Health Perspect, 116(10), 1433-1438.

Katsouyanni, K. 2003. *Ambient Air Pollution and Health*. Br Med Bull, 68, 143-156.

Krewski, D. 2009. *Evaluating the Effects of Ambient Air Pollution on Life Expectancy*. N Engl J Med, 360(4), 413-415.

Krewski, D., Burnett, R., Jerrett, M., Pope, C. A., Rainham, D., Calle, E., *et al*. 2005. *Mortality and Long-Term Exposure to Ambient Air Pollution: Ongoing Analyses Based on the American Cancer Society Cohort*. Journal of Toxicology and Environmental Health, Part A: Current Issues, 68(13), 1093 - 1109.

Kunzli, N., Jerrett, M., Mack, W. J., Beckerman, B., LaBree, L., Gilliland, F., *et al.* 2005. *Ambient Air Pollution and Atherosclerosis in Los Angeles.* Environ Health Perspect, *113*(2), 201-206.

Laden, F., Schwartz, J., Speizer, F. E., and Dockery, D. W. 2006. *Reduction in Fine Particulate Air Pollution and Mortality: Extended Follow-up of the Harvard Six Cities Study*. Am. J. Respir. Crit. Care Med., 173(6), 667-672.

Larrieu, S., Jusot, J. F., Blanchard, M., Prouvost, H., Declercq, C., Fabre, P., *et al.* 2007. *Short Term Effects of Air Pollution on Hospitalizations for Cardiovascular Diseases in Eight French Cities: The PSAS Program.* Sci Total Environ, *387*(1-3), 105-112.

Luginaah, I. N., Fung, K. Y., Gorey, K. M., Webster, G., and Wills, C. 2005. *Association of Ambient Air Pollution with Respiratory Hospitalization in a Government-Designated "Area of Concern": The Case of Windsor, Ontario.* Environ Health Perspect, 113(3), 290-296.

Medina-Ramon, M., Zanobetti, A., and Schwartz, J. 2006. *The Effect of Ozone and PM_{10} on Hospital Admissions for Pneumonia and Chronic Obstructive Pulmonary Disease: A National Multicity Study.* Am J Epidemiol, 163(6), 579-588.

Mindell, J., and Joffe, M. 2004. *Predicted Health Impacts of Urban Air Quality Management*. J Epidemiol Community Health, 58(2), 103-113.

Neuberger, M., Moshammer, H., and Kundi, M. 2002. *Declining Ambient Air Pollution and Lung Function Improvement in Austrian Children*. Atmospheric Environment, 36(11), 1733-1736.

Neupane, B., Jerrett, M., Burnett, R. T., Marrie, T., Arain, A., and Loeb, M. 2010. *Long-Term Exposure to Ambient Air Pollution and Risk of Hospitalization with Community-Acquired Pneumonia in Older Adults*. Am J Respir Crit Care Med, 181(1), 47-53.

Ostro, B., Broadwin, R., Green, S., Feng, W. Y., & Lipsett, M. 2006. *Fine Particulate Air Pollution and Mortality in Nine California Counties: Results from CALFINE*. Environ Health Perspect, *114*(1), 29-33.

Peng, R. D., Chang, H. H., Bell, M. L., McDermott, A., Zeger, S. L., Samet, J. M., *et al*. 2008. *Coarse Particulate Matter Air Pollution and Hospital Admissions for Cardiovascular and Respiratory Diseases Among Medicare Patients*. JAMA, 299(18), 2172-2179.

Pengelly, L. D., Szakolcai, A., Birmingham, B., Cole, D., Muller, P., Bailey, S., *et al.* 1997. *Human Health Risk Assessment for Priority Air Pollutants*: Regional Municipality of Hamilton-Wentworth.

Peters, A., Dockery, D. W., Muller, J. E., and Mittleman, M. A. 2001. *Increased Particulate Air Pollution and the Triggering of Myocardial Infarction.* Circulation, 103(23), 2810-2815.

Peters, A., von Klot, S., Heier, M., Trentinaglia, I., Hormann, A., Wichmann, H. E., *et al*. 2004. *Exposure to Traffic and the Onset of Myocardial Infarction*. N Engl J Med, 351(17), 1721-1730.

Pope, C. A., III 1989. *Respiratory Disease Associated with Community Air Pollution and a Steel Mill, Utah Valley.* American Journal of Public Health, 79, 623-628.

Pope, C. A., III, Burnett, R. T., Thun, M. J., Calle, E. E., Krewski, D., Ito, K., *et al.* 2002. *Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution.* JAMA, 287(9), 1132-1141.

Pope, C. A., III, Burnett, R. T., Thurston, G. D., Thun, M. J., Calle, E. E., Krewski, D., *et al*. 2004. *Cardiovascular Mortality and Long-Term Exposure to Particulate Air Pollution: Epidemiological Evidence of General Pathophysiological Pathways of Disease*. Circulation, 109(1), 71-77.

Pope, C. A., III, and Dockery, D. W. 2006. *Health Effects of Fine Particulate Air Pollution: Lines that Connect.* Journal of Air & Waste Management Association, 56 (6), 709-742.

Pope, C. A., III, Ezzati, M., and Dockery, D. W. 2009. *Fine-Particulate Air Pollution and Life Expectancy in the United States*. N Engl J Med, *360*(4), 376-386.

Pope, C. A., III, Thun, M. J., Namboodiri, M. M., Dockery, D. W., Evans, J. S., Speizer, F. E., *et al.* 1995. *Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults.* Am J Respir Crit Care Med, 151(3 Pt 1), 669-674.

Puett, R. C., Hart, J. E., Yanosky, J. D., Paciorek, C., Schwartz, J., Suh, H., *et al*. 2009. *Chronic Fine and Coarse Particulate Exposure, Mortality, and Coronary Heart Disease in the Nurses' Health Study*. Environ Health Perspect, 117(11), 1697-1701.

Rosenlund, M., Bellander, T., Nordquist, T., and Alfredsson, L. 2009. *Traffic-Generated Air Pollution and Myocardial Infarction*. Epidemiology, 20(2), 265-271.

Ryan, P. H. L., G.; Biagnini, J.; Bernstein, D.; Grinshpun, S.A.; Shukla, R.; Wilson, M.S.; Villareal, M.; Burkle, J.; and Lockey, J. 2005. *Is It Traffic Type, Volume, Or Distance? Wheezing in Infants Living Near Truck and Bus Traffic.* J Allergy Clin Immunol, 116, 279-284.

Sahsuvaroglu, T., and Jerrett, M. 2003. *A Public Health Assessment of Mortality and Hospital Admissions Attributable to Air Pollution in Hamilton*. Hamilton: McMaster Institute of Environment and Health.

Samet, J., and Krewski, D. 2007. *Health Effects Associated With Exposure to Ambient Air Pollution*. Journal of Toxicology and Environmental Health, Part A: Current Issues, 70(3), 227 - 242.

Samoli, E., Peng, R., Ramsay, T., Pipikou, M., Touloumi, G., Dominici, F., *et al*. 2008. *Acute Effects of Ambient Particulate Matter on Mortality in Europe and North America: Results from the APHENA Study*. Environ Health Perspect, 116(11), 1480-1486.

Schwartz, J. 2001. *Air Pollution and Blood Markers of Cardiovascular Risk.* Environ Health Perspect, 109 Suppl 3, 405-409.

Schwartz, J., Coull, B., Laden, F., and Ryan, L. 2008. *The Effect of Dose and Timing of Dose on the Association Between Airborne Particles and Survival.* Environ Health Perspect, 116(1), 64-69.

Souza, M. B., Saldiva, P. H., Pope, C. A., III, and Capelozzi, V. L. 1998. *Respiratory Changes Due to Long-Term Exposure to Urban Levels of Air Pollution: A Histopathologic Study in Humans.* Chest, 113(5), 1312-1318.

Stieb, D. M., Judek, S., and Burnett, R. T. 2002. *Meta-Analysis of Time-Series Studies of Air Pollution and Mortality: Effects of Gases and Particles and the Influence of Cause of Death, Age, and Season.* J Air Waste Manag Assoc, 52(4), 470-484.

Stieb, D. M., Szyszkowicz, M., Rowe, B. H., and Leech, J. A. 2009. *Air Pollution and Emergency Department Visits for Cardiac and Respiratory Conditions: A Multi-City Time-Series Analysis.* Environ Health, 8, 25.

Sullivan, J., Sheppard, L., Schreuder, A., Ishikawa, N., Siscovick, D., and Kaufman, J. 2005. *Relation Between Short-Term Fine-Particulate Matter Exposure and Onset of Myocardial Infarction*. Epidemiology, 16(1), 41-48.

Sunyer, J., Ballester, F., Tertre, A. L., Atkinson, R., Ayres, J. G., Forastiere, F., *et al.* 2003. *The Association of Daily Sulfur Dioxide Air Pollution Levels with Hospital Admissions for Cardiovascular Diseases in Europe (The Aphea-II study)*. Eur Heart J, *24*(8), 752-760.

Szyszkowicz, M. 2007. *Air Pollution and Emergency Department Visits for Ischemic Heart Disease in Montreal, Canada*. Int J Occup Med Environ Health, 20(2), 167-173.

Szyszkowicz, M. 2008. *Ambient Air Pollution and Daily Emergency Department Visits for Ischemic Stroke in Edmonton, Canada.* Int J Occup Med Environ Health, 21(4), 295-300.

Szyszkowicz, M. 2009. *Air Pollution and ED Visits for Chest Pain*. Am J Emerg Med, 27(2), 165-168.

United States Environmental Protection Agency (USEPA) 2009. *Provisional Assessment of Recent Studies on Health and Ecological Effects of Ozone Exposure*. Washington, DC.

Villeneuve, P. J., Burnett, R. T., Shi, Y., Krewski, D., Goldberg, M. S., Hertzman, C., *et al.* 2003. *A Time-Series Study of Air Pollution, Socioeconomic Status, and Mortality in Vancouver, Canada*. J Expo Anal Environ Epidemiol, 13(6), 427-435.

Villeneuve, P. J., Chen, L., Stieb, D., and Rowe, B. H. 2006. *Associations Between Outdoor Air Pollution and Emergency Department Visits for Stroke in Edmonton, Canada*. Eur J Epidemiol, 21(9), 689-700.

von Klot, S., Peters, A., Aalto, P., Bellander, T., Berglind, N., D'Ippoliti, D., *et al.* 2005. *Ambient Air Pollution is Associated with Increased Risk of Hospital Cardiac Readmissions of Myocardial Infarction Survivors in Five European Cities*. Circulation, 112, 3073 - 3079.

Wellenius, G. A., Schwartz, J., and Mittleman, M. A. 2006. *Particulate Air Pollution and Hospital Admissions for Congestive Heart Failure in Seven United States Cities*. Am J Cardiol, 97(3), 404-408.

World Health Organization (WHO) 2006. *Health Risks of Particulate Matter from Long Range Transboundary Air Pollution*. Bonn Office.

Wilson, A. M., Wake, C. P., Kelly, T., and Salloway, J. C. 2005. *Air Pollution, Weather, and Respiratory Emergency Room Visits in Two Northern New England Cities: An Ecological Time-Series Study.* Environ Res, 97(3), 312-321.

Wong, E. Y., Gohlke, J., Griffith, W. C., Farrow, S., and Faustman, E. M. 2003. *Assessing the Health Benefits of Air Pollution Reduction for Children.* Environ Health Perspect, 112(2) 226-232.

Zanobetti, A., and Schwartz, J. 2005. *The Effect of Particulate Air Pollution on Emergency Admissions for Myocardial Infarction: A Multicity Case-Crossover Analysis*. Environ Health Perspect, 113(8), 978-982.

Zanobetti, A., and Schwartz, J. 2006. *Air Pollution and Emergency Admissions in Boston, MA*. J Epidemiol Community Health, 60(10), 890-895.

Zanobetti, A., and Schwartz, J. 2009. *The Effect of Fine and Coarse Particulate Air Pollution on Mortality: A National Analysis.* Environ Health Perspect, 117(6), 898-903.

Zanobetti, A., Schwartz, J., Samoli, E., Gryparis, A., Touloumi, G., Atkinson, R., *et al*. 2002. *The Temporal Pattern of Mortality Responses to Air Pollution: A Multicity Assessment of Mortality Displacement.* Epidemiology, 13(1), 87-93.

Zeger, S. L., Dominici, F., McDermott, A., and Samet, J. M. 2008. *Mortality in the Medicare Population and Chronic Exposure to Fine Particulate Air Pollution in Urban Centers (2000-2005)*. Environ Health Perspect, 116(12), 1614-1619.

Appendix A

Summary of the Literature Considered to Derive Relative Risks

Appendix A: Summary of the Literature Considered to Derive Relative Risks

Table A.1: Health Studies from Literature used in Selecting Relative Risks for PM_{10}

Reference	Location	Study Type	Details	NT Mortality per 10 ug/m3		CI@ 95%
Short Term						
Bell, et al (2004) b	US	Summary Paper	NMMAPS	0.21		0.04-0.33
Bell, et al (2004) b	Europe	Summary Paper	Western Europe (APHEA)	0.40		0.2-0.6%
Bell, et al (2004) b	Europe	Summary Paper	Central Eastern Europe (APHEA)	0.80		0.06-1.8%
Bell, et al (2004) b	Europe	Summary Paper	APHEA 2	0.60		0.4-0.8
Brook, et al (2010)	World	Summary Paper	0.2 to 0.6% for 10 µg/m3 increase in PM10	0.20	0.60	-
Dominici, et al (2005)	US	Reanalysis short term	NMMAPS (original)	0.41		0.35-0.47
Dominici, et al (2005)	US	Reanalysis short term	NMMAPS (stringent GAM)	0.27		0.21-0.33
Dominici, et al (2005)	US	Reanalysis short term	NMMAPS (GLM)	0.21		0.15-0.27
Samoli, et al (2008)	US	Short Term (multicity)	Total Mortality, Lag 1	0.29		0.18-0.40
Samoli, et al (2008)	Canada	Short Term (multicity)	Total Mortality, Lag 1	0.84		0.3-1.4
Samoli, et al (2008)	Europe	Short Term (multicity)	Total Mortality, Lag 1	0.33		0.22-0.44
Zanobetti, et al (2002)	Europe	Short Term (multicity)	mean lag01	0.70		0.43-0.97
			Average	0.45		
Reference	**Location**	**Study Type**	**Details**	**CV and Respiratory Disease**		**CI@ 95%**
Short Term						
Larrieu et al (2007)	France	Short Term (multicity)	Cardiovascular ICD 10 - I00-I99	0.7		0.1-1.2

Note: values adopted as relative risks for the current study are shaded

Table A.2: Health Studies from Literature used in Selecting Relative Risks for $PM_{2.5}$

Reference	Location	Study Type	Details	NT Mortality for 10ug/m3		CI@ 95%
Short Term (Multicity)						
Bell, et al (2004) B	US/World	Summary Paper	H6C study	0.10		-
Bell, et al (2004) B	US/World	Summary Paper	ACS II	0.40		-
Brook, et al (2010)	World	Summary Paper	0.4 to 1% increase in cardio mortality for a 10 µg/m^3 increase in PM2.5 during short-term. 1.06 to 1.76% increase for long term exposure.World-wide: 0.4 to 1% increase in daily mortality for a 10 µg/m3 increase in PM2.5 within 5 days	0.40	1.00	-
Brunekreef, et al (2005)	US	Summary Paper	Fine Particles: 0.3-2.9% increase in mortality per 10 10 µg/m^3 increase. For Respiratory deaths, as high as 7.4%. Coarse Particles: 0.4 to 4.1% increase per per 10 µg/m3 increase.	0.30	2.90	-
Ostro, et al (2006)	USA	Short Term (multicity)	NT mortality	0.10		0.2-1
Zanobetti and Schwartz (2009)	US	Short Term (multicity)	all cause mortality ICD10: A00-R99	0.98		0.75-1.22
			Average	0.77		
Long Term						
Beelen, et al (2008)	Netherlands	Long term	NT mortality	1.06		0.97-1.16
Jerrett, et al (2005)	Los Angeles, US	Long term	After controlling for 44 individual covariates	17		5-30%
Jerrett, et al (2009)	USA	Long term	single poll	4.8		2.4-7.1
Jerrett, et al (2009)	USA	Long term	controlling for O3	8		4.8-11.3
Krewski, et al (2005)	US	Long Term	ACS - ongoing extended analysis	6		2-11%
Laden, et al (2006)	US	Long term	NT mortality	16		7-26%
Pope, et al (2002)	US	Long term	all cause mortality	4		1-8%
Puett et al (2009)	US	Long term	HR all cause mortality (nurses study) multipoll	1.29		1.03-1.62
Zeger, et al (2008)	US	Long term Multi City	Eastern US (>65)	6.8		4.9-8.7
Zeger, et al (2008)	US	Long term Multi City	Central US (>65)	13.2		9.5-16.9

Table A.2: Health Studies from Literature used in Selecting Relative Risks for $PM_{2.5}$ (Cont'd)

Reference	Location	Study Type	Details	CV and Resp Mortality	CI@ 95%
Zanobetti and Schwartz (2009)	US	Short Term (multicity)	Respiratory deaths (ICD10: J00-J99)	1.68	1.04-2.33
Ostro, et al (2006)	USA	Short Term (multicity)	CV deaths (ICD10 I00-I99)	0.60%	0-1.1
Bell et al 2008 Peng et al 2008	USA	Short Term (multicity)	CV Hospitalization	0.8	0.6-1.0
Long Term					
Beelen, et al (2008)	Netherlands	Long term	CV mort	1.04	0.9-1.21
Beelen, et al (2008)	Netherlands	Long term	Resp Mort	1.07	0.75-1.52
Jerrett, et al (2005)	Los Angeles, US	Long term	After controlling for 44 individual covariates	1.2-1.6%	-

Note: values adopted as relative risks for the current study are shaded

Table A.3: Health Studies from Literature used in Selecting Relative Risks for SO_2

Reference	Location	Study Type	Details	NT Mortality 10ug/m3	CI@ 95%
Short Term					
Ballester, et al (2002)	Spain	Short Term (multicity)	24 hr (with one day lag) multipoll	0.2	0.09-0.7
Ballester, et al (2002)	Spain	Short Term (multicity)	1 hr max	0.2	0-0.3
Meta analysis					
Stieb, et al (2002)	World	Metanalysis	RE pooled estimates	0.36	0.28-0.48
Reference	**Location**	**Study Type**	**Details**	**CV and Resp Disease**	**CI@ 95%**
Short Term					
Wilson, et al (2005)	US	Short term	respiratory admissions (ICD9: 460-519)	3	1-4%

Note: values adopted as relative risks for the current study are shaded

Table A.4: Health Studies from Literature used in Selecting Relative Risks for NO_2

Reference	Location	Study Type	Details	NT Mortality for 10 ppb	CI@ 95%
Short Term					
Villeneuve, et al (2003)	Vancouver, Canada	Short Term	daily mean, lag 1, all cause mort	0.2	0.45-3.6
Stieb, et al (2002)	World	Metanalysis	RE pooled estimates	1.16	0.88-1.46
			Average	0.68	
Long Term					
Beelen, et al (2008)	Netherlands	Long term	0.80-1.26% increase in mortality for a 10 µg/m^3 increase	0.68	0.63-0.73
Filleul, at al (2005)	France	Long Term	a RR of 1.14 in overall mortality for a 10 µg/m3 increase in NO2	14	3-25%
Reference	**Location**	**Study Type**	**Details**	**CV and Resp Disease**	**CI@ 95%**
Short Term					
Larrieu et al (2007)	France	Short-Term multicity	Cardiovascular ICD 10 - I00-I99	0.94	0.19-1.89

Note: values adopted as relative risks for the current study are shaded

Table A.5: Health Studies from Literature used in Selecting Relative Risks for CO

Reference	Location	Study Type	Details	NT Mortality 10 ppb	CI@ 95%
Meta Analysis					
Stieb, et al (2002)	World	Metanalysis	RE pooled estimates	1.70	1.08-1.98
Reference	**Location**	**Study Type**	**Details**	**CV and Resp Disease**	**CI@ 95%**
Short term					
Luginaah, et al (2005)	Canada	Short Term	Females (0-14 yrs) with 2-day lag, time series ICD 9: 460-519 (resp. disease)	3.53	0.05-7.22

Note: values adopted as relative risks for the current study are shaded

Table A.6: Health Studies from Literature used in Selecting Relative Risks for O_3

Reference	Location	Study Type	Details	NT Mortality for 10ppb	CI@ 95%
Short Term					
Janke, et al (2009)	UK	Short Term	multipoll, short term controlling for CO, PM10, NO2	1.57	1-2.14
Bell, et al (2004) A	US	Short Term (multicity)	95 US urban communities (1987-2000)	0.52	0.27-0.77
Bell, et al (2005)	US	Meta analysis for short term	Metanalysis results (and compared to NMMAPS)	0.87	0.55-1.18
Ito, et al (2005)	US	Meta analysis for short term	Combined estimate of 43 studies 1 hr daily max ozone, single O3	0.39	0.26-0.51
Ito, et al (2005)	US	Meta analysis for short term	Adjusting for PM	0.37	0.2-0.54
Ito, et al (2005)	US	Meta analysis for short term	combined random effects estimate 24 hour average ozone	0.8	0.55-1
Stieb, et al (2002)	World	Metanalysis	RE pooled estimates	0.5	0.32-0.38
			Average	0.72	
Reference	**Location**	**Study Type**	**Details**	**CV and Resp Disease**	**CI@ 95%**
Short Term					
Larrieu et al (2007)	France	Short Term (multicity)	a10 $\mu g/m^3$ increase in Ozone results in RR=0.1% of cardiovascular disease hospitalization (0.2 % per 10 ppb)	0.2	-

Note: values adopted as relative risks for the current study are shaded

Appendix B

Conditions of Data Sharing (City of Hamilton, Public Health Services) and Data Dissemination Policy (City of Hamilton, Corporate Services)

Appendix B: Conditions of Data Sharing (City of Hamilton, Public Health Services) and Data Dissemination Policy (City of Hamilton, Corporate Services

CONDITIONS OF DATA SHARING

Under specific terms and conditions of a data sharing agreement with the Ontario Ministry of Health and Long-term Care, the City of Hamilton Public Health Services has access to mortality and hospital discharge data via the IntelliHEALTH system. The data provided for Air Quality and Health Impact Study must be used in accordance with the following conditions in order to satisfy the requirements outlined in that agreement:

- The IntelliHEALTH citations specified below must be included as they appear wherever reference is made to the data provided.

  Mortality Data:
  Death Table, Vital Statistics, Ontario Ministry of Health and Long-Term Care. IntelliHEALTH ONTARIO, Extracted July 2010

  Hospitalization Data:
  Inpatient discharges Main Table, Inpatient Discharges, Ontario Ministry of Health and Long-Term Care. IntelliHEALTH ONTARIO, Extracted September 2010

- Data provided is to be used solely for the purposes of the proposed Air Quality and Health Impact Study and no other purpose.

- Consent must be sought from the City of Hamilton Public Health Services of any intention to publish or present a report external to the City of Hamilton containing reference to the data provided. Notification of the intention to publish or present should be given to the City of Hamilton Public Health Services at least **sixty (60) days** prior to the publication or presentation.

- Data notes as written must appear where appropriate in the full report (e.g. appendices). Where appropriate in the full report (e.g. discussion section), data notes should be used as explanations of the limitations of the data provided and the implications of the results.

- Age and sex breakdowns are provided only to allow for age and sex standardization of the data. The full data tables provided must not be reproduced in any section of the report.

MORTALITY DATA

Mortality data in these tables describes the main/primary causes of death indicated by data from death certificates from the Ontario Office of the Registrar General. Geographic information is based on place of residence from the municipality on the death certificate, not where the death occurred. City of Hamilton residents who died anywhere in Ontario are included in the data provided. The data presented are based on underlying cause of death (i.e. the disease or injury which initiated the events leading directly to death), classified by the codes of the International Statistical Classification of Diseases and Health Related Problems 9th and 10th Revisions (see ICD-9 to ICD-10 Conversion paragraph for more information). Mortality data is available for calendar years 1986-2005.

HOSPITAL DISCHARGE DATA

Hospital discharge data in these tables describes the main causes of hospitalization indicated by acute care inpatient discharges and excludes birth-related discharges of newborn and stillborn infants. Counts do not represent individuals but counts of discharge records. Some individuals are admitted to hospital several times for the same condition and some conditions are more likely to have multiple hospital admittance compared to others. Geographic information is based on patient's place of residence from the reported municipality at the time of admission. City of

Hamilton residents hospitalized in the province of Ontario are included. Data are based on most responsible diagnosis (i.e. the diagnosis with the longest duration of treatment), classified by the codes of the International Statistical Classification of Diseases and Health Related Problems 9th and 10th Revisions (see ICD-9 to ICD-10 Conversion paragraph for more information). Hospitalization data is available for calendar years 1997-2008. Factors other than need can also affect hospitalization rates so trends should be interpreted with caution.

ICD-9 TO ICD-10 CONVERSION

ICD codes are revised periodically to reflect new medical information. The 10th Revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-10) represents a considerable change from the 9th revision (ICD-9). Hospitalization data for the fiscal years 1996 to 2001 are coded based on ICD-9 and data from 2002 to 2008 are coded based on ICD-10. Hospitalization data presented for the calendar year 2002 contains both ICD-9 and ICD-10 codes. Mortality data for the calendar years 1986 to 1999 are coded based on ICD-9 and data from 2000 to 2005 are coded based on ICD-10. Changes between revisions may impact the comparability of hospitalization and mortality data based on different ICD versions if significant differences in the way the data are coded were made. Trends must be interpreted with caution. Further information about code comparability can be found here: http://www.statcan.gc.ca/pub/84-548-x/84-548-x2005001-eng.pdf

ICD-9 AND ICD-10 CODES

The following table describes the codes used to extract the mortality and hospitalization data:

Mortality Data	**ICD-9**	**ICD-10**
All Non-Traumatic	001-799	A00-R999
Disease of the Circulatory System	390-459	I00-I999
Disease of the Respiratory System	460-519	J00-J999
Hospitalization Data	**ICD-9**	**ICD-10**
All Non-Traumatic	001-799	A00-R999
Disease of the Circulatory System	390-459	I00-I999
Congestive Heart Failure	428	I500
Disease of the Respiratory System	460-519	J00-J999

Hamilton

1.1 CITY OF HAMILTON

1.2 CORPORATE SERVICES DEPARTMENT

1.3 INFORMATION SERVICES

1.4 BUSINESS APPLICATIONS SECTION

SUBJECT	**DATA DISSEMINATION POLICY**
AUTHORITY	**BUSINESS APPLICATIONS SECTION, INFORMATION SERVICES, CORPORATE SERVICES, CITY OF HAMILTON**

LICENCE POLICY STATEMENT

This is a licence policy to define what the City of Hamilton charges for the dissemination of geographic data, specifically in digital form. The licence policy ensures that consistent and equitable service is provided to the public, and ensures that costs are kept to a minimum by basing fees on cost recovery. Recovery of costs will be based on dissemination costs only and will not include the cost of acquiring, developing or maintaining the original data. When dealing with standard hard copy maps created by the City of Hamilton, from geographic data, the Corporate Pricing Policy shall apply.

APPLICATION

This licence policy defines the type of data covered, the types of charges for the data by category of data client, and provides a schedule of costs for data items, materials and custom services. The licence policy is intended to be used by staff in fulfilling requests for data from internal and external clients.

Implementation of this licence policy will commence on April 15, 2010, with the Business Applications Manager's authority to establish, revise and waive fees as appropriate, and the authority to delegate responsibility for granting exemptions from fees to the senior staff within the Business Applications Section.

1. Data Covered by Licence Policy

All geographic data which is stored on the City of Hamilton's various computer systems are covered by this policy. For the purposes of this document, "geographic data" can be defined as:

> ***"...data or information derived from data that is stored within computer systems by latitude/longitude, or other similar spatial referencing system. This includes all descriptive, or attribute data which is "linked" or "related" to the spatial referencing system by codes, keys, or similar means."***

2. Target Groups Subject to Licence Policy

For the purposes of interpreting and implementing this policy, the following categories of clients have been identified who are typical clients requesting geographic data and who would be covered by this policy:

Category "A" includes internal clients of the City of Hamilton, such as City of Hamilton departments, Police Services, Fire Department, etc.

> ***Category A clients are exempt from fees for geographic data and are not required to enter into a licence agreement but have an understanding of the data dissemination and licence policy. When the data or custom mapping is required for a capital project, data handling fees, hard copy map/publication fees and material fees will be charged. All fees may be waived at the discretion of the Business Applications Manager or the respective department Head.***

1.4.1 PRICE = DATA HANDLING FEES + MAP PLOTTING FEES + MATERIALS – SUBSIDY

Category "B" includes other municipalities, local school boards, local libraries, other local government agencies or associations, and provincial or federal government agencies involved in local programs, and local media representatives within the City of Hamilton, and includes community associations and consultants (working on behalf of the City of Hamilton, other municipalities, local school boards, local libraries, police services, other local government agencies or associations, and provincial or federal government agencies involved in local programs, and local media representatives within the City of Hamilton).

> ***Category B clients are exempt from fees for geographic data but they are required to pay data handling fees, hard copy map/publication fees and material fees charged at full cost recovery. Category B clients working in partnership with any City of Hamilton department, are exempt from handling and material fees. Category B clients are eligible for full, or partial subsidy, for all fees at the discretion of the Business Applications Manager or the respective department Head. Category B clients are required to enter into a licence agreement for use of the data.***

1.4.2 PRICE = DATA HANDLING FEES + MAP PLOTTING FEES + MATERIALS – SUBSIDY

Category "C" includes corporations, individuals, privately owned utilities (e.g. Bell, Hydro companies, Union Gas, Rogers TV, COGECO Cable, Mountain Cablevision, Southmount Cable), consultants, developers, and commercial ventures purchasing data for limited, ***non-commercial*** uses.

Category C clients must comply with the licence policy as defined herein, which includes full cost recovery for data handling fees, hard copy map/publication fees and material fees. These fees and charges shall in no case be less than full cost recovery. Category C clients are eligible for full or partial subsidy, for all fees at the discretion of the Business Applications Manager or the respective department Head. Category C clients are required to enter into a limited use licence agreement for use of the data.

1.4.3 PRICE = DATA HANDLING FEES + MAP PLOTTING FEES + MATERIALS – SUBSIDY

3. Staff Responsible for Implementation of Licence Policy

All staff within the Business Applications Section and any City of Hamilton staff who are disseminating digital data will be responsible for the implementation of, and adherence to, this licence policy.

4. Legislated Policies Affecting this Licence Policy

(1) Information contained within the geographic databases is subject to the provisions of the Municipal Freedom of Information and Protection of Privacy Act.

(2) Information which is available to the public as paper maps, plans, or reports is considered published and therefore excluded from the provision of the Municipal Freedom of Information and Protection of Privacy Act.

(3) For any electronic databases prepared under the direction or control of the City of Hamilton, the copyright in the work belongs to the City of Hamilton; the City of Hamilton has the exclusive right to use the databases in any manner or to authorize others to copy it.

(4) Responsibility to administer City of Hamilton copyright rests with Legal Services; it is advisable that each department consult with Legal in relation to City of Hamilton copyright issues.

(5) In accordance with the foregoing, direct access to the databases is restricted to employees of the City of Hamilton.

LICENCE POLICY DESCRIPTION

1. Licence Policy Description

This section describes various administrative policies, which affect the dissemination of geographic data.

1. Provision of data is expensive and shall be undertaken only when (a) there is a clear responsibility to inform (publish), (b) there is a benefit to City of Hamilton to do so, or (c) clients are willing to pay for the data.
2. Objectives of full cost recovery are to (a) increase fairness, by ensuring the direct beneficiaries of the data bear the cost, and not the taxpayers of the City of Hamilton, and (b) reduce operating costs.
3. The full costs of providing data to satisfy proprietary interests of individuals or corporations shall not be borne (i.e. subsidised) by the taxpayers of the City of Hamilton.
4. The cost of making data available for purchase to the public or corporations shall reflect the full cost of collecting, compiling, preparing, producing and disseminating the data (the "marginal" cost of disseminating), but not the original cost of acquiring the data.
5. Fees and charges may be reduced or waived where appropriate (e.g. where health and safety issues are involved) by the Manager of Business Applications.
6. Full cost recovery shall be the baseline for establishing external client charges; partial or full subsidy can be justified under the following circumstances:
   - when data is provided to other municipalities, local school boards, police services, other local government agencies or associations, and provincial or federal government agencies involved in local programs, and local media representatives within the City of Hamilton;
   - when data is provided to an individual or corporation under contract to the City of Hamilton, other municipalities, local school boards, police services, other local government agencies or associations, and provincial or federal government agencies involved in local programs, and local media representatives within City of Hamilton.
7. Commercialisation of databases as the City of Hamilton works may be achieved through (a) donation, (b) assignments, (c) loan, and/or (d) licence.
8. Donation or assignment of electronic databases shall require approval of City Council; a loan or licence shall not require Council approval, provided the City of Hamilton's copyright and ownership in the data are protected in the loan or licensing agreement.
9. Data shall be provided as a "non-exclusive" licence (City of Hamilton retains the right to access and distribute its data through other licensees).
10. Complete pro-forma licensing agreements, shall be executed for each dissemination product as described in section 2., Data Licence Agreements, below.
11. Where City of Hamilton is not the original copyright owner of the data (e.g. the data has been licensed to the City of Hamilton, such as Teranet Inc. data), the terms and conditions of the licence shall apply to any "sub-licence".
12. Revenues received by Business Applications for geographic data and hardcopy mapping shall be placed in a Business Applications general revenue account. Revenues generated

via hardcopy mapping at the Departmental level will be collected and shall remain with the Department producing the output.

13. Provision of "hard copy" products (e.g. whiteprints, paper maps, etc.) which have been derived from data or databases shall be at the discretion of Departments; there is an implied copyright on all products; however an effort should be made to place copyright statements on the original documents, as well as other disclaimers and acknowledgements.
14. The cost of making “hard copy” maps available for purchase to the public or corporations shall reflect the full cost of assembling, compiling, preparing, producing and reproducing the data (the "marginal" cost), but not the original cost of acquiring the data.

2. Data Licence Agreements

Data Licence Agreements are required each time data is provided to a client. The Data Licence Agreement shall reflect the policies described wherein. The Data Licence Agreement must be executed between the Licensee and the owner before data is provided or used. It is important to note that there may be different terms and conditions depending on the source of the data. Often there are provisions which must be "passed through" from the original owner to the Licensee.

When a client wishes to re-use the data provided under an existing licence agreement, for a new project, a new licence agreement shall be prepared and an administration fee charged to cover the time for staff to produce the new licence agreement.

One standard form of Data Licence shall be available, with customized conditions for use depending on the nature of the data provided. Where the City of Hamilton is not the sole-owner, a multi-party licence agreement will be used, naming all parties involved. A Data Licence Agreement shall be prepared and signed by all parties prior to delivery of the product. A sample of the Data Licence Agreement is attached as Schedule “A”.

3. Schedule of Costs

The costs of the data dissemination as defined in the above licence policy shall be implemented with the Manager of Business Applications authority to establish, revise and waive fees as appropriate. The current schedule of fees is attached as Schedule “B”.

Hamilton

City of Hamilton
Corporate Services Department
Information Services
Business Applications Section

Schedule "A"

Data Licence Agreement

THIS AGREEMENT dated the **25** day of **October, 2011.**

BETWEEN:

SENES Consulting Limited ("Licensee")

AND

City of Hamilton

WHEREAS City of Hamilton is the owner of digital files containing geographic information covering the City of Hamilton (hereinafter the "Database");

AND WHEREAS City of Hamilton has agreed to grant a restricted, non-transferable, non-exclusive licence to **SENES Consulting Limited** subject to the terms and conditions of this Licence Agreement;

NOW THEREFORE City of Hamilton and the Licensee covenant and agree as follows:

1. Grant of Licence

City of Hamilton hereby grants to the Licensee a restricted, non-transferable, non-exclusive licence to use electronic files of the City of Hamilton's Geographic Database as itemized in Addendum 'A' (hereinafter called the "Licensed Database") royalty-free for the sole purpose of fulfilling its responsibilities and obligations under **Air Quality**. This licence shall commence on execution of this Licence Agreement by all parties and expire on completion of the Project, subject to early termination.

2. Fee Payable

The Licensee shall pay to the City of Hamilton a fee of **$0.00** for the granting of the licence hereunder. The Licensee shall pay the fee to the City of Hamilton within thirty (30) days of receiving an invoice therefore.

3. Restricted Use

The Licensee is authorized to use the Licensed Database solely for its own internal operation and for the sole purpose of fulfilling its responsibilities and obligations under **Air Quality**. The Licensee acknowledges that the Licensed Database is protected by copyright and that the only right, which the Licensee obtains, to the Licensed Database is the right of use in accordance with the terms of this Licence Agreement. Where the Database or portion thereof, is used in combination with other data to produce derived works for distribution to individuals, associations and corporations, it must be provided in a non-digital format. Any third party requiring access to the Licensed Database for the purpose of producing such derived works must execute an agreement, in a form acceptable to the City Solicitor, with the City of Hamilton prior to being given access to the Licensed Database.

The Licensee may engage contractors to perform work on the Licensed Database. The Licensee shall ensure that contractors do not copy the Licensed Database or use the Licensed Database for any purpose

other than providing services for the Licensee with respect to the Licensee's responsibilities and obligations under **Air Quality**, or in any manner in which the Licensee is prohibited under this Licence Agreement.

4. No transfer

The Licensee shall not sub-licence, assign, or otherwise transfer any of the rights, duties, or obligations hereunder without the express written approval of the City, which approval may be unreasonably withheld. Any attempt by the Licensee to sub-licence, assign or transfer any of the rights, duties or obligations hereunder are void, unless same is conducted in accordance with this section. The Licensed Database and any works derived therefrom shall not be sold or distributed to third parties in any manner by the Licensee.

5. No warranty

City of Hamilton (including its officials, officers, directors, employees, representatives, affiliates, volunteers, and agents) shall not be obliged to update the files or the Licensed Database or to make any changes thereto at the request of the Licensee. Further, City of Hamilton (its officials, officers, directors, employees and agents) make no representation or warranty of any kind, express or implied, with respect to the information provided pursuant to this Licence Agreement ("Information"). The Information will be made available to the Licensee on an "as is, where is" basis, without any representations or warranties, express or implied, of accuracy, completeness, currency, usefulness, merchantability, or fitness for any purpose, or those arising by law or usage of trade or course of dealing. The entire risk as to the use, dissemination and/or reliance upon the Information is assumed by the Licensee. Licensee acknowledges and agrees that the City of Hamilton (including its officials, officers, directors, employees and agents) shall not have any liability to any party whatsoever for any claims, actions, loss, damage, including without limitation, loss of revenue or profit or savings, loss of or damage, or any indirect or incidental, special or consequential damages arising from or relating in any way to the use and/or reliance upon information contained herein, including without limitation, for infringement of any party's proprietary rights.

6. Indemnity

Licensee shall indemnify, defend and hold City of Hamilton (including its officials, officers, directors, employees, representatives, affiliates, volunteers and agents) [collectively referred to as the "Indemnitees"] harmless against any and all claims, demands, costs, penalties, fines, fees, damages (including indirect, special or consequential damages) or causes of action, including, without limitation, proprietary or personal injury (including death) that arise from, either directly or indirectly, or relate to, the use, dissemination and/or reliance upon any of the Information, including, without limitation, any act or omission of the Licensee (including its officers, directors, partners, employees, contractors and agents), in connection with this Licence Agreement, as well as any patent, trademark or copyright infringement.

The rights to indemnity contained herein shall survive the early termination or expiry of this Licence Agreement.

7. Default

If the Licensee fails to comply with any of the terms or conditions of this Licence Agreement, City of Hamilton may terminate this Licence Agreement and all rights of the Licensee created hereunder.

8. Effects of termination

Upon the termination of this Licence Agreement for whatever cause, all rights and privileges granted to the Licensee hereunder will immediately terminate and the Licensee shall immediately return to the City of Hamilton, or destroy, the Licensed Database and all related copies and materials. City of Hamilton reserves the right to require proof from the Licensee of the destruction of the Licensed Database and related copies and materials.

9. Governing Law

This Licence Agreement is governed by the laws of the Province of Ontario and the laws of Canada applicable therein.

10. Enurement

Subject to s.4, this Licence Agreement is binding upon and inures to the benefit of the parties and their respective successors and permitted assigns.

11. Notices

Any notice, instruction or other communication required or permitted to be given to any party pursuant to this Licence Agreement must be in writing and will be deemed to have been sufficiently given if delivered personally or sent by pre-paid registered mail or by facsimile to the corresponding address show below:

If to the Licensee:

1.5 SENES CONSULTING LIMITED C/O: TALAR SAHSUVAROGLU (TSAHSUVAROGLU@SENES.CA)

1.6 121 GRANTON DR, UNIT 12

Richmond Hill, Ontario
L4B 3N4

If to City of Hamilton:

1.7 SUPERVISOR, BUSINESS APPLICATIONS (CITIZEN SERVICES)

1.8 CITY OF HAMILTON, BUSINESS APPLICATIONS SECTION

1.9 P.O. BOX 586, STN LCD1

1.10 HAMILTON, ONTARIO L8N 3K7

1.11 PHONE # - (905) 546-2424 EXT. 4267

1.12 FAX # - (905) 546-2333

or to such other address as any party may from time to time notify the others in accordance with this section.

Any such communication will be deemed to have been received and delivered on the date of delivering, if delivered, or on the fifth business day after mailing thereof, if sent by pre-paid registered mail, or on the date of transmission, if sent by facsimile.

IN WITNESS WHEREOF the parties have duly executed this Licence Agreement as of the date first written above.

2.0 SENES CONSULTING LIMITED

3.0

_____
(signature)

Name: **Talar Sahsuvaroglu**

Title: **Environmental Scientist**

Date: **25 October 2011**

City of Hamilton

(signature)

Name: **Mr. Dave Salter**

Title: **Supervisor, Business Applications (Citizen Services)**

Date:

4.0 PREPARED BY: ST, BUSINESS APPLICATIONS, CITIZEN SERVICES, INFORMATION SERVICES

Addendum 'A'

Licensed Database, transferred to licensee in electronic format, in file(s) as named hereafter;

For the purposes of this contract you have been determined as a **Category B** client.

Enter a description of data provided here:
Need details as to what is provided, in what format, and geographic extent (e.g. DWG, shapefile, feature names, ortho file names, "area bounded by…", etc.).

Data...

City Boundary; Shapefile and jpg

Municipal (Community boundary) Shapefile and jpg

Hamilton

City of Hamilton
Corporate Services Department
Information Services
Business Applications Section

Schedule "B"

GIS Data Dissemination Fees

4.1.1.1 Digital GIS Data Handling Fees

All digital data requesters shall be charged a handling fee of $75.00 per hour, (minimum 1 hour charge) to cover the cost of staff time spent in discussions with the requester, and time spent in the preparation and delivery of the data. The hourly rate will be further accumulated to the initial costs when the client requests modifications or updates for an existing project.

Data is available in the following formats:

Standard Formats - Intergraph's GeoMedia *.MDB warehouse
Export formats - *.DGN, *.DXF, *.DWG or Shapefiles

Hard Copy Map Plotting Fees

All hard copy map requesters shall be charged a plotting fee of $75.00 per hour, (minimum 1 hour charge) to cover the cost of staff time spent in discussions with the requester, and time spent in the preparation and delivery of the map. The hourly rate will be further accumulated to the initial costs when the client requests modifications or updates for an existing map.

Fees will also include the cost of the paper per linear foot:

Standard Bond 24lb paper	$2.00 per linear foot
Photo Quality Bond	$7.50 per linear foot

Note: a minimum paper cost of $10.00 per plot will apply. Plots are printed on a 42" plotter.

All pricing is subject to applicable taxes.

Appendix C

Summary of Data Used in This Study

Appendix C: Summary of Data Used in This Study

Table C.1: Air Quality Data for PM_{10}

	PM10 Geometric Mean (ug/m^3)			
	Station 29300	Station 29324	Station 29314	**Average**
1997	19.1	17.3	-	**18.2**
1998	21.4	19.6	-	**20.5**
1999	21.4	17.3	-	**19.4**
2000	22.5	-	18.7	**20.6**
2001	21.6	-	16.7	**19.2**
2002	20.5	-	14.6	**17.6**
2003	24.2	-	18.2	**21.2**
2004	16.7	-	16.8	**16.8**
2005	21.4	-	18.4	**19.9**
2006	15.8	-	12.6	**14.2**
2007	20.6	-	13.9	**17.2**
2008	16.5	-	13.0	**14.8**
2009	13.8	-	12.2	**13.0**

Table C.2: Air Quality Data for $PM_{2.5}$

	$PM_{2.5}$ Mean (ug/m^3)			
	Station 29000	Station 29114	Station 29118	**Average**
1997	-	-	-	-
1998	12.4	13.1	-	**12.8**
1999	11.7	9.8	-	**10.8**
2000	11.7	8.5	-	**10.1**
2001	11.1	8.1	-	**9.6**
2002	13.0	8.9	-	**11.0**
2003	10.6	9.6	-	**10.1**
2004	8.9	9.3	8.4	**8.8**
2005	10.0	9.8	9.6	**9.8**
2006	9.1	8.1	8.2	**8.5**
2007	8.9	7.8	8.3	**8.3**
2008	8.3	7.3	7.6	**7.8**
2009	6.8	6.3	6.1	**6.4**

Table C.3: Air Quality Data for SO_2

SO_2 Mean (ppb)				
	Station 29000	Station 29114	Station 29118	**Average**
1997	5.3	5.2	4.9	**5.1**
1998	6.3	6.6	3.8	**5.6**
1999	6.6	5.5	4.5	**5.5**
2000	5.1	5.8	4.0	**5.0**
2001	6.0	5.3	4.0	**5.1**
2002	4.9	4.8	2.6	**4.1**
2003	5.0	5.3	4.0	**4.8**
2004	4.0	-	-	**4.0**
2005	5.3	-	-	**5.3**
2006	4.8	3.3	-	**4.1**
2007	4.2	3.5	-	**3.9**
2008	4.3	3.0	-	**3.7**
2009	3.3	3.0	-	**3.2**

Table C.4: Air Quality Data for NO_2

NO_2 Mean (ppb)				
	Station 29000	Station 29114	Station 29118	**Average**
1997	18.6	15.4	19.5	**17.8**
1998	22.4	16.8	23.4	**20.9**
1999	21.6	14.8	21.8	**19.4**
2000	21.8	15.4	21.0	**19.4**
2001	22.5	14.8	19.5	**18.9**
2002	20.9	15.4	19.0	**18.4**
2003	21.3	14.5	18.0	**17.9**
2004	16.8	-	-	**16.8**
2005	19.3	-	-	**19.3**
2006	17.0	11.6	-	**14.3**
2007	17.0	11.9	-	**14.5**
2008	14.7	10.5	-	**12.6**
2009	13.6	9.9	-	**11.8**

Table C.5: Air Quality Data for CO

	CO Mean (ppm)	
	Station 29000	**Average**
1997	0.7	**0.7**
1998	1.1	**1.1**
1999	0.8	**0.8**
2000	0.8	**0.8**
2001	0.7	**0.7**
2002	0.6	**0.6**
2003	0.71	**0.7**
2004	0.44	**0.4**
2005	0.3	**0.3**
2006	0.32	**0.3**
2007	0.22	**0.2**
2008	0.19	**0.2**
2009	0.19	**0.2**

Table C.6: Air Quality Data for O_3

O_3 Mean (ppb)				
	Station 29000	Station 29114	Station 29118	**Average**
1997	18.1	22.2	18.6	**19.6**
1998	19.1	24.1	19.3	**20.8**
1999	19.5	24.1	20.0	**21.2**
2000	17.0	22.6	16.9	**18.8**
2001	18.8	24.2	18.6	**20.5**
2002	20.4	27.7	20.5	**22.9**
2003	21.7	28.4	22.0	**24.0**
2004	20.1	24.6	19.2	**21.3**
2005	23.3	28.2	21.2	**24.2**
2006	23.2	27.5	20.9	**23.9**
2007	24.8	29.2	23.0	**25.7**
2008	25.1	29.0	23.3	**25.8**
2009	24.3	27.2	21.8	**24.4**

Table C.7: Health Baseline Data

	Total NT Mortality	Respiratory Hospital Admissions	Cardiovascular Hospital Admissions
1997	3,867	2,736	7,457
1998	3,934	3,265	7,311
1999	3,930	3,328	7,563
2000	3,928	3,500	7,715
2001	3,857	3,232	7,994
2002	3,765	3,040	7,847
2003	3,992	3,168	7,103
2004	3,725	3,269	7,077
2005	3,934	3,623	6,515
2006	-	3,239	6,082
2007	-	3,188	6,335
2008	-	3,053	5,917
2009	-	-	-

Appendix D

Summary of Results

Appendix D: Summary of Results

Table D.1: Annual Non-Traumatic Mortality Results from Current Model

Annual Non-Traumatic Mortality						
	PM_{10}	**$PM_{2.5}$**	**SO_2**	**NO_2**	**CO**	**O_3**
1997	31.7	-	7.1	46.9	4.6	54.7
1998	36.3	38.6	7.9	55.8	7.4	59.0
1999	34.2	32.5	7.8	51.8	5.3	60.0
2000	36.4	30.5	7.0	51.8	5.3	53.3
2001	33.2	28.5	7.1	49.7	4.6	57.0
2002	29.7	31.7	5.6	47.2	3.8	62.0
2003	38.1	31.0	6.8	48.7	4.8	69.1
2004	28.1	25.3	5.4	42.5	2.8	57.1
2005	35.2	29.6	7.6	51.6	2.0	68.6
Average	**33.7**	**31.0**	**6.9**	**49.6**	**4.5**	**60.1**

Table D.2: Annual Respiratory Hospital Admission Results from Current Model

Annual Respiratory Hospital Admissions						
	PM_{10}	**$PM_{2.5}$**	**SO_2**	**NO_2**	**CO**	**O_3**
1997	60.7	-	42.1	138.7	-	87.2
1998	81.5	-	54.5	193.6	-	110.5
1999	78.4	-	55.2	183.5	-	114.6
2000	87.8	-	52.2	193.0	-	107.0
2001	75.4	-	49.4	173.9	-	107.8
2002	65.0	-	37.4	159.3	-	112.9
2003	81.8	-	45.3	161.4	-	123.7
2004	66.7	-	39.2	155.9	-	113.1
2005	87.7	-	58.0	198.7	-	142.4
2006	56.0	-	39.4	131.6	-	125.5
2007	66.9	-	36.8	130.9	-	132.8
2008	54.9	-	33.4	109.3	-	127.9
Average	**71.9**	**-**	**45.3**	**160.8**	**-**	**117.1**

Notes:

PM_{10}, NO_2 and O_3 results adjusted to account for possible risk rate overestimation (Sahsuvaroglu and Jerrett (2003))

$PM_{2.5}$ and CO were not evaluated for this endpoint as no appropriate risk rates were available

Table D.3: Annual Cardiovascular Hospital Admission Results from Current Model

Annual Cardiovascular Hospital Admissions						
	PM_{10}	**$PM_{2.5}$**	**SO_2**	**NO_2**	**CO**	**O_3**
1997	95.0	-	24.4	125.0	5.9	29.3
1998	104.9	74.6	26.0	143.4	9.1	30.5
1999	102.4	65.0	26.7	137.9	6.8	32.1
2000	111.3	62.3	24.4	140.7	7.0	29.1
2001	107.2	61.4	26.0	142.3	6.3	32.8
2002	96.4	68.7	20.5	136.0	5.3	35.9
2003	105.4	57.4	21.6	119.7	5.7	34.1
2004	83.0	50.0	18.1	111.6	3.5	30.1
2005	90.7	50.9	22.2	118.2	2.2	31.5
2006	60.4	41.1	15.7	81.8	2.2	29.0
2007	76.4	42.2	15.6	86.0	1.6	32.5
2008	61.2	36.7	13.8	70.1	1.3	30.5
Average	**91.2**	**55.5**	**21.2**	**117.7**	**4.7**	**31.4**

Notes:

SO_2 and CO results adjusted to account for possible risk rate overestimation (Sahsuvaroglu and Jerrett (2003))

Table D.4: Summary of Health Outcome Results from Current Model

	NT Acute Exposure Mortality **Avg. Incidences per Year** **1997 - 2005**	**Respiratory Hospital Admissions** **Avg. Incidences per Year** **1997 - 2008**	**Cardiovascular Hospital Admissions** **Avg. Incidences per Year** **1997 - 2008**
PM_{10}	34	72	91
$PM_{2.5}$	31	-	56
SO_2	7	45	21
NO_2	50	161	118
CO	5	-	5
O_3	60	117	31
Total	**186**	**395**	**322**