

**AIR POLLUTION-RELATED BURDEN OF  
ILLNESS IN TORONTO: 2004 UPDATE.**  
Technical Report

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**Prepared for:**

**Environmental Protection Office**

**Toronto Public Health**

**Community and Neighbourhood Services**

**City of Toronto, Ontario**



# **AIR POLLUTION-RELATED BURDEN OF ILLNESS IN TORONTO: 2004 UPDATE.**

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## **Executive Summary**

In the last decade, there has been growing use of burden of illness estimates as an indicator of air quality impact at all levels of government (Federal, Provincial and Municipal), by non-governmental agencies and community groups. Its greatest application seems to have been in the planning mode: to stimulate action; to address perceived errors; and to avoid harm from planned changes which will have environmental consequences. The population in general has noted that governments can be called into action much more by drawing attention to threats to community health than by focussing on adverse impacts on the natural environment. Thus to a properly educated and convinced community, the burden of illness study based on local air quality and health data, but using risk coefficients from the literature is a cost-effective and reliable approach to the problem of risk evaluation and risk communication.

Toronto Public Health has previously used the BOI methodology to estimate the air pollution-related burden of illness for the City of Toronto associated with several criteria pollutants (Pengelly et al., 2000). The present work has been undertaken for two reasons: first, to update the scientific foundations of the previous study; and second, to assess the air pollution burden of illness related to the most recent measurements of air quality in Toronto.

The APBIT 2000 study made use of published literature up to February 2000, and thus the starting point for the review for the current study was literature beginning in January 2000, and a few prior works which had become known to us in the interim.

For the update to the APBIT May 2000 report, a review of the recent epidemiological studies evaluating the associations between air pollution and health was conducted for the period January 2000 to September 2003. Subsequently, in light of rapid developments in this area, additional reports were selected, which led to the inclusion of other studies, as well as the rejection of some studies already included..

In the context of heavy pressure on the EPA to revise the 1987 Particulate Criteria Document, the Health Effects Institute, in the United States funded in 1996 two complementary studies which together are referred to as the National Morbidity, Mortality, and Air Pollution Study (NMMAPS: Samet 2000b, 2000c). In mid-2002, two groups of investigators independently reported findings in the literature which had a major impact on the interpretation of most of the time-series epidemiological studies reported up to that time. In this Report we have given a brief history of that finding, referred to as the "GAMs problem".

It should be noted that the focus of the NMMAPS studies and the subsequent re-analysis was the health effect of particulate air pollution. The impact of the GAMs problem is such that the only recent data currently considered acceptable for the purposes of determining human health risk estimates associated with ambient particles are either those which derive from the re-analysis reports, or those from studies which have explicitly avoided the GAMs problem.

From the literature review, we found that by far the greatest effort in the period 2000-2004 has been to examine the relationship between non-traumatic(NT) mortality and fine particles, measured in a number of different ways. As a result, we have the greatest confidence in our estimate of NT mortality associated with fine particulate air pollution in Toronto.

Examination of the literature between 2000 and 2004 has yielded only a few studies of hospital admissions in the whole population, although there have been a few studies examining morbidity in children and seniors; in children, primarily studies of asthma, and in seniors, studies of

obstructive lung disease and heart disease. Although useful, these have not allowed us to make direct comparisons with the APBIT 2000 study. As a result, 5 of the 9 coefficients we have used to estimate hospital admissions in the current study are from APBIT 2000.

There are several factors (including the changes in understanding of the relevant science) which might have led us to expect a reduction in air-pollution related mortality in Toronto. The overall non-traumatic mortality for 1999 is 13,246, which is a 20% reduction from the 1995 figure of 16615. Part of this reduction is a consequence of a different way of handling missing postal codes in the mortality data, but a reduction persists if this is accounted for. This is in spite of a 7% increase in the population of the city overall.

There has also been a change in the character of air pollution in Toronto between 1995 and 1999. It appears that there has been a large increase in the air pollution related to fossil-fuelled electrical generation (PM10, SO2, SO4), and a much smaller decrease in transportation-related primary pollutants (CO and NO2), but a large increase in ozone, in part from long-range transport.

We have examined the recent epidemiological literature, as well as recent reviews carried out for regulatory purposes, and have determined a set of coefficients of the air pollution-related burden of illness applicable to Toronto. We have also summarized recent quality-controlled air pollution data for several sites within the City, and have obtained baseline health outcome information in a form which can be linked to the air quality monitoring sites. Using this information, we have estimated the non-traumatic mortality associated with air pollution in Toronto for the year 1999, the most recent year for which there was adequate information. We have quantified the air pollution risk in the City of Toronto taking into account the most recent scientific evidence available, and have estimated that **approximately 1700 premature deaths each year, and between 3000 and 6000 hospital admissions** are associated with the criteria pollutants O3, NO2, SO2, CO and PM10 breathed by the public at large.

In comparing this result to that previously obtained in a similar exercise in 2000, we find that in spite of substantial revisions to earlier studies which have tended to reduce the estimate of burden of illness of air pollution in urban centres, using the same methods, the estimate of premature mortality as a result of the present work is consistent with the lower bound estimate of 700 premature deaths made in 2000, ignoring the impact of chronic exposure to particles. However, based on the much greater scientific strength of the revised and re-affirmed chronic exposure studies, we believe that a more supportable estimate of non-traumatic mortality attributable to air pollution in Toronto is 1700. The estimates of 3000 to 6000 excess hospital admissions in this update are consistent with the lower and upper bounds (3300 to 7600) of the estimate made in 2000.

New studies in the literature demonstrate the importance of focussing on susceptible groups: infants, children and the elderly. Rather than examining the effect of air pollution, pollutant by pollutant on the population as a whole, future studies should examine the role of the pollutant mix on susceptible groups, and to develop air quality management strategies aimed at the sources of the most toxic components of the pollutant mix.

The results of this estimation of the air pollution-related burden of illness in the City of Toronto shows that every year there is still a major public health cost attributable to criteria pollutants from fossil-fuel combustion in the City and beyond.

***To reduce the levels of air pollution where people live, governments at all levels must sustain a commitment to: 1) reduce our dependency on fossil energy, particularly coal-fired electrical generation, and 2) support and sustain public transit in order to reduce the pollution burden created by automobiles and trucks.***



## **Acknowledgements:**

The authors wish to acknowledge the support and leadership in the field of air quality and public health shown by Dr. Sheela Basrur. Dr. Basrur has understood the importance clean air has to the health and well-being of the community, and the constant need to keep this issue in the forefront of priorities at the municipal, provincial and federal levels of government.

This work would not have been possible without the interest, support and professional guidance of Dr. Monica Campbell, with whom we have had a productive, rewarding and friendly collaboration for many years.

Air quality data were provided through the offices of Dr. Chad Cheng, of the Canadian Meteorology Service, Environment Canada; and Phil Keily and Melynda Mesbouris of the Ontario Ministry of the Environment.

Health outcome data were obtained and summarized by Monica Bienenfeld, Epidemiologist, of Toronto Public Health.

Dr. Rick Burnett, of Health Canada provided much useful discussion of technical issues, and early access to important work he and his associates carried out, which was key to the development of recent and reliable estimates of risk coefficients relating non-traumatic mortality to gaseous pollutants.

## **1. BACKGROUND**

### **1.1 Burden of Illness**

In the last decade, there has been growing use of burden of illness estimates as an indicator of air quality impact at all levels of government (Federal, Provincial and Municipal), by non-governmental agencies and community groups<sup>1</sup>. Its greatest application seems to have been in the planning mode: to stimulate action; to address perceived errors; and to avoid harm from planned changes which will have environmental consequences. The population in general has noted that governments can be called into action much more by drawing attention to threats to community health than by focussing on adverse impacts on the natural environment. There is a danger, however that a community may call for a primary research “community health study” which, for the most part, is likely to be doomed to failure by virtue of its limited statistical power (Legator and Howells-Daniel 1994). Thus to a properly educated and convinced community, the burden of illness study based on local air quality and health data, but using risk coefficients from the literature is a much more cost-effective and reliable approach to the problem of risk evaluation and risk communication.

Interest in the use of burden of illness (BOI) estimates to assess the human health risk of existing or expected levels of air pollution has been stimulated by the availability of increasingly well supported risk coefficients from the scientific literature. Data and methods have become available which allow for the quantification of adverse health effects associated with exposure to air pollution in Europe, South and Central America, the United States and Canada. These have provided numerical coefficients associating a wide range of health end points with air pollutants, both alone and in combination, and the coefficients may be used to estimate the air pollution burden of illness in a community, even if no explicit epidemiological studies have been previously conducted in that community. This can be done if air pollution levels have been measured in the community (or can be otherwise estimated), and if disease specific overall burden of illness data (e.g., hospital admissions, mortality) are also available for that community.

To further expand the use of Burden of Illness methodology, Environment Canada and Health Canada developed a model package with the assistance of a consultant (the Air Quality Valuation Model: AQVM), the methodology for which has been described (Chestnut et al., 1999). This package includes databases for air quality and population-based health outcomes for most urbanized areas of Canada, as well as “concentration-response functions” for human health effects. The AQVM moves beyond BOI estimates, however, in that it assesses economic costs associated with the health burdens, and potential benefits associated with avoidance of those burdens by reduction in pollutant levels achieved by various policy or planning strategies. A major design limitation of the AQVM arises from the fact that it is restricted to estimation of BOI associated with ozone and fine particle exposure. This model was used recently by the Ontario Medical Association (see endnote 1).

Toronto Public Health has previously used the BOI methodology to estimate the air pollution-related burden of illness for the City of Toronto associated with several criteria pollutants (Pengelly et al., 2000). The present work has been undertaken for two reasons: first, to update the scientific foundations of the previous study; and second, to assess the air pollution burden of illness related to the most recent measurements of air quality in Toronto.

### **1.2 Factors Influencing Recent Direction of Air Pollution Scientific Research**

#### The Role of the U.S. Environmental Protection Agency.

The United States Environmental Protection Agency (EPA) is a powerful Federal institution, and the consequences of its actions and regulations have very large economic consequences to many industries in the USA. Over the last 40 years in the USA, the focus of air pollution regulation by the EPA has been mainly on the reduction of the levels of ground-level (tropospheric) ozone,

largely on the basis of controlling automobile-related emissions. In the last 20 years, however, epidemiological studies from all parts of the world have demonstrated the association of fine particle pollution with mortality and morbidity in urbanized areas. Two major sources of fine particle pollution are coal-fired electric power generating stations; and diesel vehicles.

### Particles.

In 1987, the US EPA promulgated a 24-hr standard for fine particles (PM<sub>10</sub>) of 150 ug/m<sup>3</sup>. Following this, on the basis of many epidemiological studies it became clear to many in the public health field that this standard was not protective of health, and there was increasing pressure on EPA to review and revise it. There was also equally strong pressure from industry to leave it at the 1987 level. Nevertheless, in 1997 a new standard was promulgated which recognized the importance of the so-called “respirable fraction” of fine particles with an aerodynamic mass median diameter less than 2.5 micrometers (PM<sub>2.5</sub>). This standard consisted of a 24-hr concentration of 65 ug/m<sup>3</sup> for PM<sub>2.5</sub>, and the retention of the 24-hr concentration of 150 ug/m<sup>3</sup> for PM<sub>10</sub>. The standard was the subject of vigorous litigation on the part of industry on a variety of grounds, with the aim of returning to the 1987 situation. On the other hand, the American Lung Association and others litigated on the basis that the 1997 standard was not sufficiently protective of human health.

In March 2002 the US Appeals Court rejected both sets of challenges, leaving the EPA free to implement the regulation. As a result of further litigation against EPA by environmental groups in March 2003, a settlement was filed in the US District Court for the District of Columbia in May 2003, setting out the timetable for review of the 1997 standard.. EPA was required to issue the Final Criteria Document for Particulate Matter by December 19, 2003.

During this whole period, EPA was not unconscious of the need to support and review research on the effects of fine particles on health, and a substantial research effort was set in motion, a major portion of which was coordinated by the Health Effects Institute (HEI)<sup>1</sup>.

## **2. LITERATURE REVIEW**

The APBIT 2000 study made use of published literature up to February 2000, and thus the starting point for the review for the current study was literature beginning in January 2000, and a few prior works which had become known to us in the interim. In addition, we were aware of a finding in the literature which had a major impact on the interpretation of most of the time-series epidemiological studies reported up to that time. Included in the following is a brief history of that finding, referred to as the “GAMs problem”.

For the update to the APBIT May 2000 report, a review of the recent epidemiological studies evaluating the associations between air pollution and health was conducted for the period January 2000 to September 2003. The review began with searches using the following databases: Biological Sciences, Environmental Pollution and Management, Epidemiology, Medline and Toxline; with keywords including: air pollution, health, time series, particulate matter and gaseous pollutants. In order to be selected the study had to include the pollutants and end points of interest in addition to being a time-series study. Twenty-eight articles were selected which met the criteria.

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<sup>1</sup> The Health Effects Institute (HEI) is an independent, nonprofit corporation chartered in 1980 to provide high-quality, impartial, and relevant science on the health effects of pollutants from motor vehicles and from other sources in the environment. Supported jointly by EPA and industry, HEI has funded hundreds of studies, producing important research findings on the health effects of a variety of pollutants, most recently particulate air pollution.

## 2.1 Growth and Nature of the Literature

An indication of the activity in the air pollution scientific community is demonstrated in Figures 1 and 2 below. Over the last 15 years the number of studies published in English has grown by more than a factor of three, and the number of epidemiological time series studies has grown by ten times. At the time of preparation of this Report, literature for only the first two months of 2004 were available.

Figure 1.

### Air Pollution and Health Literature studies 1990-2004

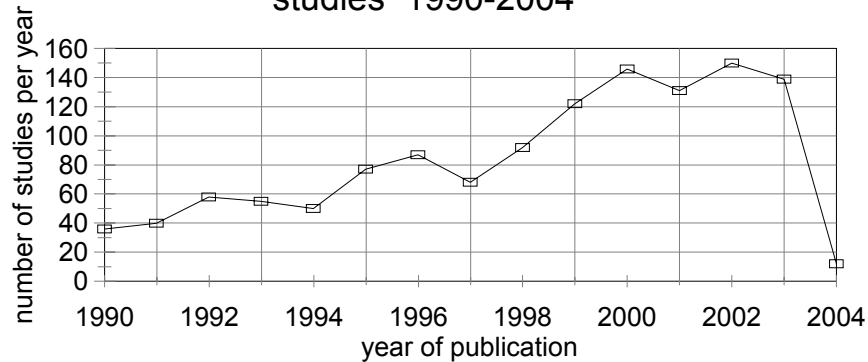
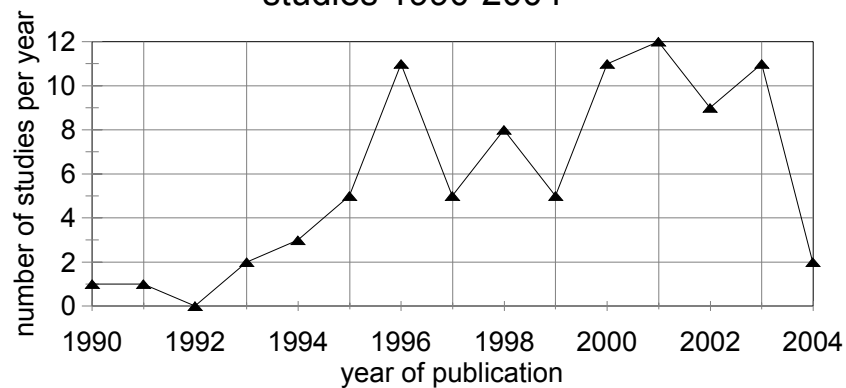


Figure 2.

### Air pollution / health time series studies 1990-2004



However, as we shall discuss below, at least some of the publications in 2003 were re-analysis of studies published in earlier years as a result of analytical problems noted in mid-2002. The consequence of this was that all time-series studies using “generalized additive models with nonparametric smoothers”; a technique introduced in 1995; were subject to revision. The technique was introduced by Schwartz, in the USA, and rapidly adopted in many studies there, but was not adopted as quickly in Europe. The first APHEA studies, a major European collaborative effort, did not use this technique.

## 2.2 NMMAPS and the “GAMs Problem”

In the context of heavy pressure on the EPA to revise the particulate Criteria Document, HEI in 1996 funded two complementary studies which together are referred to as the National Morbidity, Mortality, and Air Pollution Study (NMMAPS: Samet 2000b, 2000c). The authors concluded (in June 2000)

*“The epidemiologic evidence on PM and mortality and morbidity has prompted the promulgation of a new standard in the United States and a rethinking of guidelines for PM in Europe. Our analyses provide evidence that air pollution with particles is still adversely affecting the public’s health and strengthen the rationale for limiting concentrations of respirable particles in outdoor air.”*

In the summer of 2002 it was reported to the Health Effects Institute that epidemiological time-series study designs employing statistical Generalized Additive Models (GAMs) using non-parametric smoothing methods could result in incorrect estimates of effects of air pollution on health. These observations raised questions about the validity of risk coefficients based on GAMs methodology made up to that time. It was recognized that this could have a major impact on the NMMAPS study, as well as many other studies in the literature which used the same methodology. The Health Effects Institute responded by initiating and funding a comprehensive re-analysis of the NMMAPS studies, as well as selected studies undertaken elsewhere in the world (HEIRA 2003).

[Editor’s note: the following is an abridged summary of the HEI “Statement” at the beginning of the report HEIRA 2003: pp i-v]

Work by Dominici and colleagues (2002c) and Ramsay and coworkers (2003) called for caution in use of the S-Plus software for fitting generalized additive models (GAMs) to estimate relative rates of mortality and morbidity in time-series studies of air pollution and health.. Dominici et al. found that biased estimates of the regression coefficients and their standard errors can result when default convergence criteria are used when confounding factors are modeled with nonparametric smooth functions. Independently, Ramsay and coworkers (2003) noted that the S-Plus GAM function uses a computational approximation that, when the air pollution variable correlates with the nonlinear functions included in the model, can underestimate standard errors of the relative rates.

Concurrently, results of NMMAPS and other time-series studies were under review as part of the periodic review of the National Ambient Air Quality Standards (NAAQS) for PM. Thus understanding how these results might be changed by new analyses became a priority.

As the funding sponsor for NMMAPS, HEI asked the NMMAPS investigators to prepare reports presenting their new analyses. Two NMMAPS reports were submitted to HEI: “Mortality Among Residents of 90 Cities” by Dominici and colleagues and “Morbidity and Mortality Among Elderly Residents of Cities with Daily PM Measurements” by Schwartz and colleagues. A Special Panel of the HEI Health Review Committee reviewed these reports.

In addition, in the summer of 2002, EPA identified additional key studies from the US, Canada, and Europe that were cited in the draft of the Air Quality Criteria for Particulate Matter and had used GAM in their analyses. The EPA requested that the investigators who had conducted those studies also carry out and report revised analyses. The agency asked that they (1) reanalyze the original data using the same nonparametric approach (GAMs) that was used originally, but with stricter convergence criteria; and (2) examine the sensitivity of the findings obtained with GAMs when using parametric models. The latter would also estimate more accurate standard errors. EPA requested that HEI review the resulting short communication reports of the revised analyses and write a Commentary on the effect of different analytic approaches on the results. HEI agreed to take on this effort.

[ Ed. Note: It is to the great credit of the HEI and all of the investigators involved that a report of all of these findings was made available in May 2003.]

## Results of the Re-Analysis.

Overall, for the NMMAPS data, GAMs with stricter convergence criteria and GLMs with natural cubic splines resulted in lower estimates of effect than those from the original analyses conducted with GAM and default convergence criteria. In individual cities, the revised effect estimates for mortality typically decreased and standard errors increased. Across the 90 cities, the revised mean effect on mortality decreased substantially from 0.41% (increase per 10 ug/m<sup>3</sup> increase in PM<sub>10</sub> concentration at lag 1) to 0.27% when using GAM with stricter criteria and to 0.21% when using GLM with natural cubic splines: an overall decrease of nearly 50%.

The overall decreases in effect estimates for hospitalizations for cardiovascular diseases and for chronic obstructive pulmonary disease were smaller (approximately 8% to 10%); a small but clear association continued to be found. The effect estimate on pneumonia hospitalizations was substantially reduced. As in the original studies, revised results for PM<sub>10</sub> morbidity and mortality did not change substantially when copollutants were included in the models.

## Other Studies

Nineteen primary authors submitted 21 short communication reports presenting results from analyses originally reported in 37 published original articles and reports. Differences between the original and revised effect estimates varied substantially across and within studies. Overall, GAMs with stricter convergence criteria and GLMs with natural cubic splines yielded lower effect estimates but largely continued to identify an association of PM with mortality and morbidity, in particular for cardiovascular and respiratory diseases. The overall impact of the other revised analyses included:

- While the number of studies showing an association of PM with mortality was slightly smaller, the PM association persisted in the majority of studies.
- In some of the large number of studies in which the PM association persisted, the estimates of PM effect were substantially smaller.
- In the few studies in which investigators performed further sensitivity analyses, some showed marked sensitivity of the PM effect estimate to the degree of smoothing and/or the specification of weather.

## General Conclusions

The impact of using more appropriate convergence criteria on the estimates of PM effect in the revised analyses varied greatly across the studies. In some studies, stricter convergence criteria had little impact, and in a few the impact was substantial. **In no study were conclusions based on the original analyses changed in a meaningful way by the use of stricter criteria** (editorial emphasis).

These revised analyses have renewed the awareness of the uncertainties present in estimates of short-term air pollution effects based on time-series data. Neither the appropriate degree of control for time, nor the appropriate specification of the effects of weather, has been determined for time-series analyses. In the absence of adequate biological understanding of the time course of PM and weather effects, and their interactions, the Panel recommends exploration of the sensitivity of future time-series studies to a wider range of alternative degrees of smoothing and to alternative specifications of weather variables.

## Impact Calculations (Burden of Illness)

Common practice has come to use effect estimates from observational air pollution studies to estimate the impact of air pollution on a large population such as an entire country. If effect estimates from the NMMAPS 90 cities mortality study were applied, the revised impact would be approximately half of the estimated impact derived using the original effect estimates. This example reinforces the need to qualify estimates of impact by specifying the assumptions and uncertainties on which the estimates are based.

### **2.3 Consequences of the “GAMs problem” to BOI estimates for Toronto.**

It should be noted that the focus of the NMMAPS studies and the subsequent re-analysis was the health effect of particulate air pollution. The impact of the GAMs problem is such that the only recent data currently considered acceptable for the purposes of determining human health risk estimates associated with ambient particles are either those which derive from the re-analysis reports, or those from studies which have explicitly avoided the GAMs problem.

The impact of the problem on studies of the burden of illness from gaseous pollutants was only of importance to EPA (and consequently to HEI) to the extent that gaseous pollutants (referred to as co-pollutants) might act as surrogates for particles or confounders to the particulate / health outcome relationship. There is no doubt that the GAMs problem impacts on the recent studies of

the health effects of gaseous pollutants, but much less attention has been paid to this issue.

In time-series studies of air pollution and mortality since 1995, GAM has been the most widely applied method, because it allows for nonparametric adjustments for nonlinear confounding effects of seasonality, trends, and weather variables (Schwartz 1994). The report by Schwartz *et al.* in 1995 was likely the first to use this technique in relating health outcomes to air pollution (Schwartz *et al.*, 1995). The consequence of this is that many of the risk coefficients used in the APBIT 2000 study were derived from studies published within the last 10 years which used GAMs.

## **2.4 Science Review for APBIT 2004 Update**

Twenty of the 28 papers obtained in the initial search are components of the HEI Re-Analysis document (HEIRA 2003), and are some of the original publications which were re-analysed in that document. The re-analysis examined one major document: The National Morbidity, Mortality and Air Pollution Study (NMMAPS Part I and Part II; Samet *et al.* 2000b and 2000c), and a group of original publications of 37 selected time-series studies. Some of the selected time-series studies were also components of the NMMAPS study. In the publications selected for re-analysis, 25 focussed specifically on mortality, 8 on morbidity, and 4 on both.

Many studies included both single and multi-pollutant models. For the initial 28 papers, an analysis matrix was constructed which allowed for rapid comparison of key methodological, analytical and outcome components among studies. If the multi-pollutant model yielded a significant impact on the single pollutant effect it was reported in the summary table. This is similar to the process used in APBIT 2000. Subsequently, in light of rapid developments in this area, additional reports were selected, which led to the inclusion of other studies, as well as the rejection of some studies already included..

In addition, a very useful broad and comprehensive summary of the work primarily pertaining to particulate pollution may be found in the U.S. EPA Fourth External Review Draft of Air Quality Criteria for Particulate Matter (June, 2003): Volume II (USEPA 2003), available on the USEPA web site.

### **2.4.1 Premature Non-traumatic Mortality**

#### **2.4.1.1 Particles**

##### **2.4.1.1.1 Acute (Short-term Exposure) Response**

In the APBIT 2000 study we defined the “unit incremental health risk per cent ( $\Delta H\%$  /unit)” as the per cent change in health outcome, per unit of pollutant (APBIT 2000; Section 3.3.1). The value of  $\Delta H\%$  /unit used for non-traumatic mortality associated with  $PM_{10}$  was 0.08%/( $\mu\text{g}/\text{m}^3$ ). This value was the result of a review carried out in support of the Canadian National Ambient Air Quality Objectives for Particulate Matter (APBIT 2000 ref NAAQO 1999b). The figure of 0.08%/( $\mu\text{g}/\text{m}^3$ ) was a mean derived from 23 studies ( at least one of which used the GAMs method) in several countries. It is of interest that the study that used GAMs (Schwartz *et al.*, 1996) reported the numerically identical coefficient for mortality associated with  $PM_{10}$  of 0.08%/( $\mu\text{g}/\text{m}^3$ ).

The corresponding value determined from NMMAPS 2000 is 0.041, and after revision has been reduced to 0.027 or 0.021, depending on method. It must be borne in mind that the NMMAPS is a single study of 90 US cities. On the other hand, the revision of the study (Burnett *et al.*; 2000) of eight Canadian cities (done in conjunction with the NMMAPS re-analysis) gives statistically significant values between 0.043 and 0.095, again depending on method (Burnett and Goldberg 2003).

In summarizing the results of his own re-analysis of data, Joel Schwartz provides a useful interpretation of the whole re-analysis exercise (Schwartz 2003a):

“These results are similar to most of the other re-analyses of time-series studies that have been reported. That is, the main conclusions about association, confounding, and effect modification have not been changed. In most cases there are modest changes in size of the effect estimates although in a few cases they increased, and in a few instances (notably the 90 cities NMMAPS mortality estimates) the change in effect size estimates were more substantial (Dominici et al 2002). Moreover, these results are similar to the results of the APHEA1 study (Katsouyanni et al 1997), a large multicity study that did not use GAM models to control for season and weather, and hence was not affected by the problems with the GAM software. This general stability provides confidence that the overall conclusions previously reached about this extensive literature should remain unchanged.”

This can be seen in the results of studies explicitly relating PM<sub>10</sub> to non-traumatic mortality in Table 1 (following)

Table 1. Comparison of  $\Delta H\%$  /unit values for non-traumatic mortality and PM<sub>10</sub> from the HEI Re-Analysis: for NMMAPS II, p19; for “selected papers” from Appendix A, p 272.

STUDY	PRE-REVISION	POST-REVISION
Samet et al., (NMMAPS II, 2000); Dominici et al., HEIRA (2003) p.19	0.041	0.021 - 0.27
Burnett et al.(2000); HEIRA (2003) p.85	0.087	0.053 - 0.070
Fairley (1999); HEIRA (2003) p.97	0.16	0.156 - 0.166
Hoek et al.(2000); HEIRA (2003)p.133	0.018	0.019
Katsouyanni et al.(2001); HEIRA (2003) p. 157	0.062	0.041 - 0.059
Klemm et al. (2000); HEIRA (2003) p.165	0.08	0.04 - 0.07
Lippmann et.al., (2000); Ito (HEIRA 2003) p. 143	0.034	0.013 - 0.018 (p>0.05)
Moolgavkar (2000); HEIRA (2003) p.183	0.048	0.045 - 0.047
Schwartz (2000); HEIRA (2003a) p.211	0.067	0.055 - 0.66
Wichmann et al.,(2000); Stölzel et al., HEIRA (2003) p.231	0.126	0.105 - 0.123
Zanobetti et al. (2002); HEIRA (2003) p.249	0.07	0.057 - 0.67

The range in the post-revision coefficients reflects the results of different strategies being used to deal with the effects of time and other confounding factors: the degree of temporal smoothing used in the original analyses, the number of smoothed terms in the models, and the degree of nonlinear collinearity (concurvity) among the smoothed terms. The relative importance of these and other explanations for variability of results among all the studies remains unclear.

For the purposes of the present work, we have chosen to select the lowest in the range given above for 10 of the studies (excluding Lippmann, which was not statistically significant), and then to take the mean of these lowest values. This gives a coefficient of 0.06 % change in NT mortality /  $\mu\text{g}/\text{m}^3$  PM<sub>10</sub>.

#### 2.4.1.1.2 Mortality Associated with Chronic, as Distinct from Short-term Exposure.

In our previous report (APBIT 2000) we took note of the evidence in the literature that seemed to demonstrate a greater risk of premature mortality associated with chronic (long-term: of the order of years) exposure to particulate pollution in comparison to acute (short-term: a few days) exposure. Pope and co-workers reported in 1995 a study which they found a unit incremental mortality risk ( $\Delta H\% / \mu\text{g}/\text{m}^3$ ) of 0.7 for fine particles, nearly ten times the figure of 0.08 we used for the acute risk in APBIT 2000. At that time, Pope *et al.*'s study, as well as another study of chronic particle effects by Dockery *et al.* were under challenge by industry, and a major re-analysis was just being completed. We felt at that time to use the chronic coefficients would not be scientifically conservative, and thus they were not included in the mortality estimates.

To make the current "Update" as consistent as possible with the earlier work, we again include the chronic estimates, and the data are provided in the calculations shown in the Appendix. The most recent (1999) mortality data, the corresponding 1999 air quality data, and the revised risk coefficients from the re-analysis (HEI 2000) of the two studies (Pope *et al.*, and Dockery *et al.*) were used. We estimate that 447 premature deaths are associated with chronic sulphate exposure, and 1236 premature deaths with chronic  $\text{PM}_{2.5}$  exposure. However, we must caution (as we did in the 2000 report) that these two estimates should not be summed, as in theory the sulphate air quality measure is part of the  $\text{PM}_{2.5}$  measure, because sulphate particles are usually less than  $1 \mu\text{m}$  in aerodynamic size.

The U.S. EPA states (USEPA 2003):

*Long-term PM exposure durations, on the order of months to years, as well as on the order of a few days, are statistically associated with serious human health effects (indexed by mortality, hospital admissions/medical visits, etc.). More chronic PM exposures, on the order of years or decades, appear to be associated with life shortening well beyond that accounted for by the simple accumulation of the more acute effects of short-term PM exposures (on the order of a few days). Substantial uncertainties remain regarding the magnitude of and mechanisms underlying chronic health effects of long-term PM exposures and the relationship between chronic exposure and acute responses to short-term exposure.*

*Recent investigations of the public health implications of such chronic PM exposure-mortality effect estimates were also reviewed. Life table calculations by Brunekreef (1997) found that relatively small differences long-term exposure to airborne PM of ambient origin can have substantial effects on life expectancy. For example, a calculation for the 1969-71 life table for U.S. white males indicated that a chronic exposure increase of  $10 \mu\text{g}/\text{m}^3$  PM was associated with a reduction of 1.3 years for the entire population's life expectancy at age 25.*

*Also, new evidence of associations of PM exposure with infant mortality and/or intrauterine growth retardation and consequent increase risk for many serious health conditions associated with low birth weight, if further substantiated, would imply that life shortening in the entire population from long-term PM exposure could well be significantly larger than that estimated by Brunekreef (1997).*

Our preliminary estimate of premature mortality in the 2004 "update" of 700 premature deaths attributable to the air pollution mixture in Toronto using 1999 data, was based on recent studies and revisions of older work which examined the acute response to air pollution, which was the method employed in APBIT 2000. Scientific understanding has advanced substantially in the short period of time between the 2000 APBIT study and the present, and in addition has withstood challenges by industrial and regulatory stakeholders. The two major studies of chronic exposure to fine particles have been carefully validated and their original conclusions supported, if not strengthened by the re-analysis.

Our final approach to the estimate of premature mortality associated with fine particles is to use the chronic mortality associated with  $\text{PM}_{2.5}$  (1236), in place of the acute mortality figure associated with  $\text{PM}_{10}$  of 177. This when added to the premature mortality associated with the

gases (519, see below) yields a total estimate of pollution-related premature mortality of 1755. Although not directly comparable in method to the estimate made in 2000, it does represent a closer reflection of the development of scientific understanding in the intervening period.

#### 2.4.1.2. Gases

Since the identification of the GAMs problem is relatively recent (Dominici 2002), and the major focus since that time has been on the burden of illness related to fine particulate pollution, relatively little attention has been paid to studies directed explicitly to determine the role of gaseous pollutants. A few studies which were part of the HEI Re-analysis examined gases as co-pollutants of the relationships derived for particles, and some coefficients may be derived from them. In addition, a small number of studies in the last decade either did not use GAMs, or used them in a way that was relatively unaffected by the problems identified by Dominici *et al.*, and Ramsay *et al.*

##### Stieb *et al.* Meta-Analysis

In April, 2002, Stieb *et al.*, from Health Canada published the results of a comprehensive, systematic and analytic synthesis of studies from around the world that link both pollutant gases and particles with daily mortality in which 175 publications are cited. Stieb *et al.* found statistically significant coefficients relating the pollutants PM<sub>10</sub>, CO, NO<sub>2</sub>, O<sub>3</sub> and SO<sub>2</sub> to mortality in single pollutant models, but noted that effect sizes were generally reduced in multipollutant models, remaining significantly different from zero for PM<sub>10</sub> and SO<sub>2</sub>.

Stieb *et al.* (2003) revisited their original meta-analysis in light of the “GAMs problem” noting that the prevalence of the GAM based estimates had increased dramatically since these methods were first applied in this area in 1996. They classified estimates of effect size from primary studies according to whether they were GAM based, assuming that any estimate that was based on non parametric smoothing functions of time or weather would be potentially affected. A summary of results from both studies is given in Table 2 below, where effect size has been expressed per unit of pollutant..

Table 2. Percent increase excess all-cause non-traumatic mortality per unit pollutant ( $\Delta$  H% / unit), single and multi-pollutant models (after Stieb *et al.*, 2002, 2003), also Burnett *et al.* 2004.

Pollutant	PM <sub>10</sub>	CO	NO <sub>2</sub>	O <sub>3</sub>	SO <sub>2</sub>
unit	$\mu\text{g}/\text{m}^3$	ppm	ppb	ppb	ppb
$\Delta$ H%/unit ( <b>mixed, 2002</b> ) single pollutant	0.064	1.545	0.117	0.051	0.096
multipollutant	0.038	0.636 *	0.038 *	0.019 *	0.085
$\Delta$ H%/unit ( <b>non-GAM, 2003</b> ) single poll.	0.042	4.273	0.042	0.045	0.096
multipollutant	0.032	0.000 *	0.021 *	0.026 *	0.085
$\Delta$ H%/unit ( <b>GAM, 2003</b> ) single poll.	0.070	1.455	0.142	0.054	0.106
multipollutant	0.042	0.636 *	0.046 *	0.013 *	0.085
$\Delta$ H%/unit ( <b>Burnett <i>et al</i> 2004</b> ) single poll.		0.670	0.100	0.090	0.074
multipollutant		0.190 *	0.075	0.085	0.046

Note: 1. \* indicates coefficient is not statistically significant.  
2. Concentrations of pollutants 24 hr avg, except for O<sub>3</sub> which is 24 hr max.

It can be seen from the re-analysis that non-GAM effect sizes are less than GAM effect sizes in general, with some exceptions. Pooled estimates for NO<sub>2</sub> and PM<sub>10</sub> from both single and multipollutant models were reduced based on non-GAM vs. GAM-based studies (69 and 55% reductions for NO<sub>2</sub> for single and multipollutant models, respectively, and 39 and 21% reductions for PM<sub>10</sub>). In the case of O<sub>3</sub>, a 16% reduction was observed based on single pollutant estimates, while a doubling of effect size was seen based on multipollutant estimates. A three fold increase in single pollutant effect size was seen for CO, but it should be borne in mind that multipollutant estimates for NO<sub>2</sub>, CO and O<sub>3</sub> were not statistically significant. There was an essentially no difference between non-GAM and GAM-based estimates for SO<sub>2</sub>. In many ways the results of the re-analysis by Stieb *et al.* are very similar to those found in the re-analysis of the NMMAPS and associated studies. The additional benefit of the Stieb *et al.* re-analysis is that it includes explicit data for the gaseous pollutants.

### Burnett *et al* Multipollutant Analyses

Two earlier multipollutant studies by Burnett *et al* (Burnett *et al* 1998, 2000) made use of GAMs with nonparametric smoothing functions, and as a result were potentially subject to the problem described above. For this, among other reasons, Burnett and co-workers have undertaken a very recent study of air pollution-related mortality in Canadian cities which utilizes different statistical methods not subject to the same problems (Burnett *et al.* 2004). Burnett (personal communication January 7, 2004) provided us with a table supplemental to those given in the publication, which shows coefficients for the relationship between NT mortality and the four gaseous pollutants in a statistical model which includes all four together (See Appendix 1.). These coefficients can be compared with the results of the initial and revised meta-analysis of Stieb *et al* in Table 2. In the recent model (Burnett *et al.* 2004) the single-pollutant coefficients for NO<sub>2</sub> and O<sub>3</sub> are approximately twice those found for non-GAM single-pollutant results from the meta-analysis, and were much larger and statistically significant, in contrast with the multipollutant coefficients in the meta-analysis. For CO and SO<sub>2</sub>, Burnett *et al.* 2004 found lower values in the single pollutant analyses: in the multi-pollutant analysis the CO coefficient was small, and not statistically significant (as in the meta-analysis), but the SO<sub>2</sub> coefficient although reduced by half, was significant, as it was in the meta-analysis.

We have chosen to use the coefficients for gaseous pollutants from the Burnett *et al.* 2004 multipollutant model in the current assessment of air pollutant related mortality for Toronto.

#### **2.4.1.3** Relative burden of illness associated with gases and particles

There is a significant body of scientific discussion about the attribution of the air pollution burden of illness to gases and particles; divided roughly into three groups: “primarily particles”; “primarily gases”; and “the pollutant mix”. Added to this discussion is another issue: are gases “confounders” of the “particle effect”, or are they “surrogates” of the particle effect? (e.g., Sarnat *et al.*, 2001) The evidence seems to be that the air pollution “mix” has an adverse impact on human health, and the relative strength of the various components as found in different studies may well reflect the unique character of air pollution in the area of study. This in fact was the motivation for the 90-city NMMAPS study, in order to “even out” the varying characteristics of pollution across the United States. For example, it is well established that sulphates form a larger proportion of the fine particle pollution in the urbanized portion of Eastern North America than in the West (Schwartz 2003b).

Katsouyanni *et al.* (2001) carried out an analysis of this problem, and have provided insight to it in their discussion:

“Since the early reports of associations between ambient particles and daily deaths, questions have arisen about the potential for those effects to be confounded by other air pollutants. We have addressed that issue in our analyses using two-pollutant models. We found no evidence for the effects of PM<sub>10</sub> to be confounded by SO<sub>2</sub> or ozone. BS effects were not confounded by SO<sub>2</sub> either but were higher with simultaneous control for ozone.

These results are supported by the similar findings of Schwartz (EHP 2000) who has recently reported on a multicity analysis of ten U.S. locations, and Samet *et al* (NMMAPS 1999) and Schwartz *et al* (JECH 1996) who examined two- and three-pollutant models in the 20 largest U.S. cities. On the basis of these large studies, we believe that confounding by SO<sub>2</sub> or ozone can be dismissed as an explanatory factor for observed associations with particles.”

“In contrast, we did find evidence that both the PM<sub>10</sub> and BS associations were moderately confounded by NO<sub>2</sub>. These results are different from those reported in the United States, where Samet *et al* (NMMAPS 1999) find no evidence of confounding by NO<sub>2</sub>.”

“In the analysis by Levy *et al* (EHP 2000) effect modification has also been investigated. Larger increases in mortality were found in populations with a proportion of persons over 65 years of age greater than 13% (0.77%) than in those with a smaller proportion of elderly (0.64%). The most important positive predictor of the effect size was the ratio PM<sub>2.5</sub>/PM<sub>10</sub>. In univariate analysis, there was some effect modification by NO<sub>2</sub>, but in a multivariate model, including ten effect modifiers among which was the ratio PM<sub>2.5</sub>/PM<sub>10</sub>, little effect modification by NO<sub>2</sub> was observed.”

“The most obvious explanation for [these findings] lies in the difference in relative source contribution between European and U.S. cities. In the United States, there are few diesel cars. In contrast, in many European cities, diesel cars approach 50% of all cars. Therefore, motor vehicles are a larger source of urban particles in Europe than in the United States, where they are the predominant source of urban NO<sub>2</sub>. Hence, it may be more difficult to distinguish between NO<sub>2</sub> and particles in Europe than in the United States. If particle measures were only standing for NO<sub>2</sub>, however, then the results of Samet *et al*. would be unexpected. Hence, it would be unwise to interpret these results as indicating that half of the particle effects are really the result of NO<sub>2</sub>.”

“Among the potential effect modifiers identified [in our own study], an important one is NO<sub>2</sub> concentration, an indicator of pollution originating from traffic. The higher the NO<sub>2</sub> concentrations (in absolute terms or relative to PM<sub>10</sub>) the larger the effect observed on mortality. This relation suggests that particles originating from vehicle exhausts are more toxic than those from other sources. Our conclusion is supported by other work. For example, Laden *et al* (EHP 2000) have examined the elemental composition of all of the PM<sub>2.5</sub> filters from the Harvard Six Cities Study. The slope (per µg/m<sup>3</sup>) of the effect for the traffic particles was twice as great as that for the long-range transport particles. In the NMMAPS project, regional differences were found in the effect estimates that were larger in the Northeast of the United States and smaller in the Southeast. The continuing pattern of regional differences in both studies suggests that further work on particle composition as an explanatory factor is warranted.”

It appears from this discussion that Katsouyanni *et al*. do not feel that the relationship between PM<sub>10</sub> and mortality is confounded by SO<sub>2</sub> or O<sub>3</sub>, and that although there is some evidence for effect modification by NO<sub>2</sub> in Europe, it is much less likely to be the case in North America.

#### **2.4.2 Morbidity (Hospital Admissions)**

As indicated above, the GAMs problem has impacted substantially on epidemiological time-series studies of air pollution and health conducted since 1995. Schwartz first described the use of GAMs in this context (Schwartz, 1994) and used it in a study of cardiovascular hospital admissions (Schwartz and Morris, 1995).

It has been noted in a previous section that of the 37 studies the HEI re-analysis examined, only 10 had to do with morbidity, and very few of these studies included pollutant gases in the analysis. Furthermore, the greater part of the morbidity studies recently carried out in the USA examined hospital admission morbidity in the elderly (age >64), partly because health care is available to this group at government cost, and administrative data are available.

The implication of this is that respiratory and cardiac hospital admission coefficients from these studies are not applicable to the population as a whole, although the information is useful in establishing the burden of illness associated with air pollution in the elderly, one of the susceptible groups.

#### 2.4.2.1 Particles

What can be seen from the re-analysis of the studies of hospital admission is that with the exception of pneumonia in the elderly, there has been little change to the risk associated with PM10 as a result of the re-analysis, compared with the substantial changes observed in the morbidity coefficients. The comparison is demonstrated in Table 4.

Table 4. Percent increase excess morbidity (health outcome) per unit PM<sub>10</sub> ( $\Delta H\% / \mu\text{g}/\text{m}^3$ )

STUDY and health outcome	ICD-9 code	PRE-REVISION	POST-REVISION
<b>Atkinson et al.,(2001):</b> HEIRA (2003) p.81			
asthma 0-14	493	0.12	0.15
asthma 15-64	493	0.11	0.10
COPD+asthma >64	490-496	0.1	0.1
all Resp Dis >64	460-519	0.09	0.1
<b>Samet et al. (NMMAPS II, 2000);</b> Schwartz et al., HEIRA (2003) p.25			
cardiovascular disease (CVD) >64	390-429	0.102	0.101
chronic obstructive pulmonary disease (COPD) >64	490-492; 494-496	0.142	0.109
pneumonia >64	480-487	0.162	0.078
<b>LeTertre et al., 2002;</b> HEIRA (2003) p 173			
cardiac disease	390-429	0.05	0.05
ischaemic heart disease >65	410-413	0.08	0.07
<b>Zanobetti et al., 2000;</b> HEIRA (2003) p. 241			
cardiovascular disease CVD >64	390-429	0.117	0.099
COPD >64	490-496 exc 493	0.181	0.171
pneumonia >64	480-487	0.19	0.171
<b>Moolgavkar 2000;</b> HEIRA (2003) p.183			
cardiovascular disease CVD (Los Angeles)	390-429	0.089	0.077
COPD	490-496 exc 493	0.11	0.078

We have attempted to find additional morbidity studies from the literature (relative to total respiratory and total cardiac hospital admissions) which have been published in 2003 and 2004 up to February, with minimal results. The study by Cakmak *et al.* presents an interesting alternative approach to the modeling problems associated with the conventional GAMs approach to dealing with smoothing the data. The results of this work, in a re-analysis of the American Cancer Society data originally studied by Pope and co-workers, provides a more robust estimate of the association between sulphate exposure and heart disease.

### 2.4.2.2 Gases

The study of Sunyer *et al.* (2003), although using a form of GAM, used the same methodology as LeTertre *et al.* (2002), which a re-analysis (LeTertre 2003) showed was insensitive to the GAMs problems used in the U.S. mortality studies, and thus we used Sunyer *et al.*'s coefficient for cardiovascular admissions and SO<sub>2</sub>.

This, as well as some recently available morbidity data which is not subject to the GAMs problem is shown in Table 5. We have included some coefficients from the APBIT 2000 study for SO<sub>2</sub> and ozone, as these are not dependent on GAMs models, and we believe are the most appropriate currently available. Fusco, and Moolgavkar used multi-pollutant models, but the others were for the most part single-pollutant models.

The single-pollutant model study by Oftedal *et al.* yields very high coefficients, particularly for SO<sub>2</sub>, and they question this themselves, indicating that SO<sub>2</sub> may be acting as a surrogate for other pollutants. We have chosen not to use the data from this study, because these coefficients are so different from those we have found in other studies in this and the previous APBIT study.

Table 5. Percent excess morbidity (health outcome) per unit pollutant ( $\Delta$  H% / unit)

STUDY	ICD-9	CO	NO <sub>2</sub>	SO <sub>2</sub>	SO <sub>4</sub>	O <sub>3</sub>
	unit	ppm	ppb	ppb	$\mu\text{g}/\text{m}^3$	ppb
<b>Fusco et al. (2001)</b>	all resp. 460-519	2.16	0.38			
<b>Oftedal et al. (2003)</b>	all resp. 460-519		0.98	5.6		
<b>Moolgavkar (2003)</b>	COPD (490-496 exc 493)	2.29	0.163			
<b>APBIT (2000)</b>	all resp: 460-519			0.276		0.11
	CVD: 390-459					0.452
<b>Cakmak et al.(2003)</b>	heart disease 390-429				1.12	
<b>Sunyer et al. (2003)</b>	CVD: 390-429			0.07		

## 3 RISK COEFFICIENTS FOR BURDEN OF ILLNESS ESTIMATION

### 3.1 Approach Selected for the Estimation of Burden of Illness in this Report.

In the previous estimates of the air pollution-related burden of illness in Toronto (APBIT 2000), we used two approaches: one we called the “HAQI” approach, and one we called the “multipollutant” approach.

The HAQI approach followed the method used in the Hamilton Air Quality Initiative Human Health Risk Assessment (HAQI 1997), in which risk coefficients were derived from a number of recently published studies and reviews throughout the world, and for each pollutant a value of  $\Delta$ H% /unit pollutant was obtained from one or several studies. The burden of illness for a given health outcome was obtained from the product of the prevalence of that outcome in Hamilton times the product of  $\Delta$ H% /unit and the concentration of the associated pollutant in Hamilton. The total burden was the sum of all individual pollutant-related burdens, taking account of the fact that only one burden should be assigned to particulate pollution, recognizing that there were several different measures of particulate.

The strength of this approach was its reliance on a number of different studies. Its weakness (at that time less appreciated than now) was that it did not take into account the potential for statistical effect modification of one pollutant by another, since many coefficients were derived from single or at most two-pollutant models.

The “multipollutant” approach used data from the studies by Burnett et al. of Canadian cities, in which city-specific regression models containing all four of the pollutants CO, NO<sub>2</sub>, SO<sub>2</sub> and O<sub>3</sub> simultaneously were examined. The results of this analysis yielded coefficients which could be used to determine the burden of illness associated with the pollutant mix. The strength of this approach was that it utilized a model which explicitly accounted for statistical pollutant interactions: its weakness was that it depended on a single study, but that study related specifically to the 6 former municipalities within Toronto.

The three multipollutant studies by Burnett et al used in APBIT 2000 all made use of GAMs with nonparametric smoothing functions, and as a result were potentially subject to the associated problems. As indicated above, we have made use of a very recent study by Burnett and co-workers which has explicitly avoided the GAMs problem, and from which we obtained the coefficients used in the current assessment of air pollutant related mortality for Toronto (Burnett *et al.* 2004). Burnett *et al.* in this publication also concluded that the association between NO<sub>2</sub> and NT mortality “was not sensitive to the adjustment for other gaseous pollutants nor particle mass measured every sixth day.” Since in Burnett’s study the gaseous pollutant coefficients are not sensitive to adjustment for PM<sub>2.5</sub> (6d), and since Katsouyanni has argued (see above) that in North America the reverse is also likely true, we have added the NT mortality burden associated with PM<sub>2.5</sub> to that associated with the four gases except for CO.

### 3.2 Summary of $\Delta H\%$ / unit values for NT mortality:

Table 3. Non-traumatic mortality per unit pollutant ( $\Delta H\%$  / unit)

Pollutant	PM <sub>10</sub>	PM <sub>2.5</sub>	CO	NO <sub>2</sub>	O <sub>3</sub>	SO <sub>2</sub>
unit	$\mu\text{g}/\text{m}^3$		ppm	ppb	ppb	ppb
NT mortality	0.060			0.075	0.085	0.046
NT mortality (chronic exp)		0.700				

As indicated above, the coefficient for PM<sub>10</sub> was obtained by selecting the lowest in the range given above (from the re-analysis) shown in Table 1, for 10 of the studies (excluding Lippmann, which was not statistically significant), and then to take the mean of these lowest values. This gives a coefficient of 0.06 % change in NT mortality /  $\mu\text{g}/\text{m}^3$  PM<sub>10</sub>. The coefficients for NO<sub>2</sub>, O<sub>3</sub>, and SO<sub>2</sub> are obtained from the multipollutant analysis of Burnett *et al.* (2004); the coefficient for CO being omitted because it was not found significant in the 4-pollutant model. The coefficient for chronic exposure to PM<sub>2.5</sub> was derived from the HEI re-analysis of the Pope ACS study (HEI 2000).

### 3.3 Summary of $\Delta H\%$ / unit values for morbidity:

Table 6. Health outcome per unit pollutant ( $\Delta H\%$  / unit)

Outcome	PM <sub>10</sub>	CO	NO <sub>2</sub>	O <sub>3</sub>	SO <sub>2</sub>
resp. adm	0.17 (1)	2.16 (2)	0.38 (2)	0.11 (1)	0.276 (1)
card. adm	0.063 (3)		0.39 (1)	0.452 (1)	0.07 (4)

1. APBIT 2000; 2. Fusco; 3 Let/Mool; 4. Sunyer 03

Respiratory hospital admissions: for PM<sub>10</sub>, O<sub>3</sub>, and SO<sub>2</sub>, APBIT 2000; CO and NO<sub>2</sub>, Fusco *et al.* 2001. Cardiovascular hospital admissions: for PM<sub>10</sub>, the mean of LeTertre (2003) and Moolgavkar (2003); for NO<sub>2</sub> and O<sub>3</sub>, APBIT 2000; and for SO<sub>2</sub>, Sunyer *et al.*, (2003). No acceptable data were available for cardiovascular admissions associated with CO.

## 4 AIR QUALITY MEASUREMENTS.

Air Quality data were obtained for the year 1999 for Ontario Ministry of the Environment sites in the six former municipalities which now make up the amalgamated City of Toronto, in a similar way to the methods described in APBIT 2000. Since data were available for fewer sites in 1999 than in 1995, some different strategies were required to fill in the data gaps. For example, at the downtown Toronto site, data for CO were limited to about two months, and so a relationship between CO and NO<sub>2</sub> was established from the 1995 data for that site which allowed for the estimation of all of the 1999 CO values from the 1999 NO<sub>2</sub> values. Gravimetric PM<sub>10</sub> data were only available from 1 site in 1999 compared to 3 sites in 1995, so the same data for gravimetric PM<sub>10</sub> were used for all former municipalities. In light of some recent studies of the background level of ozone in North America (Fiore 2002) the “threshold” for 24hr maximum O<sub>3</sub> values could be justifiably taken at 20 ppb rather than 30 ppb, so burden of illness calculations for both values were carried out. A summary of the air quality data may be found in the detailed Burden of Illness (Mortality) calculation table.

## 5 HEALTH OUTCOME DATA.

Health outcomes related to ICD-9 disease classifications were provided by Toronto Public Health: the most recent complete data were for the year 1999, which is the reason for the selection of that year for analysis. Complete tables of these data, disaggregated by former municipality are found in Appendix 2. Note that these Tables also show the changes in health outcomes for the 1999 year compared to 1995. A summary of the data for NT mortality is also found in the detailed Burden of Illness (Mortality) calculation table.

## 6 AIR POLLUTION-RELATED BURDEN OF ILLNESS

Table 7. Air-pollution related NT mortality and hospital admissions in Toronto (1999 data).

Pollutant	OUTCOME		
	Non-Traumatic Mortality	Respiratory Hospital Admissions	Cardiovascular Hospital Admissions
PM <sub>2.5</sub>	1236	597	421
CO	20	272	
NO <sub>2</sub>	249	1461	2857
O <sub>3</sub>	219	337	2648 *
SO <sub>2</sub>	30	215	104
Total	1754	2882	3382

Note: \* deleted from final estimate. See text.

### 6.1 General Comments.

In the previous study (APBIT 2000, Section 7.1) we characterized in some detail the rationale relating morbidity and mortality to air pollutants on a pollutant-by-pollutant basis, based on the results of the publications reviewed in the course of the study. In the intervening period, the principles behind that rationale have not changed: what is different, is that in some cases the magnitude of the coefficients has changed, and the nature of the biostatistical tools to determine them. Air pollution in urban environments, as well as in non-urban areas impacted by urban or urban-created pollution undoubtedly has a substantial adverse impact on public health.

The current “update” of APBIT 2000 addresses the changes in the state of scientific understanding that has taken place in the last four years, the changes in the population of the City of Toronto, and the changes in the nature and concentration of the ambient air pollution impacting the health of its citizens. The message is still the same: air pollution is not decreasing, and there is a burden of illness associated with it that can be estimated with some confidence.

### 6.2 Premature Non-traumatic mortality

From the literature review, we found that by far the greatest effort in the period 2000-2004 has been to examine the relationship between non-traumatic(NT) mortality and fine particles, measured in a number of different ways. As a result, we have the greatest confidence in our estimate of NT mortality associated with fine particulate air pollution in Toronto. In addition, the careful work of Burnett and co-workers has provided us once again with a well-supported estimate of the NT mortality associated with the gaseous pollutants in Toronto. The data are summarized in Table 7, and **our estimate is that approximately 1700 premature deaths** in Toronto are associated with its air pollution.

### 6.3 Hospital admissions for respiratory and cardiovascular disease.

Examination of the literature between 2000 and 2004 has yielded only a few studies of hospital admissions in the whole population, although there have been a few studies examining morbidity in children and seniors; in children, primarily studies of asthma, and in seniors, studies of obstructive lung disease and heart disease. Although useful, these have not allowed us to make direct comparisons with the APBIT 2000 study. As a result, 5 of the 9 coefficients we have used

to estimate hospital admissions in the current study are from APBIT 2000. At least one of these, (cardiac hospitalization associated with O<sub>3</sub> exposure) we regarded with some doubt in 2000, and this still remains the case. If we disregard the cardiac hospital admissions related to ozone, this gives 3382 associated with the other pollutants. This added to the 2882 respiratory admissions gives a total hospitalization figure of 6264.

One alternative approach would be to make use of the estimates of hospitalization from the Burnett multipollutant models used in APBIT 2000, using 1999 data and accepting that they might be subject to the GAMs problem. Given that other estimates of morbidity using GAMs seem to be relatively unaffected (see literature review above) this might be a more acceptable approach. The calculation of this estimate is given in Appendix 3, and it yields a figure of 1426 respiratory, and 1913 cardiac hospitalizations associated with air pollution based on 1999 data.

Thus **we would estimate that from 3000 to 6000 hospital admissions** may be associated with air pollution in Toronto, but we have less confidence in this estimate than we had in 2000. It is hoped that there is a renewal of epidemiological work in the near future to provide a stronger base for the hospitalization estimate.

## 7 CONCLUSIONS

### 7.1 Results of this study

We have examined the recent epidemiological literature, as well as recent reviews carried out for regulatory purposes, and have determined a set of coefficients of the air pollution-related burden of illness applicable to Toronto. We have also summarized recent quality-controlled air pollution data for several sites within the City, and have obtained baseline health outcome information in a form which can be linked to the air quality monitoring sites. Using this information, we have estimated the non-traumatic mortality associated with air pollution in Toronto for the year 1999, the most recent year for which there was adequate information. We have quantified the air pollution risk in the City of Toronto taking into account the most recent scientific evidence available, and have estimated that **approximately 1700 premature deaths each year, and between 3000 and 6000 hospital admissions** are associated with the criteria pollutants O<sub>3</sub>, NO<sub>2</sub>, SO<sub>2</sub>, CO and PM<sub>10</sub> breathed by the public at large.

### 7.2 Comparison to APBIT 2000

In comparing this result to that previously obtained in a similar exercise in 2000, we find that in spite of substantial revisions to earlier studies which have tended to reduce the estimate of burden of illness of air pollution in urban centres, using the same methods, the estimate of premature mortality as a result of the present work is consistent with the lower bound estimate of 700 premature deaths made in 2000, ignoring the impact of chronic exposure to particles. However, based on the much greater scientific strength of the revised and re-affirmed chronic exposure studies, we believe that a more supportable estimate of non-traumatic mortality attributable to air pollution in Toronto is 1700. The estimates of 3000 to 6000 excess hospital admissions in this update are consistent with the lower and upper bounds (3300 to 7600) of the estimate made in 2000.

There are several factors (including the changes in understanding of the relevant science) which might have led us to expect a reduction in air-pollution related mortality in Toronto. It can be seen from Table 8 that the overall non-traumatic mortality for 1999 is 13,246, which is a 20% reduction from the 1995 figure of 16615. Part of this reduction is a consequence of a different way of handling missing postal codes in the mortality data, but a reduction persists if this is accounted for. This is in spite of a 7% increase in the population of the city overall.

Table 8. Population changes: 1995-1999

Popu- lation	North York	York	Scar- borough	Old Toronto	Etobicoke	East York	Toronto
1999	622857	155072	596816	686265	349891	114160	2525061
1995	585256	145415	553233	650678	325597	106968	2367146

There has also been a change in the character of air pollution in Toronto between 1995 and 1999. It appears that there has been a large increase in the air pollution related to fossil-fuelled electrical generation (PM10, SO2, SO4), and a much smaller decrease in transportation-related primary pollutants (CO and NO2), but a large increase in ozone, in part from long-range transport. This is shown in Table 9.

Table 9. Average of Toronto Site Pollutants: 1995; 1999.

Pollutant	PM10	SO4	CO	NO2	SO2	O3
1995	17.3	3.3	0.9	26.6	3.3	9.6
1999	22.3	4.5	0.78	22.3	5.0	14.1
% change from 1995	29	36	-13	-16	52	47

The impact of transportation-related pollutants, especially those from diesel-powered heavy vehicles is becoming the dominant air problem in Toronto, as it is in other urban areas.

### 7.3 Implications for Policy

New studies in the literature demonstrate the importance of focussing on susceptible groups: infants, children and the elderly. Rather than examining the effect of air pollution, pollutant by pollutant on the population as a whole, future studies should examine the role of the pollutant mix on susceptible groups, and to develop air quality management strategies aimed at the sources of the most toxic components of the pollutant mix.

The results of this estimation of the air pollution-related burden of illness in the City of Toronto shows that every year there is still a major public health cost attributable to criteria pollutants from fossil-fuel combustion in the City and beyond.

***To reduce the levels of air pollution where people live, governments at all levels must sustain a commitment to: 1) reduce our dependency on fossil energy, particularly coal-fired electrical generation, and 2) support and sustain public transit in order to reduce the pollution burden created by automobiles and trucks.***

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## **APPENDICES**

9 APPENDIX 1

Additional Table to Burnett *et al.* 2004 (personal communication R. Burnett, Jan. 7, 2004)

Table 1. Percent change in non-accidental daily mortality associated with changes in gas phase ambient air pollutants evaluated at their population-weighted average concentrations based on single and selected multiple pollutant random effects model specifications. Ratio of percent change in mortality to standard error (t-value) given in parenthesis.

Pollutant (concentration)	Single Pollutant Model	Multiple Pollutant Models		
		I	II	III
NO <sub>2</sub> (22.4 ppb)	2.25 (4.45)	1.69 (3.00)	1.72 (3.51)	1.98 (3.90)
O <sub>3</sub> (30.6 ppb)	2.74 (6.08)	2.60 (6.16)	2.59 (5.33)	2.45 (6.24)
SO <sub>2</sub> (5.0 ppb)	0.37 (3.25)	0.23 (2.09)	0.23 (2.17)	NA
CO (1.02 ppm)	0.68 (3.12)	0.19 (0.73)	NA	NA
Total Burden	6.04 (NA*)	4.71 (6.94)	4.54 (6.89)	4.42 (6.83)

\*: t-value of sum of air pollution effects based on single pollutant models cannot be appropriately determined due to the collinearity among pollutants.

## APPENDIX 2.

### HEALTH OUTCOMES

Air Pollution Burden of Illness in Toronto Study; Deaths by cause, 1999 Both sexes combined, all ages combined			
Former Municipality	All non-traumatic deaths ICD-9 (001-799)	Cardiovascular deaths ICD-9 (390-459)	Respiratory deaths ICD-9 (460-519)
Scarborough (1995 data)**	2,875 (13.7% lower) (3,332)	988 (17.4% lower) (1,196)	270 (20.1% lower) (338)
Toronto (1995 data)**	3,590 (44.4% lower) (6,456)	1,307 (39.8% lower) (2,172)	359 (43.6% lower) (636)
East York (1995 data)**	674 (4.5% higher) (645)	222 (6.7% lower) (238)	60 (22.1% lower) (77)
North York (1995 data)**	3,313 (4.5% lower) (3,469)	1,253 (3.1% lower) (1,293)	324 (16.5% higher) (278)
York (1995 data)**	828 (77.7% higher) (466)	307 (66.8% higher) (184)	83 (124.3% higher) (37)
Etobicoke (1995 data)**	1,966 (12.5% lower) (2,247)	727 (15.6% lower) (861)	172 (4.4% lower) (180)
TOTAL*	13,246	4,804	1,268
# (%) missing or invalid	2,965 (18.3%)	1,136 (19.1%)	404 (24.2%)
TOTAL** (1995 data)** (% decrease)	16,211 (2.4% lower) (16,615) (2.4%)	5,940 (0.07% lower) (5,944) (0.07%)	1,672 (8.1% higher) (1,546) (-8.1%)

\* Excludes invalid (or missing) postal codes

\*\* Includes invalid/missing postal codes

Air Pollution Burden of Illness in Toronto Study; Hospital admissions by cause, 1999 Both sexes combined; All ages combined <i>except</i> CHF (65 yrs +) and asthma (0-15, 16-64, 65+)							
Former Municipality	All non-traumatic hospital admissions (001-799)	Cardiovascular hospital admissions (390-459)	Respiratory hospital admissions (460-519)	Congestive Heart Failure hospital admissions (428)	Asthma hospital admissions (493)		
AGE				\$ 65 years	0-15	16-64	65+
Scarborough* (1995 data)**	41,609 (50,749)	6,460 (7,075)	3,665 (4,765)	789	416	217	102
Toronto* (1995 data)**	45,856 (61,000)	7,493 (8,845)	4,348 (5,458)	999	284	182	119
East York* (1995 data)**	8,862 (11,256)	1,487 (1,774)	786 (1,020)	166	67	45	22
North York* (1995 data)**	44,913 (54,010)	8,191 (9,121)	3,760 (4,396)	1,146	317	168	140
York* (1995 data)**	11,311 (14,324)	1,742 (2,186)	1,010 (1,322)	260	97	34	23
Etobicoke* (1995 data)**	25,081 (29,674)	4,604 (4,843)	2,169 (2,526)	596	216	103	68
TOTAL*	177,632	29,977	15,738	3,956	1,397	749	474
# (%) missing or invalid	13,570 (7.1%)	832 (2.7%)	336 (2.1%)	62 (1.5%)	19 1.3%	14 1.8%	4 .8%
TOTAL** (1995 data)** (% decrease)	191,202 (221,013) (13.5%)	30,809 (33,844) (9.0%)	16,074 (19,487) (17.5%)	4,018	1,416	763	478

\* Excludes invalid (or missing) postal codes

\*\* Includes invalid/missing postal codes

Air Pollution Burden of Illness in Toronto Study; Hospital admissions by cause, 1999 Both sexes combined; All ages combined						
Former Municipality	Asthma (493)	Obstructive lung disease (490-492, 496)	Respiratory infection (464, 466, 480-487, 494)	Dysrhythmia (427)	Heart failure (428)	Ischaemic heart disease (410-414)
Scarborough	735	708	1,419	557	917	2,734
Toronto	585	873	1,767	708	1,186	2,925
East York	134	151	344	144	193	673
North York	625	577	1,726	837	1,293	3,438
York	154	182	415	178	304	656
Etobicoke	387	320	1,024	431	699	1,973
TOTAL	2,620	2,811	6,695	2,855	4,592	12,399
TOTAL 1995						

**APPENDIX 3.**

**BURDEN OF ILLNESS CALCULATION TABLES**

**Premature Mortality (Ozone threshold 30 ppb)**

Community	Etobicoke	York	Toronto	East York	North York	Scarborough	Toronto City	
Population	349891	155072	686265	114160	622857	596816	2525061	
N-T Mortality	1966	828	3590	674	3313	2875	13246	
AQ Sites: Gases	35003 / 35033	36030	31103	36030	34020	33003		
TEOM: PM <sub>2.5</sub>	35033	34020	35033	34020	34020	34020		
PM <sub>10</sub>	35003 / 35033	35003	35033	33003	33003	33003		
6d: PM <sub>10</sub> / SO <sub>4</sub>	35127	35127	35127	35127	35127	35127		
1999 Average Daily (effective) Concentration			ozone thresh->	30	PM <sub>10</sub> thresh->	5		
PM <sub>10</sub> ug/m <sup>3</sup> (6d) -t <sup>1</sup>	22.3	22.3	22.3	22.3	22.3	22.3		
SO <sub>4</sub> ug/m <sup>3</sup> (6d)	4.5	4.5	4.5	4.5	4.5	4.5		
PM <sub>10</sub> ug/m <sup>3</sup> (teom)	16.2	13.4	19.1	14.7	14.7	14.7		
PM <sub>2.5</sub> ug/m <sup>3</sup> (teom)	14.2	12.7	14.2	12.7	12.7	12.7		
CO	1.2	0.6	0.6	0.6	0.8	0.9		
NO <sub>2</sub>	26.5	15.8	26.9	15.8	24.3	24.6		
SO <sub>2</sub>	5.5	4.9	4.7	4.9	5	4.9		
O <sub>3</sub> avg (24hr max; minus threshold) <sup>1</sup>	8.3	14.3	5.9	14.8	21	20.3		
O <sub>3</sub> avg (24hr avg)	19.9	23.4	20.1	23.4	22.8	21.6		
ΔH% per unit	PREMATURE MORTALITY (by former mun.; total summed across mun.)							total
PM <sub>10</sub>	0.06	26.3	11.1	48.0	9.0	44.3	38.5	177
SO <sub>4</sub> acute	0.22	19.5	8.2	35.5	6.7	32.8	28.5	131
SO <sub>4</sub> chron	0.75	66.4	27.9	121.2	22.7	111.8	97.0	447
PM <sub>2.5</sub> acute	0.085	23.7	8.9	43.3	7.3	35.8	31.0	150
PM <sub>2.5</sub> chron	0.7	195.4	73.6	356.8	59.9	294.5	255.6	1236
CO	0.19	4.5	0.9	4.1	0.8	5.0	4.9	20
NO <sub>2</sub>	0.077	40.1	10.1	74.4	8.2	62.0	54.5	249
SO <sub>2</sub>	0.046	5.0	1.9	7.8	1.5	7.6	6.5	30
O <sub>3</sub>	0.085	13.9	10.1	18.0	8.5	59.1	49.6	159
TOTAL	Includes: PM <sub>10</sub> (6d), CO, NO <sub>2</sub> , SO <sub>2</sub> , O <sub>3</sub> . For explanation, see text.							1695

(see notes following next table)

## Premature Mortality (Ozone threshold 20 ppb)

Community	Etobicoke	York	Toronto	East York	North York	Scarborough	Toronto City	
Population	349891	155072	686265	114160	622857	596816	2525061	
N-T Mortality	1966	828	3590	674	3313	2875	13246	
AQ Sites: Gases	35003 / 35033	36030	31103	36030	34020	33003		
TEOM: PM <sub>2.5</sub>	35033	34020	35033	34020	34020	34020		
PM <sub>10</sub>	35003 / 35033	35003	35033	33003	33003	33003		
6d: PM <sub>10</sub> / SO <sub>4</sub>	35127	35127	35127	35127	35127	35127		
1999 Average Daily (effective) Concentration			ozone thresh->	20	PM <sub>10</sub> thresh->	5		
PM <sub>10</sub> ug/m <sup>3</sup> (6d) -t <sup>1</sup>	22.3	22.3	22.3	22.3	22.3	22.3		
SO <sub>4</sub> ug/m <sup>3</sup> (6d)	4.5	4.5	4.5	4.5	4.5	4.5		
PM <sub>10</sub> ug/m <sup>3</sup> (teom)	16.2	13.4	19.1	14.7	14.7	14.7		
PM <sub>2.5</sub> ug/m <sup>3</sup> (teom)	14.2	12.7	14.2	12.7	12.7	12.7		
CO	1.2	0.6	0.6	0.6	0.8	0.9		
NO <sub>2</sub>	26.5	15.8	26.9	15.8	24.3	24.6		
SO <sub>2</sub>	5.5	4.9	4.7	4.9	5	4.9		
O <sub>3</sub> avg (24hr max; minus threshold) <sup>1</sup>	18.3	24.3	15.9	24.8	21	20.3		
O <sub>3</sub> avg (24hr avg)	19.9	23.4	20.1	23.4	22.8	21.6		
ΔH% per unit	PREMATURE MORTALITY (by former mun.; total summed across mun.)							total
PM <sub>10</sub>	0.06	26.3	11.1	48.0	9.0	44.3	38.5	177.23
SO <sub>4</sub> acute	0.22	19.5	8.2	35.5	6.7	32.8	28.5	131.14
SO <sub>4</sub> chron	0.75	66.4	27.9	121.2	22.7	111.8	97.0	447.05
PM <sub>2.5</sub> acute	0.085	23.7	8.9	43.3	7.3	35.8	31.0	150.07
PM <sub>2.5</sub> chron	0.7	195.4	73.6	356.8	59.9	294.5	255.6	1235.91
CO	0.19	4.5	0.9	4.1	0.8	5.0	4.9	20.24
NO <sub>2</sub>	0.077	40.1	10.1	74.4	8.2	62.0	54.5	249.20
SO <sub>2</sub>	0.046	5.0	1.9	7.8	1.5	7.6	6.5	30.22
O <sub>3</sub>	0.085	30.6	17.1	48.5	14.2	59.1	49.6	219.16
TOTAL	Includes: PM <sub>10</sub> (6d), CO, NO <sub>2</sub> , SO <sub>2</sub> , O <sub>3</sub> . For explanation, see text.							1754.72

### Notes:

1. Values of threshold provided in this Table.

PM10: average from table of re-analysed studies (HEI Re-analysis)

SO4 acute : Schwartz 96

SO4 chronic : ACS re-analysis from EPA 2003 Criteria Document Table 8-13

PM2.5 chronic : ACS re-analysis from EPA 2003 Criteria Document Table 8-13

PM2.5 acute : from Burnett HEI Re-analysis

NO2 : from Burnett 2004 Supplementary Table

O3 : from Burnett 2004 Supplementary Table

SO2 : from Burnett 2004 Supplementary Table

CO : from Burnett 2004 Supplementary Table

Burden of premature mortality based on Burnett 2004 for gases, PM<sub>10</sub> (6d) only. Values of burden of mortality related to other particulate / health metrics given for reference purposes.

**Respiratory Admissions (Ozone threshold 20 ppb)**

Community	Etobicoke	York	Toronto	East York	North York	Scarborough	Toronto City	
Population	349891	155072	686265	114160	622857	596816	2525061	
Respiratory admissions	2169	1010	4348	786	3760	3665	15738	
AQ Sites: Gases	35003 / 35033	36030	31103	36030	34020	33003		
TEOM: PM <sub>2.5</sub>	35033	34020	35033	34020	34020	34020		
PM <sub>10</sub>	35003 / 35033	35003	35033	33003	33003	33003		
6d: PM <sub>10</sub> / SO <sub>4</sub>	35127	35127	35127	35127	35127	35127		
1999 Average Daily (effective) Concentration			ozone thresh->	20	PM <sub>10</sub> thresh->	5		
PM <sub>10</sub> ug/m <sup>3</sup> (6d) -t <sup>1</sup>	22.3	22.3	22.3	22.3	22.3	22.3		
SO <sub>4</sub> ug/m <sup>3</sup> (6d)	4.5	4.5	4.5	4.5	4.5	4.5		
PM <sub>10</sub> ug/m <sup>3</sup> (teom)	16.2	13.4	19.1	14.7	14.7	14.7		
PM <sub>2.5</sub> ug/m <sup>3</sup> (teom)	14.2	12.7	14.2	12.7	12.7	12.7		
CO	1.2	0.6	0.6	0.6	0.8	0.9		
NO <sub>2</sub>	26.5	15.8	26.9	15.8	24.3	24.6		
SO <sub>2</sub>	5.5	4.9	4.7	4.9	5	4.9		
O <sub>3</sub> avg (24hr max; minus threshold) <sup>†</sup>	18.3	24.3	15.9	24.8	21	20.3		
O <sub>3</sub> avg (24hr avg)	19.9	23.4	20.1	23.4	22.8	21.6		
ΔH% per unit	RESPIRATORY ADMISSIONS (by former mun.; total summed across mun.)							total
PM <sub>10</sub>	0.17	82.2	38.3	164.8	29.8	142.5	138.9	597
SO <sub>4</sub> acute		0.0	0.0	0.0	0.0	0.0	0.0	0
SO <sub>4</sub> chron		0.0	0.0	0.0	0.0	0.0	0.0	0
PM <sub>2.5</sub> acute		0.0	0.0	0.0	0.0	0.0	0.0	0
PM <sub>2.5</sub> chron		0.0	0.0	0.0	0.0	0.0	0.0	0
CO	2.16	56.2	13.1	56.4	10.2	65.0	71.2	272
NO <sub>2</sub>	0.38	218.4	60.6	444.5	47.2	347.2	342.6	1461
SO <sub>2</sub>	0.276	32.9	13.7	56.4	10.6	51.9	49.6	215
O <sub>3</sub>	0.11	43.7	27.0	76.0	21.4	86.9	81.8	337
TOTAL	Includes: PM <sub>10</sub> (6d), CO, NO <sub>2</sub> , SO <sub>2</sub> , O <sub>3</sub> . For explanation, see text.							2881

**Cardiovascular Admissions (Ozone threshold 20 ppb)**

Community	Etobicoke	York	Toronto	East York	North York	Scarborough	Toronto City	
Population	349891	155072	686265	114160	622857	596816	2525061	
cardiovasc. admissions	4604	1742	7493	1487	8191	6460	29977	
AQ Sites: Gases	35003 / 35033	36030	31103	36030	34020	33003		
TEOM: PM <sub>2.5</sub>	35033	34020	35033	34020	34020	34020		
PM <sub>10</sub>	35003 / 35033	35003	35033	33003	33003	33003		
6d: PM <sub>10</sub> / SO <sub>4</sub>	35127	35127	35127	35127	35127	35127		
1999 Average Daily (effective) Concentration			ozone thresh->	20	PM <sub>10</sub> thresh->	5		
PM <sub>10</sub> ug/m <sup>3</sup> (6d) -t <sup>1</sup>	22.3	22.3	22.3	22.3	22.3	22.3		
SO <sub>4</sub> ug/m <sup>3</sup> (6d)	4.5	4.5	4.5	4.5	4.5	4.5		
PM <sub>10</sub> ug/m <sup>3</sup> (teom)	16.2	13.4	19.1	14.7	14.7	14.7		
PM <sub>2.5</sub> ug/m <sup>3</sup> (teom)	14.2	12.7	14.2	12.7	12.7	12.7		
CO	1.2	0.6	0.6	0.6	0.8	0.9		
NO <sub>2</sub>	26.5	15.8	26.9	15.8	24.3	24.6		
SO <sub>2</sub>	5.5	4.9	4.7	4.9	5	4.9		
O <sub>3</sub> avg (24hr max; minus threshold) <sup>1</sup>	18.3	24.3	15.9	24.8	21	20.3		
O <sub>3</sub> avg (24hr avg)	19.9	23.4	20.1	23.4	22.8	21.6		
ΔH% per unit	CARDIOVASC. ADMISSIONS (by former mun.; total summed across mun.)							total
PM <sub>10</sub>	0.063	64.7	24.5	105.3	20.9	115.1	90.8	421
SO <sub>4</sub> acute		0.0	0.0	0.0	0.0	0.0	0.0	0
SO <sub>4</sub> chron		0.0	0.0	0.0	0.0	0.0	0.0	0
PM <sub>2.5</sub> acute		0.0	0.0	0.0	0.0	0.0	0.0	0
PM <sub>2.5</sub> chron		0.0	0.0	0.0	0.0	0.0	0.0	0
CO		0.0	0.0	0.0	0.0	0.0	0.0	0
NO <sub>2</sub>	0.39	475.8	107.3	786.1	91.6	776.3	619.8	2857
SO <sub>2</sub>	0.07	17.7	6.0	24.7	5.1	28.7	22.2	104
O <sub>3</sub>	0.452	380.8	191.3	538.5	166.7	777.5	592.7	2648
TOTAL	Includes: PM <sub>10</sub> (6d), NO <sub>2</sub> , SO <sub>2</sub> , O <sub>3</sub> . For explanation, see text.							6030

**MULTIPOLLUTANT TABLE USING THE COEFFICIENTS OF BURNETT ET AL. (1999)**

COMMUNITY (1999 data)	ETOBICOKE	YORK	TORONTO	EAST YORK	NORTH YORK	SCAR- BOROUGH	TORONTO	
N-T mortality	1966	828	3590	674	3313	2875	13246	
asthma	387	154	585	134	625	735	2620	
obs. lung dis	320	182	873	151	577	708	2811	
resp. infection	1024	415	1767	344	1726	1419	6695	
dysrhythmia	431	178	708	144	837	557	2855	
heart failure	699	304	1186	193	1293	917	4592	
ischemic heart dis	1973	656	2925	673	3438	2734	12399	
1999 Average Daily (effective) Concentration								
PM-10 ug/m3	22.3	22.3	22.3	22.3	22.3	22.3		
SO4 ug/m3	4.5	4.5	4.5	4.5	4.5	4.5		
CO ppm	1.20	0.60	0.60	0.60	0.80	0.90		
NO2 ppb	26.5	15.8	26.9	15.8	24.3	24.6		
SO2 ppb	5.5	4.9	4.7	4.9	5.0	4.9		
O3 ppb	18.3	24.3	15.9	24.8	21.0	20.3		
O3 ppb 24hr	19.9	23.4	20.1	23.4	22.8	21.6		
	ΔH%/unit	NT MORTALITY						
PM10	0.04	18.9	7.9	34.4	6.5	31.8	27.6	127.0
CO	4.79	113.0	23.8	103.2	19.4	127.0	123.9	510.2
asthma								
PM10	0.0374	3.2	1.3	4.9	1.1	5.2	6.1	21.9
CO	4.07	18.9	3.8	14.3	3.3	20.4	26.9	87.5
SO2	0.142	3.0	1.1	3.9	0.9	4.4	5.1	18.5
O3	0.308	23.7	11.1	36.2	9.7	43.9	48.9	173.5
obs lung dis								
PM10	0.091	6.5	3.7	17.7	3.1	11.7	14.4	57.0
CO	2.33	8.9	2.5	12.2	2.1	10.8	14.8	51.4
O3	0.364	23.2	15.5	63.9	12.9	47.9	55.7	219.0
resp infect								
PM10	0.132	30.1	12.2	52.0	10.1	50.8	41.8	197.1
NO2	0.165	44.8	10.8	78.4	9.0	69.2	57.6	269.8
SO2	0.187	10.5	3.8	15.5	3.2	16.1	13.0	62.2
O3	0.187	38.1	18.2	66.4	15.1	73.6	57.3	268.6
								1426.4
dysrhythmia								
PM10	0.105	10.1	4.2	16.6	3.4	19.6	13.0	66.9
CO	5.94	30.7	6.3	25.2	5.1	39.8	29.8	137.0
O3	0.166	14.2	6.9	23.6	5.6	31.7	20.0	102.0
heart failure								
PM10	0.059	9.2	4.0	15.6	2.5	17.0	12.1	60.4
CO	3.47	29.1	6.3	24.7	4.0	35.9	28.6	128.7
NO2	0.273	50.6	13.1	87.1	8.3	85.8	61.6	306.5
ischemic heart disease								
NO2	0.331	173.1	34.3	260.4	35.2	276.5	222.6	1002.2
SO2	0.178	19.3	5.7	24.5	5.9	30.6	23.8	109.8
								1913.4

## 10 Endnotes.

1.

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