Appendix D
Public Health Assessment

McMaster Institute of Environment and Health

A Public Health Assessment of Mortality and Hospital Admissions Attributable to Air Pollution in Hamilton

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EXECUTIVE SUMMARY

Background

Health risks from ambient air pollution exposure have emerged as a major public health issue. In this report, we estimate mortality and hospital admissions associated with ambient air pollution exposure in Hamilton. Currently, Hamilton exceeds government objectives by about 20 days per year and has some of the highest ambient air pollution in Canada. Ambient pollution exposures result from a combination of pollution from outside the region, industrial emissions, transportation sources, and local meteorology and topography. All of these factors elevate ambient air pollution exposures and make the issue of health effects particularly important in Hamilton.

Numerous epidemiological studies have found a significant association between air pollution and health effects. In 1997, Pengelly and colleagues estimated that air pollution was associated annually with a midpoint estimate of 214 non-traumatic mortalities in Hamilton. Since 1997, much has been learned about the short-term and chronic health effects of air pollution. The past six years have also seen changing ambient air pollutant levels throughout Hamilton. The combination of new research findings and changing pollution levels has created a need to update and expand on earlier work. Information from this new assessment can help local decision-makers understand the magnitude of health effects from air pollution and in taking action that protects and improves population health in Hamilton.

Methods

To estimate mortality and hospital admissions associated with ambient air pollution in Hamilton, we derived dose-response relationships based on local estimates published in the scientific literature. These estimates were applied to recent data on air pollution and health outcomes available through government sources.

Many of the acute studies pooled here used generalized additive models in their statistical analysis. A recent statistical discovery revealed a programming limitation in the statistical software used, leaving the findings from these studies in question. Reanalyses of data indicate that risk estimates may have been overestimated by as much as 42%. Adjustments were applied to study data to account for the 42% overestimation.

In accordance with past studies, health effect estimates are compared to a zero pollution level, considered by many to be practically unattainable. We thus calculated estimates using a baseline of the lowest quintile of measured pollution values. Local estimates derived from Hamilton-specific models were also conducted. Additional sensitivity analyses were based on pooled random effects models and from chronic studies from other jurisdictions.

We compared these results to earlier studies to assess how estimates of health effects have changed since the last assessment.

Results

Our results revealed a wide range in estimates of mortality and morbidity attributable to air pollution. Using the 1997 study as baseline, estimates conducted using similar methods as the initial HAQI report, resulted in an increase of 76 deaths (298 to 374), due to larger dose-response relationships in the literature and slightly higher ambient pollution levels (Table 1). Respiratory admissions increased by 463 (144 to 607), while cardiovascular admissions increased by 1743 (257 to 2000). The 42% adjustment estimated 217 deaths, 352 respiratory admissions and 1120 cardiovascular admissions. The most conservative estimate involved combining both the 42% adjustment and the 20% baseline models. Using this new methodology, mortality and morbidity estimates decreased to 96 deaths, 139 respiratory and 479 cardiovascular admissions - an average decrease of 76% from our initial estimates.

Table 1. Summary of Mortality and Morbidity Counts Using Average Dose-Response Calculations, Based on 1997 Hamilton Pollution Values.

Pollutant	NT mortality (average incidences/year) average of estimates					Respiratory admissions (incidences/year) average of estimates					CV admissions (incidences/year) average of estimates				
	P1997	CAH	Adj	M-min	M-min adj	P1997	CAH	Adj	M-min	M-min adj	P1997	CAH	Adj	M-Min	M-Min adj
PM_{10}	97	73	43	24	14	48	144	83	46	27	112	280	157	84	49
SO_2	16	53	31	27	16	28	69	40	35	20		56	31	45	26
NO_2	81	134	78	46	27	20	244	142	83	48	125	888	497	303	176
CO	3	10	6	6	3						20	118	66	65	38
O_3	102	105	61	62	36	48	150	87	75	44		659	369	329	191
Total	298	374	217	119	96	144	607	352	239	139	257	2000	1120	826	479

P1997 = HAQI report, Pengelly 1997
CAH = Current reanalysis for City of Hamilton
Adj = Adjusted value of CAH, for overestimate of 42%
M-min = Estimate calculated for pollution values of mean – min (lower quintile) for 1997
M-min adj = Application of adjusted value to M-min calculation

Interpretation

A cautionary note is required with respect to the totals given above. They should be interpreted as general aids to decision-making rather than as exact counts of death and illness. The totals may be influenced by uncontrolled confounding of co-pollutants. This may have resulted in an overestimate of mortality and hospital admission totals.

We have excluded other serious health effects. These include the development and exacerbation of asthma, reproductive abnormalities, elevated cancer rates, and less serious respiratory conditions such as infectious respiratory diseases. Thus, our mortality and admission estimates may, in fact, underestimate the total burden of illness associated with air pollution in Hamilton.

Conclusions

In this report, we have identified air pollution as a major source of mortality and hospital admissions in Hamilton. We have not linked the health estimates to specific sources, but this represents an important area for future research. It appears that gaseous pollutants most closely associated with transportation emissions have increased over the five-year study period (1995-99). The scientific evidence on the health effects of these pollutants has also advanced. The combination of increasing pollution and growing scientific knowledge leads to the conclusion that these sources should be the focus of concerted policy efforts in the realm of land use and transportation planning. Both these areas fall within the jurisdiction of the city. Future research combining the methods used in this report with source apportionment could supply more definitive guidance for priority setting in local decision-making.

CONTENTS

1. OBJECTIVES AND BACKGROUND

1.1 Objectives

The primary objective of this report is to estimate mortality and hospital admissions attributable to ambient air pollution in Hamilton, based on the most recent research and data available. A secondary objective is to compare recent findings with those reported by Pengelly et al. (1997) and other recent reports that use similar methods (Pengelly et al., 2000).

1.2 Background

Hamilton experiences some of the highest ambient air pollution exposures in Canada, exceeding government objectives by about 20 days per year. The reasons for these high exposures include the following: (1) proximity to the Ohio River Valley, where coal-fired generating stations emit pollutants that travel hundreds of kilometers to Hamilton; (2) the Nanticoke coal-fired generating station located on the northern shore of Lake Erie, which also contributes considerably to local pollution; (3) increasing transportation emissions that result from automobile and truck traffic in and around the city; (4) local point source emissions from one of the largest industrial areas in Canada; and (5) topographic and meteorological conditions that often keep the pollution close to ground level. All of these factors elevate ambient air pollution exposures and make the issue of health effects particularly important in Hamilton.

Numerous epidemiological studies have found a significant association between air pollution and health effects (see Section 4 and Appendix 1 for details). In 1997, Pengelly et al. (1997) estimated that air pollution was associated annually with a mid-point estimate of 214 non-traumatic mortalities in Hamilton. Since 1997, much has been learned about the short-term and chronic health effects of air pollution. The past six years have also seen changing ambient air pollution levels throughout Hamilton. The combination of new research findings and pollution levels has created a need to update and expand on the earlier work. Quantitative information from this new assessment can help local decision-makers to understand the size of the health effects from air pollution and to take action to improve population health in Hamilton.

2. METHODS

2.1 Overview

The methodology used in this report followed seven steps:

1. Identification of pollutants of interest,
2. Literature review to identify risk coefficients for specific pollutants and conversion into comparable values,
3. Identification and acquisition of relevant air quality data,
4. Acquisition of health outcome data,
5. Estimation of the burden of illness due to air pollution in Hamilton using available data,
6. Sensitivity analysis using Pengelly models, data specifically derived from Hamilton studies, and adjusted estimates, and
7. Analysis of the findings.

2.2 Identification of Pollutants of Interest

Based on consultations with the Health and Environmental Impacts Working Group for Clean Air Hamilton, we utilized the criteria pollutants that were indicated in the Hamilton-Wentworth Air Quality Initiative (HAQI) report in 1997, with the exception of the "air toxics." Specifically, we included particulate matter (PM_{10}), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO), and ozone (O_3). Pengelly et al. (1997) also applied this methodology to Toronto data in 2000 (i.e., using the same pollutants except for the air toxics). In addition, we estimated the mortality attributable to fine particles (i.e., $PM_{2.5}$) because these have received increasing attention in the scholarly literature as particularly harmful to pulmonary function.

2.3 Literature Review

We conducted our literature review with the Medline and PubMed search engines. We searched combinations of the words "air pollution" with the following keywords – mortality, morbidity, health effects, time-series – for articles dated 1997 and onwards, until the beginning of October, 2001. Using Medline, 2067 related articles were identified, while the search in PubMed revealed about 6900 articles.

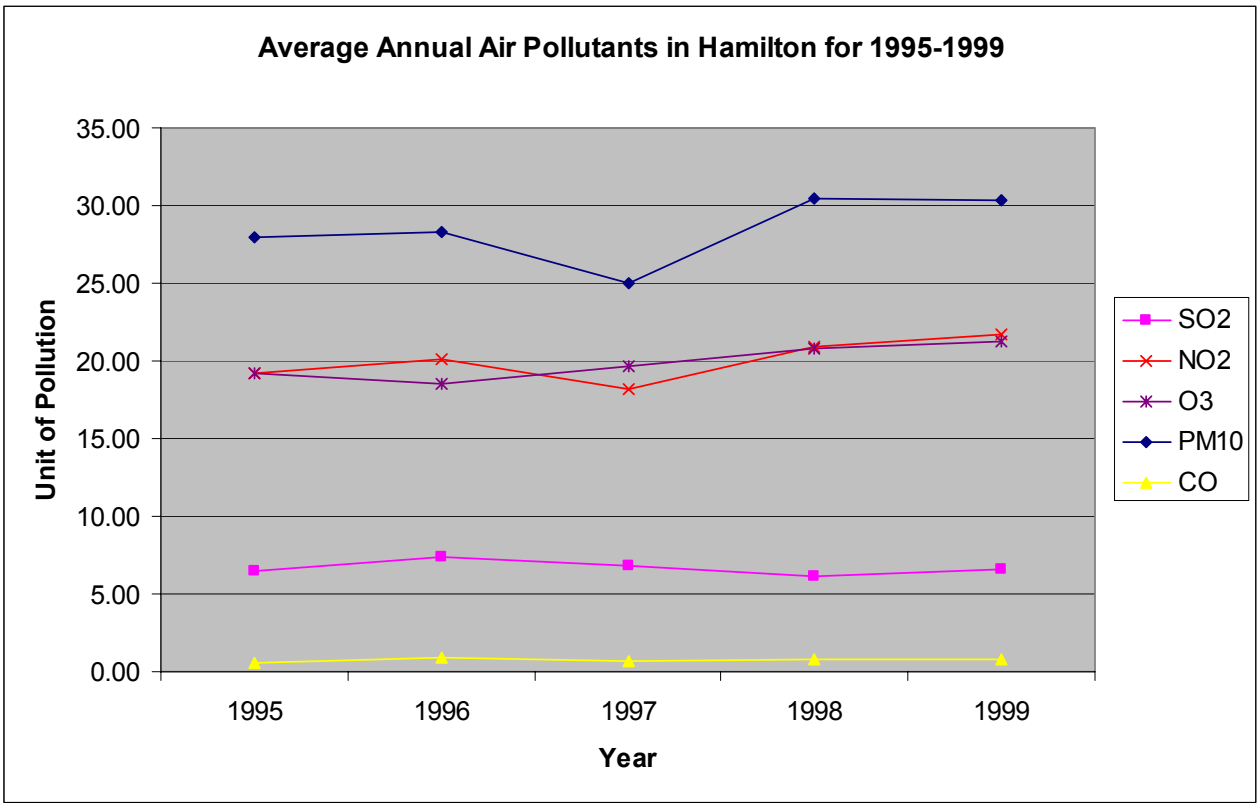
Subsequent review and selection of the articles was based on relevance, suitability of outcome measure, and significance of findings. We excluded articles that were not related to mortality or hospital admissions; those that focused on indoor air pollutants and tobacco smoke; those in languages other than English or French; and articles that specifically identified elderly or infants as study groups. Articles that made use of multipollutant models were given priority to provide maximum control for co-pollutants. While findings from single pollutant models and significant associations with the elderly population were present in the review tables, they were not included in the calculated averages. Studies including random effects and meta-analysis of previous studies as a comparative metric were selected. Research that used Hamilton estimates in particular was emphasized.

Chronic studies were included in this analysis. Based on the limited number available, this literature review included a search as far back until 1993 when the earlier chronic effect literature was published. Recent reanalyses of these articles was also included in the literature review.

2.4 Air Quality Data

Annual averages for the identified pollutants were available for multiple locations in Hamilton, courtesy of the Ministry of Environment's monitoring network. Regional arithmetic averages from all the available stations were calculated to derive the city-wide average. Figure 1 illustrates the general trends in ambient pollution for the period 1995-99. The average pollutant values for the study period can be found in Appendix 1 in tabular format.

Figure 1. Change of Average Annual Air Pollutants in Hamilton, 1995-1999



Note: SO_2, NO_2, and O_3 expressed in parts per billion (ppb), CO expressed in parts per million (ppm) and PM_{10} expressed in $\mu g/m^3$

2.5 Health Outcome Data

Annual morbidity and mortality data for Hamilton were supplied by the Central West Health Planning Information Network. The data were extracted from the Ontario "data warehouse," available through the Government of Ontario Network (GONET). The ICD-9 codes used were those indicated by the Pengelly studies, covering the area of the New City of Hamilton. Mortality data were only available for 1995 to 1997, while hospital admissions data were available for a longer period (i.e., 1995 to 1999). These tables can be found in Appendix 2.

We observed a marked increase in the number of hospital admissions, especially for cardio-vascular (CV) admissions, between 1995 and 1996. We checked the acquired data for internal errors, but the difference seems to be due to other factors not reported by the Ministry of Health.

2.6 Estimating the Mortality and Hospital Admissions Associated with Air Pollution

Following the methodology set by the Pengelly et al. 1997 and 2000 reports, we computed the relationship to estimate health outcomes as follows:

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

where:
HO = annual health outcome
B = base number of outcomes per year
ΔH% = percent change in health outcome per unit increase of pollutant
P = annual pollution average

2.6.1 Sample Calculation

The following data were utilized to calculate the premature mortality attributable to particulates (PM_{10}) for the year 1995:

- Total non-traumatic deaths in Hamilton for 1995 = 3730 deaths per year
- Percent increase in non-traumatic mortality for PM10, averaged from literature values, per unit increase = 0.076 increase in deaths per 1 µg/m3 increase in PM10 * 1/100
- Annual average of PM10 for Hamilton for 1995 = 27.9 µg/m3

$$HO = 3730 \frac{\text{deaths}}{\text{year}} \times 0.076 \frac{\text{deaths}}{\mu g/m3} \text{ per } 100 \text{ deaths} \times 27.9 \ \mu g/m3 = 79.09 \frac{\text{deaths}}{\text{year}}$$

The sample equation shows that the units cancel each other out to leave deaths per year as the final unit. Thus, following normal rounding rules, 79 premature deaths are associated with PM_{10} exposure in Hamilton for the year 1995.

2.7 Estimate Adjustments

Two adjustments were conducted on the original estimates. The first involves a recent discovery of a statistical limitation in one of the software packages used in time-series analyses. The second adjustment pertains to an achievable baseline pollution estimate.

The time-series studies summarized in this report typically have used generalized additive models (GAM) in their statistical analysis, as these models allow for control of time-varying factors through the incorporation of non-parametric smoothers of weather and other confounders. However, in light of recent findings of a programming limitation in the statistical software used in these analyses, the findings from these studies are now in question (Ramsey et al., 2003). The statistical software provided biased risk estimates because it did not assure convergence of its iterative estimation procedure. A reanalysis of National Morbidity, Mortality and Air Pollution Study (NMMAPS) data, one of the largest pooled data sets in the U.S., has revealed that the risk estimates have been overestimated by 36 - 42% (Dominici et al., 2002). The reanalysis showed that positive associations still exist, although in some cases the associations become insignificant.

All the reported findings in these types of studies now have to be reconsidered before use in policy analysis. This major statistical discovery has left scientists and policymakers wondering about the magnitude of associations between acute exposures to air pollution and health.

Adjustments were made on the summarized findings of the average dose-response estimates in this report. The values were adjusted to account for the maximum overestimation of 42%. This model is referred to as the "adjusted" model.

The second adjustment considers that in calculating risk estimates, pollution estimates are calculated in terms of comparison to a zero pollution level. The zero pollution level can be considered to be unattainable and overly idealistic. A more achievable estimate, though arbitrarily chosen, was considered to be at a baseline of 20% of pollution concentrations. Thus a separate estimate was calculated using annual pollution values of the mean minus the lower quintile, based on daily averages. These were calculated for 1997 to provide a comparison estimate. This adjustment is referred to as "baseline 20% model".

2.8 Hamilton-specific Estimates

We also calculated estimates of studies conducted in Hamilton, using the research of Burnett et al. (1998a) for gaseous air pollutants and Jerrett et al. (2001) for the particulate measure, measured with the coefficient of haze (CoH). For these estimates, multipollutant models were used for the gaseous air pollutants, while single pollutant models were available for the particulate measures. The percent risks at the mean value for relevant years were computed. Because the Poisson regression takes a log-linear form, we computed the risk estimates for each criteria pollutant as follows:

$$e^{(\beta \bar{x})}$$

where:
e is the exponential function,
β is the regression coefficient estimating the average increase in mortality associated with a unit increase in pollution, and
$\bar{x}$ is the average of the air pollutant.

3. RESULTS

3.1 Results of the Literature Review

The detailed tables containing the literature review results are contained in Appendix 3. The tables show the study location, the modeled pollutants, and the key results in a standardized format. A commentary on our findings from the literature search is presented below.

3.1.1 Carbon Monoxide (CO)

Non-traumatic mortality
Three studies have found significant associations between CO and non-traumatic mortality since the HAQI study in 1997. Burnett et al. (1998a, 1998b) and Gywnn et al. (2000) found an increase of 4.7%, 2.0%, and 4.13% per 1ppm increase, respectively. The studies all used multipollutant models.

Respiratory hospital admissions
None of the literature reported significant associations between respiratory hospital admissions and CO.

Cardiac hospital admissions
CO was related to cardiac hospital admissions, specifically for hospitalization for congestive heart failure. Schwartz (1997) examined data for Tucson, US, and reported an increase of 1.4% in admissions per 1 ppm increase. Burnett et al. (1997) calculated congestive heart failure admissions specifically for Hamilton and reported a 2.5% increase. Interestingly, the Toronto-specific estimate by the same researchers was comparatively higher at 6%.

3.1.2 Sulfur Dioxide (SO_2)

Non-traumatic mortality
Recent research shows a range of dose-response estimates for sulfur dioxide and total non-traumatic mortality. Garcia-Aymerich et al. (2000) found that in Barcelona, Spain, a 10 ppb increase in SO_2 led to a 4.2% increase in total mortality. Saez et al. (2001) found a 1.1% increase for three Spanish cities using a multipollutant model. In Madrid, Spain, Diaz et al. (1999) found a 2.1% increase in non-traumatic mortality with a single pollutant model. Taking 12 European countries into account, Katsouyanni et al. (1997) found an increase of 1.1%. Kelsall et al. (1997) considered a multipollutant model for Philadelphia, US, and found a 0.84% relative increase to the 10 ppm increase. Burnett et al. (1998a) studied SO_2 effects for 11 cities in Canada, using multipollutant models, and obtained a 3.89% increase in non-traumatic mortality for Hamilton.

Respiratory hospital admissions
Gywnn et al. (2001) associated an increase of 3.7% per 10 ppb increase in SO_2 in terms of respiratory hospital admissions. No other studies investigated this association.

3.1.3 Nitrogen Dioxide (NO_2)

Non-traumatic mortality
NO_2 has recently been significantly associated with non-traumatic mortality in a number of studies. In Rome, Italy, Michelozzi et al. (1998) found a 1.54% increase in a 10 ppb increase, while in Barcelona, Spain, Garcia-Aymerich et al. (2000) reported a 2.9% increase. Morgan (1998) in Sydney, Australia indicated the value was closer to 1.5%. However, the latter study did not take multipollutant modeling into account. Burnett et al. (1998) revealed a 1.5% increase

in non-traumatic mortality associated with a 10 ppb increase in NO_2, specifically for Hamilton, while a 2.3% increase was estimated for Toronto.

Respiratory hospital admissions:
Burnett et al. (1997a) found a 4.87% increase in respiratory admissions for Hamilton for a 10 ppb increase in NO_2.

Cardiac hospital admissions:
Three studies found significant associations between NO_2 and cardiac hospital admissions. Burnett et al. (1997a) found an 8.7% increase for the 10 ppb increase in NO_2. Morgan et al. (1998) found a lower value of 4.4%. However, a multipollutant model was not taken into account for this study. Moolgavkar (2000), in Los Angeles County, US, found a 1.7% increase, with a two-pollutant model (i.e., SO_2 and NO_2).

3.1.4 Ozone (O_3)

Non-traumatic mortality
There has been an increasing amount of research in ozone-related mortality. Recent studies showed significant associations between O_3 and non-traumatic mortality. Garcia-Aymerich et al. (2000) in Barcelona, Spain, estimated a 0.95% increase in non-traumatic mortality, while Gouveia et al. (2000) in Sao Paulo, Brazil, identified a 0.43% increase. In Philadelphia, US, Kelsall et al. (1997) found the relative risk to be at 0.94%, while in Santa Clara County, California, US, Fairley (1999) estimated a much higher risk at 2.47%. Thurston and Ito (2001) calculated this value at 0.56% in a meta-analysis study based on 12 published estimates.

Respiratory hospital admissions:
Moolgavkar et al. (1997) found a 4% increase in respiratory hospital admissions associated with a 10 ppb increase of ozone, while using a multipollutant model. Burnett et al. (1997b) found an increase of 1.5%; however, in his 1998 article (Burnett et al., 1998), this value was estimated to be 4.9%. Gywnn et al. (2000) found this value closer to 2.0%.

Cardiac hospital admissions:
Only one study, Burnett et al. (1997b) tested the ozone-admission association. They reported a 4.5% increase for cardiac hospital admissions. As this is the only study to find significant associations at such high values, this estimate should be considered preliminary.

3.1.5 Particulates

Non-traumatic mortality:
Numerous studies have calculated the percent increase in daily mortality per $10\mu g/m^3$ increase in particulate matter, in the form of TSP, PM_{10}, $PM_{2.5}$, and SO_4^{2-}.

TSP:
Alberdi Odriozola et al. (1998) and Diaz et al. (1999) conducted studies in Madrid, Spain, and found a 0.6% and 0.72% increase, respectively. In Rome, Italy, Michelozzi et al. (1998) calculated a comparable 0.66% increase. Neas et al. (1999) found a 0.56% increase in

Philadelphia using a single pollutant study. Goldberg et al. (2001) calculated increases in non-traumatic mortality in Montreal and reported a value of 0.65% for a 10 $\mu g/m^3$ in TSP in single pollutant analysis. Kelsall et al. (1997) found a 0.3% increase in Philadelphia using a multipollutant model.

PM_{10}:
Burnett et al. (1998b) estimated a 0.7% increase in non-traumatic deaths in Hamilton taking into account other pollutants, while in Montreal, Goldberg et al. (2001) calculated an increase of 0.69% in a single pollutant analysis.

In a meta-analysis, Daniels et al. (2000) found a 0.54% increase in non-traumatic deaths in 20 US cities. Samet et al. (2000) reported a 0.51% increase for 20 US cities considered. In their reanalysis of Schwartz et al. (1996) article on particulates in six US cities, Klemm et al. (2000) found a 0.8% increase associated with PM_{10}. Katsouyanni et al. (1997) reported non-traumatic mortality for PM_{10} increases equal 0.4% for the 12 European countries studied.

Primarily in European research, black smoke (BS) values were used as approximations to PM_{10} values. Saez et al. (2001) calculated a 0.64% increase for the three Spanish cities in the study, while Garcia-Aymerich et al. (2000) found this value closer to 1.1% in their single-pollutant analysis.

$PM_{2.5}$:
Goldberg et al. (2001) found a 1.96% increase in non-traumatic mortality related to the increase in $PM_{2.5}$ in Montreal. Fairley (1999) calculated a 4.46% in Santa Clara County, US. Klemm et al. (2000) estimated this increase as 1.3% in a study of six US cities. In Mexico City, Mexico, Borja-Aburto et al. (1998) recorded a 1.68% in non-traumatic mortality associated with the fine particulates. Burnett (1998) reported a 2.5% increase in Hamilton.

Respiratory hospital admissions:
PM_{10}:
Moolgavkar et al. (1997) found a 1.7% increase in respiratory hospital admissions in Los Angeles County, US. Burnett et al. (1997) calculated the relative risk at 2.1% in Hamilton, while Gywnn et al. (2000) found this value to be closer to 2.2% in New York, US.

$PM_{2.5}$:
There were no studies found to report significant associations

SO_4^{2-}:
Gywnn et al. (2000) estimated this to be 0.5% in New York, while Burnett et al. (1997) reported 2.7% for Hamilton.

Cardiac hospital admissions:
PM_{10}:
Burnett et al. (1999) found a 0.5% increase in cardiac admissions in Toronto, Canada, while Morgan et al. (1998) found this value closer to 0.76% in Sydney, Australia.

$PM_{2.5}$:
Burnett et al. (1999) calculated a 0.75% increase. Again, this was the only study that found significance, and it should be considered preliminary.

3.2 Results of Estimated and Adjusted Calculations

To calculate the final averages of the risk estimates from the literature, only multipollutant models were used. A simple averaging method for correlation studies was used to compute the overall effect from the literature (see Wolf, 1986). As well, the low and high ends of the findings are noted, as there are considerable differences in estimates of dose-response. Adjusted values were applied to the mean values. Recent pooled random effect estimates (Stieb et al., 2003) and estimates from chronic studies (Pope et al., 2002) were also included.

Notation in the following tables includes 'P1997' as the original HAQI report, Pengelly et al. (1997); 'P2000' as the City of Toronto report, Pengelly et al. (2000); 'CAH' as the current reanalysis of HAQI conducted for Clean Air Hamilton; 'Adjusted' as the current results with adjustment of 42% overestimate; 'M-min' (mean minus minimum 20%) represents the baseline 20% model; and 'M-min adj' indicates the baseline model adjusted for the 42% overestimate.

Relatively wide ranges can be observed within the estimated percent changes from increases in pollutants. For an increase of 10 $\mu g/m^3$ in PM_{10}, there was an increase ranging from 0.43% to 1.07% in non-traumatic deaths; 0.7-3.5% for respiratory admissions; and 0.5-2.3% in cardiovascular admissions. In the case of SO_2, the increase per 10ppb resulted in a range of 0.84-3.89% increase in mortality; 1.3-6.1% for respiratory admissions; and 0.2-2.1% in cardiovascular admissions. The other pollutants follow similar ranges, with the higher ranges existing for morbidity results and lower ranges in mortality estimates. Adjusted mean values were slightly higher than the low end of the estimates, except for the association between O_3 and non-traumatic mortality.

Table 1. Summary of Percent Changes per 10 Units of Pollutant: Low, Mean, High, and 42% Adjusted Mean Estimates of Calculated Values

Pollutant	NT mortality[a] (change per 10 units pollutant) range of estimates				Respiratory admissions[b] (change per 10 units pollutant) range of estimates				CV admissions[b] (change per 10 units pollutant) range of estimates			
	low	mean	high	adj mean	low	mean	high	adj mean	low	mean	high	adj mean
PM_{10} ($\mu g/m^3$)	0.43	0.76	1.07	0.44	0.7	2.1	3.5	1.22	0.5	1.4	2.3	0.8
$PM_{2.5}$(μ/m^3)	1.68	2.88	4.46	1.67								
SO_2 (ppb)	0.84	2	3.89	1.16	1.3	3.7	6.1	2.15	0.2	1.1	2.1	0.6
NO_2 (ppb)	1.5	1.9	2.3	1.10	1	4.9	9	2.84	4.4	6.55	8.7	3.8
CO (1 ppm)	2	3.68	4.95	2.13					0.4	1.95	2.5	1.1
O_3 (ppb)	0.94	1.38	1.7	0.80	1.5	2.8	4.9	1.62	1.6	4.5	7.5	2.6

NT= Non-traumatic; CV = cardiovascular;
[a] = Mortality values were calculated on the basis of 2 or 3 estimates
[b] = Morbidity values were calculated on the basis of 1 or 2 estimates; in the case of one estimate, 95% confidence intervals were used as the low and high range of estimates
adj mean = Mean estimate adjusted for 42% overestimate

Note: Because the ranges of data vary among pollutants, the 10-unit change is not directly comparable as a metric of severity in effects. For pollutants with a smaller range such as CO, a 10-unit change is proportionately larger than for PM_{10}, which has a larger range.

Table 2 compares the average values for the risk estimates found in the literature after 1997 with the literature findings from the two previous studies and the adjusted values. This identifies the trends in literature values for the estimates. Current estimates were consistently higher than the 1997 estimates, except for PM_{10} estimates for non-traumatic mortality and CO estimates for cardiovascular admissions. Adjusted values were lower than initial estimates for PM_{10} and O_3, but higher for SO_2, NO_2, and CO.

Table 2. Summary of Percent Changes per 10 Units of Pollutant, Comparing Average Estimates of Studies, Adjusted and Pooled Estimates

Pollutant	NT mortality (change per 10 units pollutant) average of estimates					Respiratory admissions (change per 10 units pollutant) average of estimates				CV admissions (change per 10 units pollutant) average of estimates			
	P1997	P2000	CAH	Adj	Pooled	P1997	P2000	CAH	Adj	P1997	P2000	CAH	Adj
PM_{10}	1	0.8	0.76	0.46	0.32	0.7	1.7	2.1	0.99	0.6	2.3	1.4	1.3
$PM_{2.5}$			1.9	1.10									
SO_2	0.6	2.25	2	1.16	0.85	0.4	2.76	3.7	1.60			1.1	0.0
NO_2	1.15	1.19	1.9	1.10	0.2	0.4	2.49	4.9	1.44		3.9	6.55	2.3
CO	1.1	3.48	3.68	2.13	0					5	6	1.95	3.5
O_3	0.3	0.4	1.38	0.80	0.3	0.8	1.1	2.8	0.64		4.52	4.5	2.6

Pooled = Pooled random effect model estimates (Stieb et al, 2003)
Adj = Mean CAH estimate adjusted for 42% overestimate

Table 3 presents the calculated mortality and morbidity estimates as incidences per year, using low, mean, high and adjusted risk estimates. Values ranged as in Table 1. Totals for all pollutants ranged from 248 to 567 annual deaths (using PM_{10} as a particulate estimate), to between 236 to 1252 respiratory and 993 to 3036 cardiovascular deaths. Adjusted mean totals were higher than the lower end estimates for all total counts.

Table 3. Summary of Low, Mean, High, and Adjusted Mean in the Mortality and Morbidity Counts Averaged for Available Years in Current Study

Pollutant	NT mortality (incidences/year) calculated estimates				Respiratory admissions (incidences/year) calculated estimates				CV admissions (incidences/year) calculated estimates			
	low	mean	high	adj mean	low	mean	high	adj mean	low	mean	high	adj mean
PM_{10}	44	77	109	45	59	176	293	102	101	284	466	165
$PM_{2.5}$	108	185	286	107								
SO_2	22	51	100	30	30	72	140	42	10	52	100	30
NO_2	108	137	166	79	59	290	532	168	629	937	1244	543
CO	6	10	14	6					26	126	162	73
O_3	68	119	178	69	88	164	287	95	227	638	1064	370
Total	248	394	567	229	236	702	1252	407	993	2037	3036	1181
Total *	312	502	744	291								

* = total has been calculated with $PM_{2.5}$ instead of PM_{10}

Table 4 compares the estimates taken from the three studies and adjusted values, calculated on current air quality and health outcome data. Detailed calculations for these estimates can be found in Appendix 3. This table shows the differences in estimated mortality and morbidity counts according to the respective study values. The adjusted estimate is lower than any of the studies for mortality, at 229 annual deaths, but higher than the initial Pengelly study for morbidity at 407 annual respiratory and 1239 cardiovascular admissions.

Table 4. Summary and Comparison of the Mortality and Morbidity Counts Using the Average Dose-response Calculated in the Three Studies with Adjusted Values, Applied to Current Hamilton Data

Pollutant	NT mortality (average incidences/year) average of estimates				Respiratory admissions (incidences/year) average of estimates				CV admissions (incidences/year) average of estimates			
	P1997	P2000	CAH	Adjusted	P1997	P2000	CAH	Adjusted	P1997	P2000	CAH	Adjusted
PM_{10}	102	81	77	45	59	142	176	102	122	466	384	223
SO_2	15	58	51	30	22	81	72	42		629	52	30
NO_2	83	86	137	79	24	147	290	168	135	338	937	543
CO	3	10	10	6					20	50	126	73
O_3	97	29	119	69	53	66	164	95		641	638	370
Total	300	264	394	229	158	436	702	407	277	2124	2137	1239

Table 5 compares the original study, the current study, adjusted risk estimate values, baseline 20% adjustments, and application of both adjustments, all calculated for 1997 values. As the values show, there is a substantial difference in total mortality and morbidity counts, depending on the assumptions underlying the calculations. Our most conservative estimate, the application of both the 42% adjustment and the baseline 20% model, estimated 96 deaths in 1997 due to PM_{10}, compared to HAQI initial estimate of 298, our initial estimate of 374, and 217 deaths if the GAM discrepancy is taken into consideration. For respiratory admissions, the most conservative estimate is only a few admissions lower than HAQI estimates (139 compared to 144, respectively), while the highest estimate stands at 607 admissions. The highest estimate for cardiovascular admissions is our initial estimate of 2000 admissions, while the most conservative estimate is 479 admissions, still higher than the 257 admissions estimated by HAQI in 1997.

Table 5. Summary of the Mortality and Morbidity Counts Using the Average Dose-response in HAQI, CAH and Both Adjustments; Applied to 1997 Hamilton Data

Pollutant	NT mortality (average incidences/year) average of estimates					Respiratory admissions (incidences/year) average of estimates					CV admissions (incidences/year) average of estimates				
	$P1997_{97}$	CAH_{97}	Adj_{97}	M-min 1997	M-min adj	$P1997_{97}$	CAH_{97}	Adj_{97}	M-min 1997	M-min adj	$P1997_{97}$	CAH_{97}	Adj_{97}	M-Min 1997	M-Min adj
PM_{10}	97	73	43	24	14	48	144	83	46	27	112	280	157	84	49
SO_2	16	53	31	27	16	28	69	40	35	20		56	31	45	26
NO_2	81	134	78	46	27	20	244	142	83	48	125	888	497	303	176
CO	3	10	6	6	3						20	118	66	65	38
O_3	102	105	61	62	36	48	150	87	75	44		659	369	329	191
Total	298	374	217	119	96	144	607	352	239	139	257	2000	1120	826	479

3.3 Results of Hamilton-specific Estimates

Hamilton-specific estimates revealed that, for NO_2 and CO, the values were comparable to the lower ranges of the literature estimates. For SO_2, estimates were slightly higher than the mean count from literature estimates, and Hamilton-specific O_3 estimates were at the higher end of the calculations (Table 6). Applying the 42% adjustment brought the Hamilton-specific total down closer to the mean of the literature estimates. The 20% baseline estimate lowered the total to 206 mortality incidences, compared to 248 for the low end of literature estimates. When both adjustments were applied, total mortality fell to 119. This Hamilton-specific value is still higher than the 96 incidences (see Table 5), which results from data averaged across all literature findings.

Table 6. Comparison of the Range of Mortality Counts Using Current Estimates with Averaged Hamilton-specific Estimates and Adjustments

Pollutant	NT mortality (incidences/year) range in estimates						
	low	mean	high	Hamilton	Adj	M-min$_{1997}$	M-min adj
PM_{10}	44	77	109				
CoH				256	148	40	23
SO_2	22	51	100	73	42	37	21
NO_2	108	137	166	108	63	45	26
CO	6	10	14	5	3	4	2
O_3	68	119	122	122	71	81	47
Total	248	394	511	564	327	206	119

CoH= coefficient of haze.

Table 7 summarizes all available calculations performed for non-traumatic mortality estimates.

Table 7. Summary and Comparison of Mortality Counts Estimated for All Available Models, Based on 1997 Hamilton Pollution Values

Pollutant	NT mortality (average incidences/year) average of estimates							
	P1997$_{1997}$	CAH$_{1997}$	Adj$_{1997}$	M-min	M-min adj	Pooled	Hamilton	Chronic
PM_{10}	97	73	43	24	14	31		
$PM_{2.5}$		110	64					232
CoH							256	
SO_2	16	53	31	27	16	22	73	
NO_2	81	134	78	46	27	14	108	
CO	3	10	6	6	3	0	5	
O_3	102	105	61	62	36	23	122	
Total	298	374	217	119	96	90		232
Total **		411	238				564	

M-min adj = Adjusted value of M-min, for overestimate of 42%
Pooled = Pooled random effect model estimates (Stieb et al, 2003)
Hamilton = Hamilton-specific dose-response estimates
Chronic = Estimates based on chronic exposures to particulates (Pope et al., 2002)
CoH= Coefficient of haze
Total ** = Totals calculated with $PM_{2.5}$ or CoH as particulate measure

4. DISCUSSION

Applying the 42% adjustment to the averaged estimates resulted in lowering the CAH estimates by almost half, from 77 to 45 deaths due to a 10μg/m^3 increase in PM_{10}, compared to 102 deaths estimated by HAQI 1997. Total mortality counts were reduced from 300 average deaths estimated by HAQI and 394 by CAH, to 229 for the adjusted model. Total respiratory admissions increased from HAQI's estimate of 158 to 407 using the conservative adjustments. The most dramatic increase, even using adjusted estimates, was for total cardiovascular admissions. HAQI estimated 277 admissions, CAH estimated 2137, while the 42% adjustment estimated 1239 admissions using averaged pollution data.

Using the 20% baseline model lowered values further below adjusted values. While SO_2 and O_3 estimates were higher than HAQI estimates for mortality, totals remained lower. Applying both adjustments lowered this value further to 96 deaths in 1997, a difference of 74% from CAH estimates. For morbidity estimates, the numbers were slightly different. Except for PM_{10}, adjusted morbidity estimates were above both HAQI and CAH estimates. Application of both adjustments resulted in only a slight decrease compared to HAQI estimates for respiratory morbidity, but almost double for cardiovascular admissions. However, in comparison to initial CAH calculations, respiratory admissions had decreased by 77%, while cardiovascular admissions had decreased by 76%.

Pooled random effects model estimates resulted in 90 deaths associated with air pollution, comparable to the 96 estimated by applying both adjustments. Chronic estimates of $PM_{2.5}$-related mortality revealed 232 counts. Chronic estimates based on cohort studies are considered to be the "gold standard" for assessing health effects related to air pollution, due to their ability to assess life expectancy and incidence, course and remission of disease (Kunzli and Tager, 2000), giving significance to this type of research. The difference between these two estimates emphasizes the importance of considering both acute and chronic exposure studies separately.

A cautionary note is required with respect to the totals given above. They should be interpreted as general aids to decision-making rather than exact counts of death and illness. Uncontrolled confounding of co-pollutants may influence the totals. Although we used multipollutant models to derive estimates, some models did not control for all criteria pollutants simultaneously. In addition, each study may contain estimation error that is not accounted for in our simple averages of effect.

The comparison of the CAH estimates to the original HAQI findings shows that the current estimates identify a larger number of health outcomes due to air pollution. This difference is pronounced in hospital admissions, hinting to the possibility of increased sensitivity in current estimates for discerning the health risks attributable to air pollution. However, calculating the estimates using the 42% overestimation adjustment and the baseline 20% model both separately and together resulted in significantly lower estimates, except for total cardiovascular admissions that remained substantially higher. The correspondence between our conservative GAM and 20% baseline model adjustment, and the pooled random effects suggests that approximately 90 to 96 deaths are associated with ambient air pollution exposure.

The Hamilton-specific estimates revealed that the total estimates of non-traumatic mortality were initially at the higher end of the range found in our literature review. With the adjustments, the values are comparable to lower end estimates. Yet these values remained slightly higher than estimates that were not based on Hamilton-specific data. It has to be noted that the inclusion of CoH does not necessarily imply an equal measure of particulates. Specifically, NO_2 and CO related mortality values were lower than the average, while SO_2 and O_3 were higher than the averages.

5. CONCLUSION

This study has estimated mortality and hospital admissions associated with ambient air pollution in Hamilton. Dose-response relationships were derived based on exposure estimates published in the peer-reviewed literature (see sections 2 and 3 as well as Appendix 3). These estimates were applied to recent data on air pollution and health outcomes available through governmental sources.

Recent scientific discoveries identified software limitations in the GAM models used in time-series modeling. Applying the adjustments to account for an approximately 42% overestimate lowered the average annual mortality rate to 229, respiratory admissions to 407, and an average 1181 incidences for cardiovascular admissions.

If further assumptions are taken into account by using a baseline 20% model of 1997 pollution values, annual mortality rates drop to 119, respiratory admissions to 239, cardiovascular admissions to 826. Applying the 42% adjustment to these values revealed mortality counts of 96 (compared to original 1997 estimates of 374), respiratory admissions at 139 (compared to 607) and cardiovascular admissions at 479 (compared to 2000). Pooled random effects model estimates reveal 90 deaths associated with air pollution, while chronic estimates of particulate pollution ($PM_{2.5}$) result in 232 deaths. This wide range shows that the possibilities of public health estimates depend on the assumptions that underlie the analysis.

Although we used multipollutant models for our estimates, there is the potential for confounding variables due to the uncontrolled effects of co-pollutants as not all models control for all criteria pollutants. Therefore, our totaled mortality estimates could exceed the actual number of deaths associated with air pollution and thus, should be viewed with caution.

The “file drawer” consideration may also lead to overestimates in the air pollution effect (Levy et al. 2000). Published research generally favours significant findings, while insignificant findings are rarely reported. Since our study relies on published articles, there may be a bias in favour of positive findings and consequently higher estimates.

Some scholars have also suggested that using short-term dose-response estimates is inappropriate for studies that assess annual estimates (McMicheal et al., 1998). The reason for this criticism stems from the notion that some of the deaths, while premature, may have occurred during the same year, regardless of pollution exposure. Following this logic, overestimates of annual mortality may accrue due to the use of short-term estimates. Because so few studies estimate

chronic effects, we had to rely mainly on short-term models for calculating our estimates. Given this limitation, it is possible that we have overestimated mortality.

Other considerations suggest our study may underestimate the total burden of illness due to air pollution in Hamilton. Our estimates only include mortality and acute health effects from air pollution. Other important health effects such as the development and exacerbation of asthma (Tenias et al., 1998; Yu et al., 2000), reproductive abnormalities (Bobak and Leon, 1999; Wang et al., 1997), elevated cancer rates (Beeson et al., 1998; Cohen, 2000) and less serious respiratory conditions such as infectious respiratory diseases (Kim et al., 1996) are excluded from this analysis.

Due to our emphasis on multipollutant models, we had insufficient data to implement random effects models that weight for statistical uncertainty in the estimates. We have, however, included estimates of meta-analyses based in the United States and Europe. These results generally show lower estimates for particulate matter and ozone than our average calculations, but the most recent estimates produce aggregate estimates that are similar to our 20% adjusted model.

In this report, we have identified air pollution as a major source of mortality and hospital admissions in Hamilton. We have not linked the health estimates to specific sources, but this represents an important area for future research. Over the past five years, SO_2 has remained fairly stable, suggesting the industrial sources are not increasing. It appears that gaseous pollutants most closely associated with transportation emissions and particulates have increased over the study period (1995-99). The scientific evidence on the health effects of these pollutants (i.e., O_2, NO_2, CO) has also advanced. The combination of increasing pollution and growing scientific knowledge leads to the conclusion that these sources should be the focus of concerted policy efforts in the realm of land use and transportation planning. Both of these areas fall within the jurisdiction of the city. Future research combining the methods used in this report with source apportionment could supply more definitive guidance for priority setting in local decision-making.

Appendix 1: Air Quality Indicators

Table 1-1. Annual Average of Air Pollutants in Hamilton

	PM_{10} ($\mu g/m^3$)	$PM_{2.5}$ ($\mu g/m^3$)	SO_2 (ppb)	CO (ppm)	NO_2 (ppb)	NO (ppb)	NO_x (ppb)	O_3 (ppb)
1995	27.90	17.58	6.43	0.60	19.25	17.00	37.00	19.18
1996	28.30	17.83	7.34	0.90	20.08	17.53	37.10	18.47
1997	24.98	15.73	6.80	0.65	18.15	14.93	33.25	19.63
1998	30.48	19.20	6.14	0.85	20.87	16.57	37.40	20.83
1999	30.30	19.09	6.56	0.75	21.65	16.50	38.23	21.20

Note: $PM_{2.5}$ is calculated as $0.63(PM_{10})$

Source: Ministry of Environment Ambient Air Pollution Reports, 1995-1999

Figure 1-2. Plot of Average PM_{10} Values for 1995-1999

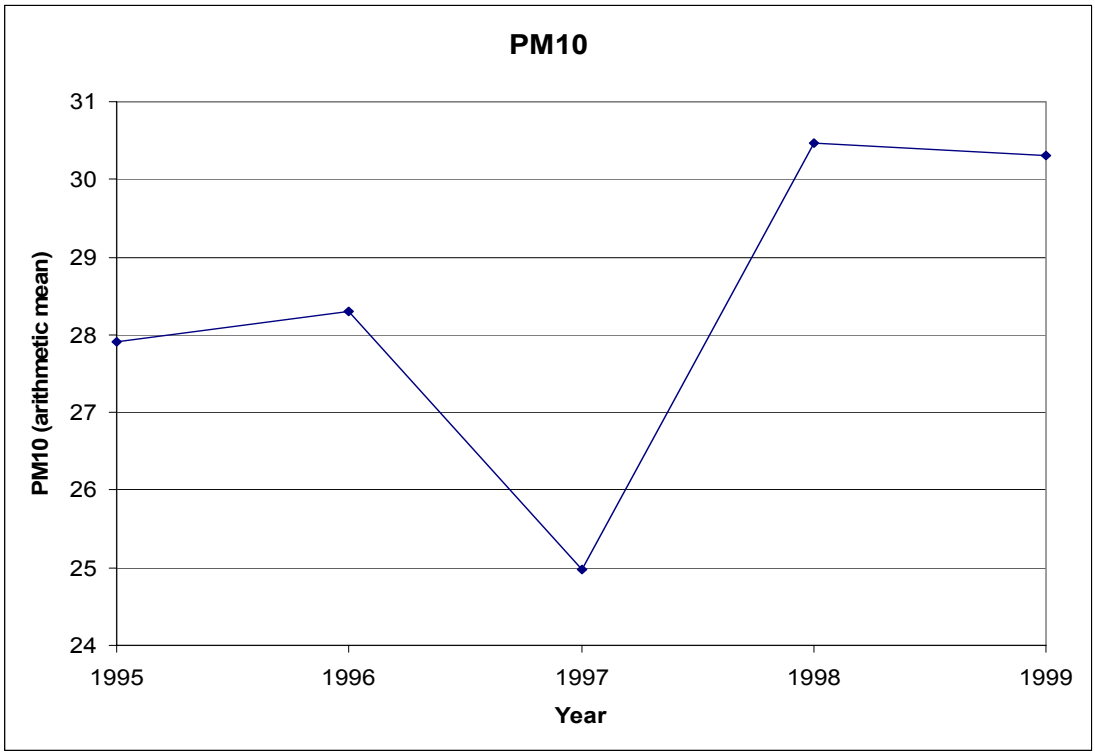


Figure 1-3. Plot of Average SO_2 Values for 1995-1999

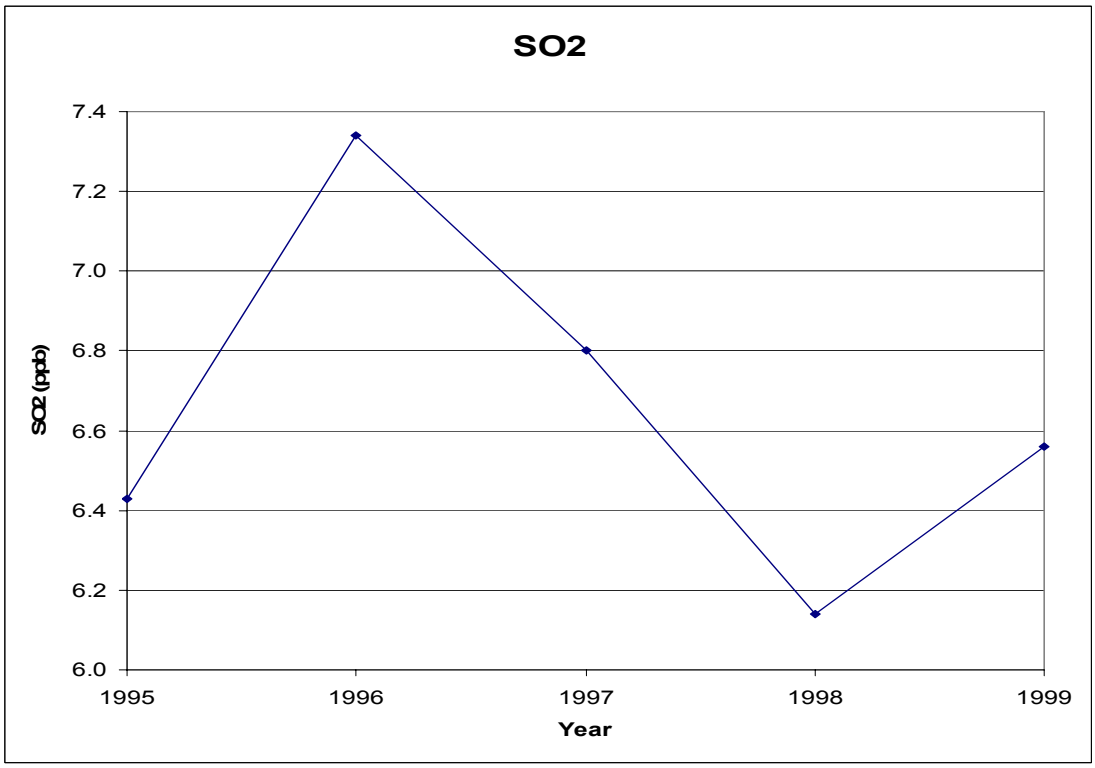


Figure 1-4. Plot of Average NO_2 Values for 1995-1999

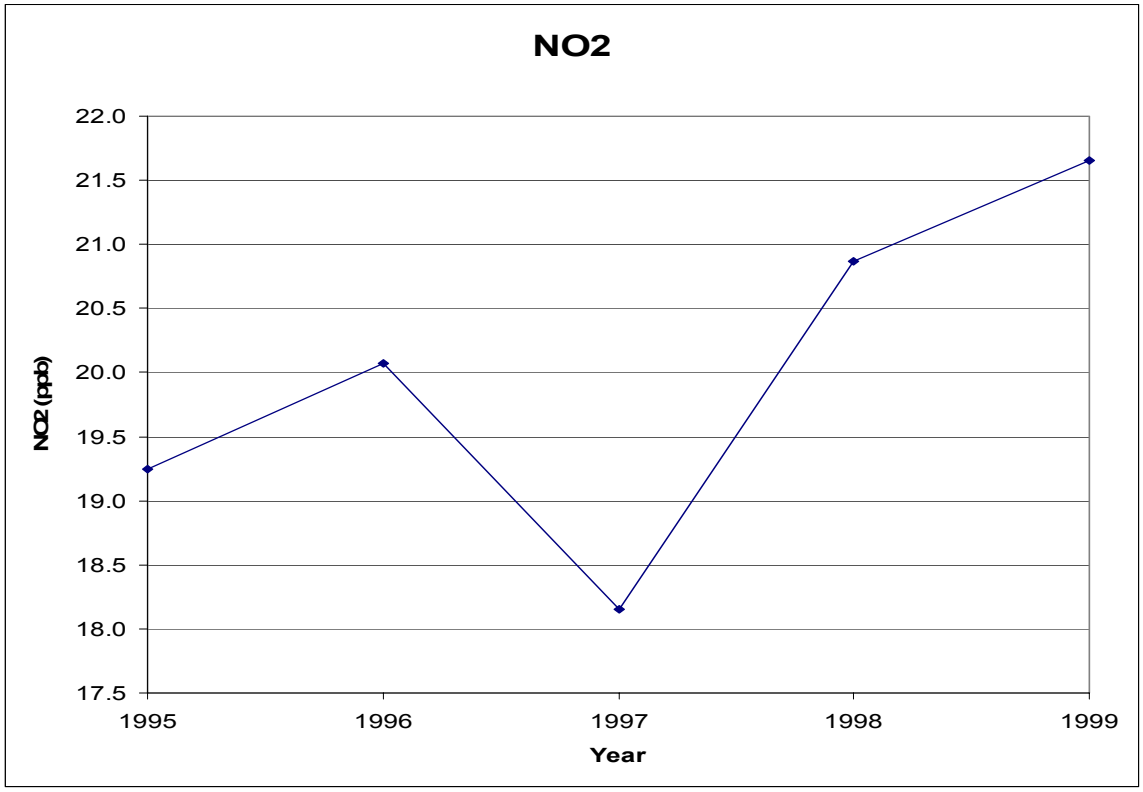


Figure 1-5. Plot of Average CO Values for 1995-1999

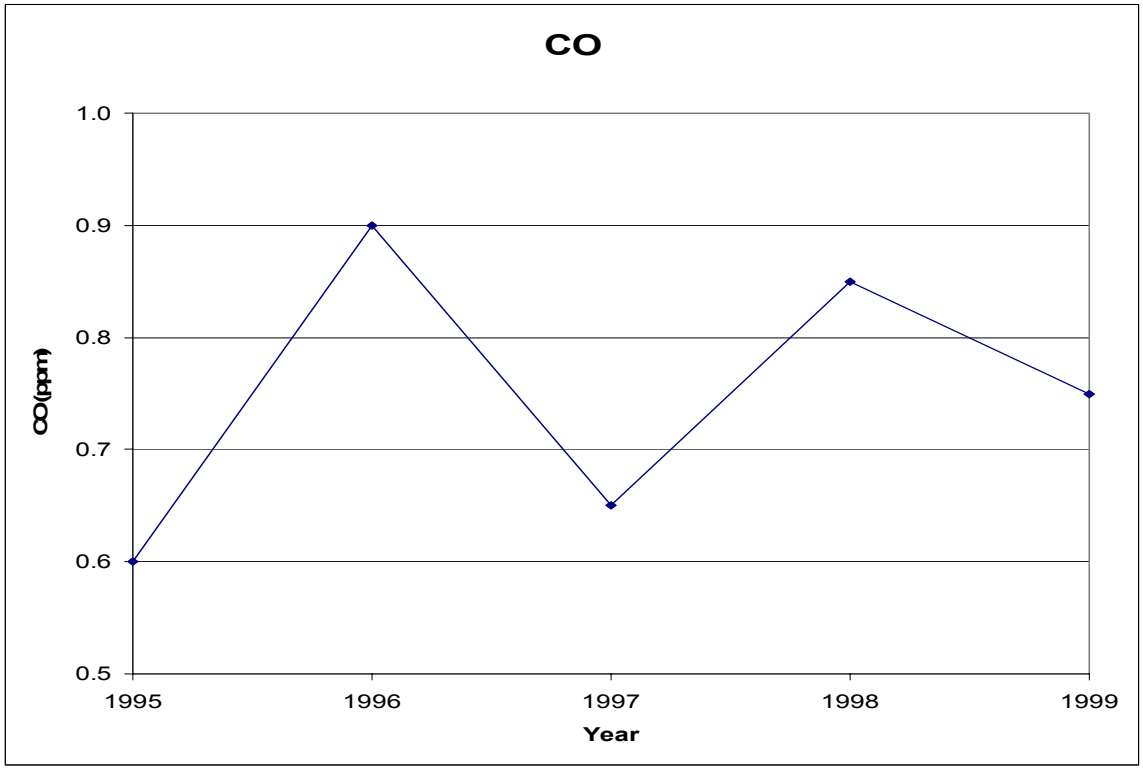
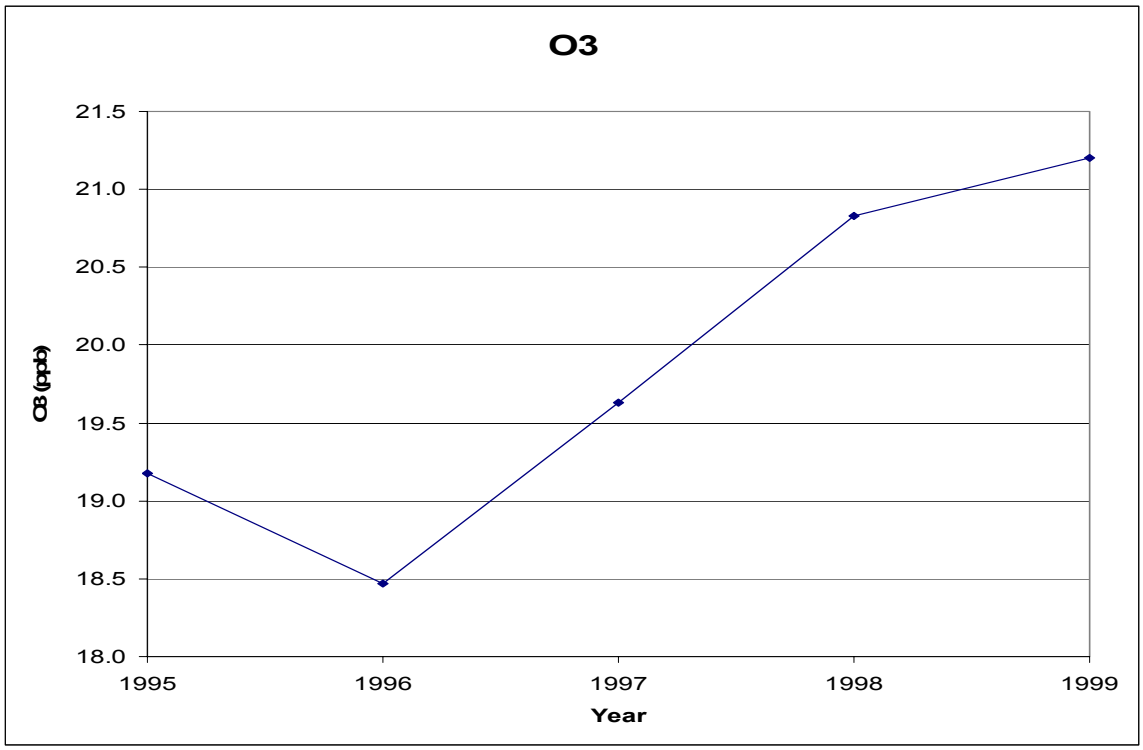


Figure 1-6. Plot of Average O_3 Values for 1995-1999



Appendix 2: Health Outcome Data

Table 2-1. Mortality and Morbidity Data for Hamilton Used in the analysis

	Non-traumatic mortality			Morbidity			
	All	CV	Resp	All	CV	CHF	Resp
1995	3,730	1,445	370	39,854	5,612	814	2,249
1996	3,694	1,422	367	41,149	7,702	1,123	3,085
1997	3,868	1,419	353	39,420	7,468	1,176	2,738
1998				40,044	7,322	1,108	3,266
1999				39,993	7,572	1,031	3,330
average	3,764	1,429	363	40,092	7,135	1,050	2,934

NT= non-traumatic
CV = cardiovascular
CHF = congestive heart failure
Resp = respiratory

Source: Central West Health Planning Information Network, 2001

Appendix 3: Detailed Literature Summary Tables

Table 3-1. Comparison of % Increases in Non-traumatic Deaths in Relation to Increases of 10 Units per Pollutant

			% change in daily mortality for each 10 unit increase in pollutant				
Reference	**Location**	**Multi Pollutant models**	**Particulates ($\mu g/m^3$)**	**SO_2 (ppb)**	**NO_2 (ppb)**	**CO (1 ppm)**	**O_3 (ppb)**
Borja-Aburto et al. (1998)	Mexico City, Mexico	$PM_{2.5}$, O_3, NO_2 (4 day lag)	$PM_{2.5}$ = 1.68 % (0.2, 3.14)				
Burnett et al. (1998a)	Estimates derived for Hamilton, Canada	CO, NO_2, SO_2, O_3		2.2%	1.5%	2.0%	1.7%
Burnett et al. (1998b)	Toronto	CO	PM_{10}= 1.5% (1.1,1.9) $PM_{2.5}$=2.5% (1.7,3.3)	3.89% (2.9, 4.86)	2.3% (1.6, 2.8)	4.95% (3.8, 6.1)	1.5% (1.2, 1.9)
Gwynn et al. (2000)	Buffalo, US	PM_{10}, CO	PM_{10} = 1.07% (0.02, 2.1)			4.1% (CI) (1.0, 7.2)	
Fairley (1999)	Santa Clara County, CA, USA	CO, NO_2, O_3, NO_3	$PM_{2.5}$ = 4.46%				2.47%
Kelsall et al. (1997)	Philadelphia, USA	TSP, SO_2, NO_2, O_3	TSP = 0.31% (0, 0.61)	0.84% (0.11. 1.57)			0.94% (0.35, 0.15)
Morgan et al. (1998)	Sydney, Australia	PM_{10}, NO_2, O_3	PM_{10}= 0.8%(0.0, 1.6)				
Saez et al. (2001)	3 Spanish Cities	SO_2, BS	BS= 0.64% (0.2, 1.1)	1.1% (0.2, 1.9)			
Chronic:							
Dockery et al. (1993)	6 US cities	yes	PM2.5: 0.68% (0.5, 0.8)				
Reanalysis of 6cities			PM2.5: 0.69% (0.6, 0.8)				
Pope et al. (1995) ACS study	151 US cities	yes	PM2.5: 0.48% (0.44, 0.51)				
Reanalysis of ACS (2000)		yes	PM2.5: 0.48% (0.45, 0.52)				
Not used in the calculation of current estimate:							
Pengelly et al. (2000)	Toronto, Canada	depending on average calculation	PM_{10}= 0.8% $PM_{2.5}$= 1.5% (0.85, 2.2)	2.2%	1.19%	3.48% (24 hr)	0.4%

Table 3-2. Comparison of % Increases in Non-traumatic Deaths in Relation to Increases of 10 Units per Pollutant for Studies Using Single-Pollutant Models and Meta-analysis Studies

		% change in daily mortality for each 10 unit increase in pollutant				
Reference	**Location**	**Particulates ($\mu g/m^3$)**	**SO_2 (ppb)**	**NO_2 (ppb)**	**CO (1 ppm)**	**O_3 (ppb)**
Alberdi Odriozola et al. (1998)	Madrid, Spain	TSP = 0.6%				
Diaz et al.(1999)	Madrid, Spain	TSP = 0.72%	2.1%			
Garcia-Aymerich et al. (2000)	Barcelona, Spain	BS = 1.1% (0.5, 1.7)	4.2% (2.2, 6.1)	2.9% (0.7, 5.1)		0.95% (0.2, 1.6)
Goldberg et al. (2001)	Montreal	TSP = 0.65% $PM_{2.5}$= 1.96%				
Gouveia et al. (2000)	Sao Paulo, Brazil	PM_{10} = 0.51% (0.1, 0.9)	4.5 % (1.1, 7.9)			0.43% (0.00, 0.85)
Katsouyanni et al. (1997)	12 European cities	PM_{10}= 0.44% (0.2, 0.6)	1.1% (0.8, 1.3)			
Michelozzi et al. (1998)	Rome, Italy	TSP = 0.66% (0.31, 1.02)		1.54% (0.14, 2.97)		
Morgan et al.(1998)	Sydney, Australia			1.5% (0.2, 2.1)		0.7% (0.0,1..3)
Neas et al. (1999)	Philadelphia, USA	TSP= 0.56% (0.27, 0.86)				
Meta-analysis articles:						
Daniels et al. (2000)	20 US cities	PM_{10} = 0.54% (0.33, 0.76)				
Klemm et al. (2000)	6 US cities (reanalysis)	PM_{10}=0.8% (0.5, 1.1)	$PM_{2.5}$=1.3% (0.9, 1.7)	$SO_4^=$=1.6% (0.9, 2.4)		
Samet et al. (2000)	20 US cities	PM_{10} = 0.51% (0.07, 0.93)				
Thurston & Ito (2001)	Combined analysis					0.56% (0.32, 1.08)

Table 3-3. Comparison of % increases in indicated morbidity values in relation to 10 unit increase per pollutant

				% change in daily morbidity for 10 unit increase in pollutant				
Reference	**Location**	**multi-pollutants**	**measure**	**Particulates ($\mu g/m^{3)}$**	**SO_2 (ppb)**	**NO_2 (ppb)**	**CO (1 ppm)**	**O_3 (ppb)**
Ballester et al. (2001)	Valencia, Spain	single pollutant	*card hosp adm*		1.1% (0.2, 2.1)			
Burnett et al. (1997a)	Hamilton	O_3, CO	*resp hosp adm*					1.5% (0.7, 2.2)
Burnett et al. (1997b)	Toronto	T, DP for PM_{10}, +SO_2, O_3 for NO_2 + PM, NO_2, CO for O_3	*card hosp admin* *resp hosp admin*	2.3% (0.3, 4.4) 2.1% (0.9, 3.3)		8.7% (3.2, 14.5) 4.9% (1.0, 9.0)	2.5% (0.2, 4.9)	4.5% (1.6, 7.5) 4.9% (2.7, 7.1)
Burnett et al. (1999)	Toronto	gaseous pollutants	*card hosp adm*	PM_{10}=0.50% $PM_{2.5}$=0.75%				
Gywnn et al. (2000)	Buffalo, NY	each gas against particulates	*resp hosp adm*	PM_{10} = 2.1%(0.7, 3.5) SO_4^{2-}= 0.5% (0.3, 0.7)	3.7% (1.3, 6.1)			2.0% (0.9, 3.0)
Morgan et al. (1998)	Sydney	single pollutant	*card hosp adm*	PM_{10} = 0.7(0.2-1.3)		4.4% (3.06-5.8)		
Morris et al. 1998	Chicago	PM_{10}, NO_2, SO_2, O_3	*chf hosp adm*				2.6% (1.0-3.9)	

Appendix 4: Detailed Calculations of Risk Estimates

Calculations for NT mortality for $PM_{2.5}$

PM 2.5	base	change per 10	%change	pollutant	outcome	42% Adj
1995	nt mort			(arith mean)		
mean	3730	1.9	0.0019	16.7	118	69
minimum		1.5	0.0015		93	54
maximum		2.5	0.0025		156	90
Pengelly 1997		N/A				
Pengelly 2000		N/A				

PM 2.5	base	change per 10	%change	pollutant	outcome	42% Adj
1996	nt mort			(arith mean)		
mean	3694	1.9	0.0019	17.0	119	69
minimum		1.5	0.0015		94	55
maximum		2.5	0.0025		157	91
Pengelly 1997		N/A				
Pengelly 2000		N/A				

PM 2.5	base	change per 10	%change	pollutant	outcome	42% Adj
1997	nt mort			(arith mean)		
mean	3868	1.9	0.0019	15.0	110	64
minimum		1.5	0.0015		87	50
maximum		2.5	0.0025		145	84
Pengelly 1997		N/A				
Pengelly 2000		N/A				

Calculations for chronic exposures to $PM_{2.5}$ and mortality

PM 2.5 Chronic exp	1995 mean	LCI	UCI	1996 mean	LCI	UCI	1997 mean	LCI	UCI	Average
Six Cities	424	311	498	427	314	502	395	290	464	415
Reanalysis of 6 cities	430	374	498	433	377	502	400	348	464	421
ACS	299	274	318	301	276	320	278	255	296	293
Reanalysis of ACS	299	280	324	301	283	327	278	261	302	293
Pope	249	62	498	251	63	502	232	58	464	244

Note: LCI = lower confidence interval
UCI = upper confidence interval

Calculations for respiratory morbidity for PM_{10}

particulates	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1995	resp			(arith mean)		
mean	2249	2.1	0.0021	27.9	132	76
min		0.7	0.0007		44	25
max		3.5	0.0035		220	127
Pengelly 1997		0.7	0.0007		44	25
Pengelly 2000		0.8	0.0008		50	29
Dominici		0.27	0.00027		17	10

particulates	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1996	resp			(arith mean)		
mean	3085	2.1	0.0021	28.3	183	106
min		0.7	0.0007		61	35
max		3.5	0.0035		306	177
Pengelly 1997		0.7	0.0007		61	35
Pengelly 2000		0.8	0.0008		70	41
Dominici		0.27	0.00027		24	14

particulates	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1997	resp			(arith mean)		
mean	2738	2.1	0.0021	25.0	144	83
min		0.7	0.0007		48	28
max		3.5	0.0035		240	139
Pengelly 1997		0.7	0.0007		48	28
Pengelly 2000		1.7	0.0017		116	67
Dominici		0.27	0.00027		18	11

particulates	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1998	resp			(arith mean)		
mean	3266	2.1	0.0021	30.5	209	121
min		0.7	0.0007		70	40
max		3.5	0.0035		349	202
Pengelly 1997		0.7	0.0007		70	40
Pengelly 2000		1.7	0.0017		169	98
Dominici		0.27	0.00027		27	16

particulates	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1999	resp			(arith mean)		
mean	3330	2.1	0.0021	30.3	212	123
min		0.7	0.0007		71	41
max		3.5	0.0035		353	205
Pengelly 1997		0.7	0.0007		71	41
Pengelly 2000		1.7	0.0017		172	99
Dominici		0.27	0.00027		27	16

Calculations for CV morbidity for PM_{10}

particulates	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1995	CV			(arith mean)		
mean	5612	1.5	0.0015	27.9	235	136
min		0.7	0.0007		110	64
max		2.3	0.0023		360	209
Pengelly 1997		0.6	0.0006		94	54
Pengelly 2000		2.3	0.0023		360	209
Dominici		0.27	0.00027		42	25

particulates	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1996	CV			(arith mean)		
mean	7702	1.5	0.0015	28.3	327	190
min		0.7	0.0007		153	88
max		2.3	0.0023		501	291
Pengelly 1997		0.6	0.0006		131	76
Pengelly 2000		2.3	0.0023		501	291
Dominici		0.27	0.00027		59	34

particulates	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1997	CV			(arith mean)		
mean	7468	1.5	0.0015	25.0	280	162
min		0.7	0.0007		131	76
max		2.3	0.0023		429	249
Pengelly 1997		0.6	0.0006		112	65
Pengelly 2000		2.3	0.0023		429	249
Dominici		0.27	0.00027		50	29

particulates	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1998	CV			(arith mean)		
mean	7322	1.5	0.0015	30.5	335	194
min		0.7	0.0007		156	91
max		2.3	0.0023		514	298
Pengelly 1997		0.6	0.0006		134	78
Pengelly 2000		2.3	0.0023		514	298
Dominici		0.27	0.00027		60	35

particulates	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1999	CV			(arith mean)		
mean	7572	1.5	0.0015	30.3	344	200
min		0.7	0.0007		161	93
max		2.3	0.0023		528	306
Pengelly 1997		0.6	0.0006		138	80
Pengelly 2000		2.3	0.0023		528	306
Dominici		0.27	0.00027		62	36

Calculations for NT mortality for SO_2

SO_2	base	change per 10	%change	pollutant	outcome	42% Adj
1995	nt mort			(arith mean)		
calculated mean	3730	2.00	0.002	6.4	48	28
Minimum		0.84	0.00084		20	12
Maximum		3.89	0.00389		93	54
Pengelly 1997		0.60	0.0006		14	8
Pengelly 2000		2.25	0.00225		54	31

SO_2	base	change per 10	%change	pollutant	outcome	42% Adj
1996	nt mort			(arith mean)		
calculated mean	3694	2.00	0.002	7.3	54	31
min		0.84	0.00084		23	13
max		3.89	0.00389		105	61
Pengelly 1997		0.60	0.0006		16	9
Pengelly 2000		2.25	0.00225		61	35

SO_2	base	change per 10	%change	pollutant	outcome	42% Adj
1997	nt mort			(arith mean)		
calculated mean	3868	2.00	0.002	6.8	53	31
min		0.84	0.00084		22	13
max		3.89	0.00389		102	59
Pengelly 1997		0.60	0.0006		16	9
Pengelly 2000		2.25	0.00225		59	34

Calculations for respiratory morbidity for SO_2

SO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1995	resp			(arith mean)		
mean	2249	3.70	0.0037	6.4	53	31
min		1.30	0.0013		19	11
max		6.10	0.0061		88	51
Pengelly 1996		1.50	0.0015		22	13
Pengelly 2000		2.76	0.00276		40	23

SO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1996	resp			(arith mean)		
mean	3085	3.70	0.0037	7.3	84	49
min		1.30	0.0013		29	17
max		6.10	0.0061		138	80
Pengelly 1996		1.50	0.0015		34	20
Pengelly 2000		2.76	0.00276		62	36

SO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1997	resp			(arith mean)		
mean	2738	3.70	0.0037	6.8	69	40
min		1.30	0.0013		24	14
max		6.10	0.0061		114	66
Pengelly 1996		1.50	0.0015		28	16
Pengelly 2000		2.76	0.00276		51	30

SO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1998	resp			(arith mean)		
mean	3266	3.70	0.0037	6.1	74	43
min		1.30	0.0013		26	15
max		6.10	0.0061		122	70
Pengelly 1996		1.50	0.0015		30	17
Pengelly 2000		2.76	0.00276		55	32

SO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1999	resp			(arith mean)		
mean	3330	3.70	0.0037	6.6	81	47
min		1.30	0.0013		29	17
max		6.10	0.0061		134	78
Pengelly 1996		1.50	0.0015		33	19
Pengelly 2000		2.76	0.00276		61	35

Calculations for cardiovascular morbidity for SO_2

SO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1995	CV			(arith mean)		
mean	5612	1.10	0.0011	6.4	40	23
min		0.20	0.0002		7	4
max		2.10	0.0021		75	44
Pengelly 1996		N/A				0
Pengelly 2000		N/A				0

SO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1996	CV			(arith mean)		
mean	7702	1.10	0.0011	7.3	62	36
min		0.20	0.0002		11	7
max		2.10	0.0021		118	68
Pengelly 1996		N/A				0
Pengelly 2000		N/A				0

SO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1997	CV			(arith mean)		
mean	7468	1.10	0.0011	6.8	56	32
min		0.20	0.0002		10	6
max		2.10	0.0021		107	62
Pengelly 1996		N/A				0
Pengelly 2000		N/A				0

SO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1998	CV			(arith mean)		
mean	7322	1.10	0.0011	6.1	49	28
min		0.20	0.0002		9	5
max		2.10	0.0021		94	54
Pengelly 1996		N/A				0
Pengelly 2000		N/A				0

SO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1999	CV			(arith mean)		
mean	7572	1.10	0.0011	6.6	55	32
min		0.20	0.0002		10	6
max		2.10	0.0021		105	61
Pengelly 1996		N/A				0
Pengelly 2000		N/A				0

Calculations for NT mortality for NO_2

NO_2	base	change per 10	%change	pollutant	outcome	42% Adj
1995	nt mort			(arith mean)		
our	3730	1.90	0.0019	19.3	137	79
min		1.50	0.0015		108	63
max		2.30	0.0023		166	96
Pengelly 1997		1.15	0.00115		83	48
Pengelly 2000		1.19	0.00119		86	50

NO_2	base	change per 10	%change	pollutant	outcome	42% Adj
1996	nt mort			(arith mean)		
our	3694	1.90	0.0019	20.1	141	82
min		1.50	0.0015		111	65
max		2.30	0.0023		171	99
Pengelly 1997		1.15	0.00115		85	50
Pengelly 2000		1.19	0.00119		88	51

NO_2	base	change per 10	%change	pollutant	outcome	42% Adj
1997	nt mort			(arith mean)		
our	3868	1.90	0.0019	18.2	134	78
min		1.50	0.0015		106	61
max		2.30	0.0023		162	94
Pengelly 1997		1.15	0.00115		81	47
Pengelly 2000		1.19	0.00119		84	49

Calculations for respiratory morbidity for NO_2

NO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1995	resp			(arith mean)		
mean	2249	4.90	0.0049	19.3	213	123
min		1.00	0.001		43	25
max		9.00	0.009		391	227
Pengelly 1996		0.40	0.0004		17	10
Pengelly 2000		2.49	0.00249		108	63

NO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1996	resp			(arith mean)		
mean	3085	4.90	0.0049	20.1	304	176
min		1.00	0.001		62	36
max		9.00	0.009		558	324
Pengelly 1996		0.40	0.0004		25	14
Pengelly 2000		2.49	0.00249		154	90

NO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1997	resp			(arith mean)		
mean	2738	4.90	0.0049	18.2	244	142
min		1.00	0.001		50	29
max		9.00	0.009		448	260
Pengelly 1996		0.40	0.0004		20	12
Pengelly 2000		2.49	0.00249		124	72

NO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1998	resp			(arith mean)		
mean	3266	4.90	0.0049	20.9	334	194
min		1.00	0.001		68	40
max		9.00	0.009		614	356
Pengelly 1996		0.40	0.0004		27	16
Pengelly 2000		2.49	0.00249		170	99

NO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1999	resp			(arith mean)		
mean	3330	4.90	0.0049	21.7	354	205
min		1.00	0.001		72	42
max		9.00	0.009		650	377
Pengelly 1996		0.40	0.0004		29	17
Pengelly 2000		2.49	0.00249		180	104

Calculations for cardiovascular morbidity for NO_2

NO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1995	CV			(arith mean)		
mean	5612	6.55	0.00655	19.3	709	411
min		8.70	0.0087		942	547
max		4.40	0.0044		477	276
Pengelly 1996		0.00	0		0	0
Pengelly 2000		4.40	0.0044		477	276

NO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1996	CV			(arith mean)		
mean	7702	6.55	0.00655	20.1	1013	587
min		8.70	0.0087		1345	780
max		4.40	0.0044		680	395
Pengelly 1996		0.00	0		0	0
Pengelly 2000		4.40	0.0044		680	395

NO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1997	CV			(arith mean)		
mean	7468	6.55	0.00655	18.2	888	515
min		8.70	0.0087		1179	684
max		4.40	0.0044		596	346
Pengelly 1996		0.00	0		0	0
Pengelly 2000		4.40	0.0044		596	346

NO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1998	CV			(arith mean)		
mean	7322	6.55	0.00655	20.9	1001	580
min		8.70	0.0087		1329	771
max		4.40	0.0044		672	390
Pengelly 1996		0.00	0		0	0
Pengelly 2000		4.40	0.0044		672	390

NO_2	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1999	CV			(arith mean)		
mean	7572	6.55	0.00655	21.7	1074	623
min		8.70	0.0087		1426	827
max		4.40	0.0044		721	418
Pengelly 1996		0.00	0		0	0
Pengelly 2000		4.40	0.0044		721	418

Calculations for NT mortality for CO

CO	base	change per 10	%change	pollutant	outcome	42% Adj
1995	nt mort			(arith mean)		
our	3730	3.68	0.00368	0.6	8	5
min		2.00	0.002		4	3
max		4.95	0.00495		11	6
Pengelly 1997		1.00	0.001		2	1
Pengelly 2000		3.48	0.00348		8	5

CO	base	change per 10	%change	pollutant	outcome	42% Adj
1996	nt mort			(arith mean)		
our	3694	3.68	0.00368	0.9	12	7
min		2.00	0.002		7	4
max		4.95	0.00495		16	10
Pengelly 1997		1.00	0.001		3	2
Pengelly 2000		3.48	0.00348		12	7

CO	base	change per 10	%change	pollutant	outcome	42% Adj
1997	nt mort			(arith mean)		
our	3868	3.68	0.00368	0.7	10	6
min		2.00	0.002		5	3
max		4.95	0.00495		13	8
Pengelly 1997		1.00	0.001		3	2
Pengelly 2000		3.48	0.00348		9	5

Calculations for respiratory morbidity for CO

CO	morbidity	change per 1	%change	pollutant	outcome	42% Adj
1995	CV			(arith mean)		
mean	814	1.95	0.0195	0.6	10	6
min		0.40	0.004		2	1
max		2.50	0.025		12	7
Pengelly 1996		2.40	0.024		12	7
Pengelly 2000		6.00	0.06		29	17

CO	morbidity	change per 1	%change	pollutant	outcome	42% Adj
1996	CV			(arith mean)		
mean	1123	1.95	0.0195	0.9	20	11
min		0.40	0.004		4	2
max		2.50	0.025		25	15
Pengelly 1996		2.40	0.024		24	14
Pengelly 2000		6.00	0.06		61	35

CO	morbidity	change per 1	%change	pollutant	outcome	42% Adj
1997	CV			(arith mean)		
mean	1176	1.95	0.0195	0.7	16	9
min		0.40	0.004		3	2
max		2.50	0.025		21	12
Pengelly 1996		2.40	0.024		20	11
Pengelly 2000		6.00	0.06		49	29

CO	morbidity	change per 1	%change	pollutant	outcome	42% Adj
1998	CV			(arith mean)		
mean	1108	1.95	0.0195	0.9	19	11
min		0.40	0.004		4	2
max		2.50	0.025		25	14
Pengelly 1996		2.40	0.024		24	14
Pengelly 2000		6.00	0.06		60	35

CO	morbidity	change per 1	%change	pollutant	outcome	42% Adj
1999	CV			(arith mean)		
mean	1031	1.95	0.0195	0.8	16	9
min		0.40	0.004		3	2
max		2.50	0.025		21	12
Pengelly 1996		2.40	0.024		20	11
Pengelly 2000		6.00	0.06		49	29

Calculations for cardiovascular morbidity for CO

CO	mortality	change per 1	%change	pollutant	outcome	42% Adj
1995	CV			(arith mean)		
mean	5612	1.95	0.0195	0.6	66	38
min		0.40	0.004		13	8
max		2.50	0.025		84	49
Pengelly 1996		2.40	0.024		81	47
Pengelly 2000		6.00	0.06		202	117

CO	morbidity	change per 1	%change	pollutant	outcome	42% Adj
1996	CV			(arith mean)		
mean	7702	1.95	0.0195	0.9	135	78
min		0.40	0.004		28	16
max		2.50	0.025		173	101
Pengelly 1996		2.40	0.024		166	96
Pengelly 2000		6.00	0.06		416	241

CO	morbidity	change per 1	%change	pollutant	outcome	42% Adj
1997	CV			(arith mean)		
mean	7468	1.95	0.0195	0.7	102	59
min		0.40	0.004		21	12
max		2.50	0.025		131	76
Pengelly 1996		2.40	0.024		125	73
Pengelly 2000		6.00	0.06		314	182

CO	morbidity	change per 1	%change	pollutant	outcome	42% Adj
1998	CV			(arith mean)		
mean	7322	1.95	0.0195	0.9	129	75
min		0.40	0.004		26	15
max		2.50	0.025		165	96
Pengelly 1996		2.40	0.024		158	92
Pengelly 2000		6.00	0.06		395	229

CO	morbidity	change per 1	%change	pollutant	outcome	42% Adj
1999	CV			(arith mean)		
mean	7572	1.95	0.0195	0.8	118	69
min		0.40	0.004		24	14
max		2.50	0.025		151	88
Pengelly 1996		2.40	0.024		145	84
Pengelly 2000		6.00	0.06		363	211

Calculations for NT mortality for O_3

O_3	base	change per 10	%change	pollutant	outcome	42% Adj
1995	nt mort			(arith mean)		
our	3730	1.38	0.00138	19.2	99	57
Random EB		0.8	0.0008		57	33
Random no EB		0.56	0.00056		40	23
Pengelly 1997		1.35	0.00135		97	56
Pengelly 2000		0.4	0.0004		29	17

O_3	base	change per 10	%change	pollutant	outcome	42% Adj
1996	nt mort			(arith mean)		
our	3694	1.38	0.00138	18.5	94	55
Random EB		0.8	0.0008		55	32
Random no EB		0.56	0.00056		38	22
Pengelly 1997		1.35	0.00135		92	54
Pengelly 2000		0.4	0.0004		27	16

O_3	base	change per 10	%change	pollutant	outcome	42% Adj
1997	nt mort			(arith mean)		
our	3868	1.38	0.00138	19.6	105	61
Random EB		0.8	0.0008		61	35
Random no EB		0.56	0.00056		42	25
Pengelly 1997		1.35	0.00135		102	59
Pengelly 2000		0.4	0.0004		30	18

Calculations for respiratory morbidity for O_3

O_3	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1995	resp			(arith mean)		
Mean	2249	2.8	0.0028	19.2	121	70
Min		1.5	0.0015		65	38
Max		4.9	0.0049		212	123
Pengelly 1997		0.9	0.0009		39	23
Pengelly 2000		1.12	0.00112		48	28

O_3	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1996	resp			(arith mean)		
Mean	3085	2.8	0.0028	18.5	160	93
Min		1.5	0.0015		86	50
Max		4.9	0.0049		280	162
Pengelly 1997		0.9	0.0009		51	30
Pengelly 2000		1.12	0.00112		64	37

O_3	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1997	resp			(arith mean)		
Mean	2738	2.8	0.0028	19.6	150	87
Min		1.5	0.0015		80	47
Max		4.9	0.0049		263	153
Pengelly 1997		0.9	0.0009		48	28
Pengelly 2000		1.12	0.00112		60	35

O_3	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1998	resp			(arith mean)		
Mean	3266	2.8	0.0028	20.8	190	110
Min		1.5	0.0015		102	59
Max		4.9	0.0049		333	193
Pengelly 1997		0.9	0.0009		61	35
Pengelly 2000		1.12	0.00112		76	44

O_3	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1999	resp			(arith mean)		
Mean	3330	2.8	0.0028	21.2	198	115
Min		1.5	0.0015		106	61
Max		4.9	0.0049		346	201
Pengelly 1997		0.9	0.0009		64	37
Pengelly 2000		1.12	0.00112		79	46

Calculations for cardiovascular morbidity for O_3

O_3	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1995	CV			(arith mean)		
Our average	5612	4.5	0.0045	19.2	485	281
Minimum		1.6	0.0016		172	100
Maximum		7.5	0.0075		808	469
Pengelly 1997		NA				0
Pengelly 2000		4.52	0.00452		487	282

O_3	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1996	CV			(arith mean)		
Our average	7702	4.5	0.0045	18.5	641	372
Minimum		1.6	0.0016		228	132
Maximum		7.5	0.0075		1069	620
Pengelly 1997		NA				0
Pengelly 2000		4.52	0.00452		644	374

O_3	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1997	CV			(arith mean)		
Our average	7468	4.5	0.0045	19.6	659	382
Minimum		1.6	0.0016		234	136
Maximum		7.5	0.0075		1098	637
Pengelly 1997		NA				0
Pengelly 2000		4.52	0.00452		662	384

O_3	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1998	CV			(arith mean)		
Our average	7322	4.5	0.0045	20.8	685	397
Minimum		1.6	0.0016		244	141
Maximum		7.5	0.0075		1142	662
Pengelly 1997		NA				0
Pengelly 2000		4.52	0.00452		688	399

O_3	morbidity	change per 10	%change	pollutant	outcome	42% Adj
1999	CV			(arith mean)		
Our average	7572	4.5	0.0045	21.2	722	419
Minimum		1.6	0.0016		257	149
Maximum		7.5	0.0075		1204	698
Pengelly 1997		NA				0
Pengelly 2000		4.52	0.00452		726	421

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