# NATIONAL AMBIENT AIR QUALITY OBJECTIVES FOR PARTICULATE MATTER

ADDENDUM TO THE SCIENCE ASSESSMENT DOCUMENT

A report by the
Federal - Provincial Working Group
on Air Quality Objectives
and Guidelines

December 1997, Revised April 1999

ADDENDUM

to

Science Assessment Document

PARTICULATE MATTER

$\leq$10μm and $\leq$2.5μm

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Editor's Note:

This addendum to the Science Assessment Document for Particulate Matter was originally intended as a rationale document for National Ambient Air Quality Objective recommendations for PM$_{10}$ and PM$_{2.5}$. As outlined in the Preface, the need for such a document has changed. As such this material, originally compiled in 1997 has been revised and re-directed towards the identification of a concentration range, which if achieved would result in substantial reduction in the risk to human health and the environment. Consequently, the analysis presented here does not utilize the most currently available ambient data, nor does it take advantage of recent improvements in our understanding of how to undertake risk and benefit analyses for ambient pollutants. This document, along with the Science Assessment Document, is intended to complete our picture of Particulate Matter impacts, risks and benefits, as of 1997.

April 15, 1999.

PREFACE

The Canadian Environmental Protection Act (*CEPA*), passed into law in 1988, replaces and builds upon the Clean Air Act and the Environmental Contaminants Act. The opening statement of the Act declares that "the protection of the environment is essential to the well-being of Canada". *CEPA* allows the Federal Government to assess substances and control their impact through national environmental quality objectives, guidelines, codes of practice, and/or regulations.

Provincial Governments have the primary responsibility in many areas of air pollution control, with Federal actions integrated with those of the provinces. The *CEPA* Federal/Provincial Working Group on Air Quality Objectives and Guidelines, consisting of representatives of federal, provincial and territorial departments of environment and health, reviews and recommends ambient air quality objectives.

Canada's *National Ambient Air Quality Objectives(s)* prescribe targets for air quality, measured at the relevant receptor (persons, vegetation, animals, materials). The *objective(s)* are national goals for outdoor air quality that protect public health, the environment, or aesthetic properties of the environment. Development of Canada Wide Standards is now the preferred risk management approach for Particulate Matter. The scientific work to date including this Addendum, is directed towards supporting development of Canada Wide Standards for Particulate Matter.

The development of National Ambient Air Quality Objectives involves a scientific review of physical and chemical properties of a substance, its sources, environmental, animal and human health effects, and environmental and human exposure assessment. The next step is integration of this information within a framework of risk assessment. The Science Assessment Document[1] contains this critical scientific evaluation, and lays the scientific groundwork for establishing the air quality objectives. Reference Levels, levels above which there are demonstrated effects on human health and/or the environment, are identified. A document outlining the process followed in reviewing and interpreting scientific information leading to the recommendation of objectives is published separately (WGAQOG, 1997).

This Addendum identifies a concentration range for Particulate Matter $\leq 10\mu m$ and $\leq 2.5\mu m$ which if met would provide for substantial reductions in the risks to human health and the environment from exposure to ambient particulate matter. The derivation of this range considers current risk and non-monetary benefits due to avoided impacts, reflecting a philosophy of environment and health protection in the context of long term risk reduction. The broad range of potential responses by the population, ecosystems and organisms in the environment are considered. Given the range of these sensitivities however, the identified range may not protect all.

It is recognized that not all locations in Canada will achieve this range. The expectation is that air quality management strategies will be implemented to facilitate the reduction of ambient air concentrations to levels approaching this range as soon as practicable. The principles of continuous improvement and non-degradation of environmental quality are advocated.

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[1] Federal-Provincial Working Group on Air Quality Objectives and Guidelines. (1998). National Ambient Air Quality Objectives for Particulate Matter. Part 1: Science Assessment Document and Identification of Reference Levels. Ottawa, Canada: Public Works and Government Services.

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Director
Bureau of Chemical Hazards
Environmental Health Directorate
Health Canada, PL 0801B3
Tunney's Pasture
Ottawa, Ontario
K1A 0L2

Chief
Science Assessment and Policy Integration Division
Atmospheric Environment Service
Environment Canada
4905 Dufferin St.
Downsview, Ontario
M3H 5T4

This document is also available on the web at: www.hc-sc.gc/ehp/ehd/catalogue/bch.htm

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1. INTRODUCTION

Under the Canadian Environmental Protection Act (*CEPA*), Part 1, Sections 8 & 9, the Ministers of Health and the Environment will formulate environmental quality objectives specifying goals toward which an environmental control effort is directed. Traditionally, National Ambient Air Quality Objectives (NAAQOs) have provided national goals for the management of air quality.

Currently, NAAQOs exist for total suspended particulate (TSP). However, particle deposition data clearly indicate that the mass of total suspended particles is not an appropriate indicator of particulate matter in relations to human health effects. Particles less than 10 μm in diameter (and possibly up to 15 μm in diameter during mouth breathing) penetrate to the lungs. For the purposes of this document, PM refers to ambient particles that are generally less than or equal to 10 μm in diameter. PM_x (e.g., x = 1, 2.5, 10, 15, 10-2.5) is used to refer to a specific size fraction of particulate matter with the aerodynamic diameter less than or equal to *x* μm.

Prior to the signing of the Harmonization Accord and the sub-agreement on Canada-Wide Standards, work was well underway to develop new objectives for particulate matter. In fact, proposed NAAQOs for particulate matter (PM10 and PM2.5) were presented to stakeholders at a workshop in December. However, in recognition of the recent designation of PM as priority substances for the development of Canada-Wide Standards, federal and provincial health and environment departments have chosen to develop Canada Wide Standards for PM, rather than continue with the NAAQO development.

The purpose of this document is to identify a concentration range, which if met would provide for substantial reduction of risk to human health and the environment from exposure to particulate matter. This exercise balances the need for a firm scientific foundation with the need for protection in the face of uncertainty and incomplete information. It requires acknowledgement that absolute safety will not be achieved. This analysis of the risks and benefits of decreasing particle concentrations will be based upon the mass concentration of particles rather than the chemical composition. It is expected that this information alone does not suffice for directing the development of air quality criteria or management plans, and that it will be used in conjunction with other information, for example, socio-economic analyses.

A brief summary of the relevant scientific literature evaluated and presented in the document titled "National Ambient Air Quality Objectives for Particulate Matter - Part 1 Science Assessment Document" is included for perspective. Information is taken from the Science Assessment Document (1997) unless otherwise cited.

2. CHARACTERIZATION OF PARTICULATE MATTER

Total suspended particulate (TSP) matter includes all airborne solid and liquid particles, except pure water, which are microscopic in size, ranging from approximately 0.005 μm to 100 μm in diameter. Particle size is considered the most important parameter in characterizing the physical behaviour of particulate matter as size affects such things as removal processes, atmospheric residence times, visibility, and is an important determinant of health and environmental effects. Particles can also be categorized by number, surface area, formation processes and chemical composition. However, particulate matter is unique among atmospheric constituents in that it is not clearly defined based on its chemical composition. It may include a broad range of chemical species including elemental and organic carbon compounds, oxides of silicon, metals, sulphates, nitrates and ammonia.

PM_{10} refers to particles generally less than 10 μm in aerodynamic diameter and are commonly referred to as *thoracic* particles because they can penetrate into the thoracic compartment (from the trachea down to and including the alveoli) of the human respiratory tract. PM_{10} is generally subdivided into a fine mode of 2.5 μm aerodynamic diameter or less ($PM_{2.5}$) and a coarse mode of particles generally larger than 2.5 μm in aerodynamic diameter. Figure 1 shows the mass distribution of particles found in the atmosphere.

Although there is some overlap, as illustrated in the figure, particles found in the fine and coarse fractions are generally distinct in terms of source, formation process, chemical composition and behaviour in the atmosphere. The type of source and/or secondary particle formation process involved will affect particle size and composition. Grinding and other mechanical processes result in the direct discharge of coarser particles to the atmosphere. Primary fine particles are formed from condensation of high temperature vapours during combustion and are discharged directly to the atmosphere. What is referred to as secondary fine particles result from: 1) reactions between gas molecules to form new particles; 2) coagulation of two particles to form one larger particle; and 3) gas-particle interactions, with gases being adsorbed and absorbed onto existing particles. These reactions involve precursor gases such as SO_2, NO_x, and VOCs.

Fine particles can be further subdivided into ultrafine (or nuclei) which are ≤ 0.1 μm, and accumulation modes, according to their volume or mass distributions. While the greatest concentration of airborne particles is found in the ultrafine mode, they contribute little to overall particle mass loading. These particles are subject to Brownian motion and coagulation processes which can quickly yield larger particles. Particles in the size range of 0.1-2.5 μm (accumulation mode) account for most of the particle surface area and much of the particle mass. The accumulation mode is so-named as atmospheric removal processes are least efficient in this size range.

Figure 1. Idealized ambient mass distribution of fine and coarse mode particle fractions [U.S. [EPA, 1996]
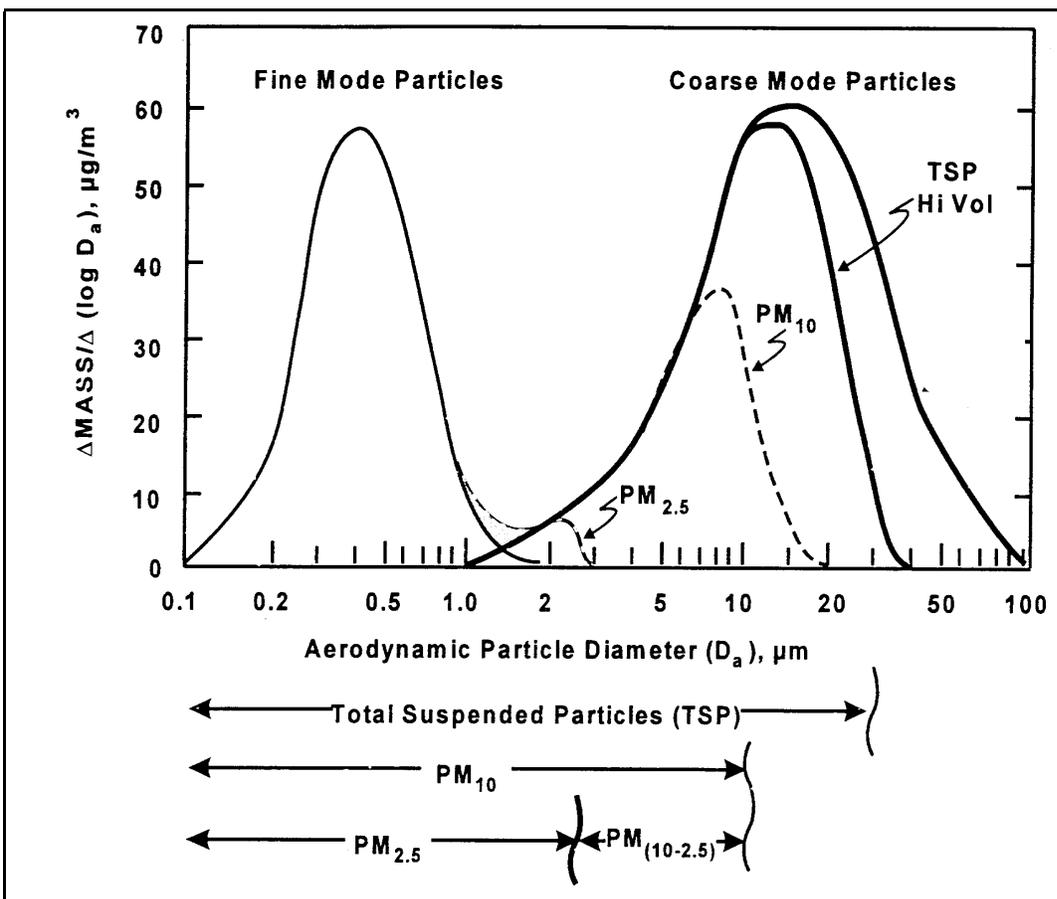
Fine particles may persist in the atmosphere from a few days to a few weeks. Particles in the accumulation mode are also efficient at scattering light, thereby contributing to visibility reduction in the atmosphere. Fine particles are primarily composed of ammonium, sulphate and nitrate, lead, elemental carbon and hundreds of different organic compounds.

Particles larger than 2.5 μm (coarse or sedimentation mode) are efficiently removed by gravitational settling and remain in the atmosphere for periods of a few hours to a few days. Although the coarse mode accounts for much of the total mass of ambient PM_{10}, it contributes little to the total number of particles. Reflecting the formation processes involved, the coarse mode is characterized by materials typical of the earth's crust (oxides of iron, calcium, silicon and aluminum) and sea spray (sodium and chloride). Wildfires, prescribed burning, residential wood combustion, power generation, wear of road surfaces, industrial facilities and diesel vehicles are examples of sources which contribute both directly to PM_{10} emissions, and indirectly through the emission of precursor gases. Windblown dust, sea spray, and mining and quarrying operations all contribute more significantly to the coarse fraction. Consideration of sources for particulate matter would not be complete without recognizing that part of the "source" which contributes to

ambient levels is transboundary, both in the form of particulate matter itself, and precursor gases.

Natural sources contribute to both fine and coarse particles in the atmosphere. Background particulate matter is generally defined as the distribution of particulate matter concentration that would be observed in the absence of anthropogenic emissions of particulate matter and/or gaseous precursor emissions. Background concentrations on an annual basis range from 4 $\mu g/m^3$ to 12 $\mu g/m^3$ PM_{10} for remote sites in North America. Based upon visual range estimates in Canada, natural background levels of $PM_{2.5}$ in western and eastern Canada are estimated to be $\leq$ 6 $\mu g/m^3$ and 10-13 $\mu g/m^3$ in southeastern Ontario. These estimates apply in the absence of natural events such as forest fires, storms etc.

Measurements

Since 1984, a national PM_{10} and $PM_{2.5}$ monitoring program has been operating under the National Air Pollution Surveillance (NAPS) network. This is primarily an urban network, with few rural sites. Particulate matter air quality data are typically collected over a 24-hour sampling period on a one-day-in-six sampling regime. By operating on this schedule, given a long enough sampling period, each day of the week is equally well sampled, and hence all conditions during the week are represented.

In urban areas, there is a diurnal pattern peaking in the morning and late afternoon, suggesting a transportation source influence. The monthly, or seasonal pattern, varies by region and is influenced by local source characteristics, prevailing meteorology and long range transport influences.

The ambient particle data exhibit a strongly skewed distribution dominated by a large number of low values, which can "mask" trends in particulate concentrations or the frequency or magnitude of "extreme" events. The current strategy of monitoring one day in six does not permit the extremes of the concentration distribution to be accurately quantified, and it is estimated that currently, peak concentrations are underestimated by ~20-30%. This is discussed further in Appendix C.

Fourteen urban sites in the NAPS dichotomous sampler network operating from 1986 to 1994 showed that 24-hour PM_{10} concentrations ranged from 5 to 175 $\mu g/m^3$, with a majority of the concentrations below 50 $\mu g/m^3$. Most of the 24-hour $PM_{2.5}$ measurements were between 2 and 30 $\mu g/m^3$.

Figure 2 illustrates TSP, PM_{10}, $PM_{2.5}$ and SO_4^{2}- mass distributions for a cross-section of Canadian cities are as box plots. At a majority of the sites, the daily variability in fine particle mass had a stronger influence on the variations in PM_{10} than did the coarse particle mass. This was most evident at the rural locations and at sites not heavily impacted by urbanization (i.e., traffic and/or construction). While this is consistent with the idea that fine particles control the variability in PM_{10}, it was not true at all sites or over all geographical areas. For example, the coarse mass dominated the PM_{10} variability at the Prairie sites and sites most heavily impacted by traffic. Comparison of the urban and rural sites that were in relatively close proximity to one another indicated that particle mass concentrations were lower at the rural locations.

This urban enhancement in concentration was greatest for the coarse particles. For example, the mean coarse particle concentration is 46% higher in Toronto compared to Egbert, while the mean concentration of fine particles is only 28% higher in Toronto.

Figure 2. Comparison of the Distributions of TSP, PM_{10}, $PM_{2.5}$ and SO_4 at 11 Urban Sites (1984-1993). The Box Plots Indicate the Median, 5th and 95th and 25th and 75th Percentiles.
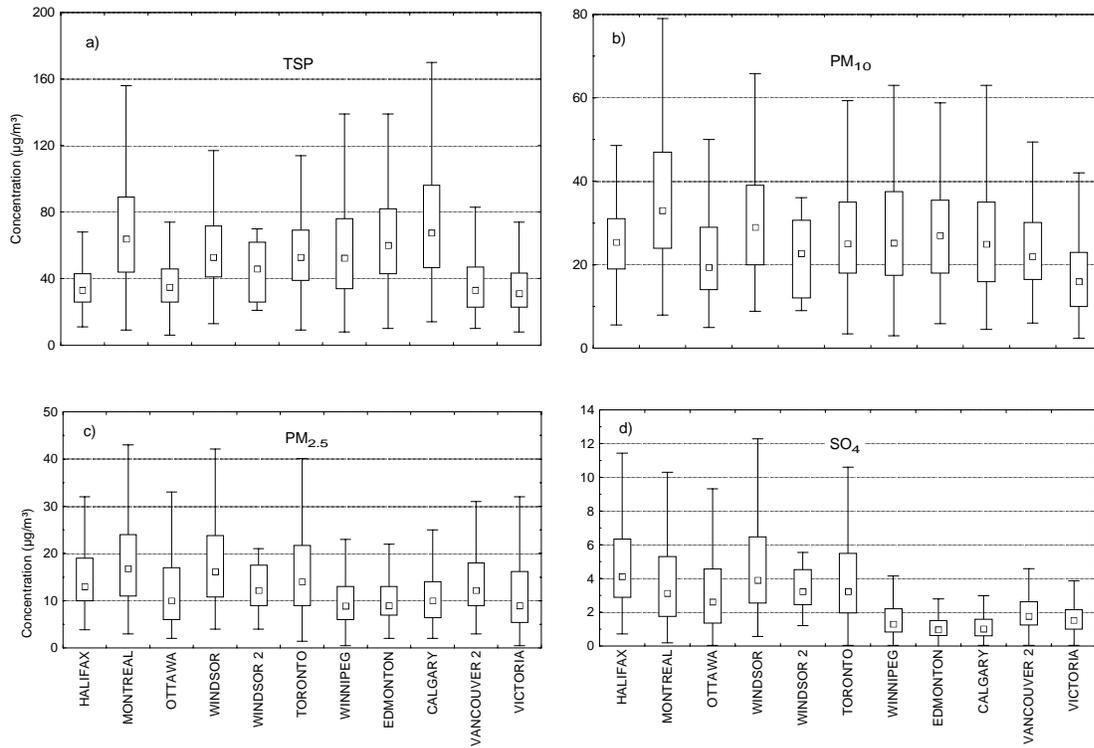
Figure 3 illustrates the variation in the $PM_{2.5}$ to PM_{10} mass ratio (1984-1993) across the country. The ratio of $PM_{2.5}$ to PM_{10} mass across Canada ranges from 0.4 - 0.6 $\mu g/m^3$. Approximately 50% of the time the ratio of $PM_{2.5}$ to PM_{10} does not vary by much more than ± 0.10 at a given site.

Figure 3. Distributions of the Ratio of $PM_{2.5}$ to PM_{10} Mass at the NAPS Dichotomous Sampler Sites. The Box Plots Indicate the Median, 5th and 95th and 25th and 75th Percentiles.
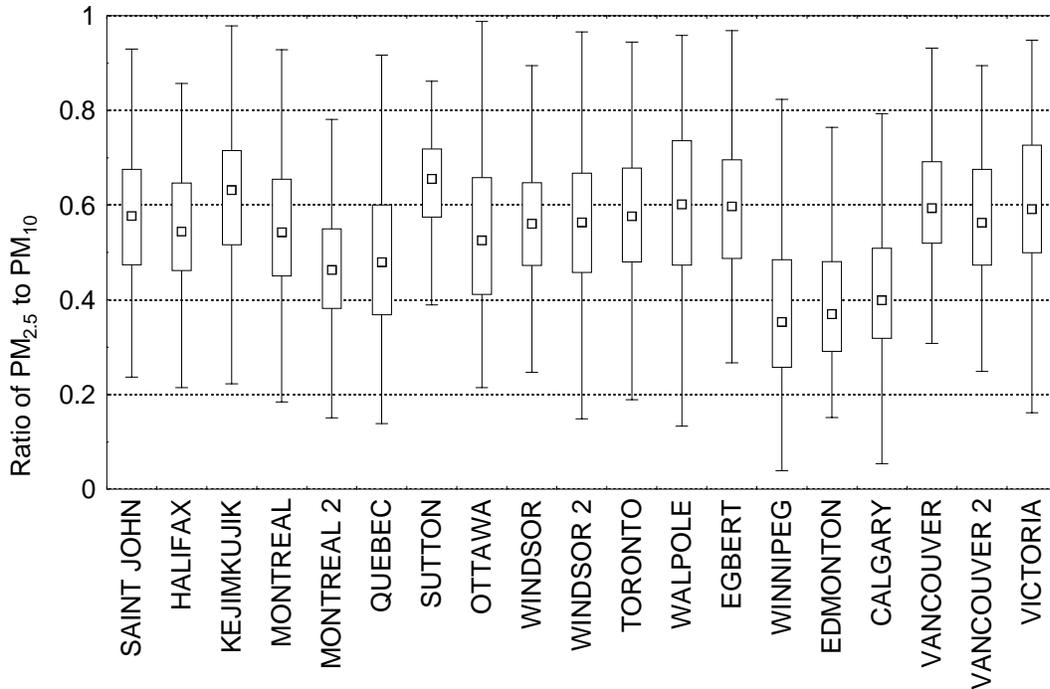
3. IMPACTS OF PARTICULATE MATTER

3.1 ENVIRONMENTAL IMPACTS

3.1.1 Aesthetic Effects (Visual Range)

There is a direct relationship between the amount of particulate matter in the atmosphere and the ability of the human eye to see through the atmosphere. Distant objects are perceived in terms of contrast, where the contrast of the distant object against the background (usually the sky) decreases as the distance between the object and the observer increases.   Visual range is the measure of transparency of the atmosphere.

Submicrometre particles are the most effective in reducing visibility; there is empirical evidence to show that there is a linear relationship between light scattering and fine particle mass.

An estimate of visual range (VR) and particulate loadings can be determined using the Koschmieder equation ($VR = 3.91 / b_{ext}$) by assuming a ratio of light scattering coefficient to fine particle mass of 3.1 m^2/g and appropriate ratios of the light extinction coefficient (b_{ext}) to the light scattering coefficient (b_{scat}) for urban and rural sites. Table 1 shows estimates of natural visibility and calculated fine particulate loadings at a number of the non urban sites in Canada. Natural visual range is considered to exist in regions not directly impacted by anthropogenic emissions. Western Canada and eastern Canada have estimated natural levels of $PM_{2.5}$ of approximately 6 µg/m^3, while southeastern Canada has a higher natural $PM_{2.5}$ level of 10 -13 µg/m^3. A 10% change in visual range is considered to be the minimum observable incremental change. Thus, in pristine parts of western and eastern Canada, a noticeable change in visual range would be observed when $PM_{2.5}$ levels exceed 6 to 7 µg/m^3 and for southeastern Canada, 14 µg/m^3. The full mathematical relationships are presented in the Science Assessment Document.

Table 1 Estimated Natural Visual Ranges in North America		
Site Location	**Visual range, km (estimated from b_{scat} (x 10^6/m))**	**Estimated $PM_{2.5}$ (µg/m^3)**
Western U.S. (Glacier National Park)	150 - 160	7.0 - 7.5
Western Canada (Waterton, AB)	210 - 350	3.2 - 5.5
Southeastern Canada (Egbert, ON)	86 - 120	9.7 - 13.0
Eastern Canada (St. Andrews, NB)	185 - 210	5.5 - 6.1

Note: VR calculated from b_{scat}/b_{ext} , and $PM_{2.5}$ calculated from $b_{scat}/PM_{2.5}$ = 3.1 m^2/g.

Figure 4. Contour plot of summer visual range (from Stuart and Hoff 1994).

Summer Visibility (RH<80%) 1951-1990
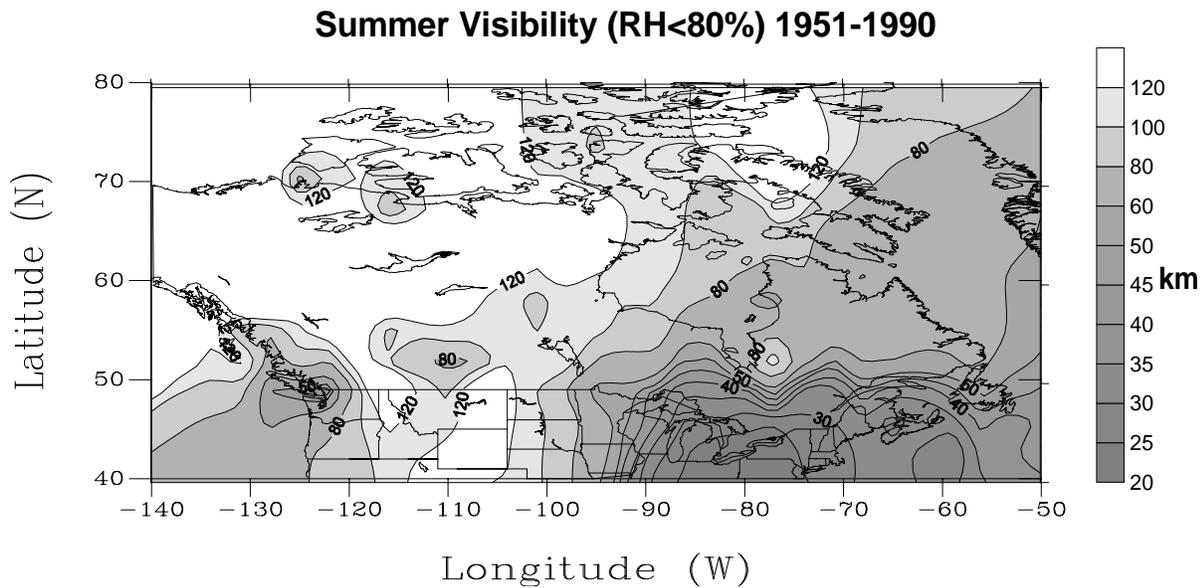
Figure 5. Contour plot of winter visual range (from Stuart and Hoff, 1994)

Winter Visibility (RH<80%) 1951-1990
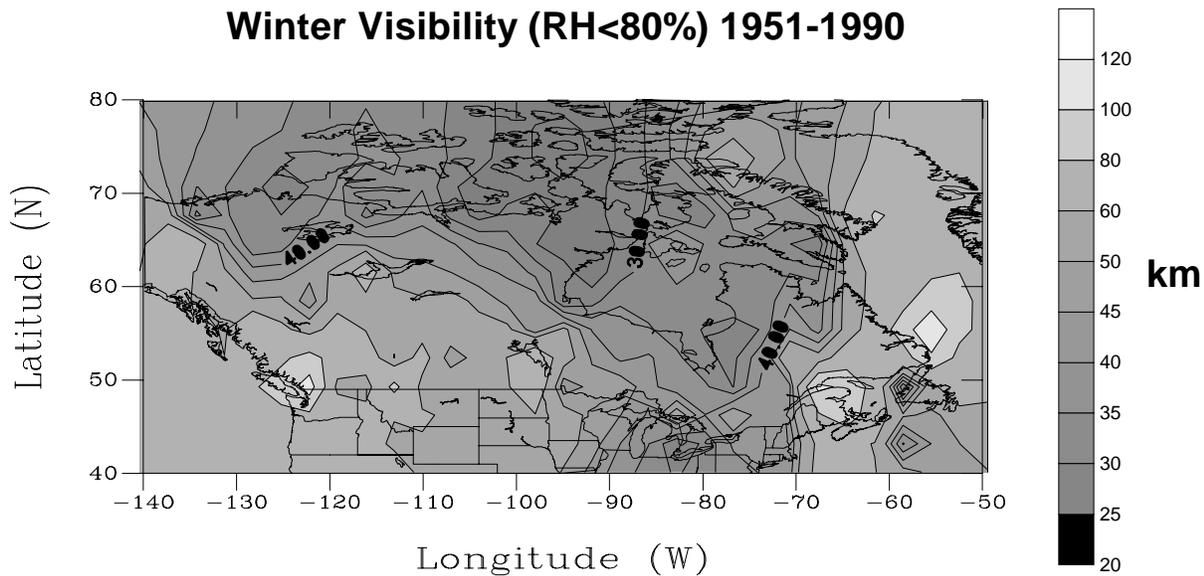
Visual range, and thus people's perception of acceptable visibility, varies by season. In Vancouver, $PM_{2.5}$ levels reach a maximum in winter, and consequently actual summer visual ranges would be greater than calculated from annual averages (1984 to 1993). Contours of visual range were determined from 140 stations across Canada, and median summer and winter visual ranges for 1951 to 1991 with relative humidity less than 80% are shown in Figures 4 and

5. For many regions of Canada, visual range is greater than 80 km. South-eastern Canada is notable, however, for significantly lower summertime visual ranges.

It is recommended that no Reference Level for $PM_{2.5}$ be identified to protect visual range, based upon the variability of perceived changes in visual range, the lack of site specific data on fine PM mass, and the inability to define a single natural background concentration.

3.1.2 Vegetation Impacts

The primary effects of particulate matter on vegetation are reduced growth and productivity, due to interference with photosynthesis, and phytotoxic impacts as a result of particle composition. The mechanisms of action include smothering of the leaf, physical blocking of the stomata, biochemical interactions, and indirect effects through soil. Particles make contact with vegetation surfaces in three ways: sedimentation, impaction and deposition. The relative efficiency of these methods depends upon the plant or soil surface, the microclimate and ambient conditions (temperature and humidity).

Given the limited amount of information available, specifically the lack of quantitative dose-effect information, it is not possible to define a Reference Level for vegetation.

3.1.3 Impacts on Materials

The deposition of particulate matter on materials can reduce their aesthetic appeal, as well as increase the rate of physical and chemical degradation. The primary effects of particulate matter on materials are to accelerate the rates of corrosion and erosion, and soiling and discolouration. Particles may act as catalysts for the conversion of SO_2 and NO_x to sulphuric acid and nitric acid which accelerate the chemical degradation of susceptible material surfaces on which they are deposited. Most information available is on the effects of particle exposure in combination with SO_2.

Given the limited amount of information available and specifically the lack of quantitative dose-effect information, it is not possible to define a Reference Level for materials.

3.2 HUMAN HEALTH IMPACTS

In evaluating the health impact of particulate air pollution, information comes from studies that have been conducted using epidemiology, controlled human exposure, and animal models. A large variance in the intensity and nature of the human response to particulate matter has been observed.

3.2.1 Epidemiological Evidence

Mortality

Daily or short-term variations in particulate matter, as PM_{10}, British Smoke (a slightly smaller particle size fraction than PM_{10}), $PM_{2.5}$, or SO_4, were significantly associated with increases in non-accidental mortality in 43 regressions carried out in 20 cities across North and South America, and Europe. The magnitude of the risk for PM_{10} was small, varying between 0.4% and 1.7% per 10 $\mu g/m^3$ increase, with a mean of 0.8% and a median also of 0.8%, for concentrations averaging 25 to 115 $\mu g/m^3$. The results were highly consistent under different PM_{10} exposure conditions. The magnitude of the increase was about the same for British Smoke as for PM_{10}. A 1% increase in daily deaths per 10 $\mu g/m^3$ increase in BS. For $PM_{2.5}$, a 1.5% increase in mortality per 10 $\mu g/m^3$ was observed (range 0.85 to 2.2% per 10 $\mu g/m^3$) at average concentrations that range 11-30 $\mu g/m^3$. The increase in $PM_{2.5}$ risk of mortality was thus about twice that for PM_{10}, while the increase for SO_4 was higher, at 2.2%. Fewer studies included more than one or two gaseous pollutants. While the increases are of very low magnitude, they signify substantial numbers of avoidable deaths due to the very large population that is exposed to PM.

Hospitalizations and Emergency Department Visits

Particulate matter of some kind has been shown to have significant associations with increased hospitalizations in most or all of the 26 studies examined. The relative risk of increased respiratory hospital admissions per 10 $\mu g/m^3$ PM_{10} was between 0.45% and 4.7%, with a median of 1.7% (n=16) at average concentrations that range 25-53 $\mu g/m^3$. The association between black and respiratory hospital admissions ranged from 0.4% to 12.3%. Results for directly measured $PM_{2.5}$, available only for Toronto and Montreal in three analyses, demonstrated positive associations with respiratory admissions; relative risks ranged from 2.5% to 9.6% per 10 $\mu g/m^3$ increase at an average concentration that ranges 12.2-18.6 $\mu g/m^3$.

Sulphates (SO_4) were associated with respiratory admissions, with increases of 2.0% to 9% for a 10 $\mu g/m^3$ increase. SO_4 appears to be a good surrogate for fine particles, primarily from combustion sources in many areas of the country. When co-regressed with ozone, a 2.7% increase per 10 $\mu g/m_3$ was demonstrated in S. Ontario, equivalent to 1.1% increase (95% CI 0.7% to 1.5%) per 10 $\mu g/m^3$ $PM_{2.5}$, based upon site-specific monitoring and conversion factors. Results for acidity (H^+) were inconsistent, with strong associations and high significance in some studies and none in others.

Effects on Lung Function, Symptoms, Restricted Activity and Days Absent from Work or School

In both normal and symptomatic and/or asthmatic children, recent epidemiology studies have shown short-term particle exposure to be associated with increased respiratory symptoms, such as cough or wheeze, and/or small reductions in lung function. Asthmatic adults also are affected by increases in the daily or short-term particle levels, mostly the fine fraction, reporting decreases in lung function and increases in respiratory symptoms.

A particularly informative series of studies was conducted in the Utah Valley where the closing and reopening of a steel mill was parallelled by marked changes in population health; respiratory admissions of children to nearby hospitals were two- to three fold higher during the winters when the steel mill was in operation in comparison to the winter during which it was closed. The number of days absent from school and the number of respiratory-related activity restrictions in adults has been found to be increased during periods with high particulate matter concentrations. Ten $\mu g/m^3$ increases in fine particles or SO_4 were associated 2.8% to 16% reductions in activity.

Longer Term and Chronic Effects

The effect of longer term and/or chronic exposure, varying between one and 16-20 years' duration, is associated with increases in mortality, lung function decrements, respiratory disease symptoms, and lung cancer. The probability of survival over a 7 to 16 years period was reduced for people living in the most polluted cities compared to the least polluted. In the Six-cities study, average mortality was increased by 9%, 14% and 35% for each 10 $\mu g/m^3$ increase in PM_{15}, $PM_{2.5}$, and SO_4, respectively. Several cross-sectional studies have reported increases in mortality, per 10 $\mu g/m^3$ increase in PM, ranging from 4.3% to 9.8% for $PM_{2.5}$, and 8.2% to 12.4% for SO_4. These cannot with certainty be ascribed to a true chronic effect, since they could equally be the result of cumulative effects of daily variations in particulate matter.

Decreases in lung function, capacity, growth and development that were shown in cohorts of children across North America after chronic or lifetime exposure to acidity, sulphate and fine particle air pollution, could be considered true chronic effects. The evidence, however, associating chronic exposure to PM with these endpoints is still inconclusive. Some studies have shown no association between exposure to PM and these chronic health endpoints. Recent evidence shows increases in the development of bronchitis and airway obstructive disease following chronic exposure to acid, sulphate, $PM_{2.5}$, coarse particles, PM_{10}, and TSP. There were indications from a long-term (20-25 years) cohort study in older adults that this increased incidence of disease, and probably also the reduced lung capacity that accompanies it, is carried over into adulthood as increased susceptibility to adverse effects of air pollutants. Chronic exposure can also increase the severity of respiratory symptoms associated with airway obstructive disease, chronic bronchitis, and asthma.

The development of lung cancer was associated with fine particulate air pollution; results are not yet available for PM_{10} or coarse particles. The association was weak by comparison to other lifestyle factors, particularly smoking, and the possibility of residual confounding cannot be dismissed.

3.2.2 Evidence From Controlled Human Exposure Studies

Controlled human exposures to acidic and inert particles have not caused significant alterations in pulmonary function in healthy individuals at relatively high levels compared to those generally experienced in the environment. However, acidity has been shown to affect the slowing of mucociliary clearance at concentrations as low as 100 $\mu g/m^3$.

The controlled human exposure studies identify asthmatics as having increased susceptibility because their pre-existing disease state increases their sensitivity to particle exposure. Asthmatics, especially children and adolescents, may experience adverse effects on pulmonary function at aerosol concentrations experienced on occasion in ambient air (~ 35 $\mu g/m^3$ of H_2SO_4 for 40 min). There has been no convincing evidence suggesting that subjects with chronic obstructive pulmonary disease (COPD) or the elderly are susceptible populations in terms of pulmonary function responses, although some data show that the pulmonary deposition of ultrafine particles (mass median aerodynamic diameter [MMAD] 0.02 - 0.24 μm) in COPD patients was higher than in healthy subjects.

However, almost all the human clinical studies regarding particle-induced health effects have been based on the observations of pulmonary function changes and subjective symptom reports. There is hardly any data published on particle-induced airway inflammatory responses. No data on changes of cardiovascular system have been documented. Several reports from clinical studies have demonstrated that ozone-induced decrements in FEV_1 do not correlate with airway inflammation parameters. "Responders" with substantial ozone-induced decrements in FEV_1 often have pulmonary tissue injury at levels similar to that seen in "non responders." If this is also the case for particles, decrements in pulmonary function may not be sensitive indicators for particle-induced lung injury. Moreover, based on the assumption that the response of pulmonary function to air pollutant may be a protective mechanism for the lungs from receiving further insults in deep airways, failure of certain subjects, such as COPD patients, to have pulmonary function responses to particles might render these patients more vulnerable to the pulmonary injury.

None of the human clinical studies has used particle generation systems that reflect the complexity of ambient particles. Based on the extremely limited clinical database available on various species of particles, acidic aerosols produce the most significant bronchoconstriction, while the toxicity of sulfate is related to acidity *per se.* The toxicity of nitrates was not considered, since previous work had shown it not to exert effects on lung function at concentrations below 1000 $\mu g/m^3$ in clinical studies. Inert particles appeared to have no effect on lung function in either healthy or asthmatic volunteers in the few studies available. Although very fine particle diesel exhaust affected neutrophil production and macrophage clearance of

microorganisms from the lung, the effects cannot be ascribed with certainty to particles, since formaldehyde and other combustion gases were also present in the inhaled mixture.

Very little work has been done on the effect of particle size specifically on airway mucociliary function. Limited studies have shown that fine particles are cleared from the lung more slowly than larger particles, and that submicrometre particles clear very slowly, taking more than one to two years in a few cases, especially in patients with obstructive lung diseases. A recent study by Peters et. al. demonstrates that symptoms and decrements of peak expiratory flow in asthmatic subjects (n = 27) were significantly associated with the 5-day mean of the number of ultrafine particles (MMAD 0.01 to 2.5 μm).

Despite the fact that the ranges of particle concentrations usually exceed those experienced by the general population, little evidence for a dose–response relationship has been documented in the clinical toxicological literature. Even at high particle concentrations in susceptible subpopulations, acidic aerosols have been found to produce only small decrements in lung function.

Overall, the clinical data does not lend much support to the observations seen in the epidemiology studies, particularly to the observations that high ambient particulate concentrations are associated with mortality within hours or a few days at most. It does indicate one susceptible subpopulation, asthmatics, who currently comprise 5 to 8 percent of the population, a percentage that has been rising in the past decade in Canada as well as in other western countries. Possible explanations for the discrepancy between clinical and epidemiological data may lie in as follows: (1) the experimental subjects can only be exposed to the tested air pollutants for short duration for practical and ethical reasons, while an urban pollution episode usually lasts a few days for general population exposure; a clinical study has shown that doubling the length of exposure to H_2SO_4 exerted greater effect on bronchial mucociliary clearance than did an order of magnitude increase in the concentration of H_2SO_4; (2) ethically it is almost impossible to investigate responses in those people most likely to be affected by air pollutants; (3) the pulmonary function parameters that are most often used in clinical studies may not be sensitive enough to indicate particle-induced adverse health effects; (4) artificial particles used in exposure chambers may not reflect the potential synergistic effects of particulate matter and aerosol mixtures; (5) in most human studies, the sizes of aerosols are above 0.5 μm. Since nanometre-sized ultrafine particles have been found in animal studies to induce acute pulmonary inflammation and death at very low concentrations, and they are present in ambient air, ultrafine particles may be a good candidate to provoke acute alveolar inflammation with release of mediators capable, in susceptible individuals, of causing cardiorespiratory responses.

3.2.3 Animal Toxicology Evidence

Studies using experimental animals have been restricted to well-defined particle species. Although not comparable to the complex ambient particle mixture, acute exposures, almost

always at concentrations well above those occurring in the environment, have been shown to cause:

- decreases in ventilatory function;

- changes in mucociliary clearance;

- increased number of alveolar macrophages and polymorphonuclear leukocytes in the alveoli;

- alterations in immunologic responses (particles with known cytotoxic properties e.g, metals, affect the immune system to a significantly greater degree);

- changes in airway defence mechanisms against microbial infections (appears to be related to composition and not the particle effect);

- increase or decrease of the ability of macrophages to phagocytize particles (related to composition);

- a range of histologic, cellular, and biochemical disturbances, including the production of proinflammatory cytokines and other mediators by the lung's alveolar macrophages, (may be related to particle size, with greater effects occurring with ultrafine particles);

- increased electrocardiographic abnormalities;

- increased mortality.

As expected, bronchial hypersensitivity to non-specific stimuli and increased morbidity from cardiorespiratory symptoms and mortality occur most likely in animals with pre-existing cardiorespiratory diseases.

The epidemiological finding of an association between a 24-hour ambient particle level below 100 μg/m^3 and mortality has not been substantiated by animal studies as far as PM$_{10}$ and PM$_{2.5}$ particles are concerned. With the exception of the ultrafine particles ($\leq$0.1 μm), none of the other particle types and sizes used in animal inhalation studies causes such acute dramatic effects, including high mortality at ambient concentrations. The lowest concentration of PM$_{2.5}$ reported to have caused acute death of rats with acute pulmonary inflammation and hypertension or chronic bronchitis, was ~250 μg/m^3 (3 days, 6 h/day), using continuous exposure to concentrated ambient particles. Some recent evidence, however, has shed some light on the potential mechanism of particle-induced cardiovascular diseases, which may provide biological plausibility for the epidemiology findings.

The particle types most likely to induce acute adverse effects include metals, organics, acids, and acidic sulphates of the fine particle mode, possibly occurring as coatings on fine or even ultrafine carrier particles. It appears that the ultrafine particle mode ($\leq$0.1 μm in size) may be of significant toxicological importance due to its large number and slow clearance rate from pulmonary interstitium.

Subchronic and chronic exposures to some particles at mass concentrations >1 mg/m^3 result in significant compromises in various lung functions similar to those seen in the acute studies, and in addition, cause:

- reductions in lung clearance;

- induction of histopathologic and cytologic changes (regardless of particle type, mass concentration, duration of exposure or species examined);

- production of chronic alveolitis and fibrosis, and

- production of lung cancer.

The interpretation of results from experimental inhalation studies in animals with particles and their significance for human exposures involves considerable uncertainties. These uncertainties relate to dosimetry of the respiratory tract, differences in the sensitivities of specific target cells, differences in cell populations in the individual airway generations of animal species, differences in metabolic activity of lung cells, and differences in the lifespan between laboratory animals and humans. A recent comparative dosimetric analysis conducted by Miller and colleagues has yielded some interesting results, namely that, based on the calculations per ventilatory unit or per alveolus, humans receive much greater numbers of particles than do rats when exposed to the same concentration of PM. The trend of differences between humans and rats is even more pronounced for the individuals with compromised lungs (smokers, asthmatics and patients with chronic obstructive pulmonary disease) compared with normal subjects. Therefore, rats exposed to 1000–1500 µg/m^3 of particles may actually have received a level of particles equivalent to 120–150 µg/m^3 in humans. Given the caution which must be exercised in extrapolating risks from animals to humans, animal studies are best used to help elucidate the mechanism(s) of particle toxicity.

The animal studies clearly show effects on the lungs resulting from the inhalation of particulate matter, effects that can be attributed to a particle effect per se, as described above. No firm conclusions can be drawn, however, from the results of the numerous animal toxicology studies to answer the question of which particle type and size is most likely to cause the adverse effects. Particle size does appear to be a very critical character, however, with smaller particles having more pronounced effects, and particle size is believed to be the most important characteristic influencing deposition in the human respiratory system.

The significance of particle size is linked also to particle number and surface area. Ultrafine particles (<0.1 µm), by virtue of their greater numbers (2.4 million particles of 0.02 µm diameter correspond in mass to 1 particle 2.5 µm in diameter), greater surface area and slow clearance from the pulmonary interstitium, may be of particular toxicological importance and may also provide an answer to the puzzle of observed epidemiological effects at low particle mass levels. Ambient monitoring of the ultrafine particle mode of the urban aerosol is very difficult, and therefore, few data are yet available to carry out epidemiological testing of the role of ultrafine particles in contributing to cardio-respiratory illness and death.

Chemical composition of the particle may also play a role. From the toxicological evidence, the

particle types most likely to induce acute adverse effects include metals, organics, acids and acidic sulphates of the fine particle mode, possibly occurring as coatings on fine or even ultrafine carrier particles. The coarse particle mode is less likely to induce acute adverse responses than are either the fine or ultrafine modes, a fact attributed to both size and composition. However, these larger particles may well contribute in some way to effects.

The impact of interactions between different constituents of air pollution has been examined in animal studies to only a limited degree, mostly focusing on particulate and one gas-phase compound only. Such combined exposures have resulted in mixed responses, showing either no effect of the combination or some synergism depending on endpoint, but overall the results are equivocal. However, realistic environmental conditions are far more complex than those utilized in experimental settings. The actual mechanism of particle induced cardiovascular response is not yet clear. Some recent studies have suggested that it may involve the oxidation of low density lipoprotein by reactive oxygen species accompanying particulate pollution. Oxidized low density lipoprotein is known to be very cytotoxic.

3.2.4 Weight of Evidence - Causality Presentation

The best evidence demonstrating an association between particulate matter and cardiorespiratory illness is provided by the mass of epidemiological data. These point to a "pyramid of effects" headed by

1. increases in mortality due to cardiorespiratory diseases,

2. increases in hospitalizations for cardiorespiratory diseases,

3. decreases in lung function in children and in asthmatic adults,

4. increases in respiratory symptoms which can lead to increases in respiratory-related activity restrictions and days lost from work or school,

5. long term or chronic effects including reduced survival, reduced lung function and capacity in children, and increases in development of chronic bronchitis and asthma in some adults.

Although the epidemiology studies are observational rather than experimental, they have been considered more relevant for development of objectives and guidelines than the animal toxicology or controlled human chamber studies for several reasons:

- they are the most direct way of assessing the adverse health outcomes of "real world" complex mixtures of pollutants to which people are exposed;

- human populations, unlike laboratory animals, are highly heterogeneous, including individuals who encompass a large range of susceptibilities, disease status and exposures, and whose responses cannot be predicted from animal toxicology studies or are not available from controlled human exposure studies due to ethic reasons;

- population studies based on large administrative databases (such as the hospital admissions study in southern Ontario based on a population of 8.7 million people) are able

to demonstrate the impacts of pollution on public health, and even to enable some partial estimate of the costs to society;

- no extrapolation is necessary when assessing the effects on public health of a particular concentration of air pollutant or of an ambient air objective, as measured by the ambient compliance monitoring network, despite our lack of knowledge about the exposures of each individual in the population. We need only know that the correlation is reasonable between the ambient monitor and the personal exposure.

- these studies were conducted under a broad range of environmental conditions in many cities on three continents, by a number of different investigators.

However, there are a number of concerns when evaluating the epidemiology evidence. One of the key issues among them is that of causality. Epidemiologic studies do not provide data on biological mechanisms of the observed associations. Associations found in a particular observational study (case-control study, cross-sectional study or survey, ecologic study or cohort study) may reflect chance, bias, or cause; rarely does a single study provide evidence of an association that is sufficiently compelling to conclude that the association is causal, either on an individual level or on a community basis. Uncontrolled bias is a frequently invoked explanation for associations found between air pollutant exposure and health. The findings of epidemiologic studies of air pollution and health need to be interpreted within the context set by understanding of mechanisms of disease pathogenesis.

Nevertheless, the criteria for evaluating causality, first proposed by Bradford Hill and modified by succeeding epidemiologists, are used here to provide a framework for considering the possibility that PM "causes " cardiorespiratory mortality and morbidity.

On Causality

In evaluating the epidemiological studies as a whole, a number of issues arise, key among them the issue of causality. Epidemiological studies do not themselves provide data on biological mechanisms that would explain the observed associations. Associations found in epidemiological studies between PM and health effects may reflect chance, bias or cause. A weight of evidence approach is used whereby multiple lines of evidence are brought together and duly considered in order to build a case for causality. On the basis of accepted criteria, the weight of evidence from the epidemiological literature that supports a causal link between particulate matter and adverse health effects is summarized as follows:

the probability of a relationship between PM and cardio-respiratory health has been ably established;

the strength of the association between exposure to PM and health outcomes can be considered relatively strong, since although the magnitude of the estimates of increased risk are generally small, they are remarkably stable among different studies and are often highly statistically significant;

a monotonically increasing (no threshold) **concentration-response curve** was observed from very low ambient levels up to much higher levels with remarkable consistency in many of the studies on acute and chronic mortality and hospitalizations;

the evidence is considered to be strong with respect to the **specificity of the effect** to respiratory and cardiac outcomes; non-respiratory effects are not associated with exposure to particulate pollution;

the **specificity of cause** is considered to be strong enough to conclude that particulate matter per se, rather than other pollutants or environmental variables, is associated with adverse health effects;

a logical **temporal relationship** exists, with exposure (e.g. daily peaks in PM), followed by effects (e.g. increased mortality and hospitalizations), although the rapidity with which mortality has been observed following incidents of high exposure remains a puzzle in terms of the mechanisms of action of particles;

positive associations between particulate air pollution and cardio-respiratory related mortality and hospitalizations, and respiratory related health effects, have been **consistently** reported in numerous studies conducted under a broad range of environmental conditions in many cities on three continents, by a number of different investigators, providing a strongly **coherent** picture of the nature of particle-induced effects.

One of the most difficult questions has been, and continues to be, the role played by other

gaseous pollutants (particularly SO_2, NO_2, CO and O_3) in the toxicity of particulate matter. Many of the available studies could not or did not consider several of these co-occuring gaseous pollutants. In analyses designed to help separate out the effects of one pollutant from another, the association of particulate matter with adverse health outcomes reported in the epidemiology literature was remarkably robust to inclusion (one at a time) of all four of the normally present gaseous air pollutants SO_2, NO_2, CO and ozone). Moreover, the magnitude of this association was often (but not always) greater than any of these other air pollutants individually or combined. The magnitude, robustness, and consistency of this association across so many locations with differing air pollutant mixtures supports the position that particulate matter of some kind is the best indicator for the effects of air pollution on adverse health outcomes. The question of which particle metric is the best indicator of toxicity remains unsettled, but current evidence suggests that some form of fine particles is the best measure of particle toxicity, although in some locations, and with respect to some endpoints, coarse particles remain important and cannot yet be entirely dismissed.

The second critical outstanding issue with respect to causality relates to the biological plausibility of the effects of particulate matter on human health. When evaluating the effects of low levels of ambient particulates, we need to clearly separate acute adverse effects from chronic effects that reflect long term levels of air pollution. The association of mortality with daily variations in particulate air pollution presents difficulties in establishing a plausible mechanism that could explain these associations, particularly the very short lag period, or in some cases no lag, between the recording of elevated particle concentrations and the occurrence of increased mortality. Several hypotheses have been put forth to explain acute particle related mortality, and although the puzzle is by no means resolved, neither is it beyond explanation. The answer may likely involve exacerbation of preexisting disease conditions and evidence is mounting for a critical role for ultrafine particles on the strength of some recent toxicological evidence that has shown that mortality in rats can be induced after exposure to relatively low concentrations of these tiny particles.

These suggested biological mechanisms still require much more research and confirmation. However, they help close a major gap in our understanding, thus providing some support for the idea of causality. Precise mechanisms of action have yet to be established. It should be noted, however, that biological plausibility is not an absolute requirement for a conclusion of causality. Epidemiological observations have often preceded the biologic knowledge of the day, as evidenced by the example of smoking and lung cancer. A fundamental purpose of epidemiology is to establish a cause with enough certainty that it will be justifiable and highly appropriate to take action to mitigate effects on public health. This point has clearly been reached with respect to particulate matter.

3.2.5 Conclusions

While it is generally accepted that statistical associations drawn from well-conducted, randomized experimental studies provide the strongest evidence for causal relationship, little

evidence is available from the non-epidemiology studies. Based on the fact that none of the human clinical studies have used particle generation systems that reflect the complexity of ambient particles, we cannot rule out a causal relationship between ambient particulate pollution and acute adverse health effects.

According to the evidence presented in the preceding sections the strength and consistency of the epidemiological evidence for mortality and morbidity effects at current levels of particulate air pollution is remarkable, robust, consistent and compelling. Although the magnitude of the estimates of increased risk are seemingly small, they were often highly statistically significant. Moreover, the adverse health effects represent a large impact on the general population, since most of the population is exposed. The evidence is considered to be strong regarding the specificity of the effect for respiratory and cardiovascular outcomes. The evidence is harder to judge, but on balance, is considered to be sufficient to conclude that particulate matter per se, rather than other pollutants and weather parameters, is associated with adverse health effects. A strong pattern of coherence between endpoints is provided both qualitatively and quantitatively by the associations shown between particulate matter and a broad range of endpoints from the least serious to the most, i.e., mortality. The time pattern of exposure and effect adds to the coherence of the picture, with the exception of the rapidity of the effects on mortality (preliminary mechanistic research may soon provide an explanation for this observation). It should be noted that biologic plausibility is not an absolute requirement for a conclusion of causality.

The Working Group is satisfied that the demonstrated association between PM and adverse health effects cannot be accounted for by confounding factors or covarying pollutants. Although the biological mechanism is not clearly elucidated, the epidemiological data support a causal hypothesis between ambient particle exposure and adverse health effects and provide a reasonable basis for preventive and public health action. Measures to reduce ambient PM_{10} and $PM_{2.5}$ concentrations will lead to improvements in the health of Canadians.

Controlled human exposure studies have shown that healthy individuals experience few or no adverse effects on pulmonary function, host defence system or particle mucocilary clearance at relatively high concentrations of soluble particles compared to ambient levels. Asthmatic individuals may experience adverse effects on airway function at concentrations equivalent to relatively high ambient levels. Effects are more pronounced in adolescents and children; they are also responsive to acidic aerosols at concentrations close to ambient levels. There is no conclusive evidence, however, of enhanced responsiveness in the elderly, or in individuals with COPD.

On a population basis, the hypothesis is that the observations reflect an exacerbation of pre-existing disease, or enhanced response of a sub-population of sensitive individuals. Suggestions that the elderly are a susceptible population, more so than young adults, remains unsolved in the absence of pathology. The mechanism of increased susceptibility in children is also unclear. Their enhanced responsiveness could be because they spend more time outdoors, provided ambient particles have greater toxic potency - which has not been proven yet. Alternatively, since children have a greater incidence of respiratory and other illnesses, their responsiveness could reflect exacerbation of existing disease or a pre-disease state. Pre-

existing disease includes COPD (chronic bronchitis, emphysema, asthma, pneumonitis), upper and lower respiratory tract infections, influenza and cardiovascular disease.

There is no clear evidence of a "threshold" level for the positive associations between particulate matter and both daily mortality and hospitalization rates. That is, any increase in ambient particulate matter is associated with a potential increase in mortality and hospitalization rates. These endpoints are emphasized as their impact can be quantified. However, they are only the tip of the iceberg with respect to other adverse health effects including exacerbation of respiratory symptoms such as bronchitis, reduced lung function, restricted activity due to illness, loss of work-days or school absences, and increased costs for medication. These particulate matter - adverse health associations are observed at concentrations currently occurring in Canada, which are low by comparison to international standards or to the concentrations observed in pollution episodes in the 1950's and 1960's in which thousands died.

The weight of evidence from the epidemiology studies indicates that exposures experienced by populations in developed countries are of sufficient adverse health consequences to have an impact on public health.

3.3 IDENTIFICATION OF REFERENCE LEVELS

The federal – provincial Working Group on Air Quality Objectives and Guidelines as part of its mandate is charged with establishing "Reference Levels", a level above which human health and environmental effects can be demonstrated. The Working Group recommends that no Reference Level for $PM_{2.5}$ be identified to protect visual range, based upon the lack of site specific data on fine PM mass, the variability of perceived changes in visual range, and the inability to define a single natural background concentration. In addition, given the limited amount of information available, specifically the lack of quantitative dose-effect information, it is not possible to define Reference Levels for the effects of particulate matter on vegetation orn materials.

For particulate matter and human health effects, the Reference Levels are derived statistically from several studies and should be interpreted as levels above which there is statistical confidence in the concentration-response relationship and a subsequent ability to provide quantification of adverse impacts (statistical LOAELs). The Reference Levels in this case should not therefore be interpreted as a threshold of effects. Below the Reference Levels, a statistically significant confidence interval cannot be established for the relationships between ambient PM and the observed health effects.

There is no clear evidence of a "threshold" level for the positive associations between particulate matter and both daily mortality and hospitalization rates, therefore, a safe level of exposure to PM cannot be identified. That is, any increase in ambient particulate matter is associated with a potential increase in mortality and hospitalization rates.

For the Reference Levels, mass concentrations of $PM_{2.5}$ and PM_{10} are recommended as the metrics of choice. Within the past several years, several large well-conducted studies have been published that support the hypothesis that some form of fine particles is generally more closely associated with respiratory illnesses than the larger particle sizes. Although most of the epidemiology studies measured PM_{10} or PM_{15}, the evidence indicated that the fine fraction of particulate matter ($PM_{2.5}$) was consistently associated with adverse health effects, and that the association was usually of greater magnitude than the associations with other particle metrics, including PM_{10}, acid, and sulphates, in studies which included both or several.

It is important to note that in humans approximately 25% to 60% of inhaled particles with a size ≤2.5 μm can be deposited in the alveolar gas exchange region, as opposed to less than 5% of larger particles (~10 μm) deposited in this region, which may render $PM_{2.5}$ more harmful in causing lung injury. There is also a strong association between adverse health effects and sulphate. Thus, based on biological deposition and available, albeit limited data, $PM_{2.5}$ is clearly the metric of choice for the fine fraction at this time.

A Reference Level for PM_{10} is also recommended, because of consistent associations demonstrated in epidemiology with mortality and hospital admissions, and due to concerns over its relationship to certain adverse endpoints (e.g., bronchitis). A few studies which have specifically examined the coarse fraction of PM_{10}, 5 - 10 μm aerodynamic diameter, in addition to PM_{10} and/or $PM_{2.5}$, have found that the coarse fraction was not associated with adverse health outcomes while the fine fraction, and often the total PM_{10} fraction as well, displayed such an association. Other studies have shown an association between the coarse fraction and adverse health outcomes. The larger particles, therefore, have not been eliminated from consideration.

The recommended Reference Levels for PM_{10} and $PM_{2.5}$ are generated based upon the weight of evidence from Canadian and U.S. studies, detailed in the Science Assessment Document, examining hospitalization and mortality relationships with air pollution. The Reference Levels for particulate matter as a 24-hour average total mass concentration are thus:

$$PM_{10} = 25 \ \mu g/m^3$$

$$PM_{2.5} = 15 \ \mu g/m^3$$

As more scientific research is conducted, the Reference Levels may change, because of better delineation of the adverse effects at lower concentrations, or better statistical analysis of the concentration-response relationships at low ambient concentrations.

4. APPROACHES TO IDENTIFYING PROTECTIVE RANGES

The Working Group developed several scientific approaches or options for identifying Air Quality Objectives. Given the decision to develop Canada Wide Standards for PM in preference to NAAQOs, the following discussion is presented in terms of identifying a concentration range or target, which if achieved would afford substantial reduction in the risks to human health and the environment. The discussion focuses solely upon human health impacts, as the supporting data for other receptor impacts were insufficient to explore quantitative risk estimates.

Determination of a No Observable Adverse Effect Level (NOAEL), or Lowest Observable Adverse Effect Level (LOAEL) or equivalent is traditionally the first step in developing air quality guidelines. This approach is discussed in sections 4.1 and 4.2 respectively.   A more rigorous approach was developed using an incremental risk analysis to estimate the number of adverse health effects resulting from increasing PM levels (section 4.3).

A summary of the targets or ranges identified as a result of these approaches is presented in Table 2. The next chapter presents the implications of these approaches in light of anticipated health benefits due to avoided impacts.

4.1 LOAEL APPROACH

One approach is identification of Particulate Matter concentrations which pose no quantifiable risk of adverse health effects. The identification of such a level is impossible for both $PM_{2.5}$ and PM_{10} since the epidemiological data show an increasing relationship with both mortality and measures of morbidity (hospital admissions, days of restricted activity, days of work loss, school absenteeism, and lung function impairment) through the entire range of monitored ambient air levels. Therefore neither a threshold nor a "No observed adverse effect level" (NOAEL) can be calculated at a population level.

Studies which have quantified the relationship between health effects and ambient PM concentrations reveal that there is a point at the lower end of the concentration distribution at which data limitations reduce the confidence in the association. From a statistical basis, this point can be considered to be a "lowest observed adverse effect level" or LOAEL. The epidemiological evidence does support the conclusion that PM has adverse health effects at concentrations below the Reference Levels; however the available data are limited in allowing understanding of the form of the relationship below this level.

In the PM Science Assessment Document a variety of studies examining associations between hospitalization and mortality were used as the basis for deriving $PM_{2.5}$ and PM_{10} statistical LOAELs. This statistical LOAEL is used as a basis for setting the Reference Levels for $PM_{2.5}$ and PM_{10}: a 24-hour PM_{10} level of 25 $\mu g/m^3$ and a 24-hour $PM_{2.5}$ level of 15 $\mu g/m^3$

The statistically derived LOAEL is a level above which the increase in the incidence of severe health effects in the population can be quantified. This leads to the recommendation that the LOAELs be considered a target for reducing health risks. Further it invites a rationale against

allowing increases in PM concentrations beyond the LOAELs in areas currently at or below them. It is also noted that additional research will likely lead to a decrease in the derived LOAELs in the future.

4.2 LOAEL WITH UNCERTAINTY FACTORS

Traditional toxicology procedures define a safe level of human exposure as some arbitrary fraction of that dose level at which no effects are observed in experimental animals or humans. In this situation, where a NOAEL has not been identified but the effects data are of sufficient quality to allow the derivation of a LOAEL, then a "virtually safe level" could be developed by the application of uncertainty factors to the LOAEL.   For the general population, uncertainty factors would account for inter and intra-species differences, potential interaction with other substances, and deficiencies in the database (for example, limited information on the mechanism of toxicity and/or toxicokinetics, the absence of chronic data or the absence of a NOAEL).

The absence of a threshold precludes the possibility that a sufficiently low level of exposure will be free of any degree of impact. Recognizing this, an arbitrarily selected factor of 2 applied to the observed effect levels may be considered appropriate to allow for the magnitude of any effect seen in the exposed group and their sensitivity compared with the general population or target group. Using this approach, the targets which would substantially reduce the risks to human health for PM_{10} and $PM_{2.5}$ are 12.5 µg/m^3 and 7.5 µg/m^3, respectively, averaged over 24-hours. These targets, however, are close to or less than the current range of PM concentrations associated with background levels. In recognition of this, "background levels" are suggested as an appropriate target for reducing human health risks.

4.3 INCREMENTAL RISK ANALYSIS

The final approach does not directly generate targets or ranges for protecting human health. Rather, it provides estimates of the number of excess hospitalizations or deaths at increasing concentrations of PM. From these results it is possible to identify a concentration or range where avoided impacts no longer substantially increase with improvements in air quality. The methodology is outlined in Appendix A. Estimates of current and potentially avoidable human health impacts and the interpretation of the analysis are presented in the Chapter Five.

The lack of a no-effect level implies that there are health outcomes, including death and hospitalizations, associated with everyday levels of ambient PM, not just the episodes of poor air quality, i.e. peak values. The majority of epidemiological studies provide an estimate of the relative risk of adverse health effects associated with a specific change in PM concentration (a 50 µg/m^3 increase in PM_{10} or a 25 µg/m^3 increase in PM2.5).   Assuming the concentration-response relationships for both PM_{10} and $PM_{2.5}$ are linear down to the LOAEL, the relative risk estimates can be used to calculate the change in adverse health impacts associated with a 1 µg/m^3 change ambient PM levels.

Briefly, the general methodology of the incremental risk analysis is:

1. Preparation of a normalized dataset for selected Canadian sites (to account for non-daily sampling of PM_{10} and $PM_{2.5}$). Datasets are prepared for current ambient air quality conditions and predicted ambient air quality conditions. The latter may be developed empirically (for example using rollback algorithms, or by air quality models.

2. For each site, sum all concentrations greater than the LOAEL to provide a cumulative estimate of the particle exposure affecting human health. Note: Though effects below the LOAELS are not robustly quantifiable at present, it is recognized that effects may be occurring at lower levels, i.e. down to background. Thus, there is a certain justification to also estimating the cumulative exposures from background estimates.

3. Average cumulative sums, for areas with more than one monitoring site as appropriate, to match the form of the incidence of the health endpoint. For example, to match Census Metropolitan Areas and/or estimate risks per million population.

4. Multiply the cumulative sums by the relative risk estimates (more specifically, by the concentration-response co-efficient based upon the relative risk estimate and the baseline incidence for the given health endpoint) for each health endpoint, to estimate the current or future risk.

Increases in PM concentrations will result in additional adverse health impacts. Conversely, decreases in PM concentrations from current ambient levels will result in human health benefits, i.e. avoided death, avoided hospitalization, fewer emergency room visits, less medication use, decreased restricted activity days, and fewer days of work lost.

For PM_{10} and $PM_{2.5}$, the incremental risk analysis resulted in the identification of an inflexion point where avoided impacts, i.e: benefits, started to drop off with improved air quality. The inflexion points are identified in Table 2 with the results of the other approaches.

Table 2. Summary of targets or ranges which would reduce risk to human health due to PM$_{10}$ and PM$_{2.5}$ exposure.

Approach	Basis	PM$_{10}$	PM$_{2.5}$
		μg/m^3 **over 24 hours**	
Lowest Observed Adverse Effect Level (LOAEL)	health based level at which statistically significant adverse effects on human health can be detected	25	15
LOAEL with a margin of safety	health based level, simulating a NOAEL, equivalent to ambient background	"Background"	"Background"
Incremental Risk Analysis	Determination of the health impacts associated with cumulative concentrations in 1 μg/m^3 increments above the LOAEL	35 – 40*	20 – 25*
* Derivation provided in Chapter 5			

5   INCREMENTAL RISK ANALYSIS: EVALUATION of APPROACHES

The Incremental Risk Analysis (IRA) approach was selected as a straightforward manner in which to assess the targets or ranges identified in the previous chapter. This analysis provides an estimate of both the current risks and future risks (or benefits due to avoided impacts) associated with reducing ambient PM. This evaluation is not intended to analyse the economic costs and benefits of alternative ways of reducing those risks.

In general, the estimation of risk is performed by summing the cumulative exposure (eg: ppb$\times$days) and applying a concentration – response co-efficient. The concentration – response co-efficients are developed from the relative risk estimates and baseline health effect incidence information. The large epidemiological database used to develop concentration-response co-efficients for adverse impacts of particulate matter allows quantification of risk for several endpoints when combined with predicted air quality levels. The concentration – response co-efficients used in this analysis, and their 95% confidence intervals are provided in Appendix A. The resulting estimates of annual outcomes reflect the sums of all daily impacts in that year. The IRA is done on a twelve month basis reflecting the fact that PM may pose a health risk at any time during the year.

5.1 CURRENT RISK ESTIMATES

The incremental risk analysis begins with an assessment of the impacts attributable to current PM concentrations. The one-in-six day sampling schedule for PM necessitated the creation of a normalized annual dataset for use in the risk and benefit estimations. The normalized lognormal ambient PM_{10} and $PM_{2.5}$ concentration distributions are discussed in Appendix B, and are based upon the 1992 through 1994 period.

The impacts attributable to current PM concentrations are estimated by initially summing all concentrations above a specified level, X, where X can be zero, the LOAELs, background, or some other concentration.   Using the LOAELS to estimate impacts involves summing all concentrations above the LOAELs, therefore, X equals 25 µg/m^3 for PM_{10} and 15 µg/m^3 for $PM_{2.5}$. For example, if the 24-hour average $PM_{2.5}$ concentration is 30 µg/m^3 for three consecutive days, the SUMX (X equals 15 μg/m^3) for that time period, (30-15)+ (30-15)+(30-15) equals 45 µg/m$^3\times$ days. The totals, referred to as SUM25 and SUM15 for PM_{10} and $PM_{2.5}$, respectively, are multiplied by the risk associated with a 1 µg/m^3 increase in PM, and then multiplied by the baseline incidence rate for a given health endpoint (e.g. the average daily Canadian non-accidental mortality rate, or the average daily respiratory hospital admissions rate). Tables 3 and 4 provide the central estimates of the number of deaths and hospitalizations resulting from current ambient PM_{10} and $PM_{2.5}$ concentrations, respectively.

Station[1]	City	Maximum 24-hour average[2]	PM$_{10}$ Cumulative Concentration (μg/m^{3}x days)	Annual Mortality
			Annual SUM25	per million people
30118	Halifax	42.4	192	2.8
30501	Kejimkujic	60.5	309	4.5
40203	Saint John	70.3	462	6.7
50104	Montreal	98.6	1446	20.9
50109	Montreal	118	3826	55.2
54101	Sutton	42.1	72	1
60104	Ottawa	68.4	1032	14.9
60204	Windsor	109.9	2148	31
60211	Windsor	104.8	3395	49
60424	Toronto	101.9	2545	36.7
60512	Hamilton	104.7	3515	50.8
61901	Walpole Is.	149.5	5536	79.9
64401	Egbert	77.5	819	11.8
70119	Winnipeg	110.6	1929	27.9
90130	Edmonton	77.6	1403	20.3
90227	Calgary	75.6	1370	19.8
100111	Vancouver	75.9	738	10.7
100303	Victoria	45.8	229	3.3

Table 3 Deaths Resulting From Current Ambient PM$_{10}$ Concentrations*

[1] Stations selected from the NAPS database; included if both PM$_{10}$ and PM$_{2.5}$ were available.

[2] Maximum values observed in a three year period (January 1992 through December 1994).

* Data from Tables A2 and A3 in Appendix A.

It is evident from Tables 3 and 4 that as maximum 24-hour average PM concentrations increase, the sum of all concentrations above *X* also increases, resulting in greater cardiorespiratory deaths and hospitalizations. However, the relationship between the number of 1 μg/m^3 increments exceeding *X* and the number of health impacts is not straightforward.   SUM*X* is city specific, and reflects the distribution of ambient concentrations. For example, the maximum 24-hour average PM$_{10}$ concentrations in Egbert and Edmonton were approximately 78 μg/m^3. The annual total of all concentration exceeding 25 μg/m^3 (SUM25) equals 819 μg/m^{3}x days and 1403 μg/m^{3}x days, respectively. Similarly, maximum 24-hour average PM$_{2.5}$ concentrations in Windsor (site #60204) and Hamilton were 61 μg/m^3, while the cumulative concentrations were 1350 μg/m^{3}x days and 2442 μg/m^{3}x days, respectively. What this implies is that for a given maximum concentration the local ambient distributions, which drive the SUM*X*, are not necessarily the same.

Station[1]	City	Maximum 24-hour Average [2]	PM$_{2.5}$ Cumulative Concentration ($\mu g/m^3 x$ days)	Annual Mortality	Annual RHA [3]	Annual CHA [4]
			Annual SUM15	per million population		
30118	Halifax	37.6	303	7.9	3.6	3
30501	Kejimkujic	46.7	375	9.8	4.4	3.8
40203	Saint John	38.3	309	8.1	3.6	3.1
50104	Montreal	69.6	946	24.7	11.2	9.5
50109	Montreal	68.9	1661	43.3	19.6	16.7
54101	Sutton	33.2	179	4.7	2.1	1.8
60104	Ottawa	53.8	709	18.5	8.4	7.1
60204	Windsor	60.6	1350	35.2	15.9	13.6
60211	Windsor	85.6	2006	52.3	23.7	20.2
60424	Toronto	66.4	1728	45	20.4	17.4
60512	Hamilton	61	2442	63.6	28.8	24.5
61901	Walpole Is.	126.6	3474	90.5	41	34.9
64401	Egbert	47.7	714	18.6	8.4	7.2
70119	Winnipeg	71.3	396	10.3	4.7	4
90130	Edmonton	56.3	321	8.4	3.8	3.2
90227	Calgary	35.6	214	5.6	2.5	2.1
100111	Vancouver	41.5	662	17.2	7.8	6.6
100303	Victoria	29.7	291	7.6	3.4	2.9

Table 4 Health Impacts Resulting from Current Ambient PM$_{2.5}$ Levels*

[1] Stations selected from the NAPS database; included if both PM$_{10}$ and PM$_{2.5}$ were available.

[2] Maximum values observed in a three year period (January 1992 through December 1994).

[3] RHA: respiratory hospital admissions.

[4] CHA: cardiac hospital admissions.

* Data from Tables A2 and A3 of Appendix A

5.2   INCREMENTAL RISK ESTIMATES

The role of the incremental risk analysis is to identify the future risks or potential benefits of avoided impacts for given reductions in ambient PM levels. Due to the current limited ability to model Particulate Matter air quality and subsequently predicted PM concentration distributions from anticipated reductions in primary PM and precursor gas emissions, an empirical algorithm was employed. The same normalized annual database (from 1992 through 1994) used to

calculate current risks, was used as a basis to estimate the incremental risks of reduced PM_{10} and $PM_{2.5}$ concentrations.

The normalized lognormal ambient PM_{10} and $PM_{2.5}$ concentration distributions (discussed in Appendix B) were adjusted using a proportional linear rollback (discussed in Appendix D) such that the maximum 24-hour average concentrations for a given site would be reduced to a specified target value. For PM_{10}, the targets range from 50 to 25 $\mu g/m^3$, and for $PM_{2.5}$, 30 to 15 $\mu g/m^3$. For example, if the maximum 24-hour average concentration of PM_{10} was 65 $\mu g/m^3$, all of the normalized daily concentrations would be reduced by 22.5% to achieve a maximum target value of 50 $\mu g/m^3$. This creates a new distribution of PM_{10} concentrations for each site. The basic assumption of the proportional linear rollback is that a broad-based emission reduction program will affect the current concentration distribution within an airshed in the same manner as it affects the representative (i.e.: normalized) maximum concentration. For this estimation of concentration distributions, there was no adjustment for background conditions.

Under the various target scenarios, all 1 $\mu g/m^3$ increments above the LOAELs are summed. Using the same methodology as described in Section 5.1 Current Risk Estimates and Appendix A, the potential health benefits resulting from a decrease in ambient PM levels are estimated. It should be noticed that the relative risk estimates (Table A1 in Appendix A) represent central risk estimates, and that the 95% confidence level has not been worked into this analysis to provide an indication of the range of predicted benefits.

The results are presented in Figure 6 for PM_{10} (illustrating the potential number of avoided deaths) and Figure 7 for $PM_{2.5}$ (illustrating the potential number of avoided impacts for deaths, respiratory hospital admissions, and cardiac hospital admissions). This analysis enables comparison of the annual incidence of health effects at current ambient $PM_{2.5}$ and PM_{10} concentrations with the potential reductions in health effects estimated under the various target scenarios. The estimates of the number avoided impacts per year at each of the specified target PM levels represent the potential benefit of decreasing current ambient concentrations.

Health benefits are realized with each 5 $\mu g/m^3$ rollback in maximum 24-hour average PM concentrations and corresponding concentration frequency distribution. When the results of the analysis were examined, there appeared to be a point at which the rate of gain in avoided impacts (benefits) began to decrease. This point was consistent across the various urban areas analysed. Figures 6 and 7 of the avoided effects for both PM_{10} and $PM_{2.5}$ show this as an inflection point (a change in the slope of the line) when concentrations are rolled back to achieve maximum 24 hour average concentrations of 35 and 40 $\mu g/m^3$ for PM_{10} and 20 and 25 $\mu g/m^3$ for $PM_{2.5}$. For perspective, the current national average 90th percentile concentrations (1992 – 1994) is 40 $\mu g/m^3$ for PM_{10} and 25 $\mu g/m^3$ for $PM_{2.5}$.

The inflection point is likely the result of consecutive rollback of ambient concentrations leading to a decrease in the difference between the mean ambient PM concentrations and a corresponding decrease in mean population exposures resulting in a levelling off of the potential benefits. Decreases in mortality and morbidity, however, are anticipated for any reduction in ambient PM levels. Benefits may be realized for not only the endpoints analysed here, but also for the other health effects associated with PM (emergency department visits, school

absenteeism, days of work lost, restricted activity days, and effects on lung function and symptoms).

An important point to grasp is that adverse health effects are occurring at ambient concentrations less than 25 $\mu g/m^3$ for PM_{10} and 15 $\mu g/m^3$ for $PM_{2.5}$. The estimated benefits from this incremental risk analysis, therefore, are conservative. The epidemiological evidence does support the conclusion that adverse health effects occur at concentrations below the lowest observed adverse effect level, however, the available data is limited in allowing understanding of the form of the relationship below this level. The actual benefits are expected to be greater than these estimates, however, our current understanding does not allow us to quantify them.

Figure 6a – e Number of Avoided Deaths Resulting From Decreased PM$_{10}$ Concentrations

Figure 6a. Number of avoided deaths resulting from decreased PM$_{10}$ concentrations
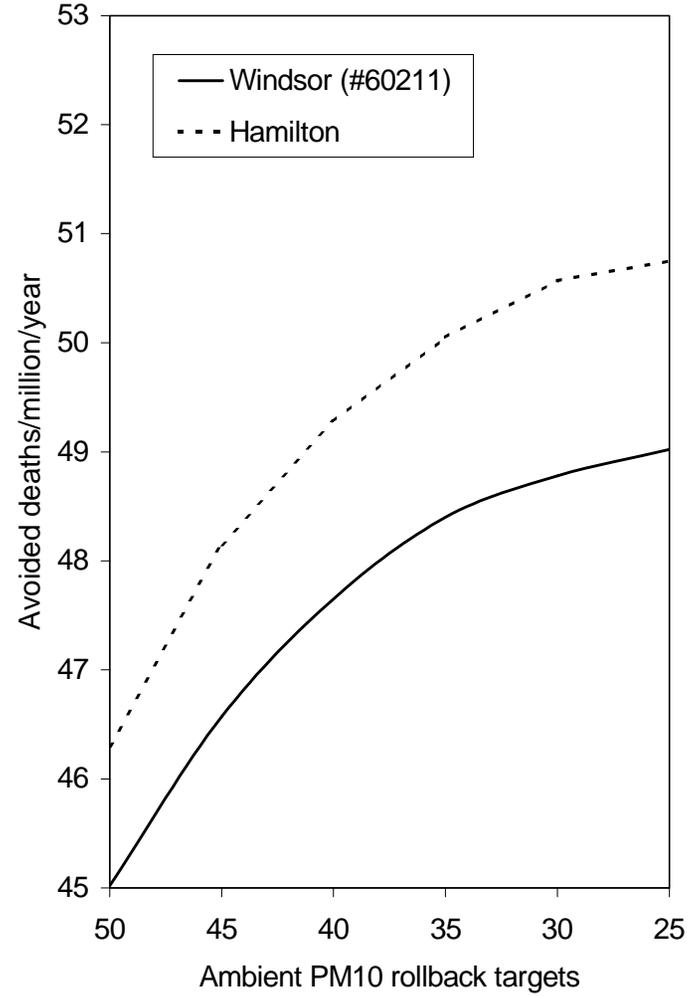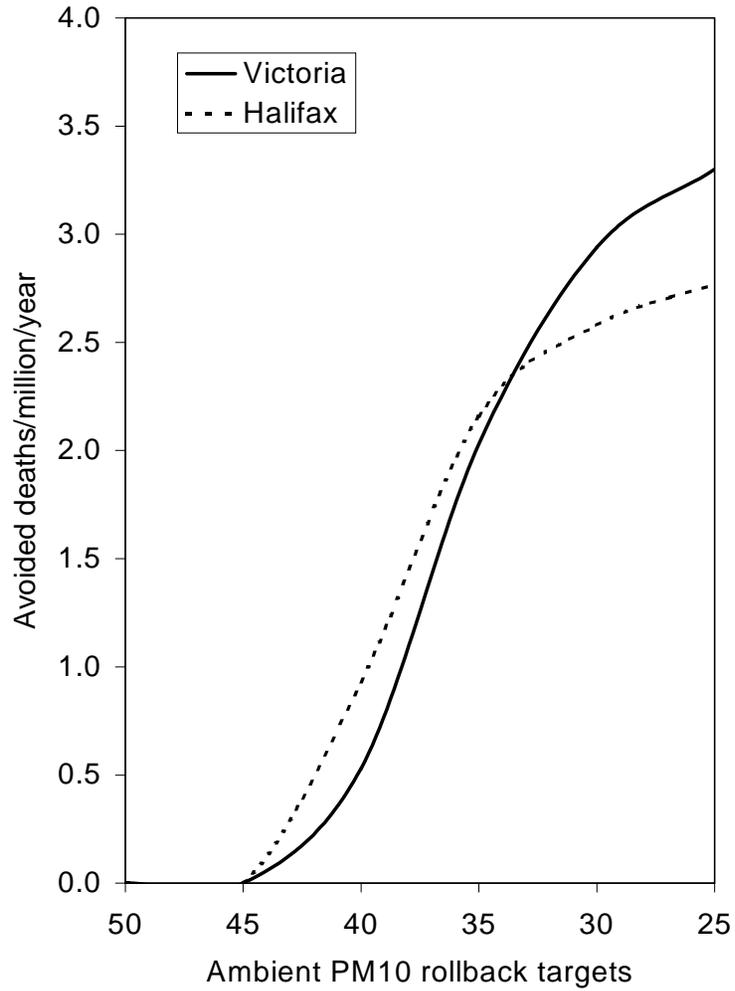
Figure 6b. Number of avoided deaths resulting from decreased PM_{10} concentrations
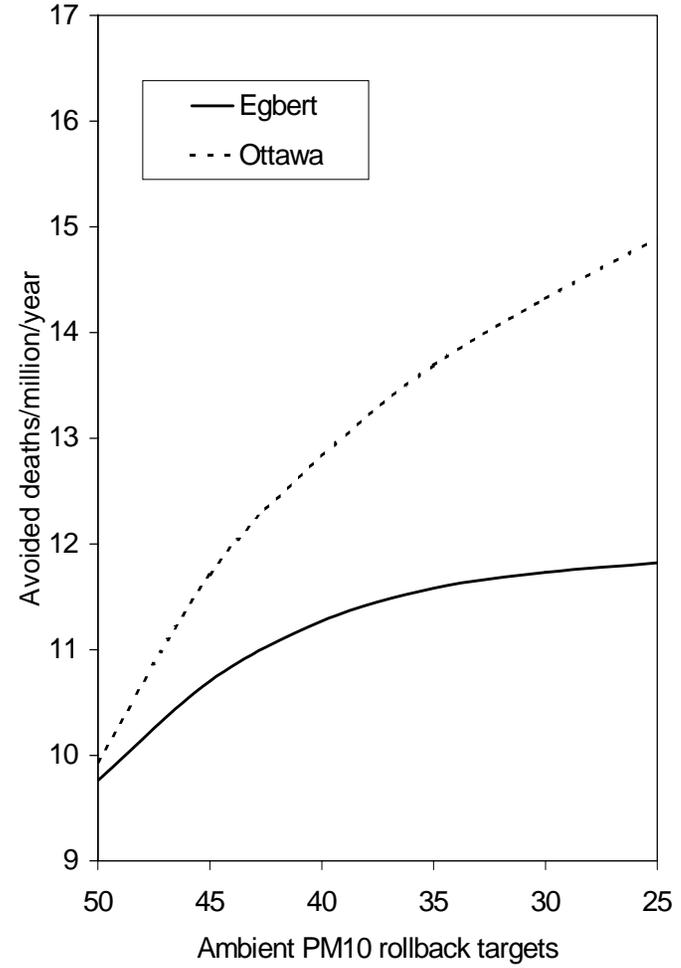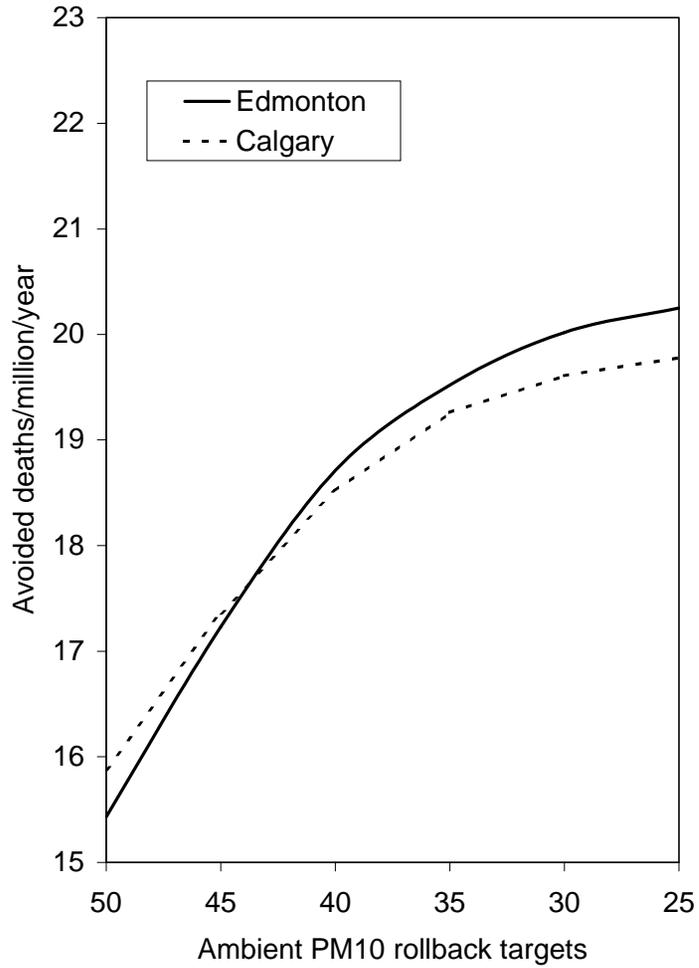
Figure 6c. Number of avoided deaths resulting from decreased PM_{10} concentrations
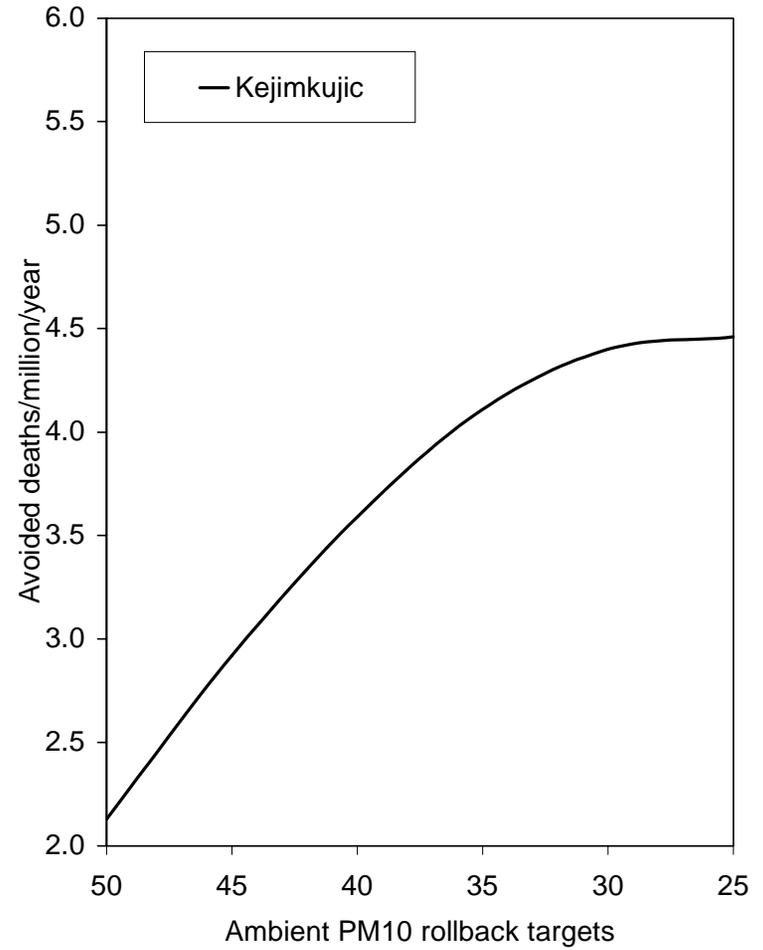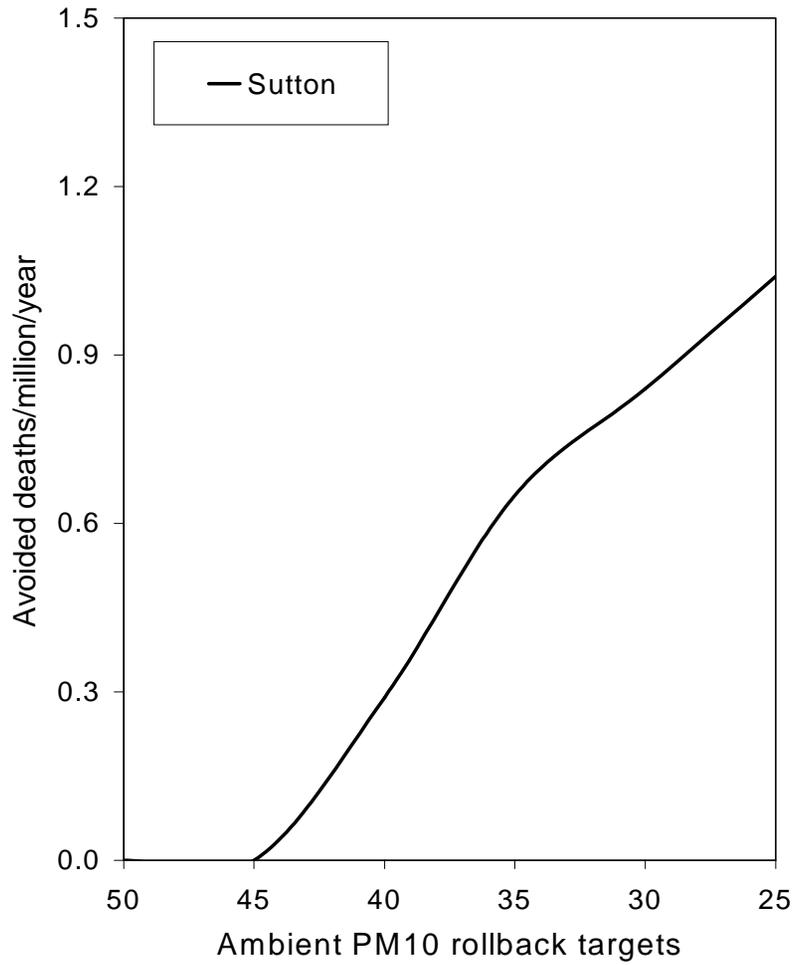
Figure 6d. Number of avoided deaths resulting from decreased PM_{10} concentrations
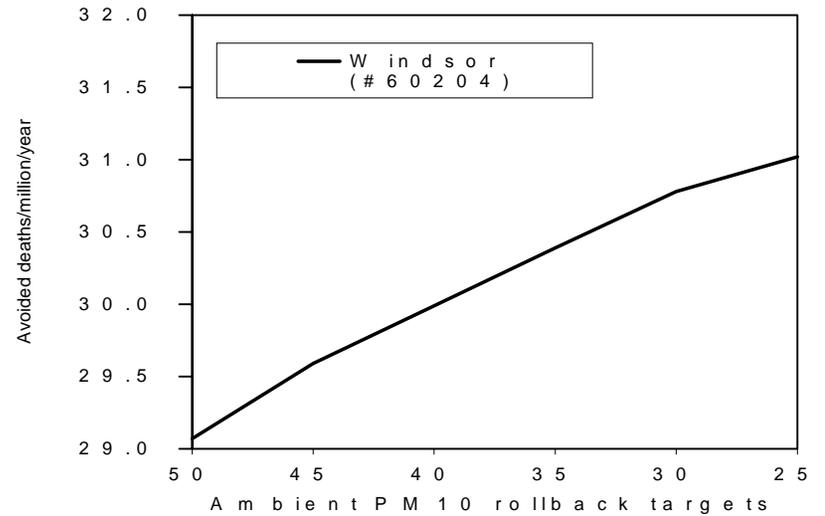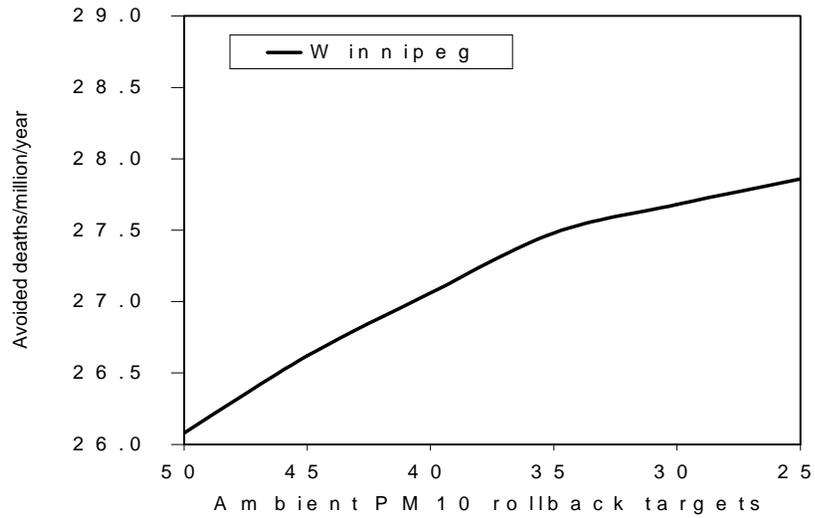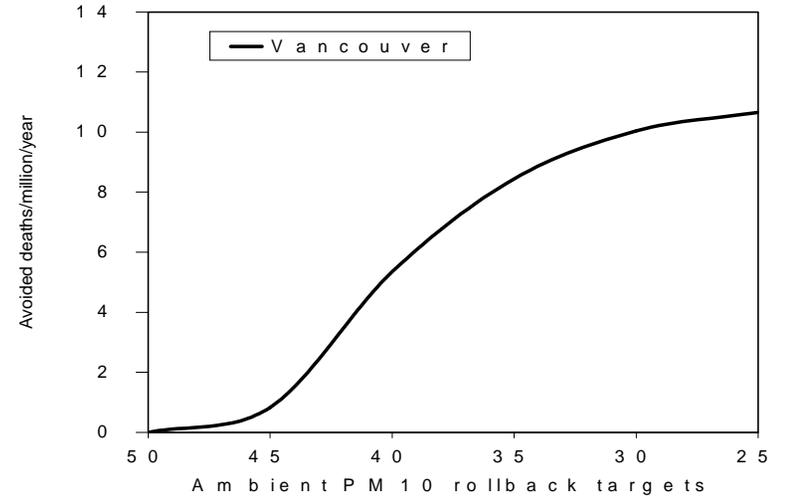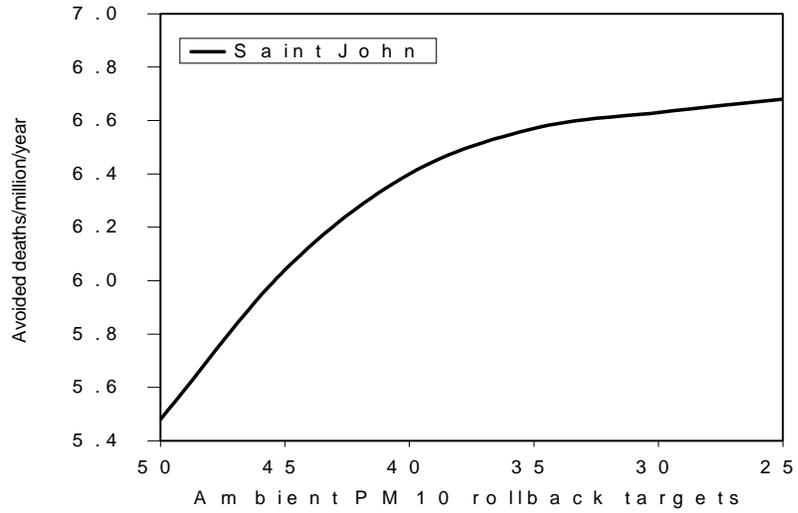
Figure 6e. Number of avoided deaths resulting from decreased PM_{10} concentrations
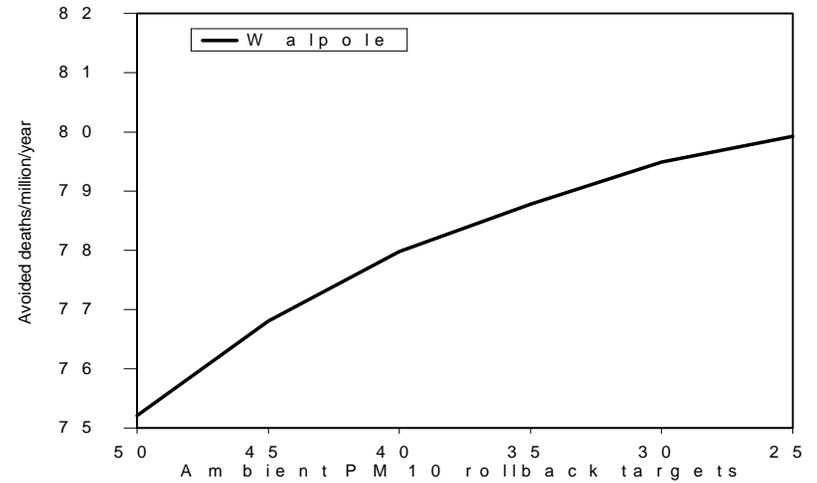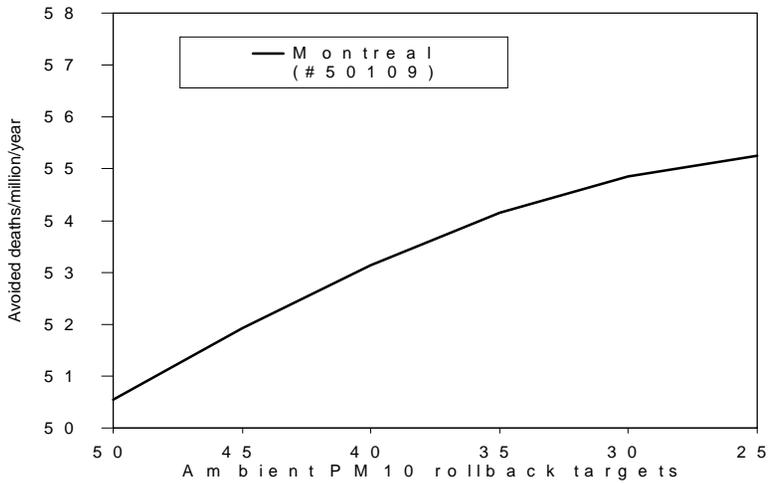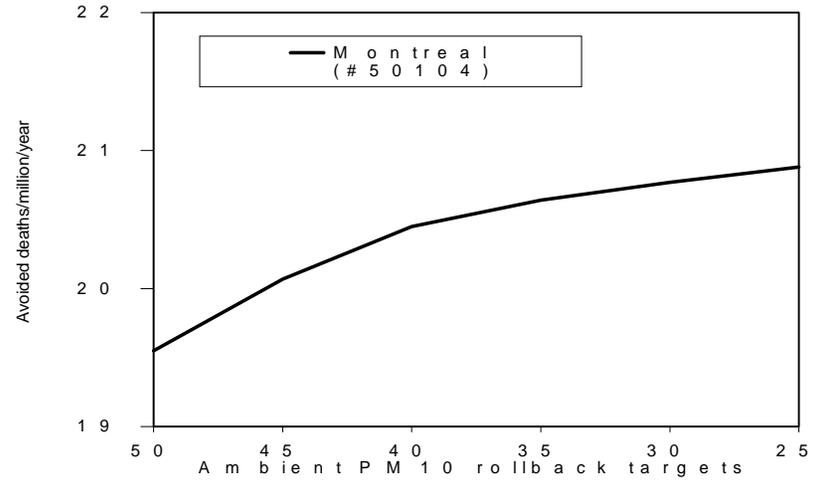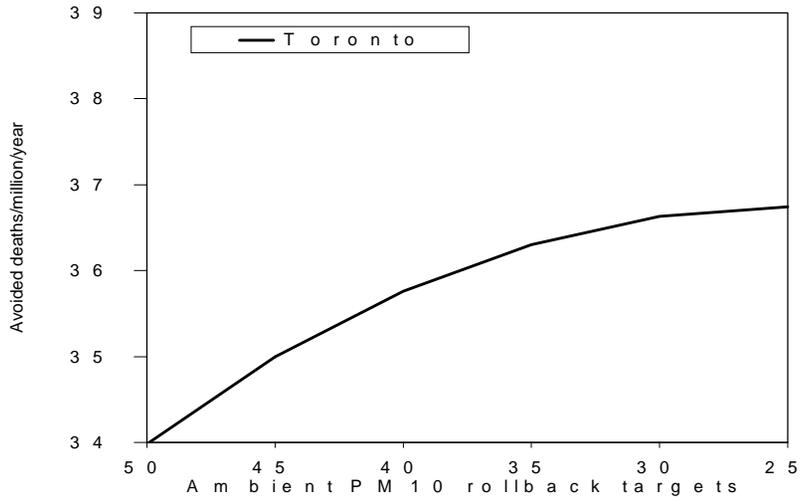
Figure 7a – i   Number of Avoided Impacts Resulting From Decreased PM2.5 Concentrations (cardiac and respiratory hospital admissions)
**Figure 7a.** Number of avoided deaths resulting from decreased PM$_{2.5}$ concentrations
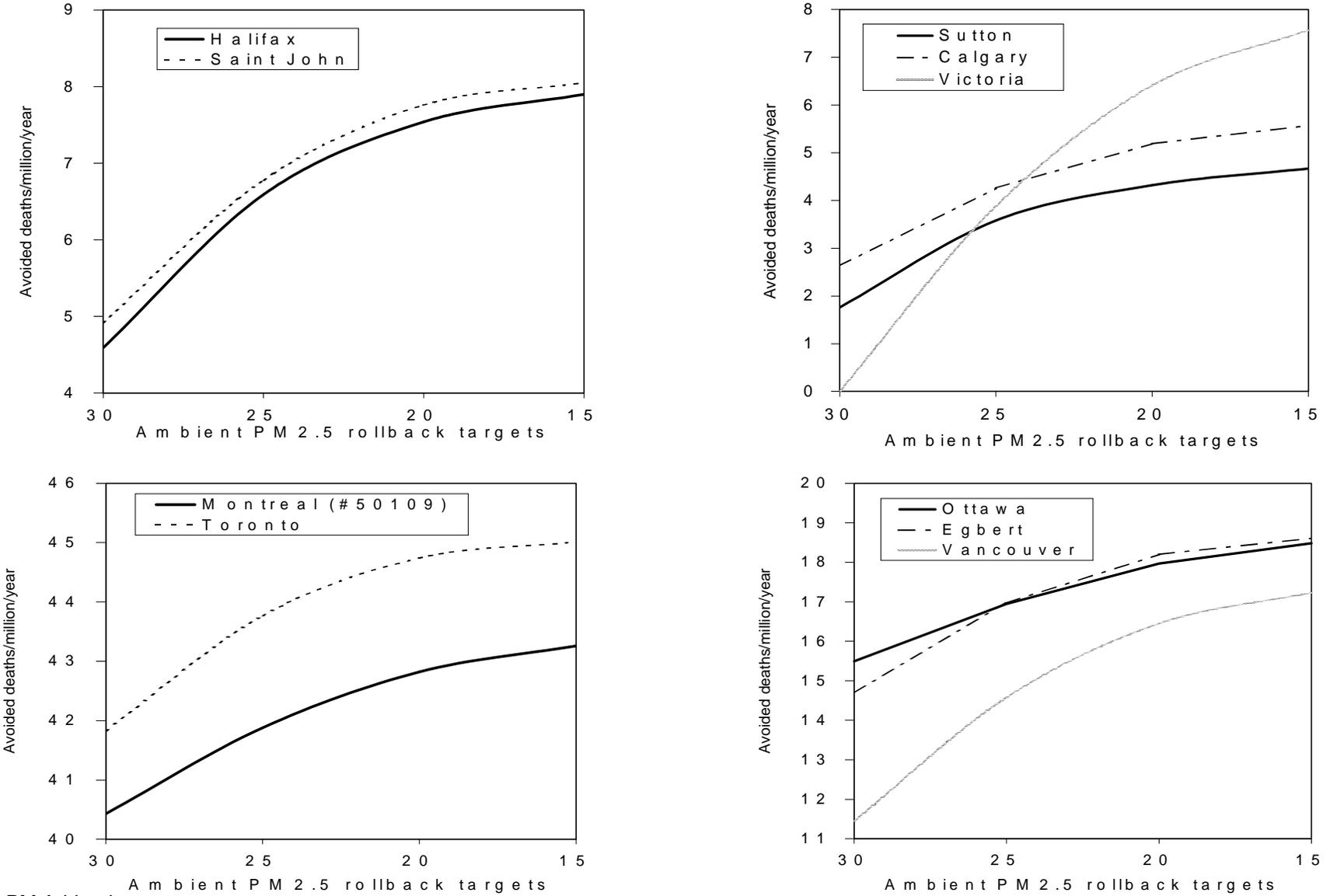
Figure 7b. Number of avoided deaths resulting from decreased PM$_{2.5}$ concentrations
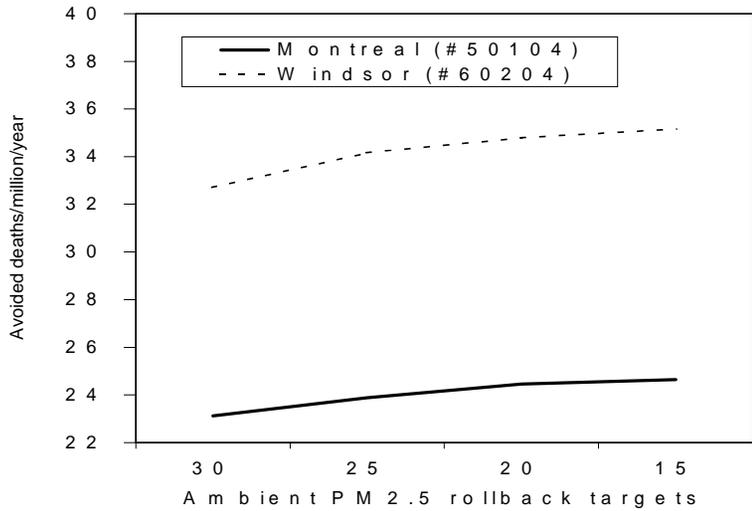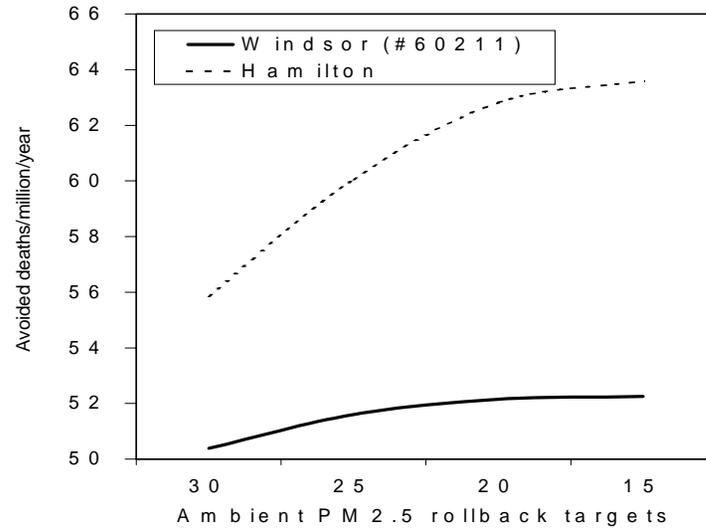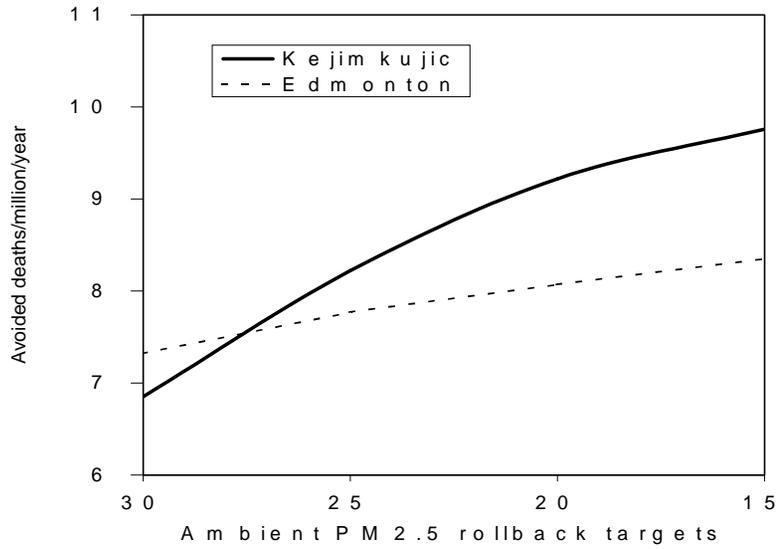
Figure 7c. Number of avoided deaths resulting from decreased PM$_{2.5}$ concentrations
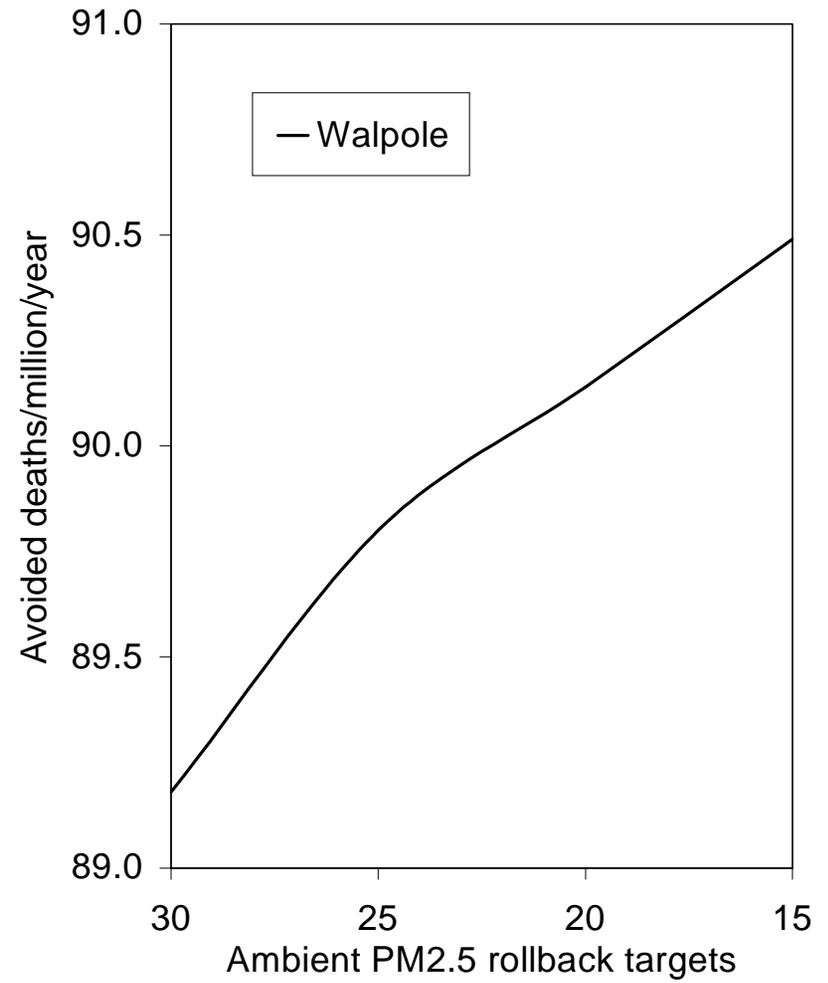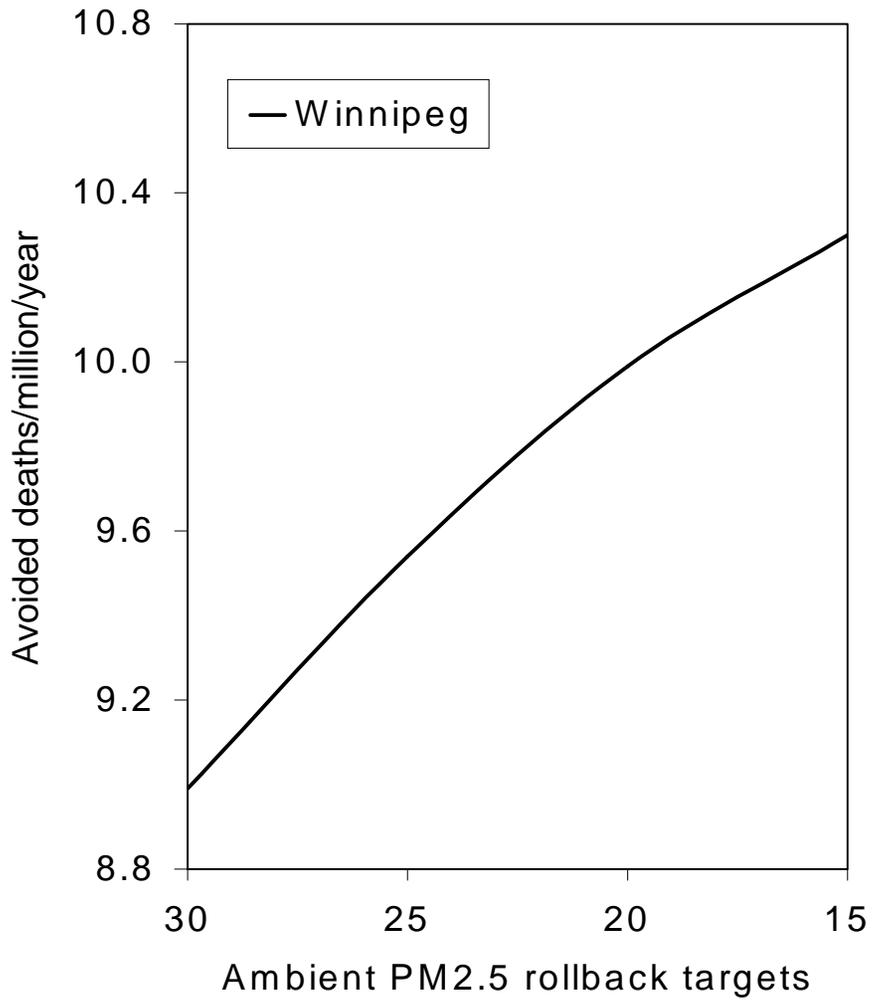
Figure 7d. Number of avoided incidences resulting from decreased PM$_{2.5}$ concentrations
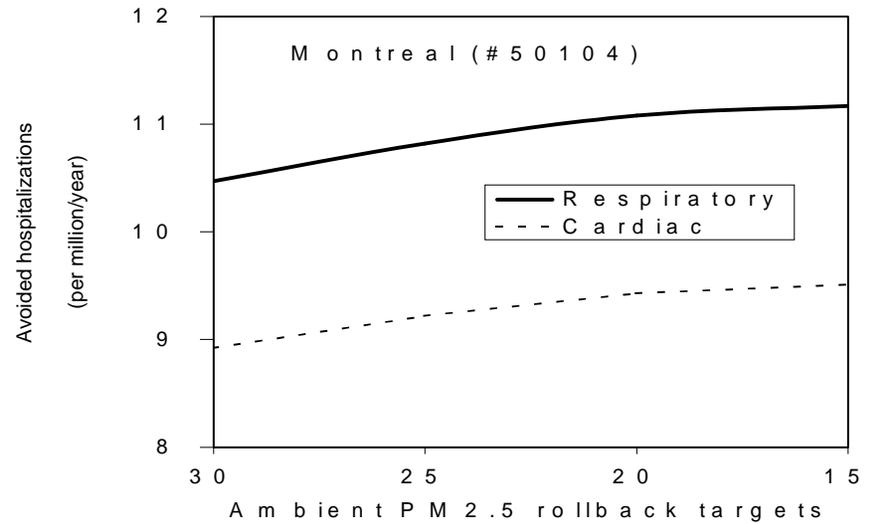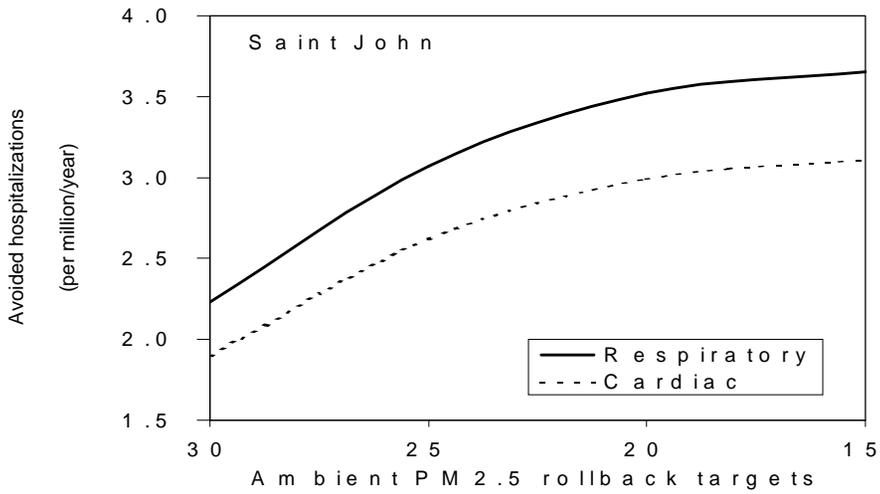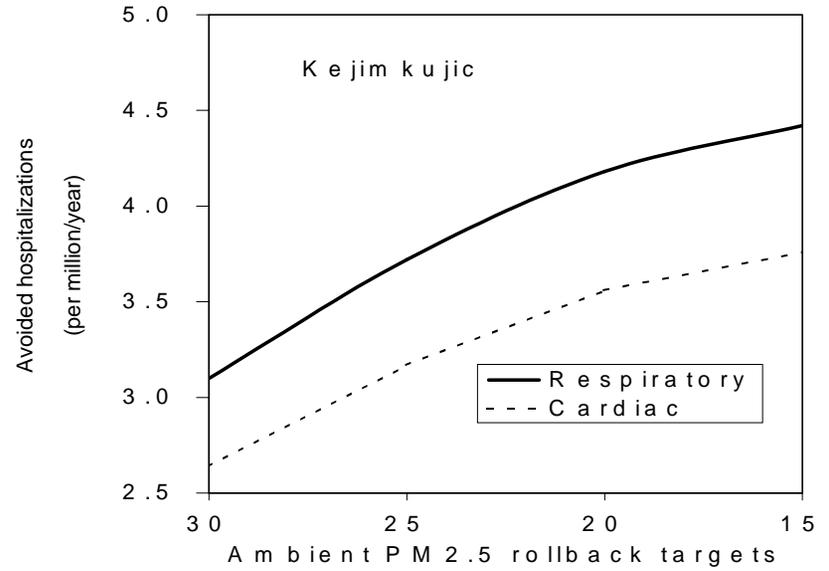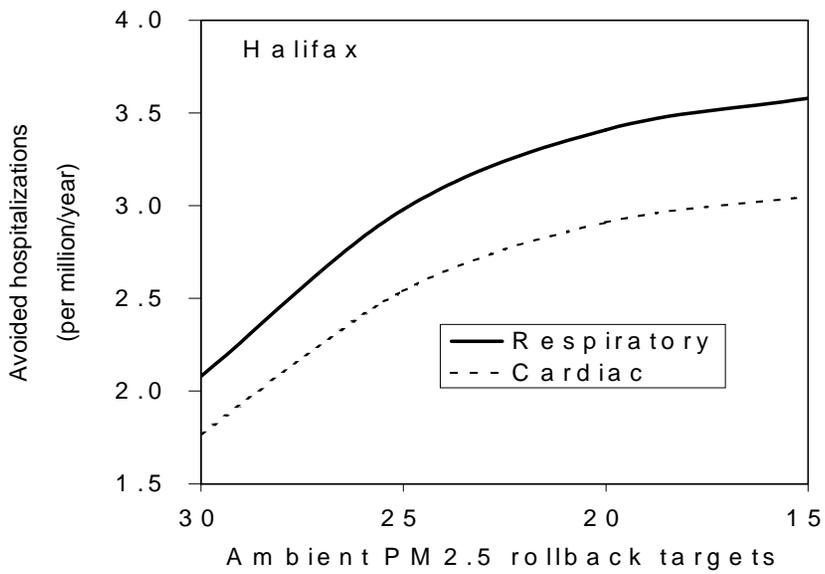
Figure 7e. Number of avoided incidences resulting from decreased PM$_{2.5}$ concentrations
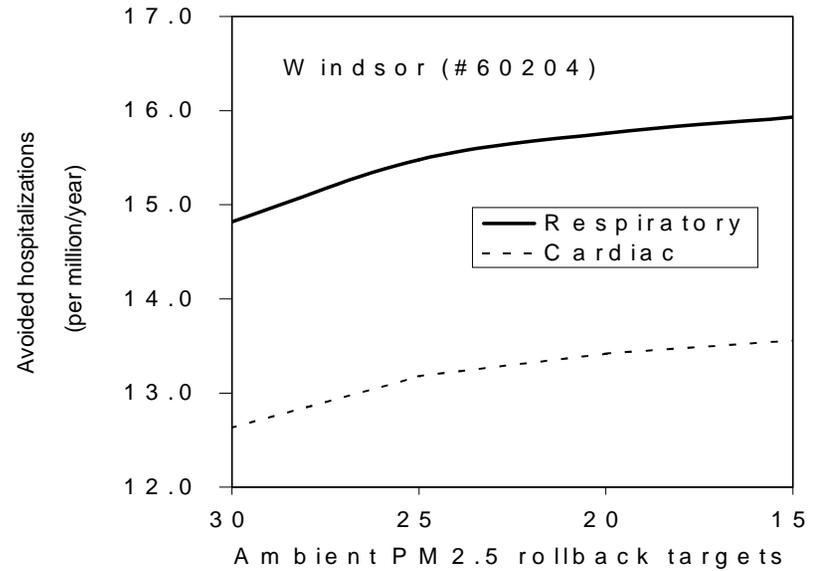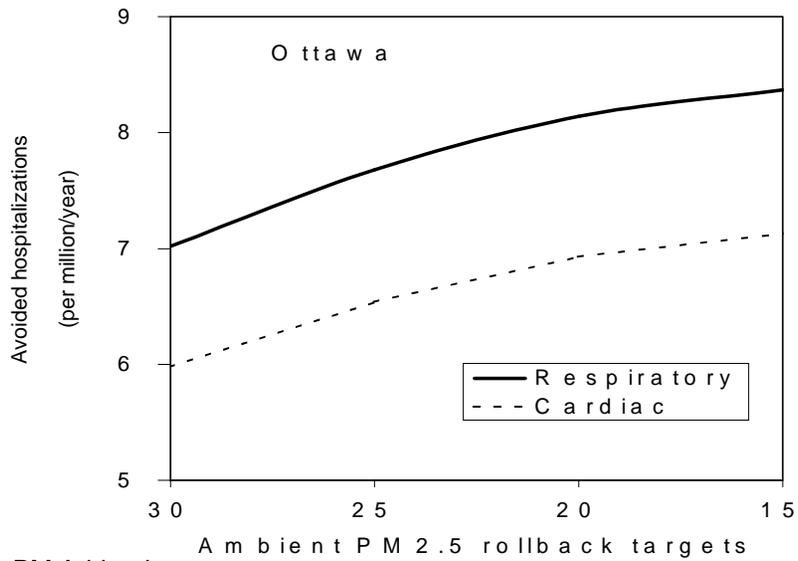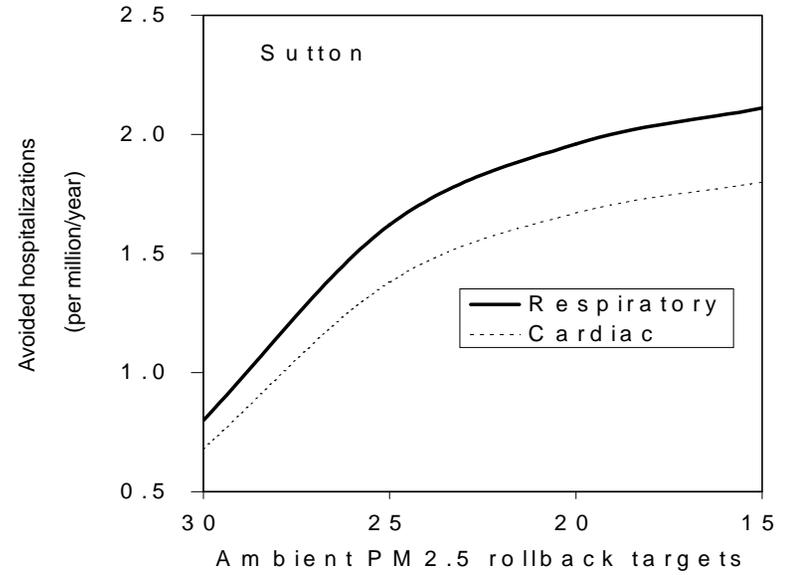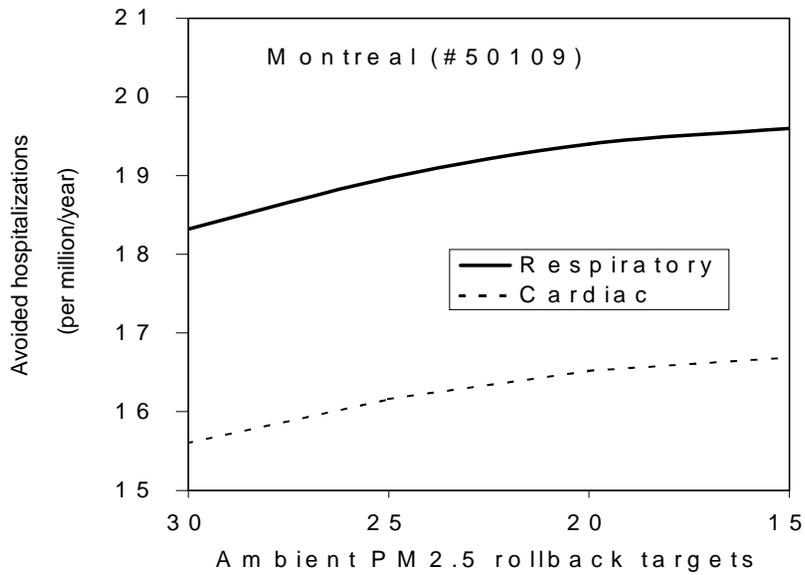
Figure 7f. Number of avoided incidences resulting from decreased PM$_{2.5}$ concentrations
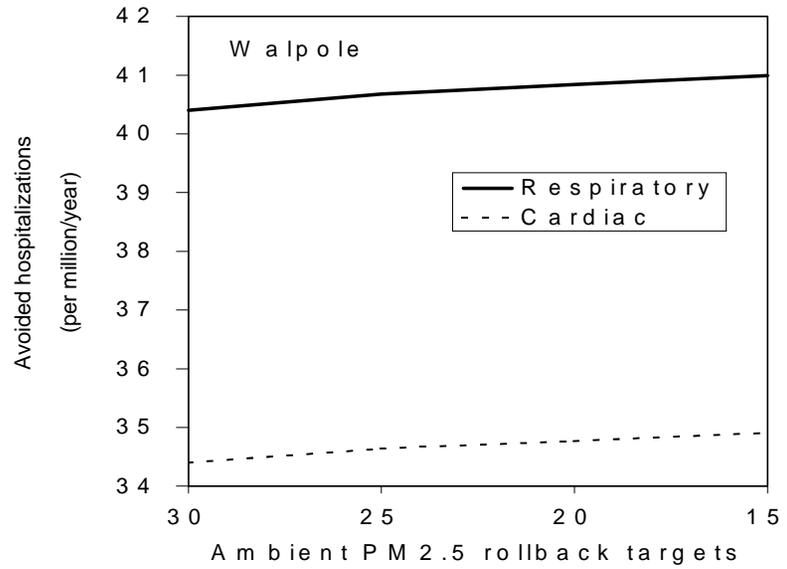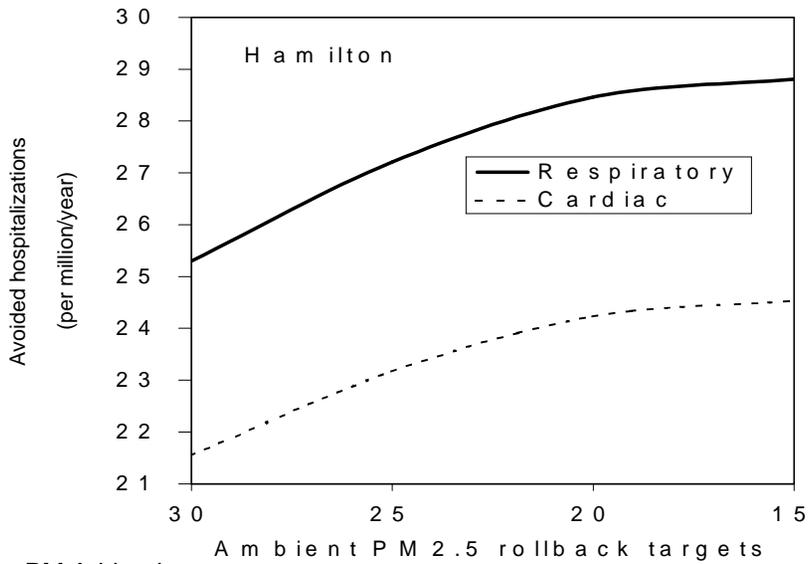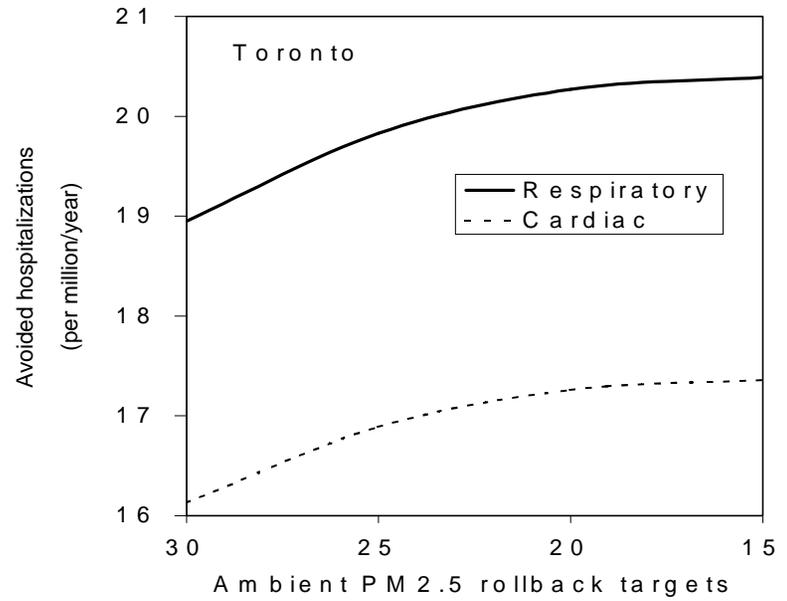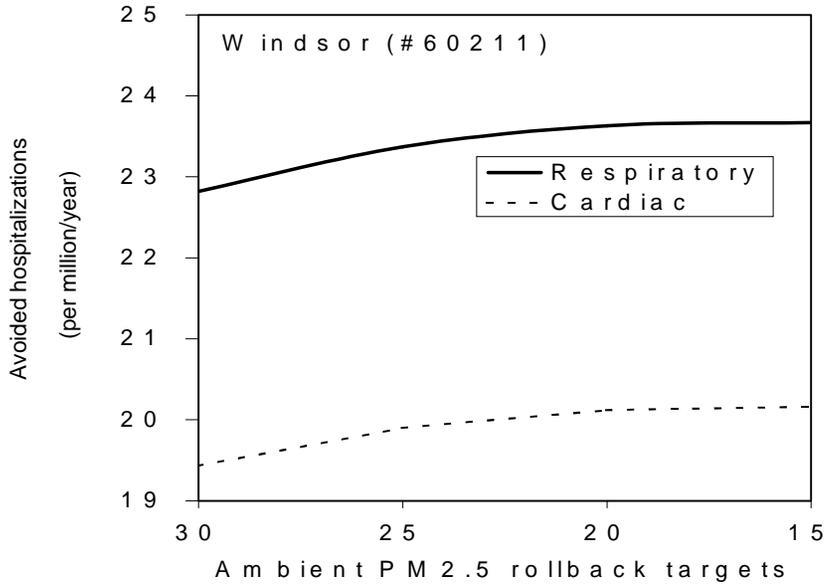
Figure 7g. Number of avoided incidences resulting from decreased PM$_{2.5}$ concentrations
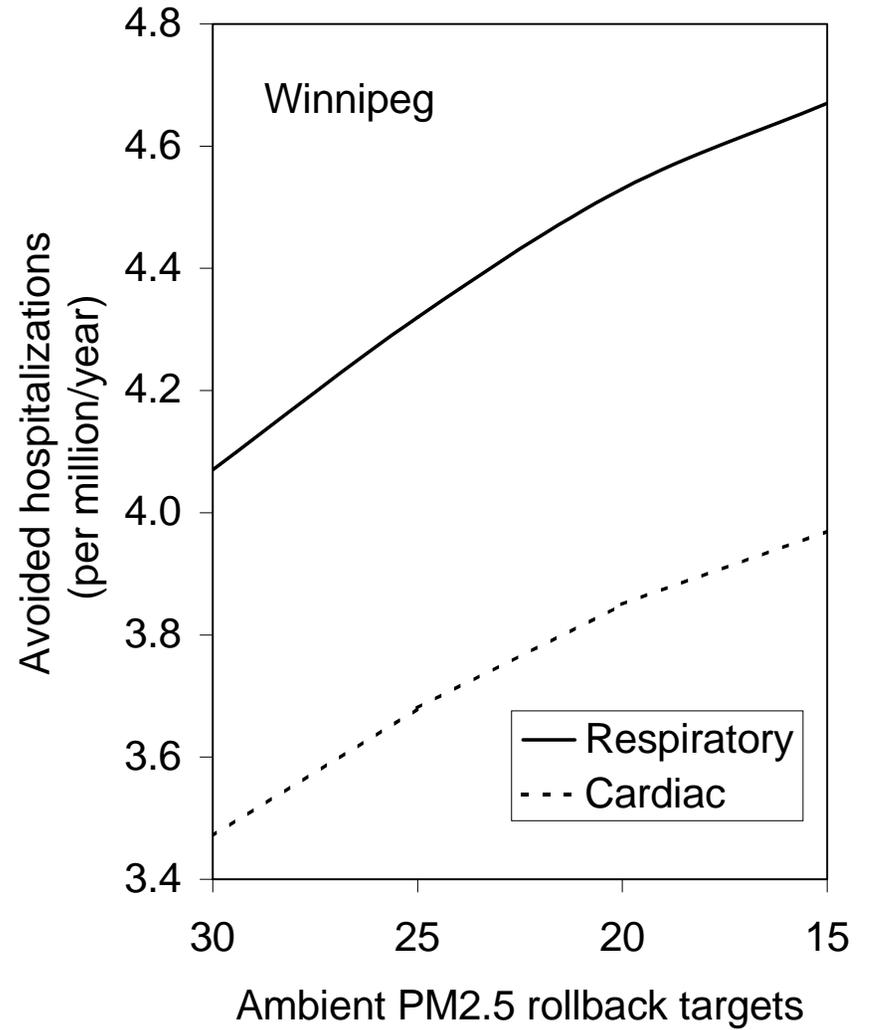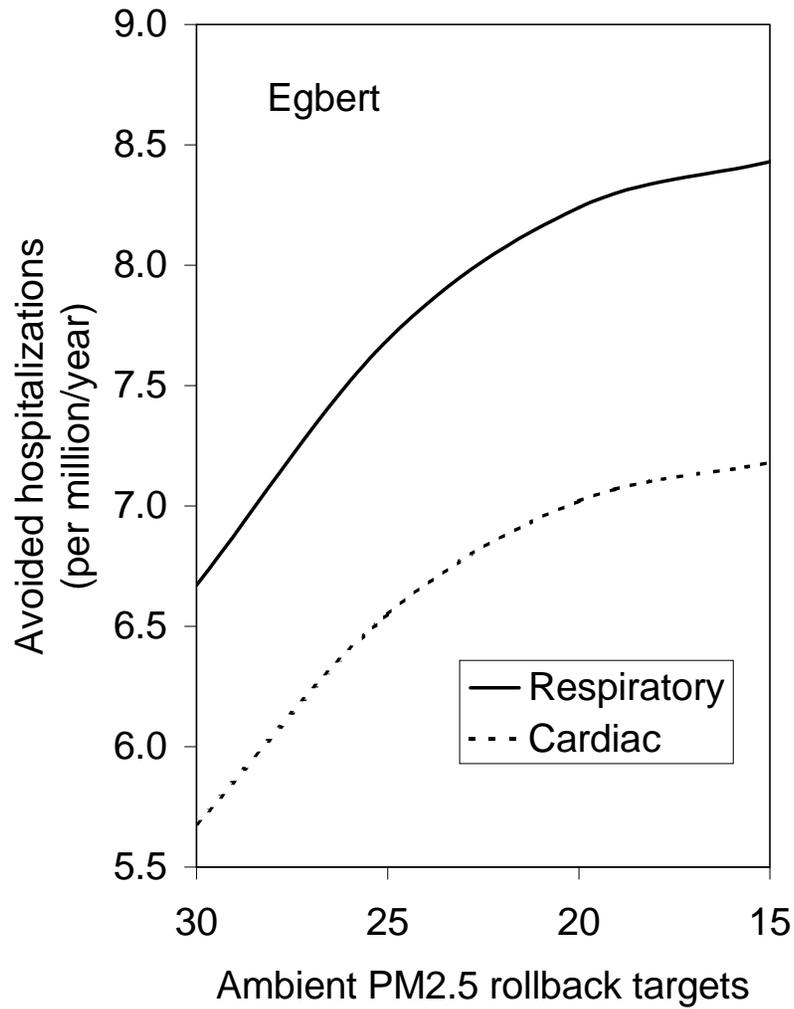
Figure 7h. Number of avoided incidences resulting from decreased PM$_{2.5}$ concentrations
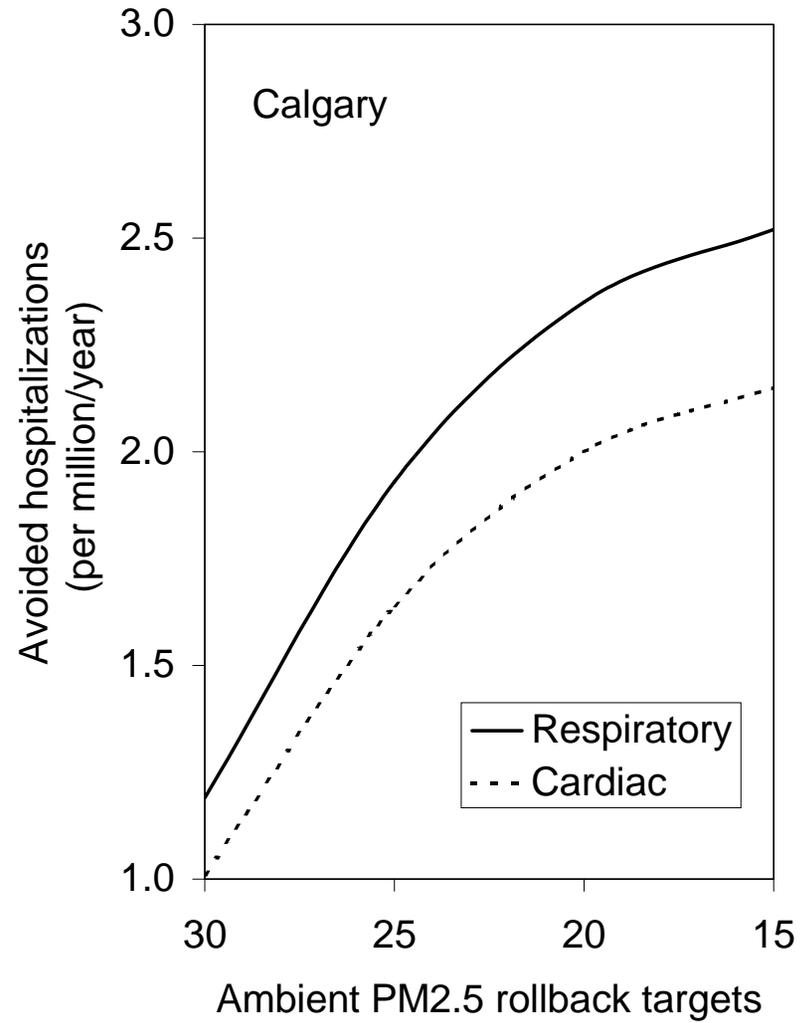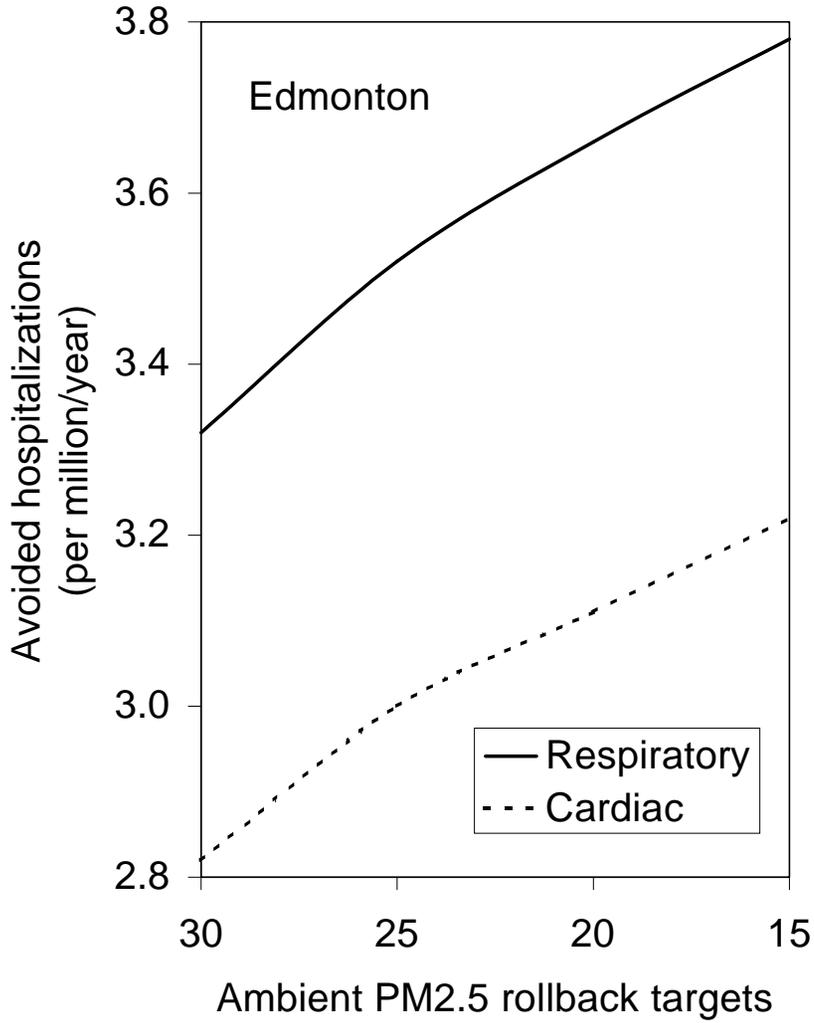
Figure 7i. Number of avoided incidences resulting from decreased $PM_{2.5}$ concentrations
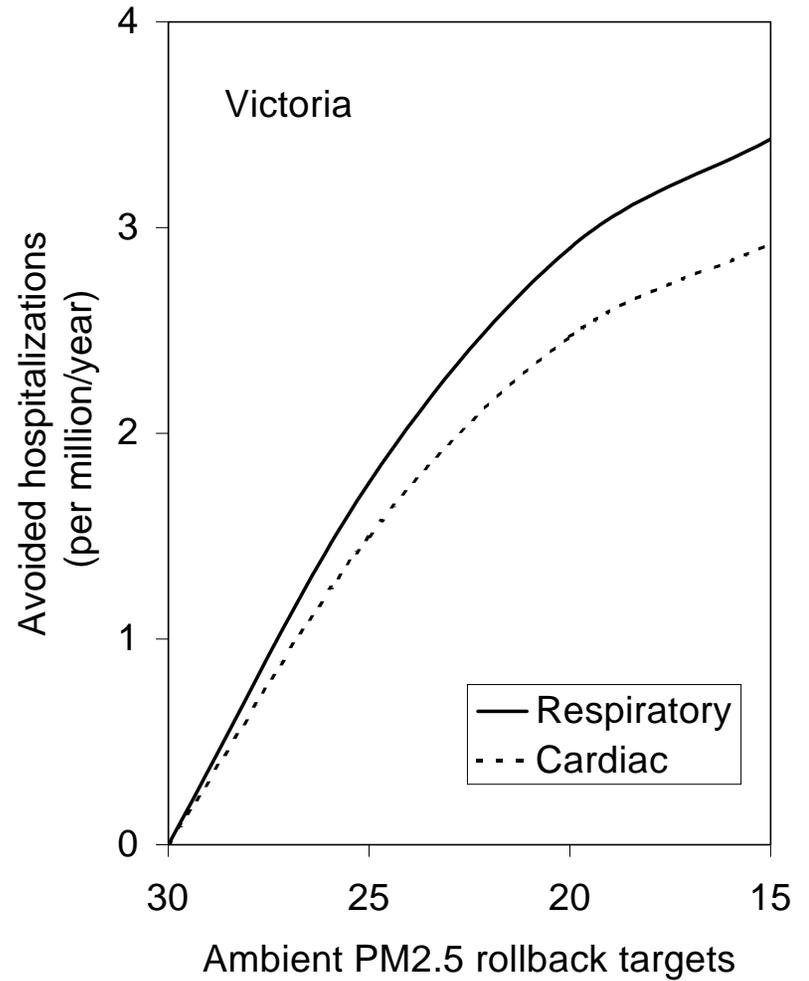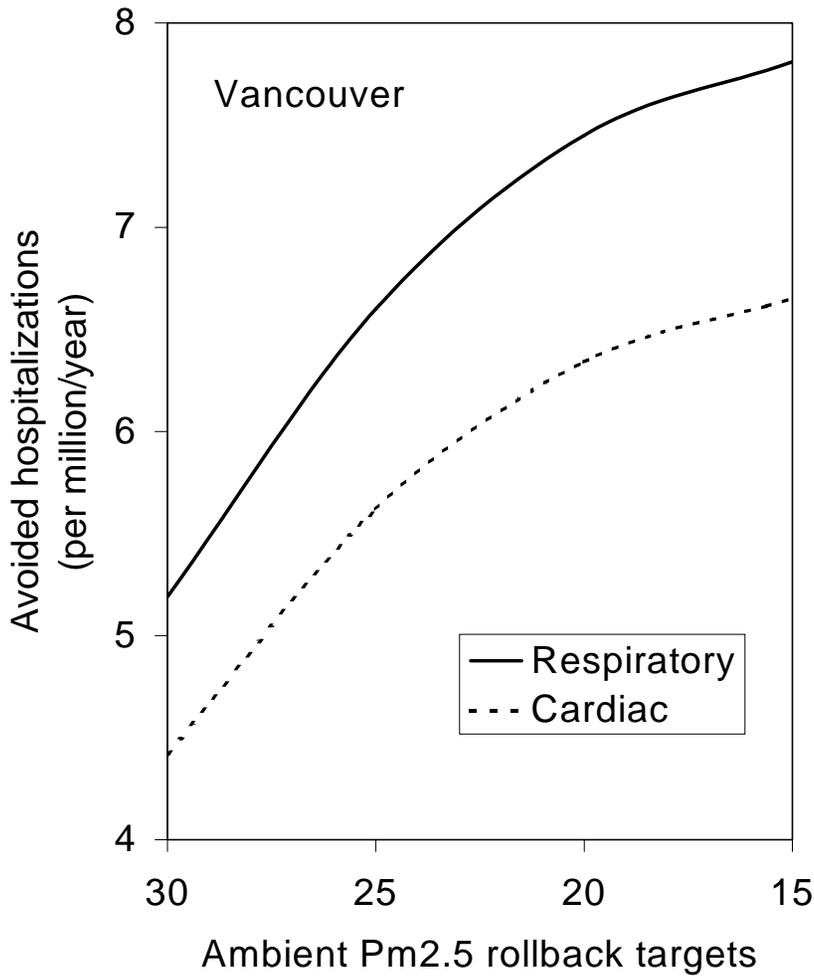
5.3 BENEFIT ESTIMATES (using the Air Quality Valuation Model)

The Air Quality Valuation Model (AQVM) is a spreadsheet model that comprises four components: concentration-response co-efficients, economic valuation, uncertainty analysis, and generation of quantitative overall benefits estimates. This model was used in parallel to the incremental risk analysis to give some additional insights to:

- the absolute number of avoided impacts that could be gained by simulating attainment of several PM_{10} target for endpoints beyond mortality and hospital admissions,

- using a different set of relative risk estimates,

- the range of uncertainty by using low, central and high estimates for relative risk,

- projecting benefits over time for the period 2000 to 2020, and

- estimating benefits for other impact levels including background concentrations.

No economic information is presented as economic valuation was considered outside the range of this exercise.

The Air Quality Valuation Model (AQVM) is a spreadsheet model designed to quantitatively estimate the human health and environmental benefits resulting from improvements in ambient air quality in Canada. The AQVM uses a damage function approach. The first step is to specify change in ambient air quality, followed by application of concentration-response functions to compute changes in physical impacts. Economic values may be applied to the physical impacts, and benefits are aggregated benefits across all affected individuals and all relevant time periods. Each stage of this process introduces uncertainty and the total uncertainty, therefore, increases as one progresses to the final estimates.

While this analysis is similar to the incremental risk analysis, it is for PM_{10} only. (Editor's Note: At time of analysis the model was not capable of estimating $PM_{2.5}$ benefits). In addition to mortality and hospitalization benefit estimates, benefits associated with reduced emergency room visits, asthma symptom days, restricted activity days, acute respiratory symptoms, child bronchitis cases and chronic bronchitis are also provided. This is not an exhaustive list of the endpoints, rather it includes the endpoints for which robust concentration-response functions have been identified in the literature. As well, the analysis is population adjusted such that it provides a national picture of the major metropolitan areas. Note that it is still not representative of the entire Canadian population, since not all urban centres are included in this analysis.

The concentration-response functions used in this analysis are presented in Table 5. The estimated benefits across the fourteen cities, of achieving the three AQO options are summarized in Tables 6 and 7. Both tables report the annual mean number of avoided events as central estimates. Low and high estimates of the annual mean number of avoided from the uncertainty analysis are shown in Table 7.

Table 5. Concentration - Response Co-efficients utilized in AQVM (AQVM Methodology Report, June, 1996)

Health Effect Category	Concentration – Response Co-efficients* (weights)
Daily mortality risk factors given a $1\mu g/m^3$ change in daily PM_{10} concentration Sources: (see section 5.3.3 and Tables 4 and 5 of AQVM Methodology Report)	L $\quad 0.9 \times 10^{-8}$ (40%) C $\quad 1.8 \times 10^{-8}$ (50%) H $\quad 6.6 \times 10^{-8}$ (10%)
Chronic bronchitis (CB) annual risk factors given a $1\mu g/m^3$ change in annual PM_{10} concentration. Source: Abbey et al. (1993)	For population 25 years and over: L $\quad 3.0 \times 10^{-5}$ (25%) C $\quad 6.1 \times 10^{-5}$ (50%) H $\quad 9.3 \times 10^{-5}$ (25%)
Respiratory hospital admissions (RHAs) daily risk factors given a $1\mu g/m^3$ change in daily PM_{10} concentration Source: Burnett et al. (1995), Pope (1991)	L $\quad 0.64 \times 10^{-8}$ (33%) C $\quad 0.78 \times 10^{-8}$ (50%) H $\quad 3.26 \times 10^{-8}$ (17%)
Cardiac hospital admissions (CHAs) daily risk factors given a $1\mu g/m^3$ change in daily PM_{10} concentration Source: Burnett et al. (1995)	L $\quad 5.0 \times 10^{-9}$ (25%) C $\quad 6.6 \times 10^{-9}$ (50%) H $\quad 8.2 \times 10^{-9}$ (25%)
Emergency room visits (ERVs) daily risk factors given a $1\mu g/m^3$ change in daily PM_{10} concentration Source: Samet et al. (1981)	L $\quad 3.2 \times 10^{-7}$ (25%) C $\quad 6.5 \times 10^{-7}$ (50%) H $\quad 9.7 \times 10^{-7}$ (25%)
Asthma sympton days (ASDs) daily risk factors given a $1\mu g/m^3$ change in daily PM_{10} concentration Source: Whittemore and Korn (1980), Ostro et al. (1991)	For population with asthma (4.7%): L $\quad 0.9 \times 10^{-4}$ (33%) C $\quad 1.6 \times 10^{-4}$ (50%) H $\quad 5.4 \times 10^{-4}$ (17%)
Restricted activity days (RADs) daily risk factors given a $1\mu g/m^3$ change in daily PM_{10} concentration Source: Ostro (1987), Ostro and Rothschild (1989)	For population aged 18 years and over: L $\quad 0.8 \times 10^{-4}$ (33.3%) C $\quad 1.6 \times 10^{-4}$ (33.4%) H $\quad 2.5 \times 10^{-4}$ (33.3%)
Days with acute respiratory symptoms (ARSs) daily risk factors given a $1\mu g/m^3$ change in daily PM_{10} concentration Source: Krupnick et al. (1990)	L $\quad 2.2 \times 10^{-4}$ (25%) C $\quad 4.6 \times 10^{-4}$ (50%) H $\quad 7.0 \times 10^{-4}$ (25%)
Children with bronchitis (B) annual risk factors given a $1 \mu g/m^3$ change in annual average PM_{10} concentration. Source: Dockery et al. (1989)	For population under age 18: L $\quad 0.8 \times 10^{-3}$ (25%) C $\quad 1.6 \times 10^{-3}$ (50%) H $\quad 2.4 \times 10^{-3}$ (25%)

* L, C and H refer to low, central and high estimates used in uncertainty analysis, according to the percentage weights given.

The PM_{10} targets that were used in this analysis were 24-hour average concentration limits of 25 $\mu g/m^3$, 35 $\mu g/m^3$ and 40 $\mu g/m^3$. It was assumed that these targets would be achieved by the year 2000 and benefits were estimated for the 2000-2020 period. The benefit estimates are based upon the normalized distribution of daily PM_{10} concentrations for the period 1992-1994, for fourteen cities. The proportional rollback method for predicting concentration distributions was used as in the Incremental Risk Analysis, for each target level (discussed in Appendix D).

The analysis took two approaches to estimating avoided impacts to provide perspective on our understanding of PM_{10} impacts in the context of the Reference Level being a LOAEL (i.e.: there is no threshold for effects) and in context of background concentrations. These issues are

addressed by summing avoided impacts from the Reference Level (25 $\mu g/m^3$) presented in Table 6; and in summing avoided impacts from an estimated average annual background (5 $\mu g/m^3$) presented in Table 7.   Background estimates are discussed in Appendix E. These two approaches are intended to bracket the anticipated range of benefits which may result from achieving lower ambient PM_{10} concentrations. The first approach provides a lower bound, in that summing benefits from the LOAEL is a conservative estimate of the future risks. The second approach provides an upper bound as it sums benefits from an estimated background concentration, which assumes that all anthropogenic sources of PM_{10} and $PM_{2.5}$ have been eliminated. However, the available data limits quantification of the relationship at lower levels. Thus, it is somewhat speculative, to provide numerical estimates. However, the absence of identified thresholds for the effects of particulate matter renders the second analysis useful for identifying the upper end of the range for potential impacts.

Specifically the setup for each approach is noted below. A theoretical graphical representation of the two approaches is included in Figure 8. Both approaches use the proportional linear algorithm (rollback) to create the new concentration distributions. If data were available for more than one monitoring station for a given city, the average of the change in the annual mean concentration was used.

> 1) SUM>25: Sums avoided impacts from the Reference Level (25$\mu g/m^3$). The rollback was applied with the restriction that individual daily measurements could not be reduced below 25 $\mu g/m^3$, the LOAEL. If the rollback reduced the 24 hour average concentration to <25 $\mu g/m^3$, then the level was set to 25 $\mu g/m^3$. This removes a substantial proportion of the benefits which would have been reduced to below 25 $\mu g/m^3$ by the rollback procedure, ie: which would have been attributable to reductions to exposures to daily concentrations in the range 5 – 25 $\mu g/m^3$. Results presented in Table 6.

> 2) SUM>5: Sums avoided impacts from average annual background (5 $\mu g/m^3$). All concentrations were reduced according to the proportional relationship to the site maximum with no consideration of a lower limit. The annual mean after rollback was not reduced below 5 $\mu g/m^3$, the estimated annual background. Results presented in Table 7.

Figure 8. Graphical representation of the current and predicted ambient PM$_{10}$ concentrations for the AQVM benefit analysis SUM>25 and SUM>5. The hatched area above the solid SUM >X line indicates the exposure (μg/m^3 × days) for which avoided impacts (benefits) are estimated.
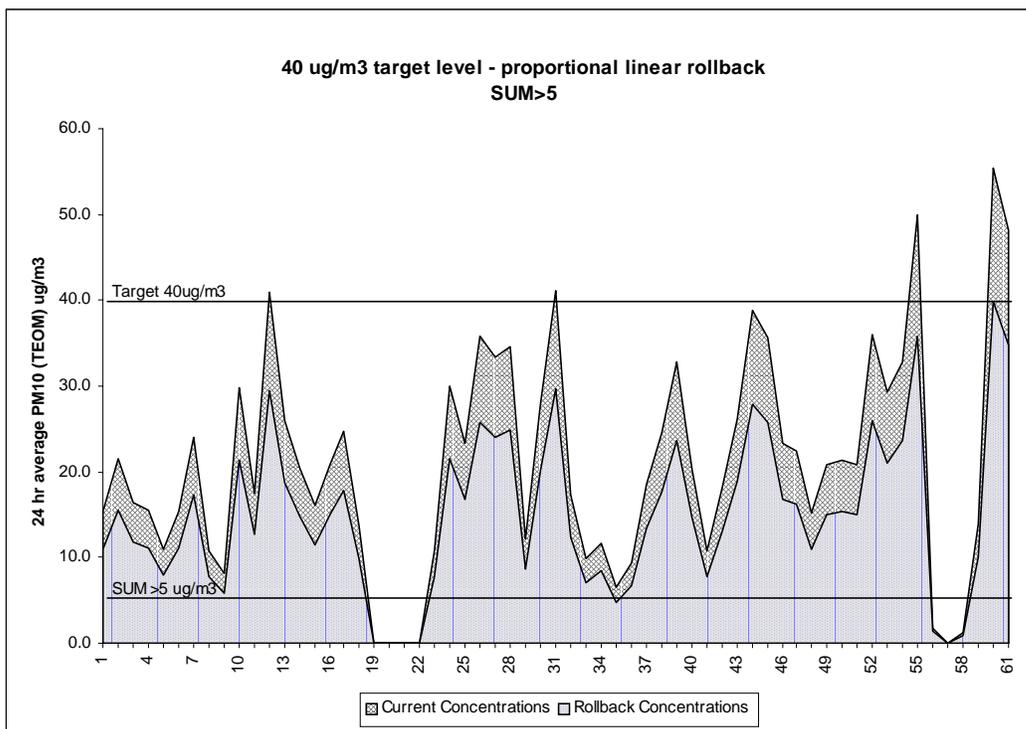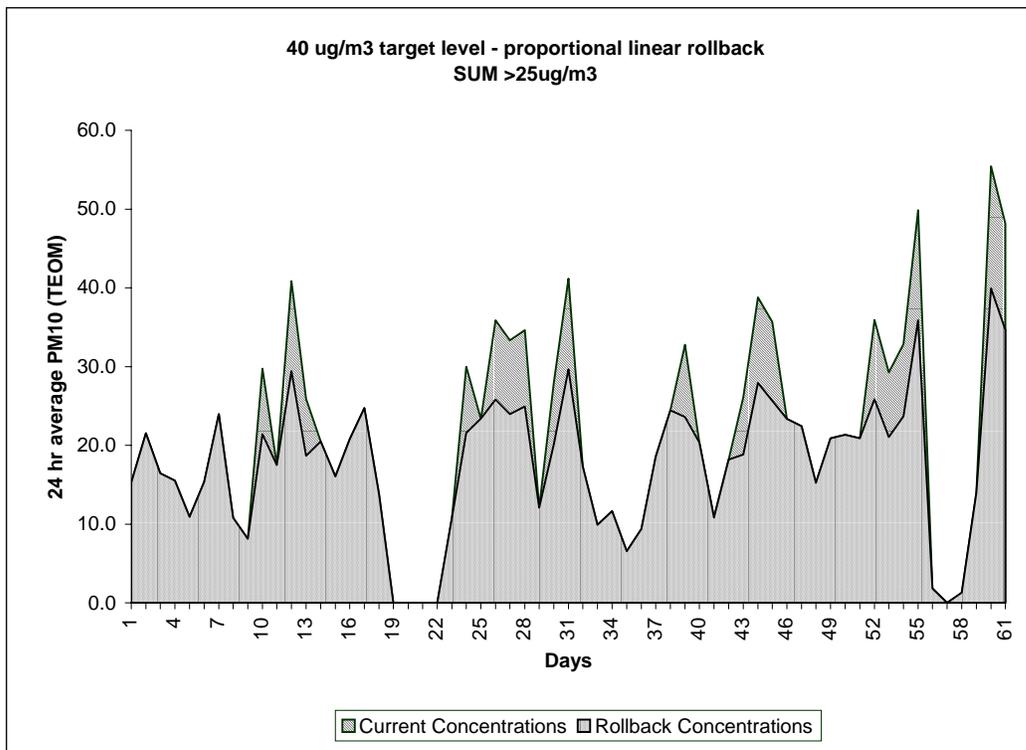
Table 6. Health benefits for the year 2020, summed >25 $\mu g/m^3$ for three 24-hour PM_{10} targets achieved by 200, for 14 cities.

	Annual Mean Number of Avoided Events For PM_{10} Targets		
	40 $\mu g/m^3$	35 $\mu g/m^3$	25 $\mu g/m^3$
Mortality	420	435	448
Chronic bronchitis cases	2,636	2,731	2,811
Respiratory hospital admissions	182	188	194
Cardiac hospital admissions	154	159	164
Emergency room visits	14,815	15,354	15,805
Asthma symptom days	175,281	181,654	186,999
Restricted activity days	2,676,008	2,772,582	2,853,723
Acute respiratory symptom days	7,900,754	8,188,758	8,430,174
Child bronchitis cases	24,881	25,806	26,578

Table 7. Health benefits for the year 2020, summed >5 $\mu g/m^3$ for three 24-hour PM_{10} targets achieved by 2000, for 14 cities.

	Annual Mean Number of Avoided Events (low / high) For PM_{10} Targets		
	40 $\mu g/m^3$	35 $\mu g/m^3$	25 $\mu g/m^3$
Mortality	1,081 (546 / 4,000)	1,224 (612 / 4,486)	1,490 (745 / 5463)
Chronic bronchitis cases	5,814 (3,351 / 10,389)	7,842 (3,759 / 11,651)	9,301 (4574 / 141801)
Respiratory hospital admissions	473 (388 / 1,976)	530 (435 / 2,217)	646 (530 / 2,699)
Cardiac hospital admissions	100 (303 / 497)	449 (340 / 558)	546 (414 / 679)
Emergency room visits	38,526 (18,966 / 57,492)	43,217 (21,276 / 64,493)	52,613 (25,902 / 78,515)
Asthma symptom days	455,810 (256,393 / 1,538,359)	511,314 (287,614 / 1,725,684)	622,484 (350,147 / 2,100,882)
Restricted activity days	6,922,957 (3,461,479 / 10,817,120)	7,763,458 (3,881,734 / 12,130,418)	9,447,037 (4,723,519 / 14,760,995)
Acute respiratory symptom days	20,583,293 (9,844,184 / 31,322,402)	23,092,347 (11,044,166 / 35,140,528)	28,117,670 (13,447,581 / 41,787,759)
Child bronchitis cases	65,736 (32,868 / 98,804)	73,813 (39,906 / 110,919)	89,987 (44,994 / 134,981)

The benefits of achieving three targets were estimated only for 14 cities with PM_{10} monitors, and based upon the normalized ambient data which underestimates current 24-hour maximum concentrations. Thus, the total benefit estimates from either approach should be interpreted as a conservative underestimate of the expected benefits for the entire population of Canada.

As expected and already demonstrated by the incremental risk analysis, the total benefits are greater for the more protective PM_{10} targets. A similar increase in benefits has been shown by the incremental risk analysis for fine particles; as the fine particle concentration limit becomes lower, the annual mean number of avoided impacts increases. The endpoints responsible for the greatest portion of the total PM_{10} benefits are mortality, chronic bronchitis and reduced activity days.

5.4 STAKEHOLDER INPUT

Given the difficulty in weighing risks and benefits in light of the scientific uncertainties, and the need to accommodate considerations of economics, technological feasibility and social factors, in setting air quality objectives, a stakeholder consultation was held in December 1997. The purpose of this consultation was to examine and discuss the new framework for establishing NAAQOs, and consult on proposed NAAQOs for Particulate Matter (PM_{10} and $PM_{2.5}$) as developed under the framework.

Since that time Canadian Environment Ministers (with the exception of Quebec) signed the Canada-Wide Accord on Environmental Harmonization and its sub-agreement on Canada-Wide Standards (CWSs). Recognizing that both NAAQOs and CWS have a role to play in the management of air quality, federal, provincial, and territorial health and environment departments have integrated the NAAQO and CWS processes. Air pollutants which have been identified by governments as needing to be managed will be targeted for either CWS or NAAQO development, not both.

In January 1998, Environment Ministers identified PM and ozone as priorities for Canada-Wide Standards. CWSs for these substances are scheduled to be presented to Ministers in the fall of 1999. By this time, the Federal – Provincial Working Group on Air Quality Objectives and Guidelines was already conducting science assessments to revise the existing objective for ozone and to develop a new objective for particulate matter. Federal, provincial, and territorial health and environment departments have agreed that NAAQOs for PM and ozone will no longer be developed. Rather, the existing science assessments will form the Risk Assessment reports for the development of CWSs for PM and ozone. These reports will be supplemented with risk and benefit analyses which will be developed by the WGAQOG. In addition, the WGAQOG will recommend ranges of levels for PM10, PM2.5 and ozone which will reduce risks.

It is still relevant to recognize the stakeholder input from the December 1997 in the context of the risk and benefit analysis, and subsequent conclusions, provided in this document. The comments received from stakeholders have been consolidated in eight themes, with WGAQOG responses included.

1) **The need for an open and transparent NAAQO development process**
Agree with keeping it open and transparent. The December workshop was the first formal opportunity for stakeholder input to the process and stakeholder desire for continued involvement will be reflected in the 2nd edition of the Protocol which will be circulated for stakeholder comment. In general, during the scientific assessment, the Working Group will consult with stakeholders at two points:
   1) draft documents will be circulated for comment, and where appropriate, workshops will be held to discuss the assessment documents more fully, and
   2) stakeholders will be asked to nominate peer reviewers for the assessment documents.
    During NAAQO development, stakeholder consultation will occur throughout the process as described in the revised Protocol.

2) **Scientific Uncertainties**
We recognize that there has been significant, open discussion on this issue in a variety of fora, and that the SAD has been rigorously peer reviewed by external experts. Scientific knowledge is continually evolving, and in time, will address many of the current uncertainties. We will proceed with publication of the Science Assessment document.

3) **Workability of NAAQOs**
NAAQOs are intended to be long term goals (for areas with poor air quality), not necessarily immediately achievable. Their goal is protection of receptors. They are intended to provide an impetus for airshed management activities, with the understanding that provinces and territories may adopt NAAQOs as they see fit. Therefore, if jurisdictions choose to develop implementation plans, it will be during this phase that recognition of regional issues, such as prevailing ambient concentrations, the role of long range transport, and sources of PM within the region, will occur. The process of developing CWSs for PM will further address achievability and implementation issues.

4) **Public Expectations**
The existence of NAAQOs does prompt public expectation that action will be taken to achieve them. WGAQOG recognizes the need for a communication strategy that clarifies that NAAQOs are long term goals designed to protect the general public and environment and offers some advice as to expected uses of AQOs. Such a communication strategy is in progress.

5) **Role of S/E/T**
The role, weighting, and degree of analysis accorded to social, economic and technological factors in the selection of the AQOs will be further developed in the Protocol in consultation with stakeholders. It is intended that such analyses will be at a national level. The information, where it exists, will provide guidance to the selection of the receptor-based AQOs, which, under CEPA, ought to be achievable, but within an unspecified timeframe.

6) **Setting of PM NAAQOs is Premature**
We do not agree t hat the setting of receptor based NAAQOs [air quality criteria] for PM is premature. There is good health based evidence that Canadians are currently experiencing adverse effects at ambient PM levels commonly encountered across Canada. Current NAAQOs do not reflect the state of scientific understanding on PM, as they target large particles (Total Suspended Particulates) rather than small particles (PM_{10} and $PM_{2.5}$). Sufficient evidence exists to link particles with adverse health effects; the lack of scientific certainty should not preclude actions to address the problem. Funds are being sought to

pursue research that will address the uncertainties. Concerns about the limited knowledge on regional sources of PM will be raised during the development of jurisdictional implementation plans.

7) Science Advisory Panels
While Science Advisory Panels (SAPs) tend to represent multi-stakeholder interests, the use of SAPs makes it much more difficult to keep science separate from policy. Therefore, SAPs will not be convened for NAAQO development. Instead, other mechanisms for stakeholder involvement will be elaborated upon in the second edition of the Protocol.

8) Harmonization with US NAAQS and Influence of Long Range Transport
Canadian NAAQOs (objectives) are not the same as U.S. NAAQSs (standards) and therefore a direct comparison of the two is inappropriate. There are initiatives underway to address PM transboundary impacts from the U.S. For example, Canada and the U.S. have signed a work plan for the development of a Joint Plan of Action on transboundary PM, under the Canada/U.S. Air Quality Accord. Canada and the U.S. are also both signatories to UN ECE Protocols, that commit them to reducing NO_x and VOCs, precursors of fine particulate matter. Consideration of the long range transport of PM will form part of the discussions around development of CWSs for PM_{10} and $PM_{2.5}$. The issue of Canadian competitiveness (a.k.a. the level playing field concern), is legitimate, and we concur with stakeholder concerns, although it is recognizably a difficult issue to address. The issue should be raised in the discussions around CWSs for PM.

6.   IDENTIFICATION of PROTECTIVE RANGES

The purpose of identifying a concentration range, which if achieved would afford substantial reduction in the risks to human health and the environment, is to provide guidance to air quality managers in developing ambient standards and management strategies. The review of human health risks and benefits has illustrated that there is a range which could be considered as suitable for achieving risk reductions. These ranges are for PM_{10} - 15 to 40 $\mu g/m^3$, and for $PM_{2.5}$ - 10 to 25 $\mu g/m^3$. Upon further reflection and examination of this range using an Incremental Risk Analysis and reviewing the absolute benefits which may be achieved at various PM_{10} target levels several points became obvious.

- The Reference Levels provide the best quantification of levels above which effects may be demonstrated, however it is recognized that effects are occurring at lower levels.

- Estimates of background particulate matter concentrations provide a lower limit on what could be achieved, in terms of reducing risk.

- An improvement from the status quo provides an upper limit upon the range. This is based upon the serious health impacts associated with current PM concentrations. The PM_{10} and $PM_{2.5}$ levels calculated from current NAAQO for Total Suspended Particulates are 50 and 25 $\mu g/m^3$, respectively. Therefore, the high end of the various approaches, the median of the 95th percentile, represents a minimal reduction in risk from current levels (PM_{10} of 40$\mu g/m^3$ and $PM_{2.5}$ of 25$\mu g/m^3$).

- The Incremental Risk Analysis provides a means of estimating the health benefits relative to the current air quality levels. The health benefits increase linearly with a reduction of PM on any given day. Applying the reduction to the data set for different Canadian cities indicated a diminishing health benefit gain (an inflexion point) within a certain narrow range above the LOAEL. The incremental analysis indicates there is an ambient level down to which there is substantial accumulation of benefits in avoided mortality and hospital admissions. At concentrations below this, there appears to be a reduced accumulation of benefits. This analysis is based on the assumption that the distribution curve for ambient levels will maintain a log-normal shape after control measures are implemented. This approach does not identify the same inflexion point for every city. Consequently, the inflexion points identify an even narrower range which could be considered suitable for achieving risk reductions: PM_{10} of 35 to 40 $\mu g/m^3$ and for $PM_{2.5}$ of 20 to 25 $\mu g/m^3$.

- The benefit estimates from AQVM support the Incremental Risk Analysis conclusions and indicate that the conclusions drawn for mortality and hospital admissions are likely to be to be true of the other health endpoints.

The WGAQOG accepts the inflexion point of the Incremental Risk Analysis as the basis for identifying a concentration range, which if achieved would afford substantial reduction in the risks to human health and the environment. The inflexion point provides a qualitative basis for balancing the reduction in benefits at lower ambient concentrations against the anticipated costs of implementing the control measures to achieve them. It is recognized that adverse impacts are experienced at and below the identified Reference Levels, however it is expected that the reductions in risk by moving from the range identified by the inflexion points, to the Reference Levels would not be significant relative to the anticipated effort of achieving such reductions. The ranges which if achieved, that would afford substantial reduction in the risks to human health and the environment are:

$$PM_{10} \quad 35 - 40 \ \mu g/m^3$$
$$PM_{2.5} \quad 20 - 25 \ \mu g/m^3.$$

7. RECOMMENDATIONS

The federal – provincial Working Group on Air Quality Objectives and Guidelines has developed several recommendations which it is suggested be considered in the development of Canada Wide Standards for PM_{10} and $PM_{2.5}$, and subsequent management strategies.

1. Ambient Monitoring: Current monitoring technologies to measure particulate matter on a mass basis have detection limits that are far lower than the range presented in Chapter 6 for both PM_{10} and $PM_{2.5}$. Instruments are also available for monitoring PM_{10}, $PM_{10-2.5}$, and $PM_{2.5}$ over daily and/or continuous time frames, and providing speciation information. However, the current ambient monitoring network is *spatially limited* in many areas of the country. PM levels that are representative of areas of high population density are poorly characterized. PM_{10} monitoring is spatially limited in some regions of the country while $PM_{2.5}$ coverage is

very limited spatially. In addition, the highest concentrations likely to occur in each of the regions are unknown due to limited network coverage and monitoring temporal resolution. The current one-in-six day *sampling schedule* leads to underestimates of peak concentrations by 20% to 30%, which limits the ability to accurately estimate health impacts on high pollution days (see discussion in Appendix B). There is a need to consistently define and achieve stable, reproducible measurements, using standardized equipment, monitoring schedules, instrument siting criteria, and QA/QC procedures.

2. The body of epidemiological evidence that allows direct comparison of the $PM_{2.5}$ and PM_{10} metrics in association with adverse health outcomes indicates greater concern should be accorded to $PM_{2.5}$, though significant concern remains for the coarse fraction of PM_{10}. It is also recommended that ambient standards developed for particulate matter maintain a realistic ratio between PM_{10} and $PM_{2.5}$ masses as observed in the ambient environment.

3. A policy of non-degradation is recommended for areas with PM concentrations below the recommended range. Progressive reduction of ambient concentrations of PM_{10} and $PM_{2.5}$ is the long term goal, in order that health impacts be reduced effectively.

4. The Working Group did consider an annual average metric for PM_{10} and $PM_{2.5.}$ However, the supporting health data links 24-hour concentrations to health endpoints, thus there is no scientific justification for the selection of a concentration based longer averaging time.

5. The collection of speciated particulate matter data on a greater temporal frequency is required to assist in further clarification of the causal mechanisms resulting in human health impacts. There is also a need to assess particle composition and size distribution to examine visibility/particle composition relationships and to identify contributing sources. The success of abatement programs can also be best assessed using continuous particulate matter data. It is recognized that continuous particulate matter monitoring only addresses some aspects of the particulate issue.

6. Some co-located PM10/PM2.5 sites should be maintained in the four representative regions of Canada. Continuous co-located monitoring of PM_{10} and $PM_{2.5}$ should be done at a few representative sites in each region. This will allow coarse particle concentrations to be calculated.

7. Development of source-receptor modelling to link health effects to particulate matter sources is needed. Recognizing the varied sources of primary and secondary PM, and the existing air quality management initiatives (provincially, federally and internationally), it would be most effective to target those sources most responsible for the observed health effects.

8. Many air quality initiatives ultimately impact on PM_{10} and $PM_{2.5}$ levels, their constituents (i.e., sulphates, nitrates, secondary organics) or their precursors (i.e., SO_2, NO_x, VOCs). From the public health point of view, of paramount importance is that of lowering the levels of any of these pollutants in the causal chain; that is, disrupting the causal chain. This overlap of pollutants, precursors, their sources and health impacts give rise to an unprecedented opportunity for comprehensive air pollution management and hence it is recommended that the various regions/provinces develop their strategies with this view in mind.

9. The recommended range has been derived to protect human health effects. Achieving these levels is also anticipated to improve visibility from current ranges, but will not achieve visibility levels associated with pristine background conditions.   Estimates of visual range (Table 8) are based upon contributions from both the fine and coarse mass fractions. It is proposed that a policy of non-degradation be adopted to protect visual range.

Table 8: Estimated Visual Range for PM Air Quality Targets

PM_{10} (µg/m^3)	$PM_{2.5}$ (µg/m^3)	Estimated Visual Range (km)	
		Urban	Rural
25	15	52	75
35	20	39	50
40	25	31	41
40	20	43	48

10. The Working Group is aware that some extreme natural events (volcanoes, forest fires, prescribed burning, etc.) will contribute to high particulate matter concentrations. While these sources will predominantly contribute to the coarse fraction of PM_{10}, combustion (burning) does contribute to $PM_{2.5}$ levels. Both fractions are implicated in adverse health effects. Local agencies charged with managing air quality may decide to exclude these events from their airshed management plans and determination of compliance.   However, many of these extreme natural events contribute to the coarse fraction of PM whose potential for public health impacts have not been disproved.

Beyond these recommendations the Working Group has provided additional information in the Appendices which is anticipated to be of use in considering the development of ambient standards and air quality management strategies for Particulate Matter.

Appendix A Incremental Risk Analysis

Appendix B Normalization of Ambient PM Data

Appendix C The Effect of Sampling Frequency on the Annual Maximum

Appendix D Proportional Linear Rollback

Appendix E Annual Average Background Concentrations for PM_{10} and $PM_{2.5}$

Appendix F Cumulative Exposure Index

Appendix G Source Apportionment Review