AIR QUALITY VALUATION MODEL
VERSION 3.0 (AQVM 3.0)
REPORT 2: METHODOLOGY

Final Report

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Any remaining errors or omissions are the sole responsibility of the authors.

ACRONYMS AND ABBREVIATIONS

AG	watershed aggregate
AOD	airway obstructive disease
AQVM	Air Quality Valuation Model
ARS	acute respiratory symptom
ASD	asthma symptom day
B	acute bronchitis (in children)
BAD	bad asthma day
CB	chronic bronchitis (in adults)
CH_4	methane
CHA	cardiac hospital admission
CO	carbon monoxide
CO_2	carbon dioxide
COH	coefficient of haze
COI	cost-of-illness
COPD	chronic obstructive pulmonary disease
CPI	consumer price index
CPUE	catch per unit effort
CRD	chronic respiratory disease
CRF	concentration-response function
CS	consumer surplus
CVM	contingent valuation method
DFA	damage function approach
DRLY	discounted remaining life years
ERV	emergency room visit
GDP	gross domestic product
GHG	greenhouse gas
HIS	U.S. Health Interview Survey
IRIS	U.S. EPA Integrated Risk Information System
g/m^3	micrograms per cubic metre
MRAD	minor restricted activity day
N_2O	nitrous oxide
NAPAP	National Acid Precipitation Assessment Program

NAPS	National Air Pollution Surveillance
NCLAN	National Crop Loss Analysis Network
NO_2	nitrogen dioxide
NO_x	nitrogen oxides
O_3	ozone
OBU	omissions, biases, and uncertainty
PCB	polychlorinated biphenyl
PM	airborne particulate matter
$PM_{2.5}$	airborne particulate matter 2.5 microns in diameter and smaller
PM_{10}	airborne particulate matter 10 microns in diameter and smaller
PM_{15}	airborne particulate matter 15 microns in diameter and smaller
POM	polycyclic organic matter
ppb	parts per billion
ppm	parts per million
PPP	purchasing power parity
RAD	restricted activity day
RHA	respiratory hospital admission
RIW	resource intensity weight
RR	relative risk
RRAD	respiratory restricted activity day
SE	standard error
SO_2	sulphur dioxide
SO_4	sulphate aerosol
TSP	total suspended particulates
U.S. EPA	United States Environmental Protection Agency
VOC	volatile organic compound
VSL	value of a statistical life
VSLY	value of a statistical life-year
W	average daily wage
WQC	Windsor-Quebec corridor
WTA	willingness to accept
WTP	willingness to pay

CHAPTER 1
INTRODUCTION

1.1 INTRODUCTION TO AQVM 3.0

The Air Quality Valuation Model Version 3.0 (AQVM 3.0) is a computational model for personal computers designed to estimate the human health and welfare benefits (or damages) associated with changes in Canada s ambient air quality. AQVM 3.0 provides a fast and easy to use model that evaluates alternative air pollution control policies in a consistent, defensible, and clearly documented manner within a framework that can also assess the implications of alternative assumptions in the benefits assessment parameters. AQVM 3.0 also can easily be updated to incorporate new scientific and economic information as it becomes available. AQVM 3.0 incorporates results from available epidemiologic and economic studies and available air quality monitoring and population data.

AQVM 3.0 uses a damage function approach to quantify the benefits from air pollution control or the damages from decrements in air quality. For annual average changes in ambient ozone, particulate matter, sulphates, carbon monoxide, sulphur dioxide, acid deposition, selected air toxics, and emissions of carbon dioxide and other greenhouse gases, AQVM 3.0 computes changes in annual impacts, and the associated economic benefits for over 20 different human health and welfare effects (see Table 2-1). The model also includes statistical analysis options that can be used to assess the uncertainty in the benefit estimates. AQVM 3.0 provides default values, based on an expert review and assessment of the available literature, for all required parameters. These default values are given as ranges for most health and welfare effects. To support sensitivity analyses, AQVM 3.0 users can change key model inputs including concentration-response functions, economic values, effect thresholds, and uncertainty parameters.

To run AQVM 3.0, the user must specify the change in ambient air pollutant concentrations expected to result from a policy. Determining these changes may require the use of separate emissions and atmospheric dispersion models. The user has the option to input absolute changes in air pollution concentrations by location or to input a selected percentage change (which can vary by province) from the baseline concentrations that are available in AQVM 3.0. The baseline air pollution concentrations in AQVM 3.0 are presented for census divisions and census metropolitan areas in Tables 3-1 and 3-2 respectively.

AQVM 3.0 covers all of Canada at the census division (CD) level. For each census division, AQVM 3.0 has baseline air quality for the selected air pollutants based on Environment Canada s

National Air Pollution Survey data for the years 1991 through 1993. AQVM 3.0 also includes baseline pollution levels for select pollutants at the census metropolitan area (CMA) level. Baseline population data for AQVM 3.0 at both the CD and CMA level come from the 1996 Canadian census. Results from AQVM 3.0 can be obtained at the census division, census metropolitan area, provincial, or national level for changes in individual health impacts (e.g., respiratory hospital admissions), changes in economic measures of benefits for individual health and welfare effects, and for the sum of all health and welfare effects. AQVM 3.0 is designed to be run for an annual change in air quality for any year between 1996 and 2035. Multiple year air quality change scenarios can be conducted by repeated model runs, one for each year in the user s scenario. Partial year changes in air quality, such as for changes only during the ozone season, can be accommodated with an adjustment in the calculation of the health and welfare effects.

Examples of the policies and regulations for which benefits have been estimated utilizing the same methodology with earlier versions of the AQVM include:

benzene in natural gas dehydrators
diesel fuel regulations
U.S. transboundary air pollution
sulphur in gasoline initiative
national acid rain strategy
ethanol gasoline
broad policy applications (ministerial presentations and speeches).

AQVM 3.0 was written for a Windows 95, Windows 97, or Windows NT 4.0 environment and will not operate using earlier versions of the Windows operating system. No special software is needed to run AQVM 3.0. It is a self-contained program with a graphic user interface that helps design benefits assessment simulations based on the data files that accompany the program. The output files from any simulation are ASCII files that can easily be saved and subsequently read into a spreadsheet or relational database software for additional analysis and formatting. The input files for population, pollution concentrations, and damage function parameters are also ASCII files that can be edited using other software. Typical run times for simulations in AQVM 3.0 range from 1 minute to 40 minutes depending on the pollutants, analyses, and reporting options selected by the user.

Consistent with earlier versions, AQVM 3.0 requires that the user select one of three available particulate matter measures (PM_{10}, $PM_{2.5,}$ or SO_4) for a given analysis. In other words, the user cannot analyse a combination of SO_4 and $PM_{2.5}$ in a single analysis. The particulate matter selection is made in AQVM 3.0 through the combined choice of a pollution and a risk/value file.

1.2 AQVM 3.0 DOCUMENTATION

The *Air Quality Valuation Model Version 3.0 (AQVM 3.0) Report 1: User s Guide* provides detailed information for installing and operating all aspects of AQVM 3.0. This *Report 2: Methodology* provides documentation on the benefit assessment methods and assumptions incorporated into AQVM 3.0. This report summarises the literature used in the assessment, how it is used, and key issues in the application of the literature to air pollution benefits analysis. This report is not intended to provide an exhaustive review of the literature and all issues in its application to benefits analysis. Recent U.S. EPA criteria documents (U.S. EPA, 1996a; 1996b) provide more detailed literature summaries, and recent similar large scale air pollution benefit analyses provide additional detail on the approaches and issues associated with using the literature for these types of applications (Chestnut, 1995a; European Commission, 1995; Rowe et al., 1995; U.S. EPA, 1997a; 1997b). This report is organized as follows.

Chapter 2: The Damage Function Approach describes the overall approach used to measure changes in health and welfare effects and the associated economic benefit measures. This chapter also describes the selection process used to identify, and lists, the health and welfare impacts that were included, and excluded, in AQVM 3.0.

Chapter 3: AQVM 3.0 Baseline Data describes the baseline air quality and population data.

Chapter 4: Concentration-Response Functions for Human Health Effects describes the interpretation of concentration-response functions and the derivation of the specific parameters used to estimate human health effects resulting from changes in ambient air quality.

Chapter 5: Economic Valuation describes the studies and economic methods used to monetize the human health and welfare effects, including visibility aesthetics, materials damage and soiling, agricultural losses, recreational fishing losses, and climate change.

Chapter 6: Uncertainty Analysis in AQVM 3.0 describes the qualitative and quantitative techniques used to estimate, report, and evaluate the uncertainty associated with reported benefit estimates.

Chapter 7: References includes the study citations.

Appendix A: Concentration-Response Functions for Sulphate Aerosol Health Effects summarises the sulphate human health concentration-response functions in AQVM 3.0, which are from the Sulphur Study report (Thurston et al., 1997b).

Appendix B: Revisions to AQVM and Response to Comments identifies the significant revisions made for AQVM 2.0, and provides responses to comments on the 1996 draft AQVM.

Appendix C: Comments on Draft AQVM Methodology Report provides comments from the peer-reviewers as well as unsolicited comments from Industry Canada.

Appendix D: Concentration-Response Functions for $PM_{2.5}$ Health Effects summarises the $PM_{2.5}$ human health concentration-response functions in AQVM 3.0.

CHAPTER 2
THE DAMAGE FUNCTION APPROACH

In this chapter we introduce the damage function approach used to quantify the benefits associated with alternative air quality scenarios (Section 2.1) and discuss the selection of the human health and welfare impacts included in AQVM 3.0 (Section 2.2). In this discussion, the terms *impacts* and *effects* refer to the physical impacts of changes in air pollution, such as changes in, or to, human health, visibility, and materials. The term *damages* refers to the economic value of adverse impacts associated with a degradation in air quality, and *benefits* refers to the value of the reduction in adverse impacts attributable to policies that improve air quality.

2.1 THE DAMAGE FUNCTION APPROACH

AQVM 3.0 is based on a damage function approach (DFA) to compute impacts and benefits, as illustrated in Figure 2-1. The DFA has been used for many types of air pollution policy assessments and is generally regarded as the preferred approach for computing benefits where the required literature and data are available and can be cost-effectively applied (Freeman et al., 1994).

The DFA involves up to a five step process.[1] In the first step, changes in emissions, by type and location, must be determined for a policy or scenario. This is typically accomplished using engineering assessments, or by specifying emission changes in terms of a stated objective (e.g., a 20% reduction in emissions of air toxic pollutants).

In the second step, the change in emissions must be translated into changes in ambient air pollution concentrations. Changes in ambient air pollution concentrations can be estimated with atmospheric dispersion models that allow for the transport of the primary emitted pollutants over space and time, and that account for secondary pollutant formation. For example, emissions of NO_x can react with other pollutants and with sunlight to create ozone and secondary particulates. Alternatively, one can employ simple rollback scenarios that assume changes in ambient concentrations are directly proportional to changes in precursor emissions.

1. In some instances some steps can be combined. For example, one may use existing data to establish the economic value of materials soiling from air pollution changes based on a relationship between air pollution levels and cleaning levels, without specifying and quantifying physical measures of soiling.

Figure 2-1
The Damage Function Approach

The first two steps in the DFA must be addressed outside of AQVM 3.0. AQVM 3.0 requires as its input an absolute or percent change in ambient air quality, by pollutant and by location, as is relevant for the analysis being conducted. Alternatively, users may simply specify the percent change in ambient air concentrations, by pollutant and by location, that is expected to result from a policy or that is the target of a policy.

The third step in a DFA is to translate the changes in ambient air pollution concentrations to changes in human health and welfare impacts. Changes in human health effects are quantified through the use of concentration-response functions. Based on input from experts at Health Canada and in the United States, several functions included in AQVM 3.0 were modified, and updated from functions developed for similar studies in the United States (Rowe et al., 1995) to incorporate studies undertaken in Canada and Canadian health data and demographics. The concentration-response functions included in AQVM 3.0 are documented in Chapter 4 for PM_{10} and ozone and in appendix A and D for sulphate and $PM_{2.5}$ respectively.

In the fourth step, the human health effects are assigned economic values using willingness-to-pay (WTP) measures based on Canadian and U.S. economic literature that addresses mortality and morbidity valuation. Nonhealth welfare impacts such as visibility aesthetics, crop damages, fishing losses, and materials damages are assigned economic values using WTP and market price measures. The selection of economic measures and values used in AQVM 3.0 is summarised in Chapter 5.

In the fifth step, benefits are computed and aggregated over the different impacts, locations, and time periods. AQVM 3.0 carries out its computations for each human health and welfare impact on a census division or census metropolitan area level on an annual average basis. Results for each location and for each impact are then aggregated to provincial and national totals for a selected year. Results can be then be obtained for individual impacts or for the total of all impacts at the census division, census metropolitan area, provincial, or national level. Benefits obtained for a scenario covering multiple years would need to be calculated one year at a time using AQVM and the appropriate air quality inputs, and then aggregated and discounted outside of AQVM to obtain present value estimates of total benefits.

Uncertainty in the model parameters is introduced at each stage of the estimation process and is recognized and incorporated in the benefits estimates. To reflect variations in the literature in terms of specific model parameters, we specify a distribution of values for health effects concentration-response coefficients and economic values. These distributions are based on a review, assessment, and synthesis of available study results and application of professional judgment. The distributions on model parameters are carried through the computation of total benefits using Monte Carlo techniques. In general, central estimates are selected to reflect our best estimate, and the high and low values are selected to be reasonably plausible alternatives to the central estimate based on the relevant literature and analyst judgment not as absolute upper and lower bounds.

Uncertainty can also be addressed quantitatively with AQVM using sensitivity analyses that examine how the impact and benefit estimates change when model parameters and other key assumptions are varied. Finally, unquantified uncertainty is addressed through the identification of key omissions, biases, and uncertainties in the assessment. These methods to address uncertainty are discussed in Chapter 6.

2.2 IMPACT IDENTIFICATION AND SELECTION FOR AQVM 3.0

There are many potential human health and welfare impacts that may be associated with air pollution. However, not all potential impacts may be quantified because of limited literature upon which to develop a defensible damage function assessment, and/or because in some cases developing such an assessment would require considerable costs. Based on the literature and other similar studies,[2] we identified potential health and welfare effects, and then selected those to be included in AQVM 3.0 based on the following criteria.

Is the literature sufficient to develop quantitative estimates? Scientific literature may suggest that there are some benefits, but the available literature may be insufficient to quantify the benefits using a damage function approach. In such cases, the potential impacts are omitted from the analysis and result in a potential understatement of the benefits of air pollution control. At what point the literature is sufficient for developing quantitative estimates is a matter of judgment. Our aim was for the estimates to be based on the weight of evidence from studies conducted using accepted methods in their respective fields, but not with the expectation that there is a consensus within each field as to how to interpret all of the available evidence. We gave greater consideration to the results of studies conducted in Canada, if available, so that the assessment would reflect Canadian population responses and baseline use of health care, which may differ from those in the Unites States and other locations. In general, health effects and economic valuation studies in Canada show results that are reasonably consistent with results from U.S. studies.

Are benefits relatively small? Some benefits may be judged to be relatively small in relation to others considered in the model. For example, if benefit B1 has a value of $100,000 per year and benefit B2 has an expected value that is orders of magnitude smaller (e.g., $100 to $1,000 per year), then B2 will have little impact on the total benefits estimate and even may be smaller than the measurement error associated with the B1 benefit estimate. Impacts that have relatively small damages are omitted or implicitly

2. See especially Rowe et al. (1995; 1996b) where extensive analyses were conducted to identify and comprehensively evaluate the ability to conduct damage function analyses for human health and environmental impacts from electricity generation and for air pollution impacts from automobiles, trucks, and buses.

assigned a zero value. Again, these omissions may result in an understatement of air pollution control benefits, but for these impacts we expect the understatement to be small.

Are the assessment costs high? For some impacts, there may be literature useful for the development of a defensible damage function approach, but this may require considerable supporting data, elaborate modelling, or other efforts that entail substantial costs. If the expected benefits of such assessments are small, then impacts with high assessment costs were omitted from AQVM 3.0, but could potentially be included in subsequent versions.

The human health and welfare impacts included in AQVM 3.0 are listed in Table 2-1. The potential impacts that were explicitly assigned zero values are listed in Part I of Table 2-2. The impacts omitted because of limited literature and/or high assessment costs are listed in Part II of Table 2-2.

Many potential terrestrial and aquatic ecosystems impacts cannot be quantified because of the lack of literature providing quantitative descriptions of how ecosystems respond to pollutants and/or the lack of supporting economic literature valuing these ecosystem impacts. In some cases, translating changes in ambient concentrations of pollutants to terrestrial and aquatic exposures would also entail considerable modelling expense.

Global climate change impacts also fall into the category of having insufficient data for economic valuation because the available studies have considerable limitations in terms of the impact coverage, methods employed, and assumptions made. Further, the available studies report considerable variability in the values to be assigned to greenhouse gas emissions (see Section 5.5.5). Therefore, we do not select any one number, or range of numbers, for inclusion in AQVM 3.0. However, because changes in greenhouse gas emissions, with some economic value assumptions per tonne of emissions, can be a dominant component of benefits for many air pollution control policies, we include greenhouse gas emissions as a model input. Reflecting the uncertainty in the economic valuation literature for global climate change AQVM 3.0 has a default value of $0/tonne. A quantitative estimate of a scenario s impact on global climate change can be obtained if the model user assigns a dollar value per tonne of equivalent carbon.

Table 2-1
Human Health and Welfare Effects in AQVM 3.0

Pollutant	Effects
Ozone	* mortality * respiratory hospital admissions * emergency room visits * asthma symptom days * minor restricted activity days * acute respiratory symptom days * agriculture crop damage (corn, soybeans, wheat, tobacco)
Particulate Matter ($PM_{10,}$ $PM_{2.5}$) or Sulphate (SO_4)	* mortality * chronic bronchitis * respiratory hospital admissions * cardiac hospital admissions * emergency room visits * asthma symptom days * restricted activity days * acute respiratory symptom days * child bronchitis * household materials soiling * visibility (change in visual range)
Carbon Monoxide (CO)	* cardiac hospital admissions
Carbon Dioxide (CO_2) Methane (CH_4)	* global climate change
Sulphur Dioxide (SO_2) Gaseous	* materials damage
Acid Deposition	* recreational fishing yield
Air Toxics (Benzene, 1,3 Butadiene, Acetaldehyde, Formaldehyde)	* cancer risk

Table 2-2
Human Health and Welfare Effects Omitted from AQVM 3.0 by Pollutant

Pollutant	Effects	Comments
Part I: Omitted because Damages Are Relatively Small or Zero		
Particulate Matter	Visibility accidents	Very small or zero value in comparison to other visibility externalities.
SO_2	Morbidity Materials (other than galvanized steel) Crops/vegetation Forests, terrestrial, groundwater	Very small damage. Limited literature.
NO_x, Nitrates	Morbidity (NO_2) Crops/vegetation, forests, surface and groundwater	Very small value in comparison to ozone and PM related health effects. Limited literature on ecosystem NO_x damages.
Ambient Ozone	Materials fabrics/dyes/ paints Other vegetation effects	Small values relative to other ozone effects and limited literature.
Part II: Omitted because of Limited Literature or High Assessment Costs		
Ambient Ozone	Forest impacts Elastomers	Preliminary analysis possible. Elastomer damages likely to be relatively small.
Air Toxics	Noncancer health risk Crops/vegetation, forests, groundwater Fish/aquatics	Limited literature. Literature is inconclusive as to whether damages would be substantive compared to cancer risks.
Ambient Ozone	Chronic respiratory illness	Literature is limited or inconclusive.
Ambient Ozone, PM_{10}, Air Toxics, CO	Terrestrial wildlife	Literature is limited, although damages are expected to be small relative to human health impacts.
Carbon Monoxide	Mortality and other morbidity	Limited literature for impacts at or below existing ambient levels.
CO_2/Greenhouse Gases	Human health Crops/vegetation Forests Water resources Biodiversity Land use Infrastructure	Significant uncertainty related to level of effects and damage quantification.
Acid Deposition	Cultural/historic materials Crops/vegetation Forests	Values may be small. Economic literature is limited.

CHAPTER 1
AQVM 3.0 BASELINE POPULATION AND AIR QUALITY DATA

This chapter presents the baseline population and air quality data incorporated into AQVM 3.0. Tables 3-1 and 3-2 present the age-specific baseline populations, and baseline concentrations for PM_{10}, $PM_{2.5}$, SO_4, ozone, CO, and SO_2, for each census division (CD) and census metropolitan area (CMA) respectively. Table 3-3 presents the population growth rates for 1996 through 2035 for each province. Tables 3-4 and 3-5 present the baseline air toxics and visual range data for each CD and CMA respectively. Tables 3-6 and 3-7 present the CDs and CMAs that, respectively, have air quality monitors for the various pollutants.[1] Table 3-8 presents the baseline production and average price data for corn, soybeans, wheat, and tobacco by province.[2]

To provide a visual reference, Figures 3-1 to 3-9 show the census divisions for which the analysis is conducted, and identify which census divisions have air quality monitors (Statistics Canada, 1992).[3]

The baseline population data for CDs and CMAs in AQVM 3.0 (see Tables 3-1 and 3-2) are from the 1996 Canadian Census (Statistics Canada, 1997a,b). The age-specific group totals: total population, population under age 20, population age 20 and older, population age 25 and older, and population age 65 and older, were developed to correspond with the populations specified in AQVM 3.0's concentration-response-functions (see Chapter 4). Age-specific population data was directly available for each CD, but only for a subset of the CMAs. For CMAs lacking age-specific data, the reported all age population was allocated into the different age-specific categories using ratios developed at the provincial level using the CD data. The number of households for both the CDs and CMAs was estimated using an average Canadian household size of 2.6 (P. De Civita, Environment Canada, personal communication, 1999).

AQVM 3.0 also includes provincial population growth factors for the CDs and CMAs (see Table 3-3). The population growth factors for 1996-2015 were estimated from provincial population projections (George et al., 1994). The population growth factors for 2016-2035 were developed based on national population projections (George et al., 1994). The population growth

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1. For SO_4, CO, acetaldehyde, 1,3 butadiene, and formaldehyde information linking CDs with monitors to corresponding CMAs was not received resulting in the indication of no monitors in the CMAs for their pollutants.

2. In AQVM 3.0 this agricultural information is used to create baseline populations that reflect the total value of the crop (i.e., quantity × price) in the baseline period.

3. These figures reflect the CDs in the 1991 Census. In the 1996 Census CDs 11 and 13 in British Columbia (Figure 3-9) were merged into other existing CDs.

factors are applied uniformly within each province (i.e., no adjustments are made for the different age groups or specific CDs or CMAs).

Baseline Air Quality for Census Divisions

The baseline air quality data for the CDs comes from air pollution monitoring data from 1991 to 1993 for ozone (132 monitors), TSP (92 monitors), PM_{10} (35 monitors), SO_4 (20 monitors), $PM_{2.5}$ (20 monitors), CO (54 monitors), and SO_2 (72 monitors) (see Table 3-1) obtained from the National Air Pollution Surveillance (NAPS) program database (Dann, 1996).[4] Concentrations over the three year period at each monitor were averaged to develop a baseline concentration for each monitor.

The baseline ozone concentrations are the average of the daily high hour reading (measured in ppb) for the May to September ozone season. The PM_{10}, SO_4, $PM_{2.5}$, and SO_2 baseline concentrations are year-round averages (measured in $\mu g/m^3$) of 24-hour concentrations. The CO baseline concentrations are the annual average of the daily high hour reading (measured in ppm).

The concentrations of these pollutants at individual monitoring locations were mapped to individual census divisions, with the exception of SO_4 and $PM_{2.5}$. When a CD contained more than one monitor, the readings from all the monitors were averaged to develop a baseline concentration. For CDs without mapped monitors, either a single monitor value, or the average of several monitor values, were used to assign a baseline concentration, which is discussed separately. The assignments were made on a case by case basis according to proximity or similarity of environment (e.g., urban, rural, or remote). In general, the closest available monitor located in a similar environment was assigned to a CD without a mapped monitor.

Because some CDs do not have monitors in them (or even close by) for some pollutants, the estimation of baseline ambient concentrations for some pollutants for some locations may entail a high degree of error. Therefore, AQVM 3.0 has an option for users to run the analysis with only those CDs that have a monitor for the pollutant of interest (see *Report 1: User s Guide* for selection of this option). For example, selecting this option may include a CD for the particulate matter calculations because a particulate monitor is located in the CD, but may not include the same CD for air toxics calculations because an air toxics monitor is not located in the CD.

Although effort was made to match similar types of locations (e.g., urban, rural, remote) when assigning air pollutant concentrations from CDs with monitors to CDs without a mapped monitor, there remains a chance of bias because, in general, air pollution monitors are located in more populated areas where air pollution concentrations are expected to be higher than in less populated areas. The baseline concentrations, however, only affect the benefits calculations if the user is applying the percentage change in air pollutant option or is selecting a nonzero health effects threshold for a pollutant. When running either of these options, the user may want to

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4. In an analysis of monitoring data from cities across Canada Dann (1994) found the following relationship of mean PM_{10} to mean TSP: $PM_{10}/TSP = 1.24 - 0.385 \times \log(TSP)$. Over the range of observed values this equation was roughly equivalent to a PM_{10}/TSP of 0.5. We used this ratio to convert TSP data to PM_{10} estimates.

conduct a sensitivity test to evaluate the effect of potential bias by limiting the analysis to those CDs with monitors.

Baseline SO_4 concentrations for 20 dichotomous sampler monitors were mapped by Environment Canada to the cities where the monitors were located (T. Dann, Environment Canada, personal communication, 1998). Data for all the monitors in a city were averaged to create the baseline concentration used in AQVM 3.0. This baseline concentration was applied to all the CDs located within the identified city.

Baseline $PM_{2.5}$ concentrations were developed for the CDs using monitoring data and observed $PM_{2.5}/PM_{10}$ ratios. $PM_{2.5}$ monitoring data was provided for the same set of monitors that were used to develop the SO_4 baseline concentrations (T. Dann, Environment Canada, personal communication, 1999). As a result, baseline $PM_{2.5}$ concentrations were initially developed for the same set of CDs covered by the SO_4 data using the same approach of averaging monitor data when more than one monitor was located in a city. For the remaining CDs, baseline $PM_{2.5}$ concentrations were developed by comparing PM_{10} and $PM_{2.5}$ data from co-located monitors. This resulted in the development of provincial $PM_{2.5}/PM_{10}$ ratios which were used to adapt the available PM_{10} information for CDs without direct $PM_{2.5}$ monitoring data.

The annual average concentrations for 1990 to 1995 of three air toxics acetaldehyde (9 monitors), 1,3 butadiene (31 monitors), and formaldehyde (9 monitors) were obtained from the NAPS program database (T. Dann, Environment Canada, personal communication, 1996). For these air toxics the available data within a province was averaged to create provincial baseline concentrations which were then applied to all the CDs in the province. Data on 1,3 butadiene concentrations were not available for Newfoundland, Prince Edward Island, Saskatchewan, the Northwest Territories, and the Yukon Territory. Newfoundland and Prince Edward Island were assigned the New Brunswick concentration. Saskatchewan was assigned the Manitoba concentration. The territories were assigned the minimum concentration in the data set. Data on acetaldehyde and formaldehyde concentrations were not available for Newfoundland, Prince Edward Island, Nova Scotia, Manitoba, Saskatchewan, Alberta, the Northwest Territories, and the Yukon Territory. They were all assigned the minimum concentration in the data set.

Baseline benzene concentrations for the CDs were developed from average concentration data for 39 monitors over the 1996-1997 period and a mapping of the monitors to specific CDs (P. De Civita, Environment Canada, personal communication, 1999). When more than one monitor was referenced to a CD an average concentration was developed. For the CDs not covered by the mapping, average concentrations were developed using the data from all the monitors in a province. These provincial average concentrations were then assigned to the CDs not covered by the original monitor mapping. For CDs in provinces in which there were no benzene monitors, the same assignment process was used as for 1,3 butadiene values (described above).

Because of the limited number of air toxics monitors, and other assessment issues, the air toxic benefit estimates must be interpreted with considerable caution (see Section 4.5 for additional discussion).

Baseline Air Quality for Census Metropolitan Areas

Baseline concentrations at the CMA level have been provided for PM_{10}, $PM_{2.5}$, ozone, and benzene based on monitoring data for 1994-1996 (P. De Civita, Environment Canada, personal communication, 1999) (see Table 3-2). Currently there are no baseline concentrations specified at the CMA level for SO_4, CO, and SO_2.

For PM_{10}, $PM_{2.5}$, and ozone, the baseline concentrations have only, to date, been applied to the CMAs linked to the monitors in mapping keys provided with the data. For benzene, the same monitoring data and approach was used to develop the CMA baseline concentrations as was used for the CDs. For the remaining air toxics, 1,3 butadiene, acetaldehyde, and formaldehyde, the provincial averages that were developed from the 1990-1995 monitoring data and used for the CDs were also used for the CMAs.

Baseline Visual Range Data

Provincial average visual range data were provided by R. Hoff of the Atmospheric Environment Service. AQVM 3.0 applies these average values equally to all CDs and CMAs within a province to specify the baseline visual range data.

Baseline Agricultural Production

Data on the average production and price of corn, soybeans, wheat, and tobacco were obtained from D. Guay of Environment Canada (personal communication, 1998) (Table 3-8). Price and production data that were provided for corn, soybeans, wheat, and tobacco(D. Guay, Environment Canada, personal Communication, 1998) and that reflect the average of annual data for 1993 through 1995, except for tobacco which uses the average of 1990 through 1995 (1990_1993 for Nova Scotia and New Brunswick) are used to specify baseline values for agricultural production in AQVM 3.0 . Note that for soybeans and wheat, price is computed as farm cash receipts divided by marketed production. Because AQVM 3.0 treats agricultural products as separate populations, the provincial production (tonnes) and average price ($/tonne) are multiplied to create a baseline “population” for the crop in the model if the crop is among the 10 most important in the province (ranked by total farm cash receipts).

Table 3-1
1996 Census Division Populations and Baseline Pollution

Province Name	Census Division	Pop under 20	Pop 20 and Older	Pop 25 and Older	Pop 65 and Older	Total Pop	House-holds	PM_{10} ($\mu g/m^3$)	$PM_{2.5}$ ($\mu g/m^3$)	SO_4 ($\mu g/m^3$)	Ozone (ppb)	CO (ppm)	SO_2 ($\mu g/m^3$)
Newfoundland	1	69,530	181,995	161,605	27,495	251,525	96,740	16.46	9.71		28.8	1.54	0.0052
Newfoundland	2	8,355	19,355	17,210	2,720	27,710	10,658	16.46	9.71		28.8	1.54	0.0052
Newfoundland	3	6,230	16,240	14,535	2,355	22,470	8,642	16.46	9.71		30.1	1.54	0.0052
Newfoundland	4	7,395	17,440	15,760	2,645	24,835	9,552	16.46	9.71		32.2	1.54	0.0052
Newfoundland	5	12,265	32,055	28,755	4,945	44,320	17,046	16.46	9.71		32.2	1.54	0.0052
Newfoundland	6	10,585	28,530	25,710	4,275	39,115	15,044	16.46	9.71		30.1	1.54	0.0052
Newfoundland	7	11,210	30,315	27,395	5,705	41,525	15,971	16.46	9.71		28.8	1.54	0.0052
Newfoundland	8	13,505	34,735	31,190	5,910	48,240	18,554	16.46	9.71		30.1	1.54	0.0052
Newfoundland	9	6,515	16,335	14,690	2,325	22,850	8,789	16.46	9.71		32.2	1.54	0.0052
Newfoundland	10	9,840	19,355	16,770	1,120	29,195	11,229	16.46	9.71		32.2	0.73	0.0052
Prince Edward Island	1	6,000	13,560	12,280	2,710	19,560	7,523	13.21	7.79		31.5	1.54	0.0017
Prince Edward Island	2	20,090	50,345	45,300	8,945	70,435	27,090	13.21	7.79		31.5	1.54	0.0017
Prince Edward Island	3	13,065	31,485	28,475	5,795	44,550	17,135	13.21	7.79		31.5	1.54	0.0017

Nova Scotia	1	4,525	12,465	11,345	2,455	16,990	6,535	9.24	5.45		39.1	1.61	0.0105
Nova Scotia	2	7,345	19,965	18,290	4,385	27,310	10,504	9.24	5.45		39.1	1.61	0.0105
Nova Scotia	3	4,795	15,700	14,510	3,615	20,495	7,883	9.24	5.45		39.1	1.61	0.0105
Nova Scotia	4	2,930	9,490	8,760	2,140	12,420	4,777	9.24	5.45		39.1	1.61	0.0105
Nova Scotia	5	5,600	16,720	15,465	3,935	22,320	8,585	9.24	7.22	2.57	39.1	1.61	0.0105
Nova Scotia	6	11,595	35,970	33,375	7,780	47,565	18,294	16.28	9.61		37.8	1.61	0.0105
Nova Scotia	7	16,500	42,700	38,915	7,410	59,200	22,769	16.28	9.61		41	1.61	0.0105
Nova Scotia	8	11,135	28,340	25,920	4,715	39,475	15,183	16.28	9.61		37.8	1.61	0.0105
Nova Scotia	9	89,395	253,575	227,970	35,210	342,970	131,912	16.28	8.95	3.3	35.4	1.61	0.0105
Nova Scotia	10	13,375	35,880	32,650	6,800	49,255	18,944	16.28	9.61		35.4	1.61	0.0105
Nova Scotia	11	8,405	25,400	23,310	6,050	33,805	13,002	16.28	9.61		37.8	1.61	0.0105
Nova Scotia	12	13,250	35,470	32,255	7,495	48,720	18,739	16.28	9.61		35.4	1.61	0.0105
Nova Scotia	13	2,860	8,045	7,430	1,765	10,905	4,194	16.28	9.61		35.4	1.61	0.0105
Nova Scotia	14	6,245	13,310	11,815	2,405	19,555	7,521	16.28	9.61		35.4	1.61	0.0105

Table 3-1 (cont.)
1996 Census Division Populations and Baseline Pollution

Province Name	Census Division	Pop under 20	Pop 20 and older	Pop 25 and older	Pop 65 and older	Total Pop	House-holds	PM10 (:g/m3)	PM2.5 (:g/m3)	SO4 (:g/m3)	Ozone (ppb)	CO (ppm)	SO2 (:g/m3)
Nova Scotia	15	6,050	14,870	13,490	2,920	20,920	8,046	20.26	11.95		35.4	1.61	0.0105
Nova Scotia	16	2,955	8,070	7,325	1,820	11,025	4,240	20.26	11.95		35.4	1.61	0.0105
Nova Scotia	17	32,735	85,130	77,125	16,945	117,865	45,333	20.26	11.95		35.4	1.61	0.0105
Nova Scotia	18	2,415	6,075	5,560	1,245	8,490	3,265	20.26	11.95		35.4	1.61	0.0105
New Brunswick	1	20,270	59,035	53,165	11,875	79,305	30,502	15.2	10.19	3.54	32.6	1.62	0.0114
New Brunswick	2	7,240	20,090	18,220	4,345	27,330	10,512	15.2	10.19	3.54	38.8	1.62	0.0114
New Brunswick	3	7,695	17,675	15,960	1,880	25,370	9,758	21.56	10.19	3.54	40	1.62	0.0114
New Brunswick	4	3,015	9,460	8,750	2,115	12,475	4,798	21.56	12.72		39.8	1.62	0.0114
New Brunswick	5	19,885	44,840	40,780	6,570	64,725	24,894	15.2	8.97		39.7	1.62	0.0114
New Brunswick	6	7,365	19,125	17,380	2,910	26,490	10,189	15.2	8.97		45.7	1.62	0.0114
New Brunswick	7	29,655	90,880	81,705	16,625	120,535	46,360	15.2	8.97		42.7	1.62	0.0114
New Brunswick	8	8,470	23,620	21,550	4,290	32,090	12,342	21.56	12.72		32.6	1.62	0.0114
New Brunswick	9	14,175	37,950	34,015	6,595	52,125	20,048	21.56	12.72		32.6	1.62	0.0114

New Brunswick	10	22,300	63,410	56,255	10,060	85,710	32,965	21.56	12.72		40	1.62	0.0114
New Brunswick	11	7,720	19,195	17,370	3,770	26,915	10,352	21.56	12.72		39.8	1.62	0.0114
New Brunswick	12	6,455	15,460	13,960	2,890	21,915	8,429	21.56	12.72		39.8	1.62	0.0114
New Brunswick	13	9,745	27,070	24,550	4,500	36,815	14,160	21.56	12.72		39.8	1.62	0.0114
New Brunswick	14	10,510	28,185	25,820	4,960	38,695	14,883	21.56	12.72		39.8	1.62	0.0114
New Brunswick	15	23,310	64,295	58,075	9,790	87,605	33,694	21.56	12.72		32.6	1.62	0.0114
Quebec	1	3,420	10,390	9,585	1,570	13,810	5,312	13.21	7.79		31.5	1.54	0.0017
Quebec	2	5,255	16,090	14,730	2,730	21,345	8,210	21.56	12.72		39.8	1.62	0.0052
Quebec	3	5,530	15,305	14,090	2,530	20,835	8,014	21.56	12.72		39.8	1.62	0.0052
Quebec	4	3,465	10,260	9,505	1,990	13,725	5,279	21.56	12.72		39.8	1.62	0.0052
Quebec	5	5,150	14,395	13,230	2,775	19,545	7,517	21.56	12.72		39.8	1.62	0.0052
Quebec	6	4,645	11,265	10,340	2,250	15,910	6,119	21.56	12.72		39.8	1.62	0.0052
Quebec	7	5,995	14,900	13,720	2,995	20,895	8,037	21.56	12.72		39.8	1.62	0.0052
Quebec	8	6,035	17,670	16,335	3,335	23,705	9,117	21.56	12.72		39.8	1.62	0.0052

Table 3-1 (cont.)
1996 Census Division Populations and Baseline Pollution

Province Name	Census Division	Pop under 20	Pop 20 and older	Pop 25 and older	Pop 65 and older	Total Pop	House-holds	PM10 (:g/m3)	PM2.5 (:g/m3)	SO4 (:g/m3)	Ozone (ppb)	CO (ppm)	SO2 (:g/m3)
Quebec	9	5,295	14,870	13,845	3,210	20,165	7,756	21.56	12.72		39.8	1.62	0.0052
Quebec	10	13,895	38,785	35,195	6,160	52,680	20,262	21.56	12.72		39.8	1.62	0.0052
Quebec	11	2,590	7,610	7,080	1,880	10,200	3,923	21.56	12.72		39.8	1.62	0.011
Quebec	12	8,730	23,395	21,450	4,570	32,125	12,356	21.56	12.72		39.8	1.62	0.011
Quebec	13	6,450	16,645	15,290	3,330	23,095	8,883	21.56	12.72		39.8	1.62	0.011
Quebec	14	6,270	16,950	15,555	3,640	23,220	8,931	21.56	12.72		39.8	1.62	0.011
Quebec	15	4,335	12,600	11,645	2,350	16,935	6,514	17.95	10.59		37.9	1.46	0.011
Quebec	16	3,225	10,215	9,540	2,060	13,440	5,169	17.95	10.59		37.9	1.46	0.011
Quebec	17	5,195	14,630	13,560	3,135	19,825	7,625	21.56	12.72		39.8	1.46	0.011
Quebec	18	5,860	17,945	16,470	3,580	23,805	9,156	22.64	13.36		32.8	1.46	0.001
Quebec	19	8,175	21,500	19,825	4,615	29,675	11,414	22.64	13.36		32.8	1.46	0.001
Quebec	20	1,720	5,165	4,775	875	6,885	2,648	22.64	13.36		32.8	1.46	0.001
Quebec	21	5,290	16,330	15,155	2,895	21,620	8,315	22.64	13.36		37.9	1.46	0.011

Quebec	22	7,610	17,210	15,810	1,420	24,820	9,546	22.64	13.36		37.9	1.46	0.001
Quebec	23	115,770	388,825	351,665	63,995	504,595	194,075	22.64	9.14		32.8	1.46	0.001
Quebec	24	13,180	38,040	34,595	6,385	51,220	19,700	22.64	13.36		32.8	1.46	0.001
Quebec	25	23,860	51,750	47,160	4,545	75,610	29,081	22.64	13.36		32.8	1.46	0.001
Quebec	26	7,480	17,595	16,000	2,905	25,075	9,644	22.64	13.36		32.8	1.46	0.001
Quebec	27	5,640	13,070	11,955	2,410	18,710	7,196	22.64	13.36		32.8	1.46	0.001
Quebec	28	5,105	13,250	12,225	2,710	18,355	7,060	22.64	13.36		32.8	1.46	0.001
Quebec	29	13,960	32,370	29,330	4,900	46,330	17,819	22.64	13.36		32.8	1.46	0.001
Quebec	30	6,065	15,235	14,065	2,975	21,300	8,192	22.37	13.2		43.1	1.46	0.001
Quebec	31	11,575	33,450	30,815	6,850	45,025	17,317	22.64	13.36		32.8	1.46	0.001
Quebec	32	6,920	17,760	16,245	3,255	24,680	9,492	22.64	13.36		32.8	1.46	0.001
Quebec	33	7,975	18,950	17,390	3,355	26,925	10,356	22.64	13.36		32.8	1.46	0.001
Quebec	34	11,500	33,675	31,340	6,475	45,175	17,375	21.64	12.77		35.3	1.46	0.001
Quebec	35	3,320	10,175	9,490	2,275	13,495	5,190	26.3	15.52		35.3	1.46	0.003

Table 3-1 (cont.)
1996 Census Division Populations and Baseline Pollution

Province Name	Census Division	Pop under 20	Pop 20 and older	Pop 25 and older	Pop 65 and older	Total Pop	House-holds	PM10 (:g/m3)	PM2.5 (:g/m3)	SO4 (:g/m3)	Ozone (ppb)	CO (ppm)	SO2 (:g/m3)
Quebec	36	16,250	50,850	47,365	11,100	67,100	25,808	26.3	15.52		35.3	1.46	0.0164
Quebec	37	35,165	105,375	96,530	18,890	140,540	54,054	21.64	12.77		35.3	1.46	0.003
Quebec	38	5,550	14,125	13,145	2,785	19,675	7,567	24.95	14.72		42.2	1.46	0.003
Quebec	39	18,260	44,650	40,690	7,470	62,910	24,196	24.95	14.72		42.2	1.46	0.003
Quebec	40	3,915	11,100	10,325	2,705	15,015	5,775	24.95	14.72		43	1.46	0.003
Quebec	41	6,395	15,555	14,435	2,890	21,950	8,442	22.37	13.2		43.1	1.46	0.003
Quebec	42	9,815	23,595	21,670	3,895	33,410	12,850	22.37	13.2		43	1.46	0.003
Quebec	43	34,020	98,410	87,610	16,655	132,430	50,935	22.37	13.2		43.1	1.46	0.003
Quebec	44	4,985	10,945	10,070	2,010	15,930	6,127	22.37	13.2		43.1	1.46	0.003
Quebec	45	9,930	28,535	26,515	5,695	38,465	14,794	22.37	13.2		43.1	1.46	0.003
Quebec	46	12,450	33,540	31,110	6,760	45,990	17,689	22.37	7.7	2.55	43.1	1.46	0.0037
Quebec	47	21,775	55,220	50,700	8,645	76,995	29,614	22.37	13.2		43.1	1.46	0.0037
Quebec	48	4,580	10,710	9,835	1,725	15,290	5,881	22.37	13.2		43	1.46	0.0037

Quebec	49	23,625	60,640	55,350	10,500	84,265	32,410	24.95	14.72		43	1.46	0.0164
Quebec	50	6,550	17,125	15,780	3,440	23,675	9,106	24.95	14.72		42.2	1.46	0.0164
Quebec	51	5,585	18,190	17,080	3,735	23,775	9,144	26.3	15.52		35.3	1.46	0.0164
Quebec	52	10,415	27,135	25,400	4,960	37,550	14,442	24.95	14.72		42.2	1.46	0.0164
Quebec	53	12,665	39,625	36,750	7,230	52,290	20,112	24.95	14.72		42.2	1.58	0.0164
Quebec	54	21,600	57,155	52,440	10,350	78,755	30,290	24.95	14.72		43	1.58	0.0037
Quebec	55	9,935	23,145	21,395	3,205	33,080	12,723	22.4	13.22		43	1.58	0.0037
Quebec	56	27,555	69,990	64,645	11,355	97,545	37,517	22.4	13.22		43.1	1.58	0.0037
Quebec	57	33,955	79,885	73,665	9,330	113,840	43,785	22.4	15.37	3.76	43	1.58	0.0037
Quebec	58	82,380	231,920	209,785	31,195	314,300	120,885	22.4	15.37	3.76	37.1	1.58	0.0037
Quebec	59	28,745	66,860	61,365	6,175	95,605	36,771	22.4	15.37	3.76	37.1	1.58	0.0037
Quebec	60	31,200	70,995	65,650	7,345	102,195	39,306	22.4	15.37	3.76	37.1	1.58	0.0037
Quebec	61	14,080	38,755	35,665	6,765	52,835	20,321	22.4	13.22		37.1	1.58	0.0037
Quebec	62	10,250	31,070	29,330	6,375	41,320	15,892	20.59	12.15		42.2	1.03	0.0015

Table 3-1 (cont.)
1996 Census Division Populations and Baseline Pollution

Province Name	Census Division	Pop under 20	Pop 20 and older	Pop 25 and older	Pop 65 and older	Total Pop	House-holds	PM10 (:g/m3)	PM2.5 (:g/m3)	SO4 (:g/m3)	Ozone (ppb)	CO (ppm)	SO2 (:g/m3)
Quebec	63	10,850	27,195	25,390	4,200	38,045	14,633	22.4	13.22		37.1	1.58	0.0037
Quebec	64	33,885	69,325	63,655	5,290	103,210	39,696	22.4	15.37	3.76	37.1	1.58	0.0037
Quebec	65	86,450	243,945	223,975	37,205	330,395	127,075	22.4	15.37	3.76	37.1	1.58	0.0037
Quebec	66	392,055	1,383,785	1,256,360	264,040	1 ,775,840	683,015	22.4	15.37	3.76	37.1	1.58	0.0037
Quebec	67	39,780	92,380	85,045	10,240	132,160	50,831	22.4	15.37	3.76	37.1	1.58	0.0037
Quebec	68	6,590	16,345	15,025	2,555	22,935	8,821	22.4	13.22		43.1	1.58	0.0037
Quebec	69	6,105	15,895	14,840	3,190	22,000	8,462	22.4	13.22		43.1	1.58	0.0037
Quebec	70	15,540	44,230	40,845	8,230	59,770	22,989	22.4	15.37	3.76	37.1	1.58	0.0037
Quebec	71	27,925	67,385	62,610	8,775	95,310	36,658	22.4	15.37	3.76	37.1	1.58	0.0037
Quebec	72	24,085	54,875	50,615	6,800	78,960	30,369	22.4	13.22		37.1	1.58	0.0037
Quebec	73	37,565	81,675	74,850	7,485	119,240	45,862	22.4	15.37	3.76	37.1	1.58	0.0037
Quebec	74	7,145	15,540	14,340	1,570	22,685	8,725	22.4	15.37	3.76	37.1	1.58	0.0037
Quebec	75	23,015	60,755	55,940	8,745	83,770	32,219	22.4	13.22		37.1	1.58	0.0037

Quebec	76	7,150	21,355	19,940	4,405	28,505	10,964	22.4	15.37	3.76	42.2	1.03	0.0015
Quebec	77	5,835	22,390	21,300	4,405	28,225	10,856	22.4	13.22		42.2	1.03	0.0015
Quebec	78	8,925	27,415	25,835	5,220	36,340	13,977	20.59	12.15		42.2	1.03	0.0015
Quebec	79	8,845	25,065	23,350	4,270	33,910	13,042	20.59	12.15		42.2	1.03	0.0015
Quebec	80	5,050	15,285	14,320	3,085	20,335	7,821	20.59	12.15		42.2	1.03	0.0015
Quebec	81	60,990	156,615	141,645	17,445	217,605	83,694	20.59	11.92	3.81	39.1	1.03	0.0015
Quebec	82	10,000	23,645	22,105	2,265	33,645	12,940	20.59	12.15		40.6	1.03	0.0015
Quebec	83	5,180	15,090	14,035	2,695	20,270	7,796	20.59	12.15		40.6	1.03	0.0015
Quebec	84	4,285	11,280	10,440	2,330	15,565	5,987	20.59	12.15		40.6	1.03	0.0015
Quebec	85	5,525	12,500	11,330	1,980	18,025	6,933	19.94	11.76		40.6	1.03	0.0015
Quebec	86	12,485	30,140	27,250	3,925	42,625	16,394	19.94	11.76		40.6	1.03	0.0015
Quebec	87	7,195	16,380	15,035	2,775	23,575	9,067	19.94	11.76		40.6	1.03	0.0015
Quebec	88	8,020	17,280	15,665	2,350	25,300	9,731	19.94	11.76		40.6	1.03	0.0015
Quebec	89	13,370	31,025	28,290	4,035	44,395	17,075	19.94	11.76		40.6	1.03	0.0015

Table 3-1 (cont.)
1996 Census Division Populations and Baseline Pollution

Province Name	Census Division	Pop under 20	Pop 20 and older	Pop 25 and older	Pop 65 and older	Total Pop	House-holds	PM10 (:g/m3)	PM2.5 (:g/m3)	SO4 (:g/m3)	Ozone (ppb)	CO (ppm)	SO2 (:g/m3)
Quebec	90	4,765	11,530	10,595	1,950	16,295	6,267	19.94	11.76		37.9	1.03	0.0015
Quebec	91	10,340	23,515	21,455	3,425	33,855	13,021	19.94	11.76		37.9	0.73	0.011
Quebec	92	8,915	19,125	17,440	2,900	28,040	10,785	17.95	10.59		37.9	0.73	0.011
Quebec	93	15,835	36,570	33,340	5,455	52,405	20,156	17.95	10.59		37.9	0.73	0.011
Quebec	94	49,180	123,165	112,335	18,300	172,345	66,287	17.95	10.59		37.9	0.73	0.011
Quebec	95	3,760	9,695	8,880	1,390	13,455	5,175	12.63	7.45		39.8	0.73	0.0052
Quebec	96	10,545	25,720	23,265	2,605	36,265	13,948	12.63	7.45		39.8	0.73	0.0052
Quebec	97	11,820	29,070	26,325	2,495	40,890	15,727	12.63	7.45		28.8	0.73	0.0052
Quebec	98	3,930	8,760	7,840	1,240	12,690	4,881	12.63	7.45		30.1	0.73	0.0052
Quebec	99	15,370	23,010	19,745	1,300	38,380	14,762	12.63	7.45		28.8	0.73	0
Ontario	1	30,870	80,420	74,040	16,705	111,290	42,804	22.15	13.07		45.5	1.52	0.006
Ontario	2	22,155	51,870	47,975	7,720	74,025	28,471	38.01	22.43		38	1.65	0.0049
Ontario	6	187,495	533,640	482,820	80,040	721,135	277,360	20.8	11.92	3.81	33.5	1.73	0.0043

Ontario	7	25,070	71,215	65,970	15,030	96,285	37,033	22.15	13.07		46.4	1.52	0.006
Ontario	9	16,490	43,340	40,370	8,870	59,830	23,012	20.8	12.27		33.5	1.73	0.0043
Ontario	10	34,240	102,115	91,260	18,485	136,355	52,444	22.15	13.07		46.4	1.52	0.006
Ontario	11	10,615	28,595	26,420	5,475	39,210	15,081	22.15	13.07		46.4	1.52	0.006
Ontario	12	32,280	86,450	79,550	18,275	118,730	45,665	25.11	14.81		42.7	1.73	0.0034
Ontario	13	6,230	18,815	17,645	4,740	25,045	9,633	22.15	13.07		46.4	1.52	0.006
Ontario	14	22,350	59,445	55,385	13,160	81,795	31,460	25.11	14.81		47.9	1.73	0.0036
Ontario	15	32,360	91,090	83,605	21,435	123,450	47,481	25.11	14.81		42.7	1.73	0.0034
Ontario	16	18,520	49,405	46,220	12,245	67,925	26,125	25.11	14.81		42.7	1.73	0.0034
Ontario	18	142,175	316,440	289,895	41,385	458,615	176,390	25.11	14.81		47.9	1.73	0.0036
Ontario	19	178,655	413,800	374,545	48,735	592,455	227,867	25.11	16.86	4.64	43.5	1.73	0.0036
Ontario	20	559,200	1,826,205	1,663,350	319,830	2,385,405	917,464	28.37	16.86	4.64	45.7	2.2	0.0048
Ontario	21	254,380	598,140	538,835	61,470	852,520	327,892	28.37	16.86	4.64	45.7	2.2	0.0048
Ontario	22	14,510	31,140	28,635	4,400	45,650	17,558	24.7	16.86	4.64	50.7	1.19	0.002

Table 3-1 (cont.)
1996 Census Division Populations and Baseline Pollution

Province Name	Census Division	Pop under 20	Pop 20 and older	Pop 25 and older	Pop 65 and older	Total Pop	House-holds	PM10 (:g/m3)	PM2.5 (:g/m3)	SO4 (:g/m3)	Ozone (ppb)	CO (ppm)	SO2 (:g/m3)
Ontario	23	48,865	122,535	110,700	20,600	171,400	65,923	24.7	16.86	4.64	50.7	1.19	0.002
Ontario	24	93,865	246,015	225,055	37,430	339,880	130,723	24.7	16.86	4.64	51.8	1.15	0.0047
Ontario	25	122,705	345,095	314,110	66,290	467,800	179,923	31.15	18.38		47	1.83	0.0083
Ontario	26	105,075	298,435	273,400	64,310	403,510	155,196	26.25	15.49		53.1	1.14	0.0043
Ontario	28	30,110	72,475	66,825	14,430	102,585	39,456	26.25	15.49		57.9	1.42	0.0041
Ontario	29	32,760	81,790	74,735	16,125	114,550	44,058	31.15	18.38		47	1.83	0.0083
Ontario	30	116,705	288,740	259,460	44,030	405,445	155,940	27.41	16.17		48.1	1.19	0.0029
Ontario	31	21,315	50,780	46,155	10,540	72,095	27,729	27.41	16.17		48.1	1.19	0.0029
Ontario	32	28,185	68,940	62,940	13,920	97,125	37,356	27.41	16.17		52	1.42	0.0041
Ontario	34	23,255	55,910	51,030	10,895	79,165	30,448	27.72	16.35		58.4	1.42	0.0041
Ontario	36	31,380	78,260	71,425	15,890	109,640	42,169	27.72	16.35		53.5	1.95	0.0061
Ontario	37	95,935	254,395	228,725	45,620	350,330	134,742	27.72	16.6	5.02	48.6	1.95	0.0061
Ontario	38	36,835	92,140	84,390	18,660	128,975	49,606	21.54	12.71		50.5	0.84	0.0094

Ontario	39	106,575	283,020	255,115	48,365	389,595	149,844	27.41	16.17		52	1.42	0.0041
Ontario	40	17,825	42,400	39,170	10,305	60,225	23,164	21.54	12.71		54.5	0.84	0.0094
Ontario	41	19,040	46,635	43,270	10,350	65,675	25,260	21.54	12.71		56.7	0.84	0.0094
Ontario	42	23,885	63,745	59,060	14,820	87,630	33,704	21.54	12.71		56.7	0.84	0.0094
Ontario	43	95,345	234,520	216,030	42,755	329,865	126,871	21.54	10.45	3.15	46.3	1.19	0.0029
Ontario	44	12,800	37,665	35,180	8,865	50,465	19,410	20.77	12.25		46.3	1.19	0.0029
Ontario	46	3,525	11,805	11,230	3,435	15,330	5,896	20.77	12.25		42.8	0.88	0.001
Ontario	47	26,385	69,850	64,115	14,275	96,235	37,014	20.77	12.25		42.8	0.88	0.001
Ontario	48	23,405	61,430	55,920	10,985	84,835	32,629	20.77	12.25		42.8	0.88	0.001
Ontario	49	9,920	29,975	28,060	7,090	39,895	15,344	20.77	12.25		42.8	0.88	0.001
Ontario	51	3,285	8,130	7,540	1,855	11,415	4,390	20.77	12.25		42.1	0.73	0.0077
Ontario	52	7,095	18,370	16,805	2,850	25,465	9,794	20.77	12.25		42.1	0.73	0.0077
Ontario	53	44,305	119,755	107,255	19,295	164,060	63,100	20.77	12.25		42.1	0.73	0.0077
Ontario	54	10,635	27,175	24,980	5,565	37,810	14,542	20.77	12.25		36.8	0.73	0.001

Table 3-1 (cont.)
1996 Census Division Populations and Baseline Pollution

Province Name	Census Division	Pop under 20	Pop 20 and older	Pop 25 and older	Pop 65 and older	Total Pop	House-holds	PM10 (:g/m3)	PM2.5 (:g/m3)	SO4 (:g/m3)	Ozone (ppb)	CO (ppm)	SO2 (:g/m3)
Ontario	56	27,940	65,310	59,100	9,790	93,250	35,865	15.91	9.39		34	0.73	0.0077
Ontario	57	34,015	91,430	83,555	17,075	125,445	48,248	20.3	11.98		37.9	0.73	0.0017
Ontario	58	43,115	114,500	103,785	20,140	157,615	60,621	20.93	12.35		34	1.09	0.001
Ontario	59	6,980	16,190	14,720	3,370	23,170	8,912	20.93	12.35		35.8	1.09	0.001
Ontario	60	21,590	41,745	37,455	6,165	63,335	24,360	15.91	9.39		34	0.73	0.0077
Manitoba	1	4,300	11,945	11,190	2,785	16,245	6,248	22.11	8.62		32.1	1.37	0.001
Manitoba	2	17,070	30,970	27,875	4,810	48,040	18,477	22.11	8.62		32.1	1.37	0.001
Manitoba	3	13,555	26,915	24,335	6,330	40,470	15,565	22.11	8.62		32.1	1.37	0.001
Manitoba	4	3,290	7,120	6,630	1,870	10,410	4,004	19.96	7.78		32.8	1.37	0.001
Manitoba	5	4,070	10,690	9,985	3,025	14,760	5,677	19.96	7.78		32.8	1.37	0.001
Manitoba	6	3,210	7,295	6,720	1,985	10,505	4,040	19.96	7.78		32.8	1.37	0.001
Manitoba	7	16,075	41,130	36,735	8,725	57,205	22,002	19.96	7.78		32.8	1.37	0.001
Manitoba	8	5,185	9,715	8,840	2,350	14,900	5,731	19.96	7.78		32.8	1.37	0.001

Manitoba	9	7,075	16,120	14,695	3,375	23,195	8,921	22.11	8.62		32.1	1.37	0.001
Manitoba	10	3,155	5,750	5,295	625	8,905	3,425	22.11	8.62		32.1	1.37	0.001
Manitoba	11	163,380	456,680	411,465	84,820	620,060	238,485	22.11	8.94	1.74	32.1	1.37	0.001
Manitoba	12	5,610	13,110	12,185	2,150	18,720	7,200	22.11	8.62		32.1	1.37	0.001
Manitoba	13	10,905	28,510	26,180	4,755	39,415	15,160	22.11	8.62		32.1	1.37	0.001
Manitoba	14	5,055	12,005	10,965	1,935	17,060	6,562	22.11	8.62		32.1	1.37	0.001
Manitoba	15	6,090	16,395	15,345	4,975	22,485	8,648	19.96	7.78		32.8	1.37	0.001
Manitoba	16	3,140	7,545	6,945	2,125	10,685	4,110	19.96	7.78		32.8	1.37	0.001
Manitoba	17	6,325	17,650	16,455	5,230	23,975	9,221	19.96	7.78		32.8	1.37	0.001
Manitoba	18	6,480	15,785	14,585	3,995	22,265	8,564	22.11	8.62		32.1	1.37	0.001
Manitoba	19	6,795	7,940	6,875	785	14,735	5,667	22.11	8.62		32.1	1.37	0.001
Manitoba	20	3,160	8,285	7,710	2,380	11,445	4,402	19.96	7.78		32.8	1.37	0.001
Manitoba	21	8,175	14,970	13,255	1,820	23,145	8,902	15.91	6.2		28.8	0.73	0
Manitoba	22	15,940	19,640	16,540	995	35,580	13,685	15.91	6.2		28.8	0.73	0

Table 3-1 (cont.)
1996 Census Division Populations and Baseline Pollution

Province Name	Census Division	Pop under 20	Pop 20 and older	Pop 25 and older	Pop 65 and older	Total Pop	House-holds	PM10 (:g/m3)	PM2.5 (:g/m3)	SO4 (:g/m3)	Ozone (ppb)	CO (ppm)	SO2 (:g/m3)
Manitoba	23	4,255	5,420	4,670	305	9,675	3,721	15.91	6.2		28.8	0.73	0
Saskatchewan	1	9,870	22,430	20,435	5,335	32,300	12,423	16.89	6.59		33.3	1.74	0.0008
Saskatchewan	2	6,600	16,515	15,335	4,695	23,115	8,890	16.89	6.59		33.3	1.74	0.0008
Saskatchewan	3	4,935	11,540	10,860	3,385	16,475	6,337	16.89	6.59		33.3	1.74	0.0008
Saskatchewan	4	3,685	8,625	7,975	2,235	12,310	4,735	16.89	6.59		33.3	1.74	0.0008
Saskatchewan	5	10,060	24,995	23,425	7,490	35,055	13,483	16.89	6.59		32	1.74	0.0008
Saskatchewan	6	65,730	154,860	139,280	27,565	220,590	84,842	16.89	6.59		32	1.74	0.0008
Saskatchewan	7	14,260	35,035	32,260	8,855	49,295	18,960	16.89	6.59		32	1.74	0.0008
Saskatchewan	8	8,970	22,680	20,950	5,880	31,650	12,173	16.89	6.59		32	1.74	0.0008
Saskatchewan	9	10,130	28,420	26,450	8,900	38,550	14,827	17.62	6.87		35.2	0.98	0
Saskatchewan	10	6,125	14,630	13,740	4,575	20,755	7,983	17.62	6.87		35.2	0.98	0
Saskatchewan	11	69,200	162,780	144,005	27,590	231,980	89,223	17.62	6.87		35.2	0.98	0
Saskatchewan	12	7,980	16,470	15,310	3,790	24,450	9,404	17.62	6.87		35.2	0.98	0

Saskatchewan	13	8,125	16,720	15,355	3,980	24,845	9,556	17.62	6.87		35.2	0.98	0
Saskatchewan	14	11,940	28,865	26,630	7,865	40,805	15,694	17.62	6.87		35.2	0.98	0
Saskatchewan	15	25,980	54,690	49,815	11,920	80,670	31,027	17.62	6.87		35.2	0.98	0
Saskatchewan	16	12,340	25,410	23,225	6,110	37,750	14,519	17.62	6.87		35.2	0.98	0
Saskatchewan	17	14,340	24,195	21,535	4,135	38,535	14,821	17.62	6.87		35.2	0.98	0
Saskatchewan	18	14,900	16,200	13,710	1,320	31,100	11,962	15.91	6.2		28.8	0.73	0
Alberta	1	18,285	44,040	40,045	8,670	62,325	23,971	27.79	10.84		38.3	2.56	0.003
Alberta	2	39,065	86,130	76,515	15,535	125,195	48,152	27.79	10.84		38.3	2.56	0.003
Alberta	3	13,355	24,410	22,215	5,065	37,765	14,525	27.79	10.84		38.3	2.56	0.003
Alberta	4	3,760	8,290	7,595	1,780	12,050	4,635	27.79	10.84		38.3	2.56	0.003
Alberta	5	14,235	29,345	26,945	5,500	43,580	16,762	27.79	10.84		38.3	2.56	0.003
Alberta	6	248,215	632,645	571,720	78,035	880,860	338,792	27.79	8.56	1.3	38.3	2.56	0.003
Alberta	7	12,880	28,290	25,900	5,930	41,170	15,835	25.95	10.12		39.9	2.27	0.0027
Alberta	8	41,450	92,135	83,290	14,550	133,585	51,379	25.95	10.12		39.9	2.27	0.0027

Table 3-1 (cont.)
1996 Census Division Populations and Baseline Pollution

Province Name	Census Division	Pop under 20	Pop 20 and older	Pop 25 and older	Pop 65 and older	Total Pop	House-holds	PM10 (:g/m3)	PM2.5 (:g/m3)	SO4 (:g/m3)	Ozone (ppb)	CO (ppm)	SO2 (:g/m3)
Alberta	9	6,195	11,995	10,885	1,735	18,190	6,996	25.95	10.12		40.6	2.04	0.0023
Alberta	10	23,615	56,395	52,235	13,030	80,010	30,773	24.25	9.46		40	2.04	0.0023
Alberta	11	261,470	637,420	574,115	88,240	898,890	345,727	24.25	10.23	1.59	39.9	2.27	0.0027
Alberta	12	19,920	36,580	33,275	5,415	56,500	21,731	24.25	9.46		39.9	2.27	0.0027
Alberta	13	19,510	43,055	39,605	7,820	62,565	24,064	24.25	9.46		39.9	2.27	0.0027
Alberta	14	9,165	18,285	16,595	2,010	27,450	10,558	24.25	9.46		39.9	2.27	0.0027
Alberta	15	7,740	23,070	19,465	2,655	30,810	11,850	27.79	10.84		38.3	2.56	0.003
Alberta	16	12,930	23,570	20,860	670	36,500	14,039	15.91	6.2		39	0.98	0.0028
Alberta	17	22,345	32,365	28,205	2,970	54,710	21,042	15.91	6.2		39	0.98	0.0028
Alberta	18	5,385	9,635	8,635	750	15,020	5,777	15.91	6.2		39	0.98	0.0028
Alberta	19	26,440	53,215	47,270	6,545	79,655	30,637	15.91	6.2		39	0.98	0.0028
British Columbia	1	16,365	40,010	36,695	6,320	56,375	21,683	20.07	11.84		37.1	2.13	0.0001
British Columbia	3	16,090	41,990	39,030	8,570	58,080	22,339	20.07	11.84		37.1	2.13	0.0001

British Columbia	5	8,795	24,110	22,640	5,570	32,905	12,656	20.07	11.84		37.1	2.13	0.0001
British Columbia	7	17,535	58,400	55,075	18,045	75,935	29,206	20.07	11.84		37.1	2.13	0.0001
British Columbia	9	67,015	155,380	142,050	30,420	222,395	85,537	20.65	12.18		38.1	2.13	0.0001
British Columbia	15	451,465	1,380,200	1,252,230	216,425	1,831,665	704,487	20.65	13.86	1.88	32.2	2.13	0.0054
British Columbia	17	71,000	247,015	226,095	57,470	318,015	122,314	15.58	10.03	1.71	31	2.13	0.0013
British Columbia	19	20,170	50,810	47,475	10,690	70,980	27,300	15.58	9.19		28.7	2.13	0.0013
British Columbia	21	30,975	90,805	84,250	20,920	121,780	46,839	15.58	9.19		28.7	2.13	0.0013
British Columbia	23	9,300	22,355	20,495	3,700	31,655	12,175	15.58	9.19		28.7	2.13	0.0001
British Columbia	25	28,445	69,195	64,205	10,755	97,640	37,554	15.58	9.19		28.7	2.13	0.0001
British Columbia	27	5,340	14,605	13,645	2,935	19,945	7,671	15.58	9.19		32.2	2.13	0.0001
British Columbia	29	6,480	18,435	17,480	4,295	24,915	9,583	15.58	9.19		32.2	2.13	0.0001
British Columbia	31	8,240	21,170	18,660	1,745	29,410	11,312	28.28	16.69		32.7	2.13	0.0001
British Columbia	33	34,325	84,490	76,550	12,850	118,815	45,698	28.28	16.69		37.1	2.13	0.0001
British Columbia	35	34,850	101,685	93,705	23,355	136,535	52,514	20.07	11.84		37.1	2.13	0.0001

Table 3-1 (cont.)
1996 Census Division Populations and Baseline Pollution

Province Name	Census Division	Pop under 20	Pop 20 and older	Pop 25 and older	Pop 65 and older	Total Pop	House-holds	PM10 (:g/m3)	PM2.5 (:g/m3)	SO4 (:g/m3)	Ozone (ppb)	CO (ppm)	SO2 (:g/m3)
British Columbia	37	19,795	51,810	48,135	11,555	71,605	27,540	20.07	11.84		37.1	2.13	0.0001
British Columbia	39	13,230	34,885	32,635	7,150	48,115	18,506	20.07	11.84		37.1	2.13	0.0001
British Columbia	41	21,045	45,440	41,440	5,655	66,485	25,571	20.07	11.84		32.7	2.13	0.0001
British Columbia	43	4,915	9,690	8,775	595	14,605	5,617	15.58	9.19		28.7	2.13	0.0001
British Columbia	45	1,305	2,630	2,370	255	3,935	1,514	15.58	9.19		28.7	2.13	0.0001
British Columbia	47	8,165	16,620	14,895	1,495	24,785	9,533	15.58	9.19		28.7	0.73	0.0001
British Columbia	49	14,990	28,635	25,575	2,565	43,625	16,779	15.58	9.19		28.7	0.73	0.0001
British Columbia	51	14,520	27,125	24,340	2,910	41,645	16,017	25.49	15.04		32.7	0.73	0.0001
British Columbia	53	31,925	67,060	59,800	5,645	98,985	38,071	25.49	15.04		32.7	0.73	0.0001
British Columbia	55	19,280	37,185	33,230	3,740	56,465	21,717	15.91	9.39		28.8	0.73	0.0001
British Columbia	57	430	970	870	70	1,400	539	15.91	9.39		28.8	0.73	0.0001
British Columbia	59	2,110	3,730	3,245	130	5,840	2,246	15.91	9.39		28.8	0.73	0.0001
Yukon	1	9,410	21,350	19,290	1,350	30,760	11,831	15.91	6.2		28.8	0.73	0
Northwest Territories	4	6,165	7,065	5,980	260	13,230	5,089	15.91	6.2		28.8	0.73	0

Northwest Territories	5	3,390	3,475	2,850	145	6,865	2,640	15.91	6.2		28.8	0.73	0
Northwest Territories	6	10,740	19,500	17,260	1,025	30,240	11,631	31.61	12.33		28.8	0.73	0
Northwest Territories	7	3,635	5,390	4,740	350	9,025	3,471	15.91	6.2		28.8	0.73	0
Northwest Territories	8	2,430	2,650	2,240	150	5,080	1,954	15.91	6.2		28.8	0.73	0

Table 3-2
1996 Census Metropolitan Area (CMA) Populations and Baseline Pollution

Province	CMA	CMA Name	Pop under 20	Pop 20 and Older	Pop 25 and Older	Pop 65 and Older	Total Pop	House-holds	PM_{10} (g/m^3)	$PM_{2.5}$ (g/m^3)	SO_4 (g/m^3)	Ozone (ppb)	CO (ppm)	SO_2 (g/m^3)
Newfoundland	1	St. John s	47,520	126,530	111,690	17,390	174,050	66,942				22.46		
Newfoundland	10	Grand Falls-Windsor	5,740	14,638	13,060	2,197	20,378	7,838						
Newfoundland	11	Gander	3,386	8,635	7,704	1,296	12,021	4,623						
Newfoundland	15	Corner Brook	7,872	20,073	17,909	3,013	27,945	10,748						
Newfoundland	25	Labrador City	2,950	7,523	6,712	1,129	10,473	4,028						
Prince Edward Island	105	Charlottetown	16,653	40,571	36,640	40,571 7,422	57,224	22,009						
Prince Edward Island	110	Summerside	4,657	11,344	10,234	2,075	16,001	6,154						
Nova Scotia	205	Halifax	86,685	245,825	220,725	33,585	332,510	127,888	13.92	8.56		37.12		
Nova Scotia	210	Kentville	6,681	18,409	16,708	18,409 3,286	25,090		9,650					
Nova Scotia	215	Truro	11,743	32,359	29,368	5,776	44,102	16,962						
Nova Scotia	220	New Glasgow	10,133	27,922	25,342	4,984	38,055	14,637						
Nova Scotia	225	Cape Breton	31,379	86,470	78,478	15,435	117,849	45,327						
New Brunswick	305	Moncton	28,555	84,930	76,145	14,455	113,485	43,648						
New Brunswick	310	Saint John	34,750	90,965	82,205	15,865	125,715	48,352	12.14	8.49		35.37		
New Brunswick	320	Fredericton	21,159	57,792	52,151	9,966	78,950	30,365						
New Brunswick	328	Bathurst	6,811	18,604	16,788	3,208	25,415		9,775					
New Brunswick	330	Campbellton	4,528	12,369	11,166	2,133	16,897	6,499						
New Brunswick	335	Edmundston	6,063	16,561	14,944	2,856	22,624		8,702					
Quebec	403	Matane	4,475	12,643	11,554	2,064	17,118	6,584						
Quebec	404	Rimouski	12,577	35,527	32,247	35,527 5,800	48,104	18,502						
Quebec	405	Rivière-du-Loup	5,851	16,527	15,105	2,698	22,378		8,607					
Quebec	406	Baie-Comeau	8,313	23,482	21,461	3,833	31,795	12,229						
Quebec	408	Chicoutimi - Jonquière	45,385	115,070	104,940	17,285	160,455	61,713	30.34					
Quebec	410	Alma	7,944	22,429	20,508	3,663	30,383	11,686						
Quebec	411	Dolbeau	3,978	11,236	10,269	1,834	15,214	5,852						

Table 3-2 (cont.)
1996 Census Metropolitan Area (CMA) Populations and Baseline Pollution

Province	CMA	CMA Name	Pop under 20	Pop 20 and Older	Pop 25 and Older	Pop 65 and Older	Total Pop	House-holds	PM_{10} (g/m^3)	$PM_{2.5}$ (g/m^3)	SO_4 (g/m^3)	Ozone (ppb)	CO (ppm)	SO_2 (g/m^3)
Quebec	412	Sept-Îles	7,322	20,683	18,903	3,376	28,005	10,771						
Quebec	421	Québec	164,360	164,537	460,120	78,180	677,180	258,419	21.21	9		38.27		
Quebec	428	Saint-Georges	6,950	19,634	17,944	3,205	26,584	10,225						
Quebec	430	Thetford Mines	7,258	20,502	18,738	3,347	27,760	10,677						
Quebec	433	Sherbrooke	38,570	108,810	97,195	18,015	147,380	56,685						
Quebec	435	Magog	5,578	15,756	14,400	2,572	21,334	8,205						
Quebec	437	Cowansville	3,151	8,900	8,134	1,453	12,051	4,635						
Quebec	440	Victoriaville	10,572	29,866	27,295	4,875	40,438	15,553						
Quebec	442	Trois-Rivières	35,235	104,715	95,735	18,370	139,950	53,827	18.94					
Quebec	444	Shawinigan	15,648	44,203	40,399	7,216	59,851	23,020	32.57					
Quebec	446	La Tuque	3,442	9,723	8,886	1,587	13,165	5,063						
Quebec	447	Drummondville	17,025	48,094	43,954	7,851	65,119	25,046						
Quebec	450	Granby	15,392	43,480	39,738	7,098	58,872	22,643						
Quebec	452	Saint-Hyacinthe	13,080	36,947	33,768	6,032	50,027	19,241						
Quebec	454	Sorel	11,245	31,764	29,030	5,185	43,009	16,542	22.71					
Quebec	456	Joliette	8,992	25,399	23,213	4,146	34,391	13,227						
Quebec	459	Saint-Jean-sur-Richelieu	21,185	55,275	50,845	8,915	76,460	29,408						
Quebec	462	Montréal	836,730	2,439,795	2,270,205	403,135	3,276,525	1,279,433	29.07	11.69		44.67		
Quebec	465	Salaberry-de-Valleyfield	10,344	29,219	26,704	4,770	39,563	15,217						
Quebec	468	Lachute	3,005	8,488	7,758	1,386	11,493	4,420						
Quebec	480	Val-d Or	8,536	24,112	22,037	3,936	32,648	12,557						
Quebec	485	Rouyn-Noranda	10,222	28,874	26,389	4,714	39,096	15,037						
Quebec	505	Ottawa - Hull	50,002	177,504	160,895	24,641	227,506	87,502				38.04		
Ontario	501	Cornwall	16,840	45,343	41,275	7,714	62,183	23,917	22.69			44.84		
Ontario	502	Hawkesbury	3,143	8,462	7,703	1,440	11,605	4,463						
Ontario	505	Ottawa - Hull	205,876	562,096	509,500	78,029	767,972	295,374	16.41	8.92		40.39		
Ontario	508	SmithsSmiths Falls	4,470	12,037	10,957	2,048	16,507	6,349						
Ontario	512	Brockville	11,566	31,143	28,349	5,299	42,709	16,427						
Ontario	515	Pembroke	6,431	17,314	15,761	2,946	23,745	9,133						
Ontario	521	Kingston	36,375	107,040	95,605	18,690	143,415	55,160	15.98			47.97		

Ontario	522	Belleville	25,260	68,199	62,510	13,875	93,450	35,942						
Ontario	527	Cobourg	4,340	11,687	10,638	1,988	16,027	6,164						

Table 3-2 (cont.)
1996 Census Metropolitan Area (CMA) Populations and Baseline Pollution

Province	CMA	CMA Name	Pop under 20	Pop 20 and Older	Pop 25 and Older	Pop 65 and Older	Total Pop	House-holds	PM_{10} (g/m^3)	$PM_{2.5}$ (g/m^3)	SO_4 (g/m^3)	Ozone (ppb)	CO (ppm)	SO_2 (g/m^3)
Ontario	528	Port Hope	3,168	8,530	7,765	1,451	11,698	4,499						
Ontario	529	Peterborough	26,345	73,850	67,410	17,425	100,195	38,537				48.03		
Ontario	530	Lindsay	5,944	16,005	14,569	2,723	21,949	8,442						
Ontario	532	Oshawa	81,350	187,420	171,070	26,415	268,770	103,373				48.63		
Ontario	535	Toronto	1,124,720	3,139,025	2,852,230	467,580	4,263,745	1,639,902	24.19	14.42		51.35		
Ontario	537	Hamilton	163,685	460,685	420,345	86,590	624,370	240,142	27.39	17.97		55.18		
Ontario	539	St. Catharines - Niagara	95,645	276,760	253,460	60,670	372,405	143,233	21.69			54.47		
Ontario	541	Kitchener	109,035	273,895	246,005	41,650	382,930	147,281				52.22		
Ontario	543	Brantford	28,425	71,810	65,470	14,480	100,235	38,552						
Ontario	544	Woodstock	8,689	23,397	21,298	3,981	32,086	12,341						
Ontario	546	Tillsonburg	3,578	9,633	8,769	1,639	13,211	5,081						
Ontario	547	Simcoe	4,165	11,215	10,209	1,908	15,380	5,915				59.24		
Ontario	550	Guelph	28,325	77,100	68,960	12,480	105,425	40,548				49.24		
Ontario	553	Stratford	7,850	21,137	19,241	3,596	28,987	11,149						
Ontario	555	London	107,570	291,050	262,260	50,310	398,620	153,315	19.44					
Ontario	556	Chatham	18,163	48,905	44,517	8,320	67,068	25,795						
Ontario	557	Leamington	11,019	29,668	27,007	5,048	40,687	15,649						
Ontario	558	Strathroy	3,210	8,642	7,867	1,470	11,852	4,558						
Ontario	559	Windsor	74,460	204,215	183,230	36,080	278,675	107,183	25.52	17.5		56.26		
Ontario	562	Sarnia	24,130	62,350	56,940	12,375	86,480	33,262	20.91			50.51		
Ontario	566	Owen Sound	8,211	22,108	20,125	3,761	30,319	11,661						
Ontario	567	Collingwood	4,224	11,372	10,352	1,935	15,596	5,998						
Ontario	568	Barrie	35,800	82,900	75,715	13,155	118,700	45,654				48.14		
Ontario	569	Orillia	10,319	27,784	25,291	4,727	38,103	14,655						
Ontario	571	Midland	9,016	24,275	22,097	4,130	33,291	12,804						
Ontario	575	North Bay	17,965	46,825	42,390	8,145	64,790	24,919				45.63		
Ontario	580	Sudbury	43,270	117,235	104,945	18,860	160,505	61,733	17.49			45.92		
Ontario	582	Elliot Lake	3,680	9,908	9,019	1,686	13,588	5,226						
Ontario	584	Haileybury	3,713	9,999	9,102	1,701	13,712	5,274						
Ontario	586	Timmins	12,864	34,635	31,528	5,893	47,499	18,269						
Ontario	590	Sault Ste. Marie	22,305	61,310	55,680	11,575	83,615	32,160	17.12			41.61		
Ontario	595	Thunder Bay	32,900	92,665	83,955	17,670	125,565	48,294	17.69			35.47		

Table 3-2 (cont.)
1996 Census Metropolitan Area (CMA) Populations and Baseline Pollution

Province	CMA	CMA Name	Pop under 20	Pop 20 and Older	Pop 25 and Older	Pop 65 and Older	Total Pop	House-holds	PM_{10} (g/m^3)	$PM_{2.5}$ (g/m^3)	SO_4 (g/m^3)	Ozone (ppb)	CO (ppm)	SO_2 (g/m^3)
Ontario	598	Kenora	4,432	11,933	10,863	2,030	16,365	6,294						
Manitoba	602	Winnipeg	178,345	488,880	441,055	88,815	667,225	256,625	20.2	8.07		35.64		
Manitoba	607	Portage la Prairie	5,898	14,487	13,094	2,784	20,385	7,840						
Manitoba	610	Brandon	11,742	28,839	26,066	5,543	40,581	15,608				42.39		
Manitoba	640	Thompson	4,162	10,223	9,240	1,965	14,385	5,533						
Saskatchewan	705	Regina	57,200	136,440	122,110	22,765	193,640	74,477	18.31			32.41		
Saskatchewan	710	Yorkton	5,459	12,254	11,096	2,605	17,713	6,813						
Saskatchewan	715	Moose Jaw	10,734	24,095	21,817	5,122	34,829	13,396						
Saskatchewan	720	Swift Current	5,066	11,371	10,296	2,417	16,437	6,322						
Saskatchewan	725	Saskatoon	65,580	153,455	135,285	24,470	219,035	84,244	18.48			35.06		
Saskatchewan	735	North Battleford	5,543	12,444	11,267	2,645	17,987	6,918						
Saskatchewan	745	Prince Albert	12,853	28,853	26,125	6,133	41,706	16,041						
Saskatchewan	750	Estevan	3,900	8,756	7,928	1,861	12,656	4,868						
Alberta	805	Medicine Hat	16,906	39,664	35,773	5,599	56,570	21,758						
Alberta	810	Lethbridge	17,010	46,040	40,620	9,235	63,050	24,250						
Alberta	825	Calgary	229,385	592,250	534,305	71,480	821,635	316,013	14.12	8.66		45.85		
Alberta	830	Red Deer	18,220	41,850	36,770	5,705	60,070	23,104						
Alberta	833	Camrose	4,103	9,625	8,681	1,359	13,728	5,280						
Alberta	835	Edmonton	249,625	612,990	551,745	84,150	862,615	331,775	17.34	8.12		49.9		
Alberta	840	Lloydminster	5,664	13,289	11,985	1,876	18,953	7,290						
Alberta	845	Grand Centre	10,508	24,653	22,234	3,480	35,161	13,523						
Alberta	850	Grande Prairie	9,306	21,834	19,692	3,082	31,140	11,977						
Alberta	860	Wood Buffalo	10,796	25,328	22,843	3,575	36,124	13,894				36.71		
Alberta	865	Wetaskiwin	3,275	7,684	6,930	1,085	10,959	4,215						
British Columbia	905	Cranbrook	4,761	13,370	12,197	2,316	18,131	6,973	16.55					
British Columbia	913	Penticton	10,839	30,437	27,767	5,273	41,276	15,875						
British Columbia	915	Kelowna	34,855	101,680	93,700	23,350	136,535	52,513	16.4			43.52		
British Columbia	918	Vernon	14,538	40,821	37,241	7,072	55,359	21,292						

Table 3-2 (cont.)
1996 Census Metropolitan Area (CMA) Populations and Baseline Pollution

Province	CMA	CMA Name	Pop under 20	Pop 20 and Older	Pop 25 and Older	Pop 65 and Older	Total Pop	House-holds	PM_{10} (g/m^3)	$PM_{2.5}$ (g/m^3)	SO_4 (g/m^3)	Ozone (ppb)	CO (ppm)	SO_2 (g/m^3)
British Columbia	925	Kamloops	24,085	60,840	54,675	8,960	84,925	32,663	15.99					
British Columbia	930	Chilliwack	17,399	48,855	44,571	8,464	66,254	25,482	14.36	7.75		37.35		
British Columbia	932	Abbotsford	42,065	94,410	85,765	17,465	136,475	52,490	17.37			34.88		
British Columbia	933	Vancouver	451,470	1,380,200	1,252,235	216,435	1,831,670	704,488	13.89	7.8		40.17		
British Columbia	935	Victoria	68,080	236,200	215,780	54,400	304,280	117,031	13.67	7.57		32.96		
British Columbia	937	Duncan	9,402	26,401	24,086	4,574	35,803	13,770						
British Columbia	938	Nanaimo	23,130	62,460	57,265	12,300	85,590	32,919						
British Columbia	940	Port Alberni	7,062	19,831	18,092	3,436	26,893	10,343	10.32					
British Columbia	943	Courtenay	14,420	40,492	36,941	7,015	54,912	21,120						
British Columbia	944	Campbell River	9,239	25,944	23,669	4,495	35,183	13,532	12.13					
British Columbia	945	Powell River	5,235	14,701	13,411	2,547	19,936	7,668						
British Columbia	950	Williams Lake	10,124	28,428	25,935	4,925	38,552	14,828	20.03					
British Columbia	952	Quesnel	6,639	18,640	17,006	3,230	25,279	9,723	20.1					
British Columbia	955	Prince Rupert	4,573	12,841	11,715	2,225	17,414	6,698						
British Columbia	960	Kitimat	2,924	8,212	7,491	1,423	11,136	4,283						
British Columbia	965	Terrace	5,499	15,442	14,088	2,675	20,941	8,054						
British Columbia	970	Prince George	23,950	51,205	45,310	4,555	75,155	28,906	22.07					

Table 3-2 (cont.)
1996 Census Metropolitan Area (CMA) Populations and Baseline Pollution

Province	CMA	CMA Name	Pop under 20	Pop 20 and Older	Pop 25 and Older	Pop 65 and Older	Total Pop	House-holds	PM_{10} (g/m^3)	$PM_{2.5}$ (g/m^3)	SO_4 (g/m^3)	Ozone (ppb)	CO (ppm)	SO_2 (g/m^3)
British Columbia	975	Dawson Creek	2,922	8,203	7,484	1,421	11,125	4,279						
British Columbia	977	Fort St. John	3,945	11,076	10,105	1,919	15,021	5,777						
Yukon	990	Whitehorse	6,671	15,137	13,676	957	21,808	8,388						
Northwest Territories	995	Yellowknife	7,067	10,208	8,865	517	17,275	6,644						

Table 3-3
Provincial Population Growth Factors from 1996 Baseline

Year	Nfld.	P.E.I.	N.S.	N.B.	Que.	Ont.	Man.	Sask.	Alta.	B.C.	Yukon	N.W.T.
1996	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000	1.0000
1997	0.9995	1.0060	1.0040	1.0029	1.0089	1.0171	1.0045	1.0007	1.0138	1.0220	1.0202	1.0181
1998	0.9985	1.0119	1.0078	1.0055	1.0175	1.0341	1.0091	1.0012	1.0275	1.0432	1.0375	1.0361
1999	0.9967	1.0179	1.0113	1.0078	1.0260	1.0510	1.0138	1.0018	1.0408	1.0637	1.0519	1.0557
2000	0.9945	1.0231	1.0145	1.0097	1.0342	1.0680	1.0187	1.0023	1.0541	1.0833	1.0634	1.0753
2001	0.9918	1.0291	1.0176	1.0113	1.0423	1.0849	1.0235	1.0029	1.0674	1.1026	1.0749	1.0964
2002	0.9887	1.0343	1.0204	1.0126	1.0501	1.1016	1.0283	1.0035	1.0808	1.1214	1.0836	1.1160
2003	0.9852	1.0387	1.0231	1.0137	1.0577	1.1184	1.0329	1.0042	1.0942	1.1399	1.0951	1.1370
2004	0.9814	1.0440	1.0254	1.0145	1.0651	1.1351	1.0378	1.0050	1.1075	1.1579	1.1037	1.1566
2005	0.9772	1.0477	1.0276	1.0151	1.0725	1.1518	1.0425	1.0060	1.1209	1.1756	1.1153	1.1777
2006	0.9727	1.0522	1.0297	1.0157	1.0796	1.1685	1.0473	1.0071	1.1342	1.1931	1.1239	1.1988
2007	0.9679	1.0566	1.0315	1.0159	1.0867	1.1852	1.0521	1.0082	1.1475	1.2104	1.1326	1.2199
2008	0.9627	1.0604	1.0332	1.0159	1.0937	1.2019	1.0569	1.0095	1.1607	1.2275	1.1412	1.2395
2009	0.9576	1.0641	1.0348	1.0158	1.1005	1.2186	1.0616	1.0109	1.1740	1.2446	1.1470	1.2620
2010	0.9521	1.0678	1.0363	1.0157	1.1072	1.2353	1.0662	1.0124	1.1873	1.2615	1.1556	1.2831
2011	0.9464	1.0708	1.0376	1.0155	1.1138	1.2520	1.0709	1.0138	1.2004	1.2784	1.1614	1.3042
2012	0.9406	1.0745	1.0388	1.0154	1.1203	1.2687	1.0756	1.0152	1.2135	1.2952	1.1671	1.3268
2013	0.9345	1.0775	1.0398	1.0151	1.1267	1.2853	1.0803	1.0167	1.2265	1.3119	1.1700	1.3494
2014	0.9285	1.0805	1.0406	1.0147	1.1330	1.3020	1.0850	1.0182	1.2394	1.3285	1.1758	1.3705
2015	0.9224	1.0827	1.0414	1.0145	1.1392	1.3186	1.0897	1.0197	1.2521	1.3451	1.1787	1.3931
2016	0.9294	1.0897	1.0484	1.0215	1.1462	1.3256	1.0967	1.0267	1.2591	1.3521	1.1857	1.4001
2017	0.9364	1.0967	1.0554	1.0285	1.1532	1.3326	1.1037	1.0337	1.2661	1.3591	1.1927	1.4071
2018	0.9434	1.1037	1.0624	1.0355	1.1602	1.3396	1.1107	1.0407	1.2731	1.3661	1.1997	1.4141
2019	0.9504	1.1107	1.0694	1.0425	1.1672	1.3466	1.1177	1.0477	1.2801	1.3731	1.2067	1.4211
2020	0.9574	1.1177	1.0764	1.0495	1.1742	1.3536	1.1247	1.0547	1.2871	1.3801	1.2137	1.4281
2021	0.9644	1.1247	1.0834	1.0565	1.1812	1.3606	1.1317	1.0617	1.2941	1.3871	1.2207	1.4351
2022	0.9714	1.1317	1.0904	1.0635	1.1882	1.3676	1.1387	1.0687	1.3011	1.3941	1.2277	1.4421
2023	0.9784	1.1387	1.0974	1.0705	1.1952	1.3746	1.1457	1.0757	1.3081	1.4011	1.2347	1.4491
2024	0.9854	1.1457	1.1044	1.0775	1.2022	1.3816	1.1527	1.0827	1.3151	1.4081	1.2417	1.4561
2025	0.9924	1.1527	1.1114	1.0845	1.2092	1.3886	1.1597	1.0897	1.3221	1.4151	1.2487	1.4631
2026	0.9994	1.1597	1.1184	1.0915	1.2162	1.3956	1.1667	1.0967	1.3291	1.4221	1.2557	1.4701
2027	1.0064	1.1667	1.1254	1.0985	1.2232	1.4026	1.1737	1.1037	1.3361	1.4291	1.2627	1.4771
2028	1.0134	1.1737	1.1324	1.1055	1.2302	1.4096	1.1807	1.1107	1.3431	1.4361	1.2697	1.4841
2029	1.0204	1.1807	1.1394	1.1125	1.2372	1.4166	1.1877	1.1177	1.3501	1.4431	1.2767	1.4911
2030	1.0274	1.1877	1.1464	1.1195	1.2442	1.4236	1.1947	1.1247	1.3571	1.4501	1.2837	1.4981
2031	1.0344	1.1947	1.1534	1.1265	1.2512	1.4306	1.2017	1.1317	1.3641	1.4571	1.2907	1.5051
2032	1.0414	1.2017	1.1604	1.1335	1.2582	1.4376	1.2087	1.1387	1.3711	1.4641	1.2977	1.5121
2033	1.0484	1.2087	1.1674	1.1405	1.2652	1.4446	1.2157	1.1457	1.3781	1.4711	1.3047	1.5191
2034	1.0554	1.2157	1.1744	1.1475	1.2722	1.4516	1.2227	1.1527	1.3851	1.4781	1.3117	1.5261
2035	1.0624	1.2227	1.1814	1.1545	1.2792	1.4586	1.2297	1.1597	1.3921	1.4851	1.3187	1.5331

* Provincial population projections are only available to 2015. The country-wide projections are used for all provinces for 2016-2035.

Source: George et al. 1994.

Table 3-4
Baseline Air Toxics and Visual Range Data by Census Division

Province Name	Census Division	Acetaldehyde (µg/m^3)	Benzene (µg/m^3)	Butadiene (µg/m^3)	Formaldehyde (µg/m^3)	Visual Range (km)
Newfoundland	1	1.0219	0.96	0.1073	1.4896	50
Newfoundland	2	1.0219	0.96	0.1073	1.4896	50
Newfoundland	3	1.0219	0.96	0.1073	1.4896	50
Newfoundland	4	1.0219	0.96	0.1073	1.4896	50
Newfoundland	5	1.0219	0.96	0.1073	1.4896	50
Newfoundland	6	1.0219	0.96	0.1073	1.4896	50
Newfoundland	7	1.0219	0.96	0.1073	1.4896	50
Newfoundland	8	1.0219	0.96	0.1073	1.4896	50
Newfoundland	9	1.0219	0.96	0.1073	1.4896	50
Newfoundland	10	1.0219	0.96	0.1073	1.4896	50
Prince Edward Island	1	1.0219	0.96	0.1073	1.4896	30
Prince Edward Island	2	1.0219	0.96	0.1073	1.4896	30
Prince Edward Island	3	1.0219	0.96	0.1073	1.4896	30
Nova Scotia	1	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	2	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	3	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	4	1.0219	0.36	0.5639	1.4896	20
Nova Scotia	5	1.0219	0.36	0.5639	1.4896	20
Nova Scotia	6	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	7	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	8	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	9	1.0219	2.93	0.5639	1.4896	20
Nova Scotia	10	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	11	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	12	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	13	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	14	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	15	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	16	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	17	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	18	1.0219	1.65	0.5639	1.4896	20
New Brunswick	1	1.1181	0.96	0.1073	1.5517	30
New Brunswick	2	1.1181	0.45	0.1073	1.5517	30
New Brunswick	3	1.1181	0.96	0.1073	1.5517	30
New Brunswick	4	1.1181	0.96	0.1073	1.5517	30
New Brunswick	5	1.1181	1.47	0.1073	1.5517	30
New Brunswick	6	1.1181	0.96	0.1073	1.5517	30
New Brunswick	7	1.1181	0.96	0.1073	1.5517	30
New Brunswick	8	1.1181	0.96	0.1073	1.5517	30
New Brunswick	9	1.1181	0.96	0.1073	1.5517	30
New Brunswick	10	1.1181	0.96	0.1073	1.5517	30
New Brunswick	11	1.1181	0.96	0.1073	1.5517	30

Table 3-4 (cont.)
Baseline Air Toxics and Visual Range Data by Census Division

Province Name	Census Division	Acetaldehyde (µg/m^3)	Benzene (µg/m^3)	Butadiene (µg/m^3)	Formaldehyde (µg/m^3)	Visual Range (km)
New Brunswick	12	1.1181	0.96	0.1073	1.5517	30
New Brunswick	13	1.1181	0.96	0.1073	1.5517	30
New Brunswick	14	1.1181	0.96	0.1073	1.5517	30
New Brunswick	15	1.1181	0.96	0.1073	1.5517	30
Quebec	1	2.3626	2.56	0.4389	3.4147	60
Quebec	2	2.3626	2.56	0.4389	3.4147	60
Quebec	3	2.3626	2.56	0.4389	3.4147	60
Quebec	4	2.3626	2.56	0.4389	3.4147	60
Quebec	5	2.3626	2.56	0.4389	3.4147	60
Quebec	6	2.3626	2.56	0.4389	3.4147	60
Quebec	7	2.3626	2.56	0.4389	3.4147	60
Quebec	8	2.3626	2.56	0.4389	3.4147	60
Quebec	9	2.3626	2.56	0.4389	3.4147	60
Quebec	10	2.3626	2.56	0.4389	3.4147	60
Quebec	11	2.3626	2.56	0.4389	3.4147	60
Quebec	12	2.3626	2.56	0.4389	3.4147	60
Quebec	13	2.3626	2.56	0.4389	3.4147	60
Quebec	14	2.3626	2.56	0.4389	3.4147	60
Quebec	15	2.3626	2.56	0.4389	3.4147	60
Quebec	16	2.3626	2.56	0.4389	3.4147	60
Quebec	17	2.3626	2.56	0.4389	3.4147	60
Quebec	18	2.3626	2.56	0.4389	3.4147	60
Quebec	19	2.3626	2.56	0.4389	3.4147	60
Quebec	20	2.3626	2.56	0.4389	3.4147	60
Quebec	21	2.3626	2.56	0.4389	3.4147	60
Quebec	22	2.3626	2.56	0.4389	3.4147	60
Quebec	23	2.3626	2.56	0.4389	3.4147	60
Quebec	24	2.3626	2.56	0.4389	3.4147	60
Quebec	25	2.3626	2.56	0.4389	3.4147	60
Quebec	26	2.3626	2.56	0.4389	3.4147	60
Quebec	27	2.3626	2.56	0.4389	3.4147	60
Quebec	28	2.3626	2.56	0.4389	3.4147	60
Quebec	29	2.3626	2.56	0.4389	3.4147	60
Quebec	30	2.3626	2.56	0.4389	3.4147	60
Quebec	31	2.3626	2.56	0.4389	3.4147	60
Quebec	32	2.3626	0.6	0.4389	3.4147	60
Quebec	33	2.3626	0.6	0.4389	3.4147	60
Quebec	34	2.3626	0.6	0.4389	3.4147	60
Quebec	35	2.3626	2.56	0.4389	3.4147	60
Quebec	36	2.3626	2.56	0.4389	3.4147	60
Quebec	37	2.3626	2.56	0.4389	3.4147	60
Quebec	38	2.3626	0.6	0.4389	3.4147	60

Table 3-4 (cont.)
Baseline Air Toxics and Visual Range Data by Census Division

Province Name	Census Division	Acetaldehyde (µg/m^3)	Benzene (µg/m^3)	Butadiene (µg/m^3)	Formaldehyde (µg/m^3)	Visual Range (km)
Quebec	39	2.3626	0.6	0.4389	3.4147	60
Quebec	40	2.3626	2.56	0.4389	3.4147	60
Quebec	41	2.3626	2.56	0.4389	3.4147	60
Quebec	42	2.3626	2.56	0.4389	3.4147	60
Quebec	43	2.3626	2.56	0.4389	3.4147	60
Quebec	44	2.3626	2.56	0.4389	3.4147	60
Quebec	45	2.3626	2.56	0.4389	3.4147	60
Quebec	46	2.3626	0.39	0.4389	3.4147	60
Quebec	47	2.3626	2.56	0.4389	3.4147	60
Quebec	48	2.3626	2.56	0.4389	3.4147	60
Quebec	49	2.3626	2.56	0.4389	3.4147	60
Quebec	50	2.3626	2.56	0.4389	3.4147	60
Quebec	51	2.3626	2.56	0.4389	3.4147	60
Quebec	52	2.3626	0.94	0.4389	3.4147	60
Quebec	53	2.3626	0.94	0.4389	3.4147	60
Quebec	54	2.3626	2.56	0.4389	3.4147	60
Quebec	55	2.3626	2.56	0.4389	3.4147	60
Quebec	56	2.3626	2.56	0.4389	3.4147	60
Quebec	57	2.3626	0.94	0.4389	3.4147	60
Quebec	58	2.3626	3.24	0.4389	3.4147	60
Quebec	59	2.3626	3.96	0.4389	3.4147	60
Quebec	60	2.3626	3.96	0.4389	3.4147	60
Quebec	61	2.3626	0.94	0.4389	3.4147	60
Quebec	62	2.3626	2.56	0.4389	3.4147	60
Quebec	63	2.3626	0.94	0.4389	3.4147	60
Quebec	64	2.3626	5.33	0.4389	3.4147	60
Quebec	65	2.3626	2.56	0.4389	3.4147	60
Quebec	66	2.3626	4.08	0.4389	3.4147	60
Quebec	67	2.3626	1.31	0.4389	3.4147	60
Quebec	68	2.3626	2.56	0.4389	3.4147	60
Quebec	69	2.3626	0.6	0.4389	3.4147	60
Quebec	70	2.3626	0.6	0.4389	3.4147	60
Quebec	71	2.3626	0.6	0.4389	3.4147	60
Quebec	72	2.3626	2.56	0.4389	3.4147	60
Quebec	73	2.3626	2.56	0.4389	3.4147	60
Quebec	74	2.3626	2.56	0.4389	3.4147	60
Quebec	75	2.3626	2.56	0.4389	3.4147	60
Quebec	76	2.3626	2.56	0.4389	3.4147	60
Quebec	77	2.3626	2.56	0.4389	3.4147	60
Quebec	78	2.3626	2.56	0.4389	3.4147	60
Quebec	79	2.3626	2.56	0.4389	3.4147	60
Quebec	80	2.3626	2.56	0.4389	3.4147	60

Table 3-4 (cont.)
Baseline Air Toxics and Visual Range Data by Census Division

Province Name	Census Division	Acetaldehyde (µg/m^3)	Benzene (µg/m^3)	Butadiene (µg/m^3)	Formaldehyde (µg/m^3)	Visual Range (km)
Quebec	81	2.3626	2.49	0.4389	3.4147	60
Quebec	82	2.3626	2.56	0.4389	3.4147	60
Quebec	83	2.3626	2.56	0.4389	3.4147	60
Quebec	84	2.3626	2.56	0.4389	3.4147	60
Quebec	85	2.3626	2.56	0.4389	3.4147	60
Quebec	86	2.3626	2.56	0.4389	3.4147	60
Quebec	87	2.3626	2.56	0.4389	3.4147	60
Quebec	88	2.3626	2.56	0.4389	3.4147	60
Quebec	89	2.3626	2.56	0.4389	3.4147	60
Quebec	90	2.3626	2.56	0.4389	3.4147	60
Quebec	91	2.3626	2.56	0.4389	3.4147	60
Quebec	92	2.3626	2.56	0.4389	3.4147	60
Quebec	93	2.3626	2.56	0.4389	3.4147	60
Quebec	94	2.3626	2.56	0.4389	3.4147	60
Quebec	95	2.3626	2.56	0.4389	3.4147	60
Quebec	96	2.3626	2.56	0.4389	3.4147	60
Quebec	97	2.3626	2.56	0.4389	3.4147	60
Quebec	98	2.3626	2.56	0.4389	3.4147	60
Quebec	99	2.3626	2.56	0.4389	3.4147	60
Ontario	1	2.0552	0.6	0.2664	3.7487	50
Ontario	2	2.0552	1.85	0.2664	3.7487	50
Ontario	6	2.0552	2.49	0.2664	3.7487	50
Ontario	7	2.0552	1.85	0.2664	3.7487	50
Ontario	9	2.0552	1.85	0.2664	3.7487	50
Ontario	10	2.0552	1.85	0.2664	3.7487	50
Ontario	11	2.0552	1.85	0.2664	3.7487	50
Ontario	12	2.0552	1.85	0.2664	3.7487	50
Ontario	13	2.0552	0.59	0.2664	3.7487	50
Ontario	14	2.0552	1.85	0.2664	3.7487	50
Ontario	15	2.0552	1.85	0.2664	3.7487	50
Ontario	16	2.0552	1.85	0.2664	3.7487	50
Ontario	18	2.0552	1.85	0.2664	3.7487	50
Ontario	19	2.0552	1.21	0.2664	3.7487	50
Ontario	20	2.0552	2.26	0.2664	3.7487	50
Ontario	21	2.0552	2.03	0.2664	3.7487	50
Ontario	22	2.0552	1.85	0.2664	3.7487	50
Ontario	23	2.0552	1.85	0.2664	3.7487	50
Ontario	24	2.0552	1.85	0.2664	3.7487	50
Ontario	25	2.0552	3.41	0.2664	3.7487	50
Ontario	26	2.0552	1.85	0.2664	3.7487	50
Ontario	28	2.0552	0.78	0.2664	3.7487	50
Ontario	29	2.0552	0.78	0.2664	3.7487	50

Table 3-4 (cont.)
Baseline Air Toxics and Visual Range Data by Census Division

Province Name	Census Division	Acetaldehyde (µg/m^3)	Benzene (µg/m^3)	Butadiene (µg/m^3)	Formaldehyde (µg/m^3)	Visual Range (km)
Ontario	30	2.0552	1.85	0.2664	3.7487	50
Ontario	31	2.0552	1.85	0.2664	3.7487	50
Ontario	32	2.0552	1.85	0.2664	3.7487	50
Ontario	34	2.0552	0.71	0.2664	3.7487	50
Ontario	36	2.0552	1.85	0.2664	3.7487	50
Ontario	37	2.0552	2.56	0.2664	3.7487	50
Ontario	38	2.0552	2.78	0.2664	3.7487	50
Ontario	39	2.0552	0.71	0.2664	3.7487	50
Ontario	40	2.0552	1.85	0.2664	3.7487	50
Ontario	41	2.0552	1.85	0.2664	3.7487	50
Ontario	42	2.0552	1.85	0.2664	3.7487	50
Ontario	43	2.0552	0.54	0.2664	3.7487	50
Ontario	44	2.0552	1.85	0.2664	3.7487	50
Ontario	46	2.0552	1.85	0.2664	3.7487	50
Ontario	47	2.0552	1.85	0.2664	3.7487	50
Ontario	48	2.0552	1.85	0.2664	3.7487	50
Ontario	49	2.0552	1.85	0.2664	3.7487	50
Ontario	51	2.0552	1.85	0.2664	3.7487	50
Ontario	52	2.0552	1.85	0.2664	3.7487	50
Ontario	53	2.0552	1.85	0.2664	3.7487	50
Ontario	54	2.0552	1.85	0.2664	3.7487	50
Ontario	56	2.0552	1.85	0.2664	3.7487	50
Ontario	57	2.0552	1.85	0.2664	3.7487	50
Ontario	58	2.0552	1.85	0.2664	3.7487	50
Ontario	59	2.0552	1.85	0.2664	3.7487	50
Ontario	60	2.0552	1.85	0.2664	3.7487	50
Manitoba	1	1.0219	1.48	0.251	1.4896	100
Manitoba	2	1.0219	1.48	0.251	1.4896	100
Manitoba	3	1.0219	1.48	0.251	1.4896	100
Manitoba	4	1.0219	1.48	0.251	1.4896	100
Manitoba	5	1.0219	1.48	0.251	1.4896	100
Manitoba	6	1.0219	1.48	0.251	1.4896	100
Manitoba	7	1.0219	1.48	0.251	1.4896	100
Manitoba	8	1.0219	1.48	0.251	1.4896	100
Manitoba	9	1.0219	1.48	0.251	1.4896	100
Manitoba	10	1.0219	1.48	0.251	1.4896	100
Manitoba	11	1.0219	1.48	0.251	1.4896	100
Manitoba	12	1.0219	1.48	0.251	1.4896	100
Manitoba	13	1.0219	1.48	0.251	1.4896	100
Manitoba	14	1.0219	1.48	0.251	1.4896	100
Manitoba	15	1.0219	1.48	0.251	1.4896	100
Manitoba	16	1.0219	1.48	0.251	1.4896	100

Table 3-4 (cont.)
Baseline Air Toxics and Visual Range Data by Census Division

Province Name	Census Division	Acetaldehyde (µg/m^3)	Benzene (µg/m^3)	Butadiene (µg/m^3)	Formaldehyde (µg/m^3)	Visual Range (km)
Manitoba	17	1.0219	1.48	0.251	1.4896	100
Manitoba	18	1.0219	1.48	0.251	1.4896	100
Manitoba	19	1.0219	1.48	0.251	1.4896	100
Manitoba	20	1.0219	1.48	0.251	1.4896	100
Manitoba	21	1.0219	1.48	0.251	1.4896	100
Manitoba	22	1.0219	1.48	0.251	1.4896	100
Manitoba	23	1.0219	1.48	0.251	1.4896	100
Saskatchewan	1	1.0219	1.48	0.251	1.4896	100
Saskatchewan	2	1.0219	1.48	0.251	1.4896	100
Saskatchewan	3	1.0219	1.48	0.251	1.4896	100
Saskatchewan	4	1.0219	1.48	0.251	1.4896	100
Saskatchewan	5	1.0219	1.48	0.251	1.4896	100
Saskatchewan	6	1.0219	1.48	0.251	1.4896	100
Saskatchewan	7	1.0219	1.48	0.251	1.4896	100
Saskatchewan	8	1.0219	1.48	0.251	1.4896	100
Saskatchewan	9	1.0219	1.48	0.251	1.4896	100
Saskatchewan	10	1.0219	1.48	0.251	1.4896	100
Saskatchewan	11	1.0219	1.48	0.251	1.4896	100
Saskatchewan	12	1.0219	1.48	0.251	1.4896	100
Saskatchewan	13	1.0219	1.48	0.251	1.4896	100
Saskatchewan	14	1.0219	1.48	0.251	1.4896	100
Saskatchewan	15	1.0219	1.48	0.251	1.4896	100
Saskatchewan	16	1.0219	1.48	0.251	1.4896	100
Saskatchewan	17	1.0219	1.48	0.251	1.4896	100
Saskatchewan	18	1.0219	1.48	0.251	1.4896	100
Alberta	1	1.0219	2.41	0.4307	1.4896	100
Alberta	2	1.0219	2.41	0.4307	1.4896	100
Alberta	3	1.0219	2.41	0.4307	1.4896	100
Alberta	4	1.0219	2.41	0.4307	1.4896	100
Alberta	5	1.0219	2.41	0.4307	1.4896	100
Alberta	6	1.0219	2.83	0.4307	1.4896	100
Alberta	7	1.0219	2.41	0.4307	1.4896	100
Alberta	8	1.0219	2.41	0.4307	1.4896	100
Alberta	9	1.0219	2.41	0.4307	1.4896	100
Alberta	10	1.0219	2.41	0.4307	1.4896	100
Alberta	11	1.0219	2.21	0.4307	1.4896	100
Alberta	12	1.0219	2.41	0.4307	1.4896	100
Alberta	13	1.0219	2.41	0.4307	1.4896	100
Alberta	14	1.0219	2.41	0.4307	1.4896	100
Alberta	15	1.0219	2.41	0.4307	1.4896	100
Alberta	16	1.0219	2.41	0.4307	1.4896	100
Alberta	17	1.0219	2.41	0.4307	1.4896	100

Table 3-4 (cont.)
Baseline Air Toxics and Visual Range Data by Census Division

Province Name	Census Division	Acetaldehyde (µg/m^3)	Benzene (µg/m^3)	Butadiene (µg/m^3)	Formaldehyde (µg/m^3)	Visual Range (km)
Alberta	18	1.0219	2.41	0.4307	1.4896	100
Alberta	19	1.0219	2.41	0.4307	1.4896	100
British Columbia	1	2.037	2.04	0.5348	2.4496	80
British Columbia	3	2.037	2.04	0.5348	2.4496	80
British Columbia	5	2.037	2.04	0.5348	2.4496	80
British Columbia	7	2.037	2.04	0.5348	2.4496	80
British Columbia	9	2.037	1.24	0.5348	2.4496	80
British Columbia	15	2.037	2.04	0.5348	2.4496	80
British Columbia	17	2.037	2.04	0.5348	2.4496	80
British Columbia	19	2.037	2.04	0.5348	2.4496	80
British Columbia	21	2.037	2.04	0.5348	2.4496	80
British Columbia	23	2.037	2.04	0.5348	2.4496	80
British Columbia	25	2.037	2.04	0.5348	2.4496	80
British Columbia	27	2.037	2.04	0.5348	2.4496	80
British Columbia	29	2.037	2.04	0.5348	2.4496	80
British Columbia	31	2.037	2.04	0.5348	2.4496	80
British Columbia	33	2.037	2.04	0.5348	2.4496	80
British Columbia	35	2.037	2.04	0.5348	2.4496	80
British Columbia	37	2.037	2.04	0.5348	2.4496	80
British Columbia	39	2.037	2.04	0.5348	2.4496	80
British Columbia	41	2.037	2.04	0.5348	2.4496	80
British Columbia	43	2.037	2.04	0.5348	2.4496	80
British Columbia	45	2.037	2.04	0.5348	2.4496	80
British Columbia	47	2.037	2.04	0.5348	2.4496	80
British Columbia	49	2.037	2.04	0.5348	2.4496	80
British Columbia	51	2.037	2.04	0.5348	2.4496	80
British Columbia	53	2.037	2.04	0.5348	2.4496	80
British Columbia	55	2.037	2.04	0.5348	2.4496	80
British Columbia	57	2.037	2.04	0.5348	2.4496	80
British Columbia	59	2.037	2.04	0.5348	2.4496	80
Yukon	1	1.0219	0.36	0.0526	1.4896	120
Northwest Territories	4	1.0219	0.36	0.0526	1.4896	120
Northwest Territories	5	1.0219	0.36	0.0526	1.4896	120
Northwest Territories	6	1.0219	0.36	0.0526	1.4896	120
Northwest Territories	7	1.0219	0.36	0.0526	1.4896	120
Northwest Territories	8	1.0219	0.36	0.0526	1.4896	120

Table 3-5
Baseline Air Toxics and Visual Range Data by Census Metropolitan Area (CMA)

Province	CMA	Acetaldehyde ($\mu g/m^3$)	Benzene ($\mu g/m^3$)	Butadiene ($\mu g/m^3$)	Formaldehyde ($\mu g/m^3$)	Visual Range (km)
Newfoundland	1	1.0219	0.96	0.1073	1.4896	50
Newfoundland	10	1.0219	0.96	0.1073	1.4896	50
Newfoundland	11	1.0219	0.96	0.1073	1.4896	50
Newfoundland	15	1.0219	0.96	0.1073	1.4896	50
Newfoundland	25	1.0219	0.96	0.1073	1.4896	50
Prince Edward Island	105	1.0219	0.96	0.1073	1.4896	30
Prince Edward Island	110	1.0219	0.96	0.1073	1.4896	30
Nova Scotia	205	1.0219	2.93	0.5639	1.4896	20
Nova Scotia	210	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	215	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	220	1.0219	1.65	0.5639	1.4896	20
Nova Scotia	225	1.0219	1.65	0.5639	1.4896	20
New Brunswick	305	1.1181	0.96	0.1073	1.5517	30
New Brunswick	310	1.1181	0.96	0.1073	1.5517	30
New Brunswick	320	1.1181	0.96	0.1073	1.5517	30
New Brunswick	328	1.1181	0.96	0.1073	1.5517	30
New Brunswick	330	1.1181	0.96	0.1073	1.5517	30
New Brunswick	335	1.1181	0.96	0.1073	1.5517	30
Quebec	403	2.3626	2.56	0.4389	3.4147	60
Quebec	404	2.3626	2.56	0.4389	3.4147	60
Quebec	405	2.3626	2.56	0.4389	3.4147	60
Quebec	406	2.3626	2.56	0.4389	3.4147	60
Quebec	408	2.3626	2.56	0.4389	3.4147	60
Quebec	410	2.3626	2.56	0.4389	3.4147	60
Quebec	411	2.3626	2.56	0.4389	3.4147	60
Quebec	412	2.3626	2.56	0.4389	3.4147	60
Quebec	421	2.3626	2.56	0.4389	3.4147	60
Quebec	428	2.3626	2.56	0.4389	3.4147	60
Quebec	430	2.3626	2.56	0.4389	3.4147	60
Quebec	433	2.3626	2.56	0.4389	3.4147	60
Quebec	435	2.3626	2.56	0.4389	3.4147	60
Quebec	437	2.3626	2.56	0.4389	3.4147	60
Quebec	440	2.3626	2.56	0.4389	3.4147	60
Quebec	442	2.3626	2.56	0.4389	3.4147	60
Quebec	444	2.3626	2.56	0.4389	3.4147	60
Quebec	446	2.3626	2.56	0.4389	3.4147	60
Quebec	447	2.3626	2.56	0.4389	3.4147	60
Quebec	450	2.3626	2.56	0.4389	3.4147	60
Quebec	452	2.3626	2.56	0.4389	3.4147	60
Quebec	454	2.3626	2.56	0.4389	3.4147	60
Quebec	456	2.3626	0.94	0.4389	3.4147	60
Quebec	459	2.3626	2.56	0.4389	3.4147	60

Table 3-5 (cont.)
Baseline Air Toxics and Visual Range Data by Census Metropolitan Area (CMA)

Province	CMA	Acetaldehyde (µg/m^3)	Benzene (µg/m^3)	Butadiene (µg/m^3)	Formaldehyde (µg/m^3)	Visual Range (km)
Quebec	462	2.3626	3.6	0.4389	3.4147	60
Quebec	465	2.3626	0.6	0.4389	3.4147	60
Quebec	468	2.3626	2.56	0.4389	3.4147	60
Quebec	480	2.3626	2.56	0.4389	3.4147	60
Quebec	485	2.3626	2.56	0.4389	3.4147	60
Quebec	505	2.3626	2.49	0.4389	3.4147	60
Ontario	501	2.3626	1.85	0.2664	3.7487	50
Ontario	502	2.0552	1.85	0.2664	3.7487	50
Ontario	505	2.0552	2.49	0.2664	3.7487	50
Ontario	508	2.0552	1.85	0.2664	3.7487	50
Ontario	512	2.0552	1.85	0.2664	3.7487	50
Ontario	515	2.0552	1.85	0.2664	3.7487	50
Ontario	521	2.0552	1.85	0.2664	3.7487	50
Ontario	522	2.0552	1.85	0.2664	3.7487	50
Ontario	527	2.0552	1.85	0.2664	3.7487	50
Ontario	528	2.0552	1.85	0.2664	3.7487	50
Ontario	529	2.0552	1.85	0.2664	3.7487	50
Ontario	530	2.0552	1.85	0.2664	3.7487	50
Ontario	532	2.0552	1.85	0.2664	3.7487	50
Ontario	535	2.0552	1.8	0.2664	3.7487	50
Ontario	537	2.0552	3.41	0.2664	3.7487	50
Ontario	539	2.0552	1.85	0.2664	3.7487	50
Ontario	541	2.0552	1.85	0.2664	3.7487	50
Ontario	543	2.0552	1.85	0.2664	3.7487	50
Ontario	544	2.0552	1.85	0.2664	3.7487	50
Ontario	546	2.0552	1.85	0.2664	3.7487	50
Ontario	547	2.0552	0.78	0.2664	3.7487	50
Ontario	550	2.0552	1.85	0.2664	3.7487	50
Ontario	553	2.0552	1.85	0.2664	3.7487	50
Ontario	555	2.0552	0.71	0.2664	3.7487	50
Ontario	556	2.0552	1.85	0.2664	3.7487	50
Ontario	557	2.0552	1.85	0.2664	3.7487	50
Ontario	558	2.0552	0.71	0.2664	3.7487	50
Ontario	559	2.0552	2.56	0.2664	3.7487	50
Ontario	562	2.0552	2.78	0.2664	3.7487	50
Ontario	566	2.0552	1.85	0.2664	3.7487	50
Ontario	567	2.0552	1.85	0.2664	3.7487	50
Ontario	568	2.0552	0.54	0.2664	3.7487	50
Ontario	569	2.0552	1.85	0.2664	3.7487	50
Ontario	571	2.0552	1.85	0.2664	3.7487	50
Ontario	575	2.0552	1.85	0.2664	3.7487	50
Ontario	580	2.0552	1.85	0.2664	3.7487	50

Table 3-5 (cont.)
Baseline Air Toxics and Visual Range Data by Census Metropolitan Area (CMA)

Province	CMA	Acetaldehyde (µg/m^3)	Benzene (µg/m^3)	Butadiene (µg/m^3)	Formaldehyde (µg/m^3)	Visual Range (km)
Ontario	582	2.0552	1.85	0.2664	3.7487	50
Ontario	584	2.0552	1.85	0.2664	3.7487	50
Ontario	586	2.0552	1.85	0.2664	3.7487	50
Ontario	590	2.0552	1.85	0.2664	3.7487	50
Ontario	595	2.0552	1.85	0.2664	3.7487	50
Ontario	598	2.0552	1.85	0.2664	3.7487	50
Manitoba	602	1.0219	1.48	0.251	1.4896	100
Manitoba	607	1.0219	1.48	0.251	1.4896	100
Manitoba	610	1.0219	1.48	0.251	1.4896	100
Manitoba	640	1.0219	1.48	0.251	1.4896	100
Saskatchewan	705	1.0219	1.48	0.251	1.4896	100
Saskatchewan	710	1.0219	1.48	0.251	1.4896	100
Saskatchewan	715	1.0219	1.48	0.251	1.4896	100
Saskatchewan	720	1.0219	1.48	0.251	1.4896	100
Saskatchewan	725	1.0219	1.48	0.251	1.4896	100
Saskatchewan	735	1.0219	1.48	0.251	1.4896	100
Saskatchewan	745	1.0219	1.48	0.251	1.4896	100
Saskatchewan	750	1.0219	1.48	0.251	1.4896	100
Alberta	805	1.0219	2.41	0.4307	1.4896	100
Alberta	810	1.0219	2.41	0.4307	1.4896	100
Alberta	825	1.0219	2.83	0.4307	1.4896	100
Alberta	830	1.0219	2.41	0.4307	1.4896	100
Alberta	833	1.0219	2.41	0.4307	1.4896	100
Alberta	835	1.0219	2.21	0.4307	1.4896	100
Alberta	840	1.0219	2.41	0.4307	1.4896	100
Alberta	845	1.0219	2.41	0.4307	1.4896	100
Alberta	850	1.0219	2.41	0.4307	1.4896	100
Alberta	860	1.0219	2.41	0.4307	1.4896	100
Alberta	865	1.0219	2.41	0.4307	1.4896	100
British Columbia	905	2.037	2.04	0.5348	2.4496	80
British Columbia	913	2.037	2.04	0.5348	2.4496	80
British Columbia	915	2.037	2.04	0.5348	2.4496	80
British Columbia	918	2.037	2.04	0.5348	2.4496	80
British Columbia	925	2.037	2.04	0.5348	2.4496	80
British Columbia	930	2.037	2.04	0.5348	2.4496	80
British Columbia	932	2.037	1.24	0.5348	2.4496	80
British Columbia	933	2.037	2.04	0.5348	2.4496	80
British Columbia	935	2.037	2.04	0.5348	2.4496	80
British Columbia	937	2.037	2.04	0.5348	2.4496	80
British Columbia	938	2.037	2.04	0.5348	2.4496	80
British Columbia	940	2.037	2.04	0.5348	2.4496	80
British Columbia	943	2.037	2.04	0.5348	2.4496	80

Table 3-5 (cont.)
Baseline Air Toxics and Visual Range Data by Census Metropolitan Area (CMA)

Province	CMA	Acetaldehyde (µg/m^3)	Benzene (µg/m^3)	Butadiene (µg/m^3)	Formaldehyde (µg/m^3)	Visual Range (km)
British Columbia	944	2.037	2.04	0.5348	2.4496	80
British Columbia	945	2.037	2.04	0.5348	2.4496	80
British Columbia	950	2.037	2.04	0.5348	2.4496	80
British Columbia	952	2.037	2.04	0.5348	2.4496	80
British Columbia	955	2.037	2.04	0.5348	2.4496	80
British Columbia	960	2.037	2.04	0.5348	2.4496	80
British Columbia	965	2.037	2.04	0.5348	2.4496	80
British Columbia	970	2.037	2.04	0.5348	2.4496	80
British Columbia	975	2.037	2.04	0.5348	2.4496	80
British Columbia	977	2.037	2.04	0.5348	2.4496	80
Yukon	990	1.0219	0.36	0.0526	1.4896	120
Northwest Territories	995	1.0219	0.36	0.0526	1.4896	120

Table 3-6
Census Divisions with Air Quality Monitors

Province Name	Census Division	PM_{10}	$PM_{2.5}$	SO_4	Ozone	CO	Acetaldehyde	Benzene	Butadiene	Formaldehyde	SO_2
Newfoundland	1	1			1	1					1
Newfoundland	2										
Newfoundland	3										
Newfoundland	4										
Newfoundland	5				1						
Newfoundland	6										
Newfoundland	7										
Newfoundland	8										
Newfoundland	9										
Newfoundland	10										
Prince Edward Island	1										
Prince Edward Island	2	1									1
Prince Edward Island	3				1						
Nova Scotia	1										
Nova Scotia	2										
Nova Scotia	3										
Nova Scotia	4							1			
Nova Scotia	5	1	1	1	1			1			
Nova Scotia	6										
Nova Scotia	7				1						
Nova Scotia	8										
Nova Scotia	9	1	1	1	1	1		1	1		1
Nova Scotia	10										
Nova Scotia	11										
Nova Scotia	12										
Nova Scotia	13										
Nova Scotia	14										
Nova Scotia	15										
Nova Scotia	16										
Nova Scotia	17	1									
Nova Scotia	18										
New Brunswick	1	1	1	1	1	1	1	1	1	1	1
New Brunswick	2		1	1	1			1	1		
New Brunswick	3		1	1	1						
New Brunswick	4										
New Brunswick	5				1			1			
New Brunswick	6				1						
New Brunswick	7										
New Brunswick	8										
New Brunswick	9										
New Brunswick	10	1									
New Brunswick	11										
New Brunswick	12										
New Brunswick	13										
New Brunswick	14										

Table 3-6 (cont.)
Census Divisions with Air Quality Monitors

Province Name	Census Division	PM_{10}	$PM_{2.5}$	SO_4	Ozone	CO	Acetaldehyde	Benzene	Butadiene	Formaldehyde	SO_2
New Brunswick	15										
Quebec	1										
Quebec	2										
Quebec	3										
Quebec	4										
Quebec	5										
Quebec	6										
Quebec	7										
Quebec	8										
Quebec	9										
Quebec	10										
Quebec	11										
Quebec	12										
Quebec	13										
Quebec	14										
Quebec	15										
Quebec	16				1						
Quebec	17										
Quebec	18										
Quebec	19										
Quebec	20								1		
Quebec	21										
Quebec	22										
Quebec	23	1	1	1	1	1					1
Quebec	24										
Quebec	25										
Quebec	26										
Quebec	27										
Quebec	28										
Quebec	29										
Quebec	30										
Quebec	31										
Quebec	32							1			
Quebec	33							1			
Quebec	34							1			
Quebec	35										
Quebec	36	1									1
Quebec	37	1									1
Quebec	38							1			
Quebec	39							1			
Quebec	40										
Quebec	41										
Quebec	42										
Quebec	43	1									

Table 3-6 (cont.)
Census Divisions with Air Quality Monitors

Province Name	Census Division	PM_{10}	$PM_{2.5}$	SO_4	Ozone	CO	Acetaldehyde	Benzene	Butadiene	Formaldehyde	SO_2
Quebec	44										
Quebec	45										
Quebec	46		1	1				1			
Quebec	47										
Quebec	48										
Quebec	49										
Quebec	50				1						
Quebec	51				1						
Quebec	52							1			
Quebec	53	1						1			
Quebec	54				1						
Quebec	55										
Quebec	56										
Quebec	57		1	1				1			
Quebec	58		1	1				1			
Quebec	59		1	1				1			
Quebec	60		1	1				1			
Quebec	61							1			
Quebec	62										
Quebec	63							1			
Quebec	64		1	1				1			
Quebec	65		1	1							
Quebec	66	1	1	1	1	1	1	1	1	1	1
Quebec	67		1	1				1			
Quebec	68				1						
Quebec	69							1			
Quebec	70		1	1				1			
Quebec	71		1	1				1			
Quebec	72										
Quebec	73		1	1							
Quebec	74		1	1							
Quebec	75										
Quebec	76		1	1							
Quebec	77										
Quebec	78				1						
Quebec	79										
Quebec	80										
Quebec	81	1	1	1	1	1		1			1
Quebec	82				1						
Quebec	83										
Quebec	84										
Quebec	85										
Quebec	86	1									
Quebec	87										

Table 3-6 (cont.)
Census Divisions with Air Quality Monitors

Province Name	Census Division	PM_{10}	$PM_{2.5}$	SO_4	Ozone	CO	Acetaldehyde	Benzene	Butadiene	Formaldehyde	SO_2
Quebec	88										
Quebec	89										
Quebec	90										
Quebec	91										
Quebec	92										
Quebec	93										
Quebec	94	1									1
Quebec	95										
Quebec	96	1									1
Quebec	97										
Quebec	98										
Quebec	99										
Ontario	1	1			1	1		1			1
Ontario	2										
Ontario	6	1	1	1	1	1	1	1	1	1	1
Ontario	7										
Ontario	9										
Ontario	10				1						
Ontario	11										
Ontario	12										
Ontario	13							1			
Ontario	14										
Ontario	15				1						1
Ontario	16										
Ontario	18	1			1	1					1
Ontario	19		1	1	1			1			
Ontario	20	1	1	1	1	1	1	1	1	1	1
Ontario	21		1	1				1			
Ontario	22		1	1							
Ontario	23		1	1	1						1
Ontario	24	1	1	1	1	1					1
Ontario	25	1			1	1		1	1		1
Ontario	26	1			1	1					1
Ontario	28				1			1	1		
Ontario	29							1			
Ontario	30				1	1					1
Ontario	31										
Ontario	32										
Ontario	34							1			
Ontario	36				1						
Ontario	37	1	1	1	1	1	1	1	1	1	1
Ontario	38	1			1	1		1	1		1
Ontario	39	1			1	1		1			1
Ontario	40				1						

Table 3-6 (cont.)
Census Divisions with Air Quality Monitors

Province Name	Census Division	PM_{10}	$PM_{2.5}$	SO_4	Ozone	CO	Acetaldehyde	Benzene	Butadiene	Formaldehyde	SO_2
Ontario	41				1						
Ontario	42										
Ontario	43		1	1	1			1			
Ontario	44										
Ontario	46										
Ontario	47										
Ontario	48				1	1					1
Ontario	49										
Ontario	51										
Ontario	52										
Ontario	53	1			1	1					1
Ontario	54										
Ontario	56										
Ontario	57	1			1						1
Ontario	58	1			1	1					1
Ontario	59										
Ontario	60				1						
Manitoba	1										
Manitoba	2										
Manitoba	3										
Manitoba	4										
Manitoba	5										
Manitoba	6										
Manitoba	7	1			1						
Manitoba	8										
Manitoba	9										
Manitoba	10										
Manitoba	11	1	1	1	1	1		1	1		1
Manitoba	12										
Manitoba	13										
Manitoba	14										
Manitoba	15										
Manitoba	16										
Manitoba	17										
Manitoba	18										
Manitoba	19										
Manitoba	20										
Manitoba	21										
Manitoba	22										
Manitoba	23										
Saskatchewan	1										
Saskatchewan	2										
Saskatchewan	3				1						
Saskatchewan	4										

Table 3-6 (cont.)
Census Divisions with Air Quality Monitors

Province Name	Census Division	PM_{10}	$PM_{2.5}$	SO_4	Ozone	CO	Acetaldehyde	Benzene	Butadiene	Formaldehyde	SO_2
Saskatchewan	5										
Saskatchewan	6	1			1	1					1
Saskatchewan	7										
Saskatchewan	8										
Saskatchewan	9										
Saskatchewan	10										
Saskatchewan	11	1			1	1					1
Saskatchewan	12										
Saskatchewan	13										
Saskatchewan	14										
Saskatchewan	15										
Saskatchewan	16										
Saskatchewan	17										
Saskatchewan	18										
Alberta	1										
Alberta	2										
Alberta	3										
Alberta	4										
Alberta	5										
Alberta	6	1	1	1	1	1		1	1		1
Alberta	7										
Alberta	8										
Alberta	9										
Alberta	10				1						
Alberta	11	1	1	1	1	1		1	1		1
Alberta	12										
Alberta	13										
Alberta	14										
Alberta	15										
Alberta	16				1	1					1
Alberta	17										
Alberta	18										
Alberta	19										
British Columbia	1										
British Columbia	3										
British Columbia	5										
British Columbia	7										
British Columbia	9				1			1			
British Columbia	15	1	1	1	1	1	1	1	1	1	1
British Columbia	17	1	1	1	1						1
British Columbia	19										
British Columbia	21										
British Columbia	23										
British Columbia	25										

Table 3-6 (cont.)
Census Divisions with Air Quality Monitors

Province Name	Census Division	PM_{10}	$PM_{2.5}$	SO_4	Ozone	CO	Acetaldehyde	Benzene	Butadiene	Formaldehyde	SO_2
British Columbia	27										
British Columbia	29										
British Columbia	31										
British Columbia	33	1									1
British Columbia	35	1			1						
British Columbia	37										
British Columbia	39										
British Columbia	41										
British Columbia	43										
British Columbia	45										
British Columbia	47										
British Columbia	49										
British Columbia	51										
British Columbia	53	1									
British Columbia	55										
British Columbia	57										
British Columbia	59										
Yukon	1										
Northwest Territories	4										
Northwest Territories	5										
Northwest Territories	6										
Northwest Territories	7										
Northwest Territories	8										

Table 3-7
Census Metropolitan Areas (CMAs) with Air Quality Monitors

Province	CMA	PM_{10}	$PM_{2.5}$	SO_4	Ozone	CO	Acetaldehyde	Benzene	Butadiene	Formaldehyde	SO_2
Newfoundland	1		1		1						
Newfoundland	10										
Newfoundland	11										
Newfoundland	15										
Newfoundland	25										
Prince Edward Island	105										
Prince Edward Island	110										
Nova Scotia	205	1	1		1			1			
Nova Scotia	210										
Nova Scotia	215										
Nova Scotia	220										
Nova Scotia	225	1	1								
New Brunswick	305										
New Brunswick	310	1	1		1			1			
New Brunswick	320										
New Brunswick	328										
New Brunswick	330										
New Brunswick	335										
Quebec	403										
Quebec	404		1								
Quebec	405										
Quebec	406										
Quebec	408	1	1								
Quebec	410										
Quebec	411										
Quebec	412										
Quebec	421	1	1		1						
Quebec	428										
Quebec	430										
Quebec	433										
Quebec	435										
Quebec	437										
Quebec	440										
Quebec	442	1									
Quebec	444	1	1								
Quebec	446										
Quebec	447										
Quebec	450										
Quebec	452										
Quebec	454	1	1								
Quebec	456							1			
Quebec	459										
Quebec	462	1	1		1			1			
Quebec	465							1			
Quebec	468										
Quebec	480										

Table 3-7 (cont.)
Census Metropolitan Areas (CMAs) with Air Quality Monitors

Province	CMA	PM_{10}	$PM_{2.5}$	SO_4	Ozone	CO	Acetaldehyde	Benzene	Butadiene	Formaldehyde	SO_2
Quebec	485										
Quebec	505	1	1		1			1			
Ontario	501	1			1						
Ontario	502										
Ontario	505	1	1		1			1			
Ontario	508										
Ontario	512										
Ontario	515										
Ontario	521	1			1						
Ontario	522										
Ontario	527										
Ontario	528										
Ontario	529				1						
Ontario	530										
Ontario	532		1		1						
Ontario	535	1	1		1			1			
Ontario	537	1	1		1			1			
Ontario	539	1	1		1						
Ontario	541		1		1						
Ontario	543										
Ontario	544										
Ontario	546										
Ontario	547		1		1			1			
Ontario	550				1						
Ontario	553										
Ontario	555	1						1			
Ontario	556										
Ontario	557										
Ontario	558							1			
Ontario	559	1	1		1			1			
Ontario	562	1			1			1			
Ontario	566										
Ontario	567										
Ontario	568				1			1			
Ontario	569										
Ontario	571										
Ontario	575				1						
Ontario	580	1	1		1						
Ontario	582										
Ontario	584										
Ontario	586										
Ontario	590	1			1						
Ontario	595	1			1						
Ontario	598										
Manitoba	602	1	1		1			1			
Manitoba	607										

Table 3-7 (cont.)
Census Metropolitan Areas (CMAs) with Air Quality Monitors

Province	CMA	PM_{10}	$PM_{2.5}$	SO_4	Ozone	CO	Acetaldehyde	Benzene	Butadiene	Formaldehyde	SO_2
Manitoba	610	1			1						
Manitoba	640										
Saskatchewan	705	1			1						
Saskatchewan	710										
Saskatchewan	715										
Saskatchewan	720										
Saskatchewan	725	1			1						
Saskatchewan	735										
Saskatchewan	745										
Saskatchewan	750										
Alberta	805										
Alberta	810										
Alberta	825	1	1		1			1			
Alberta	830										
Alberta	833										
Alberta	835	1	1		1			1			
Alberta	840										
Alberta	845										
Alberta	850										
Alberta	860				1						
Alberta	865										
British Columbia	905	1									
British Columbia	913										
British Columbia	915	1	1		1						
British Columbia	918		1								
British Columbia	925	1									
British Columbia	930	1	1		1						
British Columbia	932	1			1			1			
British Columbia	933	1	1		1			1			
British Columbia	935	1	1		1						
British Columbia	937										
British Columbia	938		1								
British Columbia	940	1									
British Columbia	943										
British Columbia	944	1									
British Columbia	945	1									
British Columbia	950	1	1								
British Columbia	952	1									
British Columbia	955										
British Columbia	960										
British Columbia	965										
British Columbia	970	1	1								
British Columbia	975										
British Columbia	977										
Yukon	990										
Northwest Territories	995										

Table 3-8
Baseline Agriculture Data by Province

Province	Corn		Soybeans		Wheat		Tobacco	
	(tonnes)	($/tonne)	(tonnes)	($/tonne)	(tonnes)	($/tonne)	(tonnes)	($/tonne)
Newfoundland								
Prince Edward Island			2,447	$251.84	12,011	$150.92	1,267	$5,121.75
Nova Scotia					4,454	$240.30	335	$4,902.33
New Brunswick					2,790	$221.19	114	$4,687.39
Quebec	1,973,333	$141.74	135,347	$284.69	82,425	$172.73	3,864	$5,078.62
Ontario	4,893,933	$127.55	1,722,683	$283.91	984,529	$118.49	64,480	$4,113.32
Manitoba					3,359,355	$152.92		
Saskatchewan					10,052,016	$151.11		
Alberta					5,069,005	$144.48		
British Columbia					94,888	$174.61		
Yukon Territory								
Northwest Territories								

Blank cells indicate that the crop is not one of the top ten crops (in terms of cash receipts) in that province.

CHAPTER 4
CONCENTRATION-RESPONSE FUNCTIONS FOR HUMAN HEALTH EFFECTS

Concentration-response functions are used to specify the relationship between ambient air pollution concentrations and human health responses. In Section 4.1 we describe the procedures for screening health effects to be included and for selecting concentration-response functions. In Section 4.2 we describe how potential double counting in the functions is accounted for in AQVM 3.0. In Sections 4.3 and 4.4 we discuss and specify concentration-response functions for PM_{10} and ozone induced mortality and morbidity. In Section 4.5 we discuss and specify concentration-response functions relating exposure to selected air toxics to increased risks of cancer. In Section 4.6 we specify a concentration-response function relating exposure to carbon monoxide to cardiac hospital admissions.

AQVM 3.0 also includes the sulphate concentration-response functions developed by the Sulphate Study expert panel (Thurston, 1997b). The selection and application of studies for sulphates is summarised in Appendix A. The AQVM 3.0 concentration-response functions for $PM_{2.5}$ are presented in Appendix D. In both cases, the functions were developed in a manner consistent with the methods reported in this chapter. It is important to note that because of the overlap between sulphates and particulate matter, AQVM 3.0 allows the user to select only one measure of particulate matter at a time in an analysis.

4.1 SELECTION PROCEDURES FOR CONCENTRATION-RESPONSE FUNCTIONS FOR OZONE AND PARTICULATE MATTER

4.1.1 Study Selection

Concentration-response functions for health effects related to ozone and particulate matter less than 10 microns in diameter (PM_{10}) were identified and adapted from the available epidemiologic literature. These functions allow the estimation of the change in the number of cases of each health effect that would be expected as a result of changes in ambient PM_{10} or ozone concentrations. Several factors were considered in selecting and applying concentration-response functions for use in this assessment.

First, an appropriate study design and methodology to allow estimation of a concentration-response function for changes in outdoor PM_{10} concentrations was required. Studies were expected to have data based on continuous monitoring of the relevant pollutants, careful

characterization and selection of exposure measures, and minimal bias in study sample selection and reporting. In addition, the studies had to provide concentration-response relationships over a continuum of relevant exposures. Second, studies that recognized and attempted to minimize confounding and omitted variables were preferred. For example, studies that compared two cities or regions and characterized them as high and low pollution areas were not used for quantitative purposes because of potential confounding by other factors in the respective areas and vague definition of exposure. Third, controls for the effects of seasonality and weather had to be included. This could be accomplished by stratifying and analysing the data by season, by examining the independent effects of temperature and humidity, or by using other statistical corrections.

A fourth consideration was that the study had to include a careful exploration of the primary hypothesis and preferably an examination of the robustness and sensitivity of the results to alternative functional forms, specifications, and influential data points. When studies reported the results of these alternative analyses, the quantitative estimates that were judged as most representative of the overall findings were selected for use in this assessment. Fifth, for inclusion in the PM_{10} portion of this review, the study had to provide an airborne particulate measure that could be converted into a common metric (i.e., concentrations of PM_{10}). For example, studies that failed to quantitatively characterize air pollution were not included. In addition, studies that used measures of particulate matter that could not be converted to PM_{10} could not be used. Sixth, the study had to involve relevant levels of air pollution. Thus, studies that examined only high level pollution episodes were not relied on for quantitative information.

Finally, studies that addressed clinical outcomes or changes in behaviour that would best lend themselves to economic valuation were included. Therefore, estimates for endpoints such as changes in lung function that are difficult to link to clinically significant symptoms were not included. Also, preference was given to studies that focussed on representative population groups to ensure the fullest possible coverage for the general population. For example, emergency room visit studies that included visits made by all segments of the population were preferred to studies that, for instance, examined visits only by asthmatics.

4.1.2 Conversions of Coefficients to PM_{10}

Air quality monitoring systems in North America began measuring PM_{10} in the late 1980s. Previously, TSP (total suspended particulates) was the most common measure used, but many other particulate matter measures are available in different locations and have been used in different health effects studies. Most of the differences between the measures are due to different size ranges or composition of particles measured (e.g., PM_{10} versus TSP or sulphates). A few of the measures are related to optical properties of particles, such as coefficient of haze (COH). PM_{10} is chosen as the metric for this analysis because it is the measure upon which most ambient air quality standards for particulate matter are currently based. It represents the size fraction of

airborne particulates that are small enough to be inhaled into the lungs, and is sometimes called inhalable particulate matter.[1]

An important underlying issue in interpreting available results from studies using these different, but related, measures of airborne particulate matter is whether different sizes or types of particles differ in the amount or type of adverse health effects they cause. Although it is reasonable to expect that there are differences, available information from the epidemiologic literature is not sufficient determine specifically how the particles that typically make up PM_{10} differ in terms of their expected health impacts. Some epidemiologic studies compared results using different measures of particulate matter for the same health effect and found a statistically stronger association between health effects and sulphates or acidity (e.g., Plagiannakos and Parker, 1988) Others found a stronger association with more comprehensive measures of particulates, such as TSP or PM_{10} (e.g., Dockery et al., 1992). Many studies found statistically significant associations with more than one measure of particulate matter. However, different measures of particulate matter tend to be highly correlated day to day or location to location, because they are measuring essentially the same air pollution phenomenon. It is therefore extremely difficult in epidemiologic analyses to determine which constituents of PM_{10} are responsible for the observed association with health effects, or to determine how the various constituents differ in terms of their effects on human health.

Because the purpose of this analysis is to be as comprehensive as possible in estimating the changes in health effects expected as a result of change in PM_{10} concentrations, we want to make use of all the available literature that speaks to this question, including studies that may have used measures of particulate matter that are related to, but not equivalent to, PM_{10}. If a study estimated a relationship between a health effect and some measure of particulate matter other than PM_{10}, it is necessary for this analysis to make some assumptions and interpretation to determine what the results imply in terms of the number of health effects for a change in PM_{10}. Ideally, this would be done by comparing levels of the different particulate matter measures taken at collocated monitors at each study site or sites. Unfortunately, these data are not usually available, and more average ratios between the measures from locations where these are available are therefore used. Two important assumptions are made when interpreting available results and converting estimates from one measure to another. First, it is assumed that no health effects are caused by particles larger than PM_{10}, and second, it is assumed that estimated health effects coefficients for particle measures that represent a component of PM_{10} (e.g., sulphates or $PM_{2.5}$) may be reflecting the effects of those measured constituents as well as the effects of other PM_{10} constituents with which they are correlated in the study area.

1. Concentration-response functions for sulfates and for fine particulate matter ($PM_{2.5}$) have also been selected from the epidemiologic literature and are discussed in appendices to this report. These are two significant constituents of PM_{10} (most sulphates also fall into the $PM_{2.5}$ size range). These concentration-response functions provide alternative options for AQVM 3.0 users to analyse pollution control strategies that might target different types of particulate matter.

Several studies conducted in the United States have estimated relationships between mortality and TSP. The average ratio between PM_{10} and TSP in the United States is approximately 0.55 (U.S. EPA, 1986b). Thus, if the observed relationship between mortality and TSP is really driven by the PM_{10} fraction, then the coefficient for a 100 g/m^3 change in TSP will predict the same number of deaths as the coefficient that would have been estimated for a 55 g/m^3 change in PM_{10}. Dividing the TSP coefficient by 0.55 will give the appropriate coefficient for a 100 g/m^3 change in PM_{10}.

Alternatively, studies conducted in southern Ontario, for example, have found relationships between sulphate particles and hospital admissions. If the sulphate coefficient reflects the effects of sulphates as well as of other particulates with which it is correlated, it is necessary to adjust the sulphate coefficient to predict the number of hospital admissions for a given change in PM_{10}. For example, average levels of sulphate particles in southern Ontario are about 18% of average PM_{10} levels in the same area (Dann, 1994). If an estimated coefficient for sulphates implies 10 health effects per 1 g/m^3 of sulphates, this is interpreted as 10 hospital admissions per 5.5 g/m^3 of PM_{10}. Thus, the sulphate coefficient is multiplied by 0.18 to obtain a PM_{10} coefficient that predicts the same number of hospital admissions. This conversion procedure presumes that the estimated sulphate coefficient reflects all the hospital admissions associated with PM_{10}, with which the sulphate measure is correlated. This assumption results in a lower health effect coefficient for PM_{10} than if the full PM_{10} effect were not assumed to be reflected in the sulphate coefficient. Alternative assumptions could be reasonably defended, but we are choosing to err on the side of not overstating the health effects associated with a change in PM_{10} concentrations when interpreting the results of these studies.

4.1.3 Ozone Measurement

Most of the ozone concentration-response functions presented in this report are from epidemiologic studies that used the daily high-hour ozone level measured at stationary monitors as the measure of ozone exposure for the study population. The daily high-hour ozone concentration is the highest hourly reading recorded on that calendar day. It may have occurred at any time during that 24-hour period, but ozone levels are usually highest during afternoon daylight hours. Some studies, especially the more recent ones, used average daily ozone concentrations rather than the daily high hour.

The daily high-hour ozone concentration has been used in most epidemiologic studies because clinical evidence shows that short-term exposures result in measurable health effects, and because it is suspected that peak rather than average exposures may be the culprit for acute health effects. The 1-hour measure is also consistent with the current standards set to protect human

health, which are based on a 1-hour average.[2] This does not mean, however, that only the highest hour of ozone in the day causes the measured health effect. To the extent that ozone levels for other averaging times, such as 7-hour averages or daily averages, are correlated with the daily high hour, then the functions reflect the effect of these exposures as well. In such circumstances, the daily high hour is a proxy for ozone exposure on that day, with emphasis on the peak rather than the average.

An adjustment is needed to compare results from studies based on daily high-hour ozone concentrations with results from studies based on daily average ozone concentrations. We obtained hourly ozone data for all year-round ozone monitors in Chicago and in Philadelphia, two of the cities where studies have been conducted using daily average ozone measures. We calculated the annual average of the daily ratio between the daily high hour and the daily average ozone concentrations. These averages ranged from about 2.0 to 3.0, and most of them clustered fairly closely around 2.5. We therefore use an adjustment factor of 2.5 to convert results based on daily average ozone concentrations to their equivalent in terms of the daily high hour.

One of the older studies conducted in the Los Angeles area that is used in this analysis used measures of total oxidants rather than ozone. It is typical that when ozone occurs, so do other photochemical oxidants. Current air monitoring techniques and ambient air quality standards in Canada and the United States now focus on ozone, which is by far the largest component of total photochemical oxidants. However, nonozone oxidants may also cause harmful health effects (U.S. EPA, 1992). When interpreting the results of the study based on a total oxidant measure to estimate the health effects associated with a specific amount of ozone, we presume that the effect per unit of ozone is equivalent to the measured effect per unit of oxidant. Thus, we do not adjust coefficients estimated for oxidants when applying them to calculate an effect for change in ambient ozone.

4.1.4 Evidence on Health Effects Thresholds for PM_{10}

An important uncertainty in all of the particulate matter health effect estimates is whether there is a threshold particulate matter level below which health effects no longer occur, or whether the slope of the concentration-response function diminishes significantly at lower concentrations.

Most of the epidemiologic studies reported here have estimated linear or log-linear functions that suggest a continuum of effects down to the lowest particulate matter levels observed in the study sample, and have not conducted any analyses to test whether there might be a threshold pollution concentration below which these health effects no longer occur. In fact, epidemiologic data are not typically adequate to fully explore the questions of whether thresholds exist and at what

2. The U.S. Environmental Protection Agency has just revised the U.S. national ozone standard to be based on an 8-hour average rather than a 1-hour average based on recent evidence suggesting that longer exposures at lower concentrations are also harmful to human health.

concentrations. When efforts have been made to identify a threshold, little conclusive evidence has been found that one exists (Ostro, 1984). Many recent epidemiologic studies show a consistent association between particulate matter and health effects across the entire range of measured particulate levels, including levels well below the current Canadian objectives and U.S. standards for particulate matter. For example, Schwartz and Dockery (1992a, 1992b) found a statistically significant relationship between mortality and particulate matter in two eastern U.S. cities across all four quartiles of daily particulate matter. The lowest 5 to 10 percentile levels of particulate matter in these studies were in the range of 30 to 40 $\mu g/m^3$ TSP (24-hour measurements). Burnett et al. (1995) conducted a threshold analysis in their study of hospital admissions to Canadian hospitals and particulate matter concentrations. Their preliminary results show statistically significant effects on days with PM_{10} equivalent as low as 25 $\mu g/m^3$ (Burnett et al., 1995). In all of these studies, a large share of days considered in the analyses have particulate matter concentrations well below the current Canadian air quality objective of 120 $\mu g/m^3$ for 24-hour TSP. These result do not prove that there is no threshold for particulate matter related health effects, but they show that health effects are seen across a range of concentrations that span well below typical standards.

On the basis of the evidence of health effects at relatively low particulate matter concentrations, AQVM 3.0 is designed with a default assumption that there is no threshold for PM_{10} health effects. However, recognizing the uncertainties about the existence and level of health effect thresholds, and the potential importance of threshold assumptions on the computed benefits, AQVM 3.0 allows the user to conduct sensitivity analyses to address this issue by selecting alternative threshold levels for (1) long-term exposure risks (mortality risks, chronic bronchitis, and acute bronchitis in children), and (2) short-term exposure risks (all other morbidity risks). Mortality is set up in the model to use a chronic exposure threshold, but can be switched to a short-term exposure threshold by the user if desired. The mortality concentration-response functions selected for the model are based on a combination of short-term and long-term exposure studies, so it is not clear which type of threshold would apply. See *Report 1: User s Guide* for instructions to implement this option.

4.1.5 Evidence on Health Effects Thresholds for Ozone

Lippmann (1993) cites evidence from clinical studies that pulmonary function and respiratory symptom effects have been observed at 1-hour exposures at 100 parts per billion (ppb) and above, and that some effects are seen at levels as low as 80 ppb when exposure times are increased to 6.6 hours. It remains quite uncertain, at this time, whether there is an entirely safe level of ozone for humans and, if so, what that level is. The Canadian air quality objective for 1-hour ozone concentration establishes 80 ppb as acceptable and 50 ppb as desirable.

All of the concentration-response functions presented in this report are based on epidemiologic studies conducted at various locations in North America, with the exception of the functions for ozone-related mortality that also incorporate results from studies conducted in South America

and Europe. Therefore, these concentration-response functions provide estimates of health effects associated with current levels of ozone in North America. Although some of the study locations, specifically southern portions of California, experience ozone levels on many days that are quite high relative to the Canadian objectives and U.S. standards, the range of ozone levels examined in all of the epidemiologic studies include levels that are typical of most of the populated areas of Canada. Thus, using these results to develop health effects estimates for Canada does not involve extrapolation of health effects risk estimates outside the range of ozone levels included in the original studies.

Table 4-1 shows the ozone levels for the study areas in most of the epidemiologic studies used to derive concentration-response relationships for this analysis. For the most part, the average ozone level in each of these studies was below the current Canadian 1-hour objective. This does not prove that there is no threshold for health effects from ozone, because this issue has not been directly addressed in any of these studies. However, it does demonstrate that epidemiologic studies have found statistically significant associations between ozone levels and various health effects in locations where mean ozone levels are well below the current acceptable air quality objective. Results from a large hospitalization study in the 16 largest cities in Canada suggest that effects are found on days with a daily high-hour ozone as low as 25 ppb (pers. comm., R. Burnett, Health Canada, May 16, 1995).

On the basis of the evidence from many epidemiologic studies of an association between health effects and ozone concentrations of relatively low levels, AQVM 3.0 is designed with a default assumption of no threshold for ozone health effects during the ozone season (May-September). However, recognizing the uncertainties about the existence and level of health effect thresholds, and the potential importance of threshold assumptions on the computed benefits, AQVM 3.0 allows the user to conduct sensitivity analyses to address this issue by defining an alternative threshold level applicable to all ozone mortality and morbidity risks. See *Report 1: User s Guide* for instructions to implement this option.

AQVM 3.0 is designed to calculate health effects of ozone only during the five-month ozone season (May-September). It is assumed that there are no benefits from reductions in ozone during other months of the year, either because emissions changes have minimal impact on ozone formation during the nonsummer months or because concentrations are so low that changes have no effect. This is, in effect, a threshold assumption. Like many other parameters in the model, the user has the option to change this assumption and calculate health benefits for year-round changes in ozone concentrations.

4.1.6 Specification of Concentration-Response Parameters

The concentration-response functions in the literature have been linearised around current ambient concentrations and population health status to estimate a concentration-response parameter, b,

Table 4-1
Ozone Levels in Selected Epidemiology Studies

Study	Location	Health Effect	Study Period Mean of Daily High-Hour Ozone (ppb) (standard deviation[a])
Burnett et al. (1994)	Southern Ontario	Hospital admissions (summer only)	50
Kinney and Ozkaynak (1991)	Los Angeles, California	Premature mortality	75 (45)
Thurston et al. (1992)	New York City Buffalo, NY	Respiratory hospital admissions (summer only)	61 70
Whittemore and Korn (1980)	Several Southern California communities	Asthma symptoms	30 to 150 (means in different communities)
Holguin et al. (1985)	Houston, Texas	Asthma symptoms	76 (21)
Ostro and Rothschild (1989)	United States selected cities nationwide	Minor restricted activity days	23 (12)
Krupnick et al. (1990)	Los Angeles, California	Acute respiratory symptoms	99 (89)

a. Standard deviation is not always reported by the authors.

which equals the change in the health risk factor per exposed (susceptible) individual for a 1 g/m^3 change in average PM_{10}, or a 1 ppb change in (high-hour) ozone, from current conditions.

The available epidemiologic evidence regarding health effects associated with air pollutants is subject to considerable uncertainty. Within a given study there is an estimated standard error on the estimated concentration-response parameter, and there are differences in results obtained from different studies looking at the same or similar health effects. For each concentration-response function presented in this report, low, central, and high estimates are selected. The central estimate is typically selected from the middle of the range reported in the study, or group of studies. The central estimate is interpreted as the best estimate based on the available literature.

Low and high estimates are selected from the available literature to reflect not absolute upper and lower bounds, but rather ranges that are reasonably likely to be correct, given results of available health effects studies. For example, ranges based on a single study are selected as plus and minus one standard error, not the absolute highest and lowest result obtained. When several studies are available for a given health effect, the selected range reflects the variation in results across the

studies. The reader should be aware that there is analyst judgement in selecting these ranges and that the ranges do not reflect all the uncertainty in the concentration-response functions because some of the uncertainty is not quantifiable. This is, however, an attempt to give a more realistic presentation than is given when only central parameters are reported.

Each low, central, and high parameter is also assigned a probability weight (the three weights summing to 100% for each quantified health effect). These probability weights, combined with the low, central, and high parameters, are used to estimate a probability distribution of the total health benefits estimate. Calculating a probability distribution for the total health benefit estimate provides an alternative to simply summing all the low estimates or all the high estimates to obtain total low and high estimates. Such simple summing can be misleading because it is highly unlikely that all the low estimates (or all the high estimates) are correct. When the low, central, and high parameters are based on results from different studies all judged as equally likely to be correct, an equal probability weight is given to the low, central, and high parameters. When only one study is selected, the range used is plus and minus one standard error from the mean results of the study. When a standard error is used, the probability weight given to the central parameter is 50%, with 25% each to the high and low parameters. In some cases less weight has been given to a high or low parameter based on analyst judgement that there is reason to suspect that particular parameter is less likely to be correct than the others. These probability weights are to a large extent based on analyst judgement rather than precise quantitative data. For a more extensive discussion of the selection and implementation of this uncertainty analysis, see Chapter 6 in this report and Rowe et al. (1995).

4.2 SUMMARY OF SELECTED CONCENTRATION-RESPONSE FUNCTIONS

Table 4-2 and Table 4-3 list the selected concentration-response functions for each of the PM_{10} and ozone health effects categories, respectively. Sections 4.3 and 4.4 explain the derivations of the parameters for these functions.

Some of the health effects categories reported in the literature may overlap. For example, acute respiratory symptoms days (ARSs) probably include some days that are also restricted activity days (RADs). To avoid double counting health effects, we make some adjustments in the concentration-response functions. We assume that all pollution-related respiratory and cardiac hospital admissions (RHAs and CHAs, respectively) involve an initial emergency room visit (ERV). We also assume that all pollution-related RADs are also acute ARSs. As a result, the following subtractions are made to calculate net PM_{10} concentration-response functions for each of these categories:

$$\text{Net ERVs} = \text{Total ERVs} - (\text{RHAs} + \text{CHAs}) \quad (4\text{-}1)$$

$$\text{Net ARSs} = \text{Total ARSs} - \text{RADs} . \quad (4\text{-}2)$$

Table 4-2
Human Health Effects from PM_{10}

Health Effect Category	Concentration-Response Parameter (weights)		
Annual mortality risk factors given a 1 g/m^3 change in annual average PM_{10} concentration Sources: Schwartz et al. (1996), Pope et al. (1995).	Low Central High	4.4×10^{-6} 12.1×10^{-6} 28.2×10^{-6}	(22%) (67%) (11%)
Chronic bronchitis (CB) annual risk factors given a 1 g/m^3 change in annual average PM_{10} concentration Source: Abbey et al. (1993).	For population 25 years and over: Low Central High	 3.0×10^{-5} 6.1×10^{-5} 9.3×10^{-5}	 (25%) (50%) (25%)
Respiratory hospital admissions (RHAs) daily risk factors given a 1 g/m^3 change in daily PM_{10} concentration Sources: Burnett et al. (1995), Pope (1991).	Low Central High	0.64×10^{-8} 0.78×10^{-8} 3.26×10^{-8}	(33%) (50%) (17%)
Cardiac hospital admissions (CHAs) daily risk factors given a 1 g/m^3 change in daily PM_{10} concentration Source: Burnett et al. (1995).	Low Central High	5.0×10^{-9} 6.6×10^{-9} 8.2×10^{-9}	(25%) (50%) (25%)
Net emergency room visits (ERVs) daily risk factors given a 1 g/m^3 change in daily PM_{10} concentration Source: Stieb et al. (1995).	Low Central High	2.96×10^{-8} 3.66×10^{-8} 14.3×10^{-8}	(25%) (50%) (25%)
Asthma symptom days (ASDs) daily risk factors given a 1 g/m^3 change in daily PM_{10} concentration Sources: Whittemore and Korn (1980), Ostro et al. (1991).	For population with asthma (6%): Low Central High	 1.62×10^{-4} 1.72×10^{-4} 1.82×10^{-4}	 (33%) (34%) (33%)
Restricted activity days (RADs) daily risk factors given a 1 g/m^3 change in daily PM_{10} concentration Sources: Ostro (1987), Ostro and Rothschild (1989).	For nonasthmatic population (94%) 20 years and older: Low Central High	 0.8×10^{-4} 1.6×10^{-4} 2.5×10^{-4}	 (33.3%) (33.4%) (33.3%)
Net days with acute respiratory symptoms (ARSs) daily risk factors given a 1 g/m^3 change in daily PM_{10} concentration Source: Krupnick et al. (1990).	For nonasthmatic population (94%): Low Central High	 1.62×10^{-4} 3.44×10^{-4} 5.18×10^{-4}	 (25%) (50%) (25%)
Children with acute bronchitis (B) annual risk factors given a 1 g/m^3 change in annual average PM_{10} concentration Source: Dockery et al. (1996).	For population under age 20: Low Central High	 0.57×10^{-3} 1.42×10^{-3} 2.27×10^{-3}	 (25%) (50%) (25%)

Table 4-3
Human Health Effects from Ozone

Health Effect Category	Concentration-Response Parameter (weights)		
Daily mortality risk factors given a 1 ppb change in daily high-hour ozone concentration Sources: (See Section 4.3.2).	Low Central High	0 4.3×10^{-9} 7.4×10^{-9}	(33%) (34%) (33%)
Respiratory hospital admissions (RHAs) daily risk factors given a 1 ppb change in daily high-hour ozone concentration Source: Burnett et al. (1997).	Low Central High	0.6×10^{-8} 1.1×10^{-8} 1.6×10^{-8}	(25%) (50%) (25%)
Net emergency room visits (ERVs) daily risk factors given a 1 ppb change in daily high-hour ozone concentration Sources: Stieb et al. (1995); Burnett et al. (1997).	Low Central High	2.6×10^{-8} 4.7×10^{-8} 6.9×10^{-8}	(25%) (50%) (25%)
Asthma symptom days (ASDs) daily risk factors given a 1 ppb change in daily high-hour ozone concentration Sources: Whittemore and Korn (1980), Stock et al. (1988).	For population with asthma (6%): Low Central High	 1.06×10^{-4} 1.88×10^{-4} 5.20×10^{-4}	 (33%) (50%) (17%)
Minor restricted activity days (MRADs) daily risk factors given a 1 ppb change in daily high-hour ozone concentration Source: Ostro and Rothschild (1989).	For nonasthmatic population (94%): Low Central High	 1.93×10^{-5} 4.67×10^{-5} 7.40×10^{-5}	 (25%) (50%) (25%)
Net days with acute respiratory symptoms (ARSs) daily risk factors given a 1 ppb change in daily high-hour ozone concentration Source: Krupnick et al. (1990).	For nonasthmatic population (94%): Low Central High	 5.07×10^{-5} 9.03×10^{-5} 13.0×10^{-5}	 (25%) (50%) (25%)

For ozone, the health effects categories expected to overlap are (1) RHAs and ERVs, and (2) minor restricted activity days (MRADs) and ARSs. As a result, the following subtractions are made to calculate net ozone concentration-response functions for each of these categories:

$$\text{Net ERVs} = \text{Total ERVs} - \text{RHAs} \quad (4\text{-}3)$$

$$\text{Net ARSs} = \text{Total ARSs} - \text{MRADs} . \quad (4\text{-}4)$$

4.3 PREMATURE MORTALITY

4.3.1 Premature Mortality and PM_{10}

Over the last few decades, many epidemiologic studies have found statistically significant associations between concentrations of particulate matter and mortality among the general population. The earliest studies focussed on relatively rare episodes of extremely high pollution concentrations in the 1940s and 1950s in the United States and in the United Kingdom (U.S. EPA, 1982a). More recent studies have found an association at concentration levels typical

of most metropolitan areas in North America [e.g., Dockery and Pope (1994) review this literature].

The earliest studies of this type were cross-sectional studies examining annual mortality rates across U.S. cities with different average particulate matter levels, often including 100 or more cities (e.g., Lave and Seskin, 1977; Evans et al., 1984; Ozkaynak and Thurston, 1987, Lipfert, 1994). More recently, many time-series studies have found statistically significant associations between daily mortality and daily fluctuations in particulate matter concentrations in a wide range of cities (e.g., Pope et al., 1992; Schwartz and Dockery, 1992a,b). Very recently, two prospective studies using individual-specific data and tracking mortality for a study sample in multiple cities over multiple years found associations between survival rates and particulate matter concentrations (Dockery et al., 1993; Pope et al., 1995).

Some skepticism remains about whether these studies reflect a true causal relationship, primarily because a specific biological mechanism to fully explain and verify this relationship has not been demonstrated in clinical or laboratory research (Utell and Samet, 1993). However, epidemiologic studies are consistently finding a statistically significant association between air pollution and mortality, using different study designs and locations, and over a wide range of particulate matter concentrations, including levels well below the current Canadian objectives or U.S. standards. In addition, recent controlled animal exposure studies have begun to suggest plausible mechanisms by which severe effects, including death, may occur after concentrated ambient air pollution exposure (e.g., Godleski et al., 1996). Godleski et al. s work, it should be noted, was conducted at higher than ambient concentrations (up to 30 times), for very short periods (3 days). Future research may shed more light on the mechanism by which particulate matter increases mortality risk, and such findings might suggest necessary revisions in the way we are estimating particulate matter health effects in AQVM 3.0. In the meantime, we are using results from available studies in a way that represents a reasonable interpretation of the available evidence.

4.3.2 Summary of Selected Quantitative Evidence

This section does not provide a detailed review of all available literature, but focuses on results available in the literature that are best suited for the purposes of this analysis. The study selection process relied on study selection criteria discussed in Section 4.1, and incorporated results from prospective cohort and time-series studies. From both perspectives the results show an association between mortality and particulate matter, and results from both types of studies are relied upon in selecting a range of concentration-response parameters for use in this analysis.

An important question that has been raised with regard to the time-series mortality results is whether they may simply represent an increase in mortality in a population subgroup that is already very ill and close to death. This is sometimes called mortality displacement. Although it is reasonable that some of the deaths associated with short-term exposures to particulate matter may be individuals who would have died within a short time even in the absence of air pollution

exposure, there is no evidence that such deaths dominate the time-series results. Spix (in press) reports, for example, that the results of his test for mortality displacement were not statistically significant. He reports that the mortality effect was minimally reduced on days following a 15-day period of high daily average mortality. In addition, Schwartz (1998) reports that although pollution-related mortality for chronic bronchitis may involve displacement of about 3 months, there is little evidence for any short-term displacement for pneumonia, cardiovascular, or all-cause mortality related to air pollution exposure. Schwartz found that modeling longer exposure times generated larger, not smaller, daily mortality effects. If mortality displacement was occurring, the size of the effect would be reduced, not increased, by modeling longer exposure times.

In addition, long-term exposure studies show a significant difference in life expectancy across cities with different air pollution concentrations. If the only effect of air pollution exposure on mortality were a shift of time of death by a few days, then no difference in life expectancy would be expected across cities with different pollution levels. Because annual average pollution concentrations are correlated with short-term concentrations, some of the mortality linked to long-term exposure is also likely to be reflected in the time-series studies. Lippmann and Thurston (1996) report, for example, that the U.S. prospective cohort studies imply an average life span shortening of about 2 years between the most polluted and least polluted cities. They also note that these studies imply an amount of excess annual deaths that exceeds that implied by the cumulative results of the time-series studies.

Long-Term Exposure Studies

Two types of long-term exposure studies have found statistically significant associations between mortality rates and air pollution levels in the United States. The first type is an ecologic cross-sectional study design in which mortality rates for various locations are analysed to determine if there is a statistical correlation with average air pollutant levels in each location. Such studies have consistently found measurably higher mortality rates in metropolitan areas with higher average levels of the particulate matter. However, concern persists about whether these studies have adequately controlled for potential confounding factors. Lipfert (1994), Ozkaynak and Thurston (1987), and Evans et al. (1984) provide examples of ecologic cross-sectional studies. These studies each conducted a thorough examination of data for 100 or more U.S. metropolitan areas, including average TSP or sulphate concentrations for each city, with special emphasis on the effects of including or excluding potential confounding factors such as occupations, education, or migration. The findings of these studies varied in terms of pollutants found to be significant and the magnitudes of the effects.

A second type of long-term exposure study is a prospective cohort study in which a sample is selected and followed over time in each location. In 1993, Dockery et al. published results for a 15-year prospective study based on samples of individuals in six cities. In 1995, Pope et al. published results of a 7-year prospective study conducted in collaboration with the American Cancer Society based on samples of individuals in 151 cities in the United States. These studies

are similar in some respects to the ecologic cross-sectional studies because the variation in pollution exposure is measured across locations rather than over time. These studies rely on the same type of pollutant exposure data as that used in the ecologic studies, average pollutant levels measured at stationary outdoor monitors in a given location. However, the mortality data are for identified individuals, which enables much better characterization of the study population and other health risks than when area-wide mortality data are used. Because they used individual-specific data, the authors of the prospective studies were able to control for mortality risks associated with differences in body mass, occupational exposures, smoking (present and past), alcohol use, age, and gender.

Dockery et al. (1993) found a mortality-rate ratio of 1.26 over the 15-year study period for the most polluted to least polluted city; this ratio applied to several measures of particulate matter, SO_2, and NO_2. Pope et al. (1995) found a mortality-rate ratio of 1.15 for highest to lowest average sulphate concentrations over 151 U.S. cities, and 1.17 for highest to lowest median $PM_{2.5}$ concentrations over 50 U.S. cities during a 7-year study period. All findings were statistically significant. Abbey et al. (1991) did not find any evidence of premature mortality associations with air pollution in a smaller, nonsmoking cohort in California.

The Pope at al. study in particular represents a very important contribution to the study of mortality and particulate matter because of the prospective design and the very large number of study locations included. The findings of a significant association between mortality and particulate matter in this study are very supportive of some of the findings in previous single-year cross-sectional studies. The Pope et al. (1995) prospective cohort study results reflect the analysis of data from over 295,000 subjects and 50 metropolitan areas for the 7-year period from 1982 to 1989. By comparison the Dockery et al. (1993) prospective cohort study analysed data from 8,111 adults from six Eastern United States cities over a 14-year period. The Pope et al. (1995) study developed risk ratios from Cox proportional hazard models in which the median fine particulate concentration for a metropolitan area over the period from 1979 to 1983 was entered as an independent variable, along with socioeconomic variables accounting for, among other factors, a subject s education, smoking status, and alcohol consumption. In addition, meteorological controls were included to account for relatively hot or cold conditions.

The prospective studies provide strong evidence that long-term exposures to higher average particulate matter concentrations are associated with statistically significantly higher risks of premature mortality. However, the prospective studies have been criticized by some (Lipfert, 1995; Vedal, 1997) for their inability to account for potentially different historical levels of air pollution than indicated by the data available for the published analyses. These studies were also criticized for not more directly or fully accounting for potential confounding by differences in diet, physical activity, and socioeconomic status. Nevertheless, these studies still support an association between life expectancy and annual average particulate levels.

The results of the two prospective studies are summarised in Table 4-4, along with the results from selected annual cross-sectional studies, for comparison purposes. To facilitate some

quantitative comparison, elasticities were calculated for each of the studies. Elasticities give the percentage change in mortality for every 1% change in the pollutant measure, evaluated at the mean of the mortality rates and pollution measures in each study. This, to some extent, controls for the differences in particulate matter measures and length of study periods. The elasticity estimates were further converted to percent change in mortality per g/m^3 of PM_{10}. The elasticities and mortality effects vary by an order of magnitude among these studies, though most of the variability is introduced by one study (Dockery et al., 1993).

Table 4-4
Comparison of Long-Term Exposure Mortality Study Results

Study	Time Period	Number of Cities	Particulate Measure	Particulate Mean (g/m^3)	Estimated Elasticity[a]	% Change in Mortality per 10 g/m^3 PM_{10}[b]
Pope et al. (1995)	1982-1989	50 151	$PM_{2.5}$ Sulphate	20.2 11.0	0.118 0.079	3.5% 1.8%
Dockery et al. (1993)	1974-1989	6	PM_{10} $PM_{2.5}$	29.9 18.0	0.251 0.222	8.4% 7.4%
Lipfert et al. (1994)	1980	149	TSP	68.4	0.033	0.9%
Ozkaynak and Thurston (1987)	1980	98	$PM_{2.5}$ Sulphate	23.1 11.1	0.060 0.086	1.6% 2.0%
Evans et al. (1984)	1960	98	TSP Sulphate	121 10.3	0.049 0.038	0.7% 0.9%

a. Elasticity is the percentage change in mortality for each 1% change in the pollution measure, estimated at the mean pollution measure. This is calculated from the reported relative risk results for the prospective studies using the formulas relative risk = exp(b × PM); change in probability of death per unit PM (M/ PM) = b × Pr × (1 - Pr), where Pr is the probability of death in the study; and elasticity = (M/ PM) × (mean PM/mean M).
b. Conversions for this table are based on $PM_{10} = 1.67 \times PM_{2.5}$ and $PM_{10} = 4 \times$ sulphate (based on U.S. data where these studies were conducted).

Time-Series Studies of Acute Exposure

In recent years, numerous studies of air pollution and human mortality have indicated effects of acute air pollution exposures on daily mortality using time-series methods. The primary strength of time-series studies is that health and pollution variations in the same population (e.g., for a single city) are followed over time, so that the study population acts as its own control population. Time-series statistical models use these respective day-to-day variations in exposure and effects data to determine whether mortality or morbidity counts rise and fall as air pollution concentrations rise and fall from day to day in the study area. This obviates the need to separately analyse comparison populations and the need to statistically adjust for differences across population characteristics (e.g., race, income, education). Such time-series studies, though, have the limitation that they focus only on short-term effects and do not reflect any potential adverse

consequences of chronic exposures to air pollution (chronic exposures are commonly evaluated using cross-sectional methods that incorporate both short- and long-term exposure effects).

4.3.3 Selection of PM_{10} Annual Mortality Concentration-Response Parameters

In this analysis, we consider both time-series epidemiologic studies of daily mortality counts and cross-sectional studies of annual mortality rates. Results from a study employing time-series epidemiologic methods are used here to develop the low concentration-response parameter estimate of the impact on premature mortality associated with a change in the level of PM_{10}, while a prospective cross-sectional study is employed to derive the high estimate. The central estimate of premature mortality cases associated with a change in the level of PM_{10} is based on a weighted mean of the low and high parameters from the time-series and prospective cross-sectional studies employed. Consistent with the procedure developed by the sulphate panel (Thurston, 1997b), we give a two-thirds to one-third relative weighting of the time-series and cross-sectional studies, respectively, in developing the central concentration-response parameter estimate. Numbers of premature deaths are calculated in every city by applying these percentages to the prevailing annual Canadian baseline nonaccidental death rate (6,700/million persons/year).[3]

The Schwartz et al. (1996) time-series study is used to develop the low concentration-response parameter for PM_{10}-related mortality because it is a recent analysis that pooled results across six North American cities and used PM_{10} as a measures of particulate matter. The low premature mortality parameter was derived from the mean mortality effect (estimated for a 10 g/m^3 change in PM_{10}) minus one standard deviation as follows: (0.80-0.15)% change in mortality per 10 g/m^3 PM_{10} = 0.065% change in mortality per g/m^3 PM_{10}.

Numerous cross-sectional studies in the literature, as described above, have indicated that, after controlling for other confounders, places with higher particulate matter concentrations have higher annual mortality rates. In this analysis, we employ the recent Pope et al. (1995) study in developing our high mortality concentration-response parameter estimate. This study, while confirmatory of earlier cross-sectional studies, is chosen because it analysed individuals, and could therefore better control for potential confounders (such as smoking) on an individual level (rather than at the aggregate city level, as in prior studies). Also, the Pope et al. study incorporated a large study sample across a large number of North American cities.

The high PM_{10} premature mortality concentration-response parameter estimate was derived from the Pope et al. (1995) study by calculating the mean mortality effect plus one standard deviation (for the most polluted areas versus the least polluted) and then dividing by the difference in average PM_{10} concentrations between most and least polluted areas during the study, as follows:

3. Obtained for 1991 from the World Health Organization (1994), *World Health Statistics Annual, 1993.*

mean result plus one standard error = (1.17 + 0.04) relative risk per 45.3 g/m^3 PM_{10} = ln(1.21) ÷ 45.3 × 100 = 0.42% change in total mortality per g/m^3 PM_{10}.[4]

Following the weighting procedure described above, the central premature mortality concentration-response parameter is thus (0.065% × 0.67) + (0.42% × 0.33) = 0.18%. The central parameter is given two-thirds weight, and the low parameter is given twice the weight of the high parameter, reflecting the larger body of time-series literature that the low parameter is drawn from. The expected value for the distribution thus equals the central estimate. The estimated percentage changes in mortality responses per g/m^3 annual average PM_{10} are thus (selected probability weights in parentheses):

Low = 0.065% (22%)
Central = 0.18% (67%)
High = 0.42% (11%) .

Changes in daily premature mortality in Canada per g/m^3 change in 24-hour PM_{10} are calculated based on the average annual Canadian nonaccidental mortality rate of 6,700 per 1,000,000 people and the low, central, and high percentage changes in PM_{10}-related premature mortality selected above. For example, the central concentration-response parameter is 0.18% of 6,700 divided by 1,000,000. The selected concentration-response parameters and calculation procedures are thus:

Low annual PM_{10} mortality risk = $4.4 \times 10^{-6} \times POP_j \times (\ PMj)$ (4-5a)
Central annual PM_{10} mortality risk = $12.1 \times 10^{-6} \times POP_j \times (\ PM_j)$ (4-5b)
High annual PM_{10} mortality risk = $28.2 \times 10^{-6} \times POP_j \times (\ PM_j)$, (4-5c)

where:

POP_j = total population in area j
PM_j = change in annual average PM_{10} in area j.

Evidence on Who Is at Risk

The results of the Philadelphia study (Schwartz and Dockery, 1992a) provide estimates of elevated mortality risks separately for those over and under 65 years old. These results suggest that about 90% of the premature deaths associated with particulate matter occur in the over-65 group. This finding is consistent with the results of an early cross-sectional mortality study (Lave and Seskin, 1977). Ostro et al. (1996) found that about 80% of the premature deaths associated

4. The Pope et al. (1995) study reports a relative risk of 1.17 for an increment of 24.5 g/m^3 in annual median $PM_{2.5}$ from the least to the most polluted city. Based on our analysis of available monitor data for $PM_{2.5}$ in the United States, the average ratio of annual median to annual mean concentrations is 0.9. This is therefore equivalent to an increment in mean $PM_{2.5}$ of about 27.2 g/m^3. Since $PM_{2.5}$ averages about 60% of PM_{10} in urban areas (Dockery et al., 1993), this is equal to approximately 45.3 g/m^3 increment in PM_{10}.

with particulate matter were in the over-65 group in their Santiago, Chile, study. In the United States, about 70% of all deaths are individuals 65 years old or older, so it appears that risks associated with air pollution exposure fall in somewhat greater proportion to the elderly.

The results from Pope et al. (1995) show that the greatest association is with deaths associated with cardiopulmonary illness and lung cancer, and that elevated mortality risks are similar for both smokers and nonsmokers in higher pollution locations. Some of the time-series studies (e.g., Schwartz and Dockery, 1992a) have also found significant cause-specific mortality associations, indicating that most pollution-associated deaths are cardiopulmonary related. Some of those at risk therefore probably suffer from chronic diseases that might be expected to shorten life expectancy even in the absence of air pollution. This does not, however, rule out the possibility that some of these chronic illnesses could themselves be related to air pollution exposure.

As discussed in Chapter 5, the age of the individual at risk of premature mortality may have some bearing on the monetary value of changing that risk. For the purposes of this analysis, it is presumed from evidence in Ostro et al. (1996) and Schwartz and Dockery (1992a) that 85% of the individuals at risk of premature mortality associated with PM_{10} are 65 years old or older.

4.3.4 Premature Mortality and Ozone

Recent epidemiologic studies from locations around the world have greatly expanded the available evidence examining the relationship between fluctuations in ambient ozone levels and observed daily mortality. Although many studies have found a statistically significant relationship between mortality and ozone in models that include a measure of particulate matter, some of the studies have not. Assessing the underlying nature of the relationship by comparing study results is complicated by the different daily ozone concentration measures used. These measures have included daily high-hour, 8-hour average, and 24-hour average values.

The studies selected for this analysis are summarised in Table 4-5 and met several important criteria. First, all of the studies have appeared in the published, peer reviewed literature. Second, all the selected ozone results are taken from a model that includes a variable accounting for the influence of particulates. This helps limit the potential confounding effects that can arise and impact the parameter estimates when a model specifies only one air pollution measure. Third, all of the analyses are based on year-round data and are for all ages of the population. These are the study selection criteria developed by the U.S. Environmental Protection Agency for the analysis of this literature as part of the regulatory impact analysis conducted regarding the recent changes made to the national ambient air quality standards for ozone in the United States (U.S. EPA, 1997b).

Table 4-5
Ozone Mortality Daily Time-Series Studies Used in Analysis

Study Authors (year)	Time Period	Location	Estimated Beta[a] (standard error)
Kinney et al. (1995)	1985-1990	Los Angeles, CA	0.0000 (2.08×10^{-4})
Samet et al. (1997); Moolgavkar et al. (1995)[b]	1973-1988	Philadelphia, PA	2.9×10^{-4} (0.69×10^{-4})
Loomis et al. (1996)	1991-1992	Mexico City	-2.01×10^{-4} (1.84×10^{-4})
Ostro et al. (1996)	1989-1991	Santiago, Chile	-1.90×10^{-4} (1.93×10^{-4})
Verhoeff et al. (1996)	1986-1992	Amsterdam	9.49×10^{-4} (10.5×10^{-4})
Hoek et al. (1997)	1986-1991	Rotterdam	6.89×10^{-4} (2.69×10^{-4})
Anderson et al. (1996)	1987-1992	London	4.16×10^{-4} (1.00×10^{-4})
Ito and Thurston (1996)	1985-1990	Chicago, IL	6.74×10^{-4} (2.39×10^{-4})

a. The estimated beta is the Poisson coefficient, which is interpreted as the percentage change in daily mortality per unit change in ozone. If not reported by the authors, these are calculated from the reported relative risks and statistical confidence intervals based on the formula: $RR = e^{X}$. Results based on 8-hour or 24-hour ozone concentrations were adjusted to daily high-hour equivalent using a peak to mean ratio of 2.5. For example, if an RR is reported for a 10 ppb change in 24-hour average ozone, we consider this equivalent to a 25 ppb change in daily high-hour ozone.
b. To avoid giving undue weight to the Philadelphia results, which are basically two analyses of the same data, we calculated a fixed effects weighted mean result from the two studies and included that single result in the random effects analysis for all the study locations.

In four of the eight locations listed in Table 4-5, the estimated relationship between ozone and mortality was not found to be statistically significant. All of these studies were credible analyses meeting the study selection criteria, so the lack of significant findings in several cases cannot be ignored. However, there is one potentially important factor to consider in comparing these results. The studies that do not find a significant relationship tend to be those with a smaller number of observations. Three of the four studies in this case relied on 2 years of data or less. Although the Los Angeles study covers 6 calendar years, the data included only every sixth day (because of the PM_{10} monitoring schedule in the area). Similarly, because of missing data, the Santiago study had about 2 years of data.

For this analysis, the results from the nine daily time-series studies in Table 4-5 were combined in a random effects model to estimate a central weighted average concentration-response parameter. The concentration-response parameters from the studies were combined using weights

developed on the basis of the inverse of the variance associated with a study s parameter estimate. The random effects pooling method is consistent with the presumption that the true underlying relationship between ozone and daily nonaccidental mortality may vary from location to location (U.S. EPA, 1997a). This contrasts with a fixed effects model, which hypothesizes that the study results from different locations are in fact different estimates of one true underlying relationship that is constant across locations. The random effects pooling method builds on the results of the fixed effects model, so whether the results are consistent with fixed effects model assumptions is tested statistically before the random effects model is applied.

In estimating the random effects model the studies results must first be converted into a common parameter. For this analysis, the relative risk results from the studies were converted into their equivalent concentration-response parameter estimates and associated standard errors assuming that the results had been generated through a Poisson modelling process where the relative risk is the result of the following equation:

$$\text{Relative Risk} = e^{(X)}, \qquad (4\text{-}6)$$

where:

	=	estimated ozone parameter (beta) in the Poisson regression
X	=	the change in the level of the pollutant associated with the relative risk.

We calculated two pooled results from the studies in Table 4-5. The first, for all eight locations and including all the results, was 2.34×10^{-4} (s.e. = 1.08×10^{-4}). The second, for the four locations where a statistically significant ozone effect was found Philadelphia, Chicago, London, and Rotterdam was 4.02×10^{-4} (s.e. = 0.80×10^{-4}).

To calculate the daily ozone mortality concentration-response parameters, the percentage impact results are multiplied by the average Canadian daily nonaccidental mortality rate of 18.4 per million. The central concentration-response parameter is based on the random effects estimate for all eight locations. The low concentration-response parameter is assigned a value of zero to reflect that in four of the eight study locations represented in Table 4-5 a statistically significant relationship between ozone and nonaccidental daily mortality was not found. The high concentration-response parameter reflects the random effects result for the four locations where a statistically significant relationship between ozone and mortality was found. The resulting concentration-response parameters for daily ozone-related nonaccidental mortality are as follows:

$$\text{Low daily ozone mortality risk} = 0 \qquad (4\text{-}7a)$$

$$\text{Central daily ozone mortality risk} = 4.3 \times 10^{-9} \times POP_j \times (O_j) \qquad (4\text{-}7b)$$

$$\text{High daily ozone mortality risk} = 7.4 \times 10^{-9} \times POP_j \times (O_j), \qquad (4\text{-}7c)$$

where:

POP_j = total population in area j
O_j = change in daily high-hour ozone in area j.

Because about half of the selected studies found a statistically significant relationship between ozone and mortality and half did not, we selected equal probabilities weights for each of the selected risk values: 33% for the low estimate of zero ozone mortality, 33% for the high estimate based on the studies that found a significant relationship between ozone and mortality, and 34% for the central estimate based on a weighted average of all nine study results.

4.4 ACUTE AND CHRONIC MORBIDITY FOR PM_{10} AND OZONE

In this section we describe the development of the concentration-response functions for selected morbidity effects. Epidemiologic studies have found associations for PM_{10} and ozone with morbidity effects ranging from elevated hospital admissions rates to small differences in lung function measurements. The studies selected as the basis for quantitative estimates in this report provide evidence for a ranges of illnesses and symptoms likely to have some economic significance; this means symptoms that are noticeable to the subject and that can be expected to have some impact on the individual s well-being. For this reason, studies that evaluate only short-term effects on lung function have not been included. Although this may be a medically relevant health endpoint, it cannot at this time be translated into changes in symptoms or illness that can be readily valued.

4.4.1 Chronic Respiratory Disease: PM_{10} Evidence

For at least the past two decades, there has been some evidence suggesting that higher ambient particulate matter exposures are associated with higher rates of chronic respiratory disease. Much of this evidence, however, has been based on cross-sectional analyses, comparing disease or symptom prevalence rates in different communities with different average pollution levels (e.g., Ferris et al., 1973; 1976; Hodgkin et al., 1984; Portney and Mullahy, 1990). These studies can suggest a possible association, but are difficult to use for quantitative estimates of specific concentration-response functions because they look at differences in prevalence rather than just new cases of chronic illness.

Recently published articles (Abbey et al., 1991; 1993) reported results of a 10-year cohort study conducted at Loma Linda University in California with a large sample of nonsmoking adults. The follow-up evaluations in this study allowed for the development of information on changes in chronic respiratory disease incidence over time and exposure measures for the 10-year period. Thus, new cases of disease were analysed in relation to pollution exposure for a matching time period. This study provides, for the first time, a concentration-response function for new cases of

chronic respiratory disease. However, uncertainties about the nature of the exposure that leads to chronic illness, and lag times between exposure and illness onset, still exist with these findings. This difficulty stems primarily from uncertainty about how to characterize the relevant exposure units, in particular the time aspects of exposure. Chronic symptoms presumably occur as a result of long-term exposures, but cross-sectional analyses are not very enlightening about whether, for example, it is the 5-year average, the 20-year average, or the number of times a given concentration is exceeded that is the relevant exposure measure. Application of the concentration-response function from Abbey et al. in this analysis therefore requires some assumptions on this that are explained below.

The Loma Linda University Study

In the first stage of the Loma Linda University study, a large sample (approximately 7,000) of Seventh Day Adventists (selected because they do not smoke) was interviewed in 1977. Health histories, current respiratory symptoms, past smoking and passive smoking exposure, and residence location histories were obtained. Hodgkin et al. (1984) compared the chronic respiratory disease status of respondents who had lived for at least 11 years in either a high or a low pollution area in Southern California. After adjusting for sex, race, age, education, occupational exposure, and past smoking history, residents of the higher pollution area had a rate of chronic obstructive pulmonary disease (COPD, including chronic bronchitis, asthma, and emphysema) that was 15% higher than did residents in the low pollution area. Using the same 1977 Loma Linda sample, Euler et al. (1987) also report results showing a statistically significant association between past TSP exposure, based on residence ZIP-code history, and the prevalence of chronic respiratory disease.

Abbey et al. (1991, 1993) performed a cohort study with the Seventh Day Adventist sample in 1987, which provides better quantitative concentration-response information. Nearly 4,000 subjects who had been interviewed in 1977 were interviewed again in 1987. All were 25 years old or more in 1977. Estimates of air pollutant exposures were developed based on subjects reported residence locations over the 10-year period and pollutant measures from stationary outdoor monitors at each location over the 10-year period.

Several different health outcomes were examined in 1987, including new cases of emphysema, chronic bronchitis, or asthma, among those who had not reported any definite symptoms of these diseases in 1977. Disease definition was based on self-reported symptoms using the standardized respiratory symptoms questionnaire developed by the National Heart and Lung Institute for the United States. Respondents were classified as having *definite* symptoms of emphysema, chronic bronchitis, or asthma if they met specific criteria for the disease diagnosis. Having definite symptoms of any one of these three was defined as definite airway obstructive disease (AOD). Having definite chronic bronchitis was defined as having symptoms of cough and/or sputum production on most days for at least 3 months/year, for 2 years or more. Emphysema and asthma required a physician s diagnosis as well as associated symptoms. Respondents with some

respiratory symptoms, but who did not meet the full criteria for that disease, were classified as *possible*.

Logistic models were estimated for mean concentrations of TSP and for frequency of hours above selected concentration levels (60, 75, 100, 150, and 200 g/m^3). The regressions included independent variables for past and passive smoking exposure, possible symptoms in 1977, childhood respiratory illness, gender, age, and education. A statistically significant association was reported between long-term TSP exposure levels and AOD, as well as for chronic bronchitis alone. Statistically significant TSP measures were mean levels for the 10-year period and the number of hours at a concentration of 100 g/m^3 and higher. Statistically significant results for asthma were obtained only for number of hours at a concentration of 150 g/m^3 and higher.

Threshold Evidence for Chronic Respiratory Disease

The same uncertainty exists regarding the potential existence and level of a threshold for chronic effects of long-term particulate matter exposure as for health effects associated with short-term exposures, but some additional comments are warranted. There is no clear a priori reason to expect that a threshold for short-term exposures would necessarily be the same, higher, or lower than a threshold for long-term exposures.

Two studies conducted to date provide some suggestive evidence that there may be a threshold level for chronic respiratory effects associated with particulate matter exposures. As noted above, Abbey et al. (1991, 1993) report no significant relationship between any chronic respiratory effects and hours above a concentration of 60 or 75 g/m^3 TSP, but do report a significant association for the number of hours above 100 g/m^3. They also report a significant association with mean TSP levels, and report that about 25% of the sample was exposed to mean TSP levels of 75 g/m^3 or less. These results do not prove one way or another whether it is mean exposure or peak exposure, or some combination of the two, that causes the elevated risk, nor do they prove the existence of a threshold. In this analysis, a concentration-response function for chronic bronchitis based on average particulate matter exposures is selected, but this does not exclude the possibility that it is the peak levels associated with a given average level that actually cause the risk rather than chronic exposure to low or moderate TSP levels.

The Abbey et al. results suggest that if hourly levels of TSP do not exceed 100 g/m^3, there does not appear to be an elevated risk of developing chronic respiratory disease. However, hourly peaks of TSP above 100 g/m^3 are quite common in urban areas even when annual average TSP concentrations are well below 70 g/m^3 (the current Canadian acceptable objective of annual average TSP). Given this, and that the current Canadian acceptable objective for TSP is 120 g/m^3 for a 24-hour average, these findings suggest that if a threshold exists it is well below the current Canadian acceptable objectives for particulate matter.

Chestnut et al. (1991) report that lung function is lower in locations with quarterly TSP levels above 60 g/m^3 TSP. This translates to about 33 g/m^3 PM_{10}. A quarterly average can exceed

33 g/m^3, although annual averages are below this level. In any case, 33 g/m^3 is well below the current U.S. federal annual average PM_{10} standard of 50 g/m^3.

Neither of these studies provides definitive information on whether a chronic effects threshold exists or, if it does, what it would be in terms of annual average particulate matter levels, except that it appears to be well below current federal standards.

Chronic Bronchitis Risk Estimates from Abbey et al. (1993)

The chronic bronchitis (CB) results from Abbey et al. (1993) are selected for quantification in this analysis to avoid any ambiguity in terms of the meaning of the health endpoint. The reported relative risks for AOD are slightly higher than those for chronic bronchitis alone, so focusing on chronic bronchitis is conservative in terms of not overstating the estimated chronic respiratory risks associated with TSP. The estimates based on these findings are conservative for several other reasons as well. One is that the estimates do not reflect any mortality due to chronic respiratory disease that may have occurred during the 10-year period. Subjects are in the sample only if they were alive in 1987. Another reason is that the estimates used in this analysis reflect only the development of new cases. The authors report evidence that increased severity of AOD is associated with TSP exposure for those who reported definite symptoms in 1977. Thus, it appears that TSP exposure both aggravates existing cases and causes new cases.

Two uncertainties in the quantitative estimates based on Abbey et al. (1993) should be noted. First, the authors report that a few subjects who initially described symptoms that are classified as chronic bronchitis did not continue to report these symptoms in follow-up evaluations. This suggests reversibility in the symptoms for some subjects that is not consistent with how chronic bronchitis is defined in the economics studies that have estimated monetary values for reducing risks of developing chronic bronchitis. This does not invalidate the relative risk for self reported symptoms in relation to pollution exposure, but it raises some questions regarding monetary valuation of these cases. This is discussed further in Chapter 5. The second uncertainty is how long a change in PM_{10} exposure must exist before a change in chronic bronchitis incidence occurs. The estimates are annualized here based on the assumption that the change in risk begins as soon as the change in air pollution exposure begins. This probably overstates the change in new chronic bronchitis cases in the first few years after a reduction in PM_{10} concentrations, but there is not enough information from the study to determine what the lag between changes in exposure and changes in risk may actually be.

Abbey et al. (1993) report a relative risk of 1.36 for a new case of chronic bronchitis at a 10-year average exposure of 60 g/m^3 TSP versus zero TSP. This result is based on multiple logit regression analyses using mean TSP levels for the 10-year period. The relative risk is the ratio of the risk for a person in the exposed population to the risk for a person in the nonexposed population. To calculate the average risk per individual per unit of TSP, the estimated logit coefficient rather than the reported relative risk is needed. The following standard relationship between the logit coefficient and relative risk is therefore used:

$$\text{Exp}(b \times \quad \text{TSP}) = \text{RR} , \tag{4-8}$$

where:

b = the estimated logit coefficient for mean TSP
TSP = the TSP increment
RR = the relative risk.

Substituting the reported relative risk of 1.36 for a 60 g/m^3 increment of TSP into Equation 4-8, the estimated logit coefficient of 0.00512 per g/m^3 TSP is obtained. The logit coefficient can be used to calculate the probability of developing chronic bronchitis in 10 years per unit of TSP as follows:

$$\text{Pr}/ \quad \text{TSP} = b \times \text{Pr} \times (1 - \text{Pr}) , \tag{4-9}$$

where:

Pr = probability an individual comes down with chronic bronchitis in 10 years.

Substituting the mean incidence of new cases of chronic bronchitis in the study sample (234 new cases out of 3,310 subjects) and the estimated logit coefficient into Equation 4-9 gives the predicted probability of new cases in 10 years as a function of the 10-year average TSP level, as shown in Equation 4-10. The plus and minus figure in parentheses is one standard error, estimated assuming the logit coefficient is significant at the 95% confidence level. Standard errors were not reported by the authors, but the relative risk was reported with a $p < 0.05$. The 95% confidence interval was therefore estimated for a presumed $p = 0.05$, and the resulting confidence intervals were used to calculate high and low estimates.

$$\text{New cases/1,000,000/10 years} = 338 (\pm 171) \times \text{TSP}_{10\text{yr}} . \tag{4-10}$$

This result is first divided by 0.55 to convert to PM_{10} and then divided by 10 to obtain an estimate of the average annual number of new cases. This annual average probably overstates the new cases expected in the first few years after a change in PM_{10} levels occurs, but probably understates the number of new cases after several years of higher exposure. The exact lag between the change in exposure and the elevated risk is not known, but the Abbey et al. results indicate that it is within 10 years.

We apply the risk estimates to the adult population age 25 and over because this is the minimum age in the Abbey et al. study group. Chronic bronchitis takes a while to develop, and these risk estimates may not apply to younger individuals. The calculation procedure, including the concentration-response parameters, used in this analysis to obtain the number of new chronic bronchitis cases each year is as follows:

$$\text{Low annual cases of CB} = 3.0 \times 10^{-5} \times \text{POP}_{25j} \times (\Delta \text{PM}_j) \quad (4\text{-}11a)$$
$$\text{Central annual cases of CB} = 6.1 \times 10^{-5} \times \text{POP}_{25j} \times (\Delta \text{PM}_j) \quad (4\text{-}11b)$$
$$\text{High annual cases of CB} = 9.3 \times 10^{-5} \times \text{POP}_{25j} \times (\Delta \text{PM}_j), \quad (4\text{-}11c)$$

where:

CB	=	adult chronic bronchitis
POP_{25j}	=	population age 25 and over years in area j
ΔPM_j	=	change in annual average PM_{10} in area j.

The probability weight selected for the central estimate is 50%, with the low and high each given 25% weights.

4.4.2 Hospital Admissions: PM_{10} and Ozone

Recent evidence suggests an association between RHAs and ambient particulate matter and ozone concentrations. In addition, recent evidence suggests a relationship between cardiac hospital admissions (CHAs) and ambient particulate matter levels.

PM_{10} and Respiratory Hospital Admissions

For this analysis, quantitative estimates of the relationship between PM_{10} and RHAs are developed from the results of the Burnett et al. (1995) and Pope (1991) studies. Burnett et al. (1995) examined the relationship between RHAs and sulphate from 1983 through 1988 in Ontario. The analysis utilized air pollution data from a large network of monitors throughout Ontario and counts of the daily RHAs for all ages and specific age groups, excluding elective admissions, from 168 acute care hospitals located in Ontario below the 47th parallel. The association was evaluated using time-series regression models that controlled for day-of-week effects, slow moving serial correlations associated with seasonal patterns, and differences between hospitals. The model results were ultimately used to estimate the percentage of excess respiratory admissions associated with sulphates.

Pope et al. (1991) analysed monthly frequencies of RHAs for all ages, as well as for specific age defined population subsets, with respect to mean monthly PM_{10} concentrations from 1985 through 1989 in two Utah valleys. The study utilized linear regression models that controlled for temperature and autocorrelation to examine the association. Ozone concentrations were low during the winter season when RHAs and PM_{10} were elevated, so that unlike with the eastern North American studies, there should be little problem with covarying ozone concentrations affecting the PM_{10} parameter estimates.

For a central and low concentration-response parameter estimate of RHAs associated with particulate matter, we use the sulphate results from Burnett et al. (1995) from a model that also

controlled for ozone in the regression, thereby reducing the chance of overstating the combined effect of these two collinear pollutants. We give greater weight to the results of this study because it utilises Canadian air pollution and hospital admissions data. For a high estimate, we use the Pope (1991) results.

Specifically, Burnett et al. (1995) report 3.5% of excess RHAs are associated with a 13 g/m^3 increase in sulphate based on the results from the model that also controlled for ozone. The average number of RHAs per day for the study period was 16.0 per million population. As a result, 3.5% of the 16.0 daily RHA are attributed to a 13 g/m^3 increase in sulphate. Therefore, the daily RHA concentration-response parameter per g/m^3 sulphate is $0.035 \times (16.0 \times 10^{-6}) \div 13 = 4.31 \times 10^{-8}$. This sulphate-based result is converted to its PM_{10} equivalent assuming a ratio of sulphate to PM_{10} of 0.18 in Ontario (Dann, 1994). As a result, multiplying by 0.18 gives the daily RHA concentration-response parameter per g/m^3 of PM_{10} (the standard error associated with the initial estimate can be calculated based on the reported 95% confidence interval). Thus, the central and low estimates for RHAs from PM_{10} are as follows, with the low estimate representing the central estimate minus one standard error:

$$\text{Central daily RHA for } PM_{10} = 7.8 \times 10^{-9} \times PM_{10} \times POP_j \quad (4\text{-}12a)$$

$$\text{Low daily RHA for } PM_{10} = 6.4 \times 10^{-9} \times PM_{10} \times POP_j, \quad (4\text{-}12b)$$

where:

POP_j = total population in area j
PM_{10} = change in daily (24-hour) PM_{10}.

We apply a 50% probability to the central estimate, and 33% to the low.

The reported coefficients from the linear regressions in the Pope (1991) study (plus and minus one standard error) are divided by the reported population in each valley to estimate the monthly RHA concentration-response parameter per person per g/m^3 of mean monthly PM_{10}.

$$\text{Utah Valley: Monthly RHA per person} = 0.95 (\pm 0.53) \times 10^{-6} \times PM_{10} \quad (4\text{-}13a)$$

$$\text{Salt Lake Valley: Monthly RHA per person} = 1.03 (\pm 0.36) \times 10^{-6} \times PM_{10}, \quad (4\text{-}13b)$$

where:

PM_{10} = change in monthly average PM_{10} in g/m^3.

Dividing these results by 30.4 converts the estimates into an equivalent individual RHA risk factor per g/m^3 change in daily PM_{10}. This is a straightforward transformation given the linear form of the original monthly regressions. Thus, the Pope results imply the following daily relationships.

Utah Valley: Daily RHA per person = 3.13 (± 1.8) × 10^{-8} × PM_{10} (4-14a)
Salt Lake Valley: Daily RHA per person = 3.39 (± 1.2) × 10^{-8} × PM_{10} , (4-14b)

where:

PM_{10} = change in 24-hour PM_{10} in g/m^3.

Averaging the Pope results for the two study areas results in the following relationship.

$$\text{High daily RHA for } PM_{10} = 3.26 \times 10^{-8} \times (\ PM_j) \times POP_j\ , \quad (4\text{-}15)$$

where:

POP_j = population in location j
PM_j = change in daily (24-hour) PM_{10} in area j.

This averaged result from Pope et al. (1991) is used as the high concentration-response parameter, but is given a smaller probability weight of 17% because the results are so much higher than the Canadian study results.

Ozone and Respiratory Hospital Admissions

The results of Burnett et al. (1997) are used to quantify low, central, and high concentration-response parameters for the association between RHAs and ozone. The study evaluates air monitoring and hospital admissions data from a geographically diverse sample of 16 Canadian cities, all of which have at least 100,000 residents, from April 1981 through December 1991. The study was selected because of this broad geographic scope, the lengthy time period considered, and the use of Canadian admissions and air monitoring data.

Concentration-response parameters are developed from a model examining the observed number of RHAs with respect to the previous day s high hourly ozone concentration while controlling for particulate matter, with a COH measure, carbon monoxide, cyclical fluctuations, and meteorological factors. While some analyses in the study encompass data from all 16 cities, the model of interest utilises data from the 11 cities with COH information. The model results imply that a 30 ppb increase in ozone would be associated with a 2.4% increase in RHAs. This is equivalent to a 0.08% increase in RHAs per ppb. This result, combined with the observed average daily RHA rate for these cities of 1.39 per 100,000, suggests a central daily RHA concentration-response parameter of 1.1(± 0.5) × 10^{-8} per ppb daily high hour ozone. The low and the high concentration-response parameters are estimated as minus and plus one standard deviation from the central estimate. The standard deviation was in turn estimated based on the reported significance level of the initial model results. The resulting low, central, and high, daily RHA concentration-response parameters are as follows:

$$\text{Low daily RHA for ozone} = 0.6 \times 10^{-8} \times O_{3j} \times POP_j \quad (4\text{-}16a)$$
$$\text{Central daily RHA for ozone} = 1.1 \times 10^{-8} \times O_{3j} \times POP_j \quad (4\text{-}16b)$$
$$\text{High daily RHA for ozone} = 1.6 \times 10^{-8} \times O_{3j} \times POP_j\,, \quad (4\text{-}16c)$$

where:

O_3 = change in daily high hour ozone in area j
POP_j = population in area j.

We apply a 50% probability to the central estimate, and 25% probability to both the low and high estimates.

Additional studies provide support for these results. Thurston et al. (1992, 1994) found evidence of an association between RHAs during summer months and either sulphate or ozone concentrations, or both. The 1992 study, however, did not report results for models that include both ozone and sulphate, so their results for both pollutants are most likely confounded by the presence of the other correlated pollutant, while the 1994 study covered only a limited period during the expected high pollution season. However, the results are useful for rough comparison to the Burnett et al. results. Burnett et al. (1994) found that the mean sulphate concentration was associated with a 2.2% increase in RHA when only sulphate was included in the model, and that the mean ozone concentration was associated with a 6.0% increase in RHAs when only ozone was included in the model. (These percentages fell to 1.4% and 4.5%, respectively, when sulphate and ozone were both included in the model.) The single pollutant results are similar to results obtained by Thurston et al. (1992) for New York City, which were 3.5% for mean sulphate and 5.3% for mean ozone. These estimates are also reasonably consistent with the findings obtained in the Toronto study (Thurston et al., 1994). The Burnett et al. (1994) study found that 4.5% of RHAs in Ontario were associated with an increase in ozone from 0 to 50 ppb in a model that accounted for sulphates.

Bates and Sizto (1989) provide some additional evidence on the issue. They estimated a stepwise regression for RHAs during the summer months in Ontario. First they included temperature, which explained 0.89% of the variance in RHAs. Then they added sulphate, which increased the explained variance to 3.3%. When ozone was then added, the explained variance increased to 5.6%. This suggests that adding ozone to the regression explains about as much of the variance as that explained by the sulphate variable. Because ozone was added last, this is reasonably strong evidence that there is an additional effect associated with ozone. The authors also report that RHAs are about 7% higher on high ozone (80 to 200 ppb) days than on low ozone (10 to 60 ppb) days, although the authors note that there is not sufficient evidence from their analysis to attribute this entire effect to ozone.

PM_{10} and Cardiac Hospital Admissions

The association between CHAs and particulate matter has also been examined. The CHA risk factors with respect to PM_{10} are estimated based on the Burnett et al. (1995) results for sulphates. This study, described above in the section for PM_{10} and RHAs, reports that 3.3% of excess CHAs are associated with a 13 g/m^3 increase in sulphate in a model controlling for ozone. The sulphate-based results were converted to PM_{10}, as in the RHA analysis, assuming a ratio of sulphate to PM_{10} of 0.18 in Ontario (Dann, 1994). Thus, 3.3% of the average number of CHAs per day per million population (14.4) in the study area are associated with a 13 g/m^3 increase in sulphate. Dividing by 13 gives the daily CHAs per g/m^3 sulphate [$0.033 \times (14.4 \times 10^{-6}) \div 13 = 3.66 \times 10^{-8}$]. Multiplying by 0.18 converts these results from sulphates to the daily CHAs per g/m^3 PM_{10}. The central CHA concentration-response parameter for PM_{10} is thus as follows, with the low and high parameters selected as minus and plus one standard error (standard errors calculated based on the reported 95% confidence interval) from the central estimate:

$$\text{Low daily CHA for } PM_{10} = 5.0 \times 10^{-9} \times PM_{10} \times POP_j \quad (4\text{-}17a)$$

$$\text{Central daily CHA for } PM_{10} = 6.6 \times 10^{-9} \times PM_{10} \times POP_j \quad (4\text{-}17b)$$

$$\text{High daily CHA for } PM_{10} = 8.2 \times 10^{-9} \times PM_{10} \times POP_j, \quad (4\text{-}17c)$$

where:

POP_j = total population in area j
PM_{10} = change in daily (24-hour) PM_{10}.

We apply a 50% probability to the central estimate, and 25% probability to both the low and high estimates.

4.4.3 Emergency Room Visits: PM_{10} and Ozone

Studies in the United States have found an association between particulate matter and the incidence of ERVs for all causes (Samet et al., 1981) and for asthma-related diagnoses (Schwartz et al., 1993). One U.S. study (Weisel et al., 1995) and two Canadian studies (Bates et al., 1990; Stieb et al., 1996) also found an association between ozone and emergency room visits for asthma. These findings are consistent with the findings for hospital admissions in that many hospital admissions for acute events begin with a visit to the emergency department. To estimate the numbers of ERVs associated with changes in PM_{10} and ozone we follow the approach taken in the sulphate panel report in assuming the ERVs are proportional to the pollution-related hospital admissions described in the previous section. This produces estimates of all respiratory-related ERVs (and cardiovascular for PM_{10}), not just those related to asthma.

The Saint John Particle Health Effects Study (Stieb et al., 1995) provides data that indicate that for each respiratory disease hospital admission in Saint John, NB, there are 5.3 ERVs for

respiratory diseases, and for each cardiovascular disease hospital admission there are 1.4 ERVs. For example, the low ERV concentration-response parameter is thus $(5.3 \times 0.64 \times 10^{-8}) + (1.4 \times 5.0 \times 10^{-9}) = 4.1 \times 10^{-8}$. Assuming these ratios apply elsewhere in Canada, and using the above derived concentration-response parameters for hospital admissions in each category, this yields:

$$\text{Low daily ERV} = 4.1 \times 10^{-8} \times (\ PM_j) \times POP_j \quad (4\text{-}18a)$$
$$\text{Central daily ERV} = 5.1 \times 10^{-8} \times (\ PM_j) \times POP_j \quad (4\text{-}18b)$$
$$\text{High daily ERV} = 18.4 \times 10^{-8} \times (\ PM_j) \times POP_j\ , \quad (4\text{-}18c)$$

To estimate the net ERV concentration-response parameters, the corresponding low, central, and high estimates for the RHA and CHA parameters are subtracted from the ERV estimates above. This adjustment results in the following net ERV concentration-response parameters for PM_{10}:

$$\text{Low net daily ERV} = 2.96 \times 10^{-8} \times (\ PM_j) \times POP_j \quad (4\text{-}18d)$$
$$\text{Central net daily ERV} = 3.66 \times 10^{-8} \times (\ PM_j) \times POP_j \quad (4\text{-}18e)$$
$$\text{High net daily ERV} = 14.3 \times 10^{-8} \times (\ PM_j) \times POP_j\ , \quad (4\text{-}18f)$$

where:

POP_j = population in location j
PM_j = change in daily average PM_{10} in area j.

For ozone, we have estimates of respiratory hospital admissions only. Using the 5.3 ratio applied to the RHA estimates for ozone, and following the above procedure, we obtain the following concentration-response parameters for ozone-related emergency room visits:

$$\text{Low daily ERV for ozone} = 3.2 \times 10^{-8} \times\ O_{3j} \times POP_j \quad (4\text{-}19a)$$
$$\text{Central daily ERV for ozone} = 5.8 \times 10^{-8} \times\ O_{3j} \times POP_j \quad (4\text{-}19b)$$
$$\text{High daily ERV for ozone} = 8.5 \times 10^{-8} \times\ O_{3j} \times POP_j\ , \quad (4\text{-}19c)$$

To estimate the net ERV concentration-response parameters, the corresponding low, central, and high estimates for the RHA parameters are subtracted from the ERV estimates above. This adjustment results in the following net ERV concentration-response parameters:

$$\text{Low net daily ERV for ozone} = 2.6 \times 10^{-8} \times\ O_{3j} \times POP_j \quad (4\text{-}19d)$$
$$\text{Central net daily ERV for ozone} = 4.7 \times 10^{-8} \times\ O_{3j} \times POP_j \quad (4\text{-}19e)$$
$$\text{High net daily ERV for ozone} = 6.9 \times 10^{-8} \times\ O_{3j} \times POP_j\ , \quad (4\text{-}19f)$$

where:

O_3 = change in daily high hour ozone in area j
POP_j = population in area j.

As with the hospital admissions estimates, the probability weight selected for the central parameter of emergency department visits is 50%, with the low and high parameter each given 25% weights.

4.4.4 Aggravation of Asthma Symptoms: PM_{10} and Ozone

Several studies have related air pollutant concentrations to exacerbation of asthma symptoms in individuals with diagnosed asthma. Two epidemiologic studies with currently diagnosed asthmatics provide quantitative information to allow estimates of the frequency of elevated asthma symptoms as a function of ambient particulate matter concentrations (Whittemore and Korn, 1980; Ostro et al., 1991). Whittemore and Korn (1980), Holguin et al. (1985), and a reanalysis of the latter reported by Stock et al. (1988) also found a significant association of asthma symptoms with ozone concentrations, while controlling for the particulate matter effect.

All of these studies had subjects (diagnosed asthmatics) record daily asthma symptoms during the duration of the study. An elevation of asthma symptoms was defined for each subject based on each individual s manifestation of asthma symptoms. This typically meant a notable increase in symptoms, such as shortness of breath or wheezing, and/or in use of medication relative to what was normal for that individual. Daily particulate matter and ozone levels were then examined for correlations with day-to-day fluctuations in asthma symptom frequency, controlling for other factors such as weather and previous-day symptoms.

Whittemore and Korn (1980) studied asthmatics (adults and children) living in six different communities in the Los Angeles area. Each subject reported asthma symptoms during one or more 34-week periods between 1972 and 1975. A total of 443 subject periods of data was obtained (some subjects provided data for more than one period). The study used a statistical approach to estimate both individual-level and group effects.

Ostro et al. (1991) examined the association between several different air pollutants, including sulphates, $PM_{2.5}$, and acidic aerosols, and increases in asthma symptom days among adults during winter months in Denver. A significant association was found between the probability of moderate or severe asthma symptom days (measured as shortness of breath) and sulphate particulate levels, after controlling for temperature, day of week, previous-day illness, and use of a gas stove. Ozone levels were very low, near background levels, and do not create a confounding influence.

The Ostro et al. (1991) results suggest greater effects of particulate matter on asthma symptoms than those obtained by Whittemore and Korn (1980), however, this might be because Ostro et al. considered only the winter months in Denver (the high particulate matter season), when more frequent respiratory colds may cause asthmatics to be more sensitive to air pollutants. The central concentration-response parameter for PM_{10}-related ASDs is based on the Whittemore and Korn (1980) results. The high parameter is selected as the average of the Whittemore and Korn (1980)

results and the Ostro et al. (1991) results, and the low parameter is based on Whittemore and Korn (1980) minus one standard error.

The logistic model used by Whittemore and Korn (1980), Holguin et al. (1985), and Stock et al. (1988) generates an equation with a nonlinear first derivative. Thus, we need a baseline probability rate for elevated asthma symptoms to predict the frequency of days with elevated asthma symptoms per unit of ozone. The average rate of asthma symptom days for the Whittemore and Korn study sample is available, but it appears to be quite high, suggesting that the study sample over-represents fairly severe levels of asthma. The authors report that some subjects were excluded because of insufficient manifestation of asthma symptoms. More representative data on average asthma symptom frequency are, however, not available at this time. It is therefore necessary to make some reasoned assumptions about what an average rate may be.

In the Los Angeles study sample, about 26% of the sample experienced elevated asthma symptoms on any given day, but subjects who did not report any asthma symptoms during the study period were excluded, so 26% probably overstates the asthma symptom rate for a typical sample of asthmatics. If all of the excluded subjects are presumed to have had no elevated asthma symptoms during the study period and this is factored into the calculation, the average daily symptom rate is reduced to 15%. This is similar to the 15% shortness of breath frequency reported by Ostro et al. (1991). Also, Holguin et al. (1985) report an average daily asthma symptom rate of 15% for their study sample, or 13% if those excluded from the study are factored in. As a check on the plausibility of these rates as representative of the active asthmatic population we consider asthma severity information reported by the National Center for Health Statistics (1980). They report that of all active asthmatics in the United States, 55% have mild symptoms, 32% have moderate symptoms, and 13% have severe symptoms.[5] If we assume that mild means one symptom per month, moderate means one symptom per week, and severe means a symptom every other day, the average daily symptom rate would be 13%. We select this lower approximated rate to minimize the chance of overstating the expected effect of PM_{10} and ozone on the average asthmatic.

5. The 1979 Health Interview Survey report (National Center for Health Statistics, 1980) gives the frequency of bother and the severity of asthma for respondents with diagnosed asthma. We calculated a frequency of asthma symptoms by cross tabulating this descriptive information. We defined mild as those who report being bothered "some" by asthma symptoms "once in a while." We defined severe as those who report being bothered "a great deal" by asthma symptoms "often" or "all the time." We defined moderate as everything in between.

Specifically, using a logistic model with both particulate matter and ozone included in the model, Whittemore and Korn (1980) obtained a coefficient for daily (24-hour) TSP (g/m^3) of 0.00079, with a standard error of approximately 0.00034. The probability of elevated asthma symptoms on a given day (ASD) as a function of PM_{10} levels is given by the following relationship in a logistic specification:

$$\Pr/\ PM_{10} = b \times \Pr \times (1 - \Pr)\ , \qquad (4\text{-}20)$$

where:

Pr = probability of elevated asthma symptoms on a day
b = the estimated logit coefficient for PM_{10}.

Using 13% as the baseline probability that an asthmatic will experience elevated asthma symptoms on a given day, and substituting the estimated TSP coefficient (adjusted to PM_{10} by dividing by 0.55) into Equation 4-20, gives the following results from Whittemore and Korn (1980):

$$\text{Daily ASD for } PM_{10} = 1.62 \text{ (s.e. } = 0.70) \times 10^{-4} \times \ PM_{10}\ . \qquad (4\text{-}21)$$

The Ostro et al. (1991) results suggest the following relationship between elevated asthma symptoms and daily sulphate (SO_4) concentrations:

$$\text{Daily ASD for sulphate} = 0.0077 \text{ (s.e. } = 0.0038) / SO_4\ . \qquad (4\text{-}22)$$

Using the reported SO_4 mean for the study of 2.11 g/m^3 to linearise the function and converting from sulphate to PM_{10} multiplying by a conversion factor of 0.05 yields the following daily ASD concentration-response parameter per asthmatic based on the Ostro et al. (1991) results[6]:

$$\text{Daily ASD for } PM_{10} = 1.82 \ (\pm\ 0.90) \times 10^{-4} \times \ PM_{10}\ . \qquad (4\text{-}23)$$

The results from the two studies are quite comparable. We take an average of the Ostro et al. (1991) results (converted to PM_{10}) and the Whittemore and Korn (1980) results (converted to PM_{10}) for the central ASD concentration-response parameter. The low concentration-response parameter is based on Whittemore and Korn, and the high concentration-response parameter is based on Ostro et al. (1991). The resulting ASD concentration-response parameters are applied to the diagnosed asthmatic population (estimated to be 6.0% of the Canadian population, Statistics Canada, 1994) as follows:

$$\text{Low daily ASD for } PM_{10} = 1.62 \times 10^{-4} \times (\ PM_j) \times POP_{aj} \qquad (4\text{-}24a)$$
$$\text{Central daily ASD for } PM_{10} = 1.72 \times 10^{-4} \times (\ PM_j) \times POP_{aj} \qquad (4\text{-}24b)$$

6. The Ostro et al. study reports mean concentrations of $PM_{2.5}$ at the same monitor during the study period. From this we calculated a sulphate-to-$PM_{2.5}$ ratio of 0.1. This is consistent with our expectations that Denver has lower SO_2 emissions than many parts of the U.S. Data provided to us by the Colorado Department of Public Health and Environment (personal communication, Bill Kotasek, Colorado Department of Public Health and Environment, April 20, 1999) show an average $PM_{2.5}$-to-PM_{10} ratio during winter months at a downtown Denver location (the Ostro et al. data were also measured downtown) of about 0.5. Combined, this gives a sulphate-to-PM_{10} ratio of 0.05.

$$\text{High daily ASD for } PM_{10} = 1.82 \times 10^{-4} \times (\ PM_j) \times POP_{aj}\ , \qquad (4\text{-}24c)$$

where:

POP_{aj} = asthmatic population in location j (6% of POP_j)
PM_j = change in daily (24-hour) PM_{10} in area j.

Each estimate is given an equal probability weight.

In their analysis of the Los Angeles data, Whittemore and Korn (1980) obtained an ozone coefficient from the logistic model for daily high-hour ozone (ppb oxidant) of 0.00166, with a standard error of approximately 0.00072. The Holguin et al. (1985) study of the relationship between ambient ozone levels and ASDs for 51 individuals with asthma living in Houston was of similar design to the Whittemore and Korn study. Subjects kept a daily record of asthma symptoms for the period of May through October 1981. Using statistical analysis techniques similar to those used by Whittemore and Korn, Holguin et al. report an estimated ozone (daily high-hour ppb) coefficient of 0.0062, with a standard error of 0.0023 from a logistic model. In their reanalysis of the Houston data, Stock et al. (1988) report additional results obtained with a measure of fine particle exposure added to the specification. The reported ozone coefficient from the revised model was 0.0046, with a standard error of 0.0035. The lower statistical significance of ozone in the Stock et al. (1988) results appears to have resulted from a reduced number of exposure periods in the analysis due to missing data for fine particles, and a stronger effect of pollens during the remaining exposure periods.

The Houston study predicts a greater response to ozone than the Los Angeles study. One reason for this may be that the ozone measure in the Houston study was adjusted for indoor exposure. It is likely that the Whittemore and Korn estimate would have been greater if this adjustment were applied to their data. We use the Whittemore and Korn (1980) results for the central and low concentration-response parameters for ozone-related ASDs, and the Stock et al. (1988) Houston results for the high concentration-response parameter. The Stock et al. results were chosen rather than the Holguin et al. results because they account for fine particles in the analysis.

In the same logistic model described above in discussing the relationship of ASDs to PM_{10}, Whittemore and Korn (1980) obtained a coefficient for ozone (oxidant daily high-hour ppb) of 0.00166 with a standard error of approximately 0.00072. Substituting the 13% presumed average asthma symptom rate and the results for ozone from Whittemore and Korn into Equation 4-20, we get a central concentration-response parameter for ozone-related ASDs of:

$$\text{Central daily ASD for ozone} = 1.88 \times 10^{-4} \times \ O_3\ , \qquad (4\text{-}25)$$

where:

O_3 = change in daily maximum 1-hour ozone in ppb.

For a low concentration-response parameter we use Whittemore and Korn results minus one standard deviation:

$$\text{Low daily ASD for ozone} = 1.06 \times 10^{-4} \times \quad O_3 \, . \tag{4-26}$$

The Stock et al. (1988) Houston study measured attacks and peak 1-hour ozone exposure in each 12-hour period from 7 a.m. to 7 p.m. and from 7 p.m. to 7 a.m. We expect that the measured ozone effect is dominated by the daytime periods because this is when ozone exposures are at their highest. This means that if we use their model to predict asthma symptoms over the entire day by simply multiplying by two, we could seriously overstate the number. Because the Houston coefficient for the 12-hour period already exceeds the Los Angeles results by a factor of about three, we make no further adjustments when using the Houston results to predict the number of ASDs per day for each unit of ozone.

For a high concentration-response parameter we use the Stock et al. results for the Houston study:

$$\text{High daily ASD for ozone} = 5.20 \times 10^{-4} \times \quad O_3 \, POP_{aj} \, . \tag{4-27}$$

We give less weight to the Houston results because of the smaller sample size and uncertainty in interpreting the ozone exposure measure. As a result, we give a 50% probability weight to the central estimate, 33% to the low, and 17% to the high.

All of the ozone-related ASDs concentration-response parameters are intended for use with the 6% of the Canadian population that has diagnosed asthma as a susceptible population.[7]

4.4.5 Restricted Activity Days: PM_{10}

RADs include days spent in bed, days missed from work, and days when activities are partially restricted because of illness. Ostro (1987) examined the relationship between adult all-cause RADs in a two-week period and particles with diameter less than 2.5 microns ($PM_{2.5}$) in the same two-week period for 49 metropolitan areas in the United States. The RAD data were from the U.S. Health Interview Survey (HIS) conducted annually by the National Center for Health Statistics. The $PM_{2.5}$ data were estimated from visual range data from airports in each area. Because $PM_{2.5}$ has a more significant impact on visual range than do large suspended particles, a direct relationship can be estimated between visual range and $PM_{2.5}$.

Separate regression estimates were obtained for 6 years, 1976 to 1981. A statistically significant relationship was found in each year and was consistent with earlier findings relating RADs to

7. Personal communication, Dr. Charles Mustard, Chief, Respiratory Disease Division, Laboratory Centre for Disease Control, Health Canada, 1998.

TSP by Ostro (1983). The mean of the estimated coefficient for $PM_{2.5}$ across the 6 years indicated approximately 91,200 RAD each year per 1 million population for each g/m^3 increase in annual average $PM_{2.5}$, and ranged from a low of 53,200 for the 1981 coefficient to a high of 171,000 for the 1976 coefficient.

Additional work conducted by Ostro and Rothschild (1989) added ozone measures to the regressions and found the estimated relationship between RAD and $PM_{2.5}$ to be essentially unchanged, suggesting that the RAD/$PM_{2.5}$ relationship was not confounded by the exclusion of ozone levels and is independent of ozone exposures. The newer work also estimated the relationship between respiratory RAD (RRAD) and $PM_{2.5}$ for employed individuals only. It was expected that this relationship might be more stable than that between all-cause RAD and $PM_{2.5}$ for all adults for two reasons: (1) it is expected that pollution induced RADs might be predominantly related to respiratory illness, and (2) workers might define a RAD more consistently than the entire adult population. It was expected, though, that confining the data to RRAD for workers might result in a smaller total number of predicted restricted activity days for a given level of pollution because all effects might not be classified as respiratory and workers may be a healthier and therefore less sensitive group, on average, than all adults. The findings are consistent with this expectation. The average of the $PM_{2.5}$ coefficients for the 6 years suggested an annual increase of approximately 47,100 RRAD per 1 million population for each g/m^3 increase in annual average $PM_{2.5}$, and ranged from a low of 30,800 for the 1978 coefficient to a high of 54,700 for the 1980 coefficient.

The mean results over the 6 years from Ostro (1987) for all-cause RADs for all adults (mean coefficient = 0.0048) have been selected as the basis for the central concentration-response parameter for this analysis. The mean results from Ostro and Rothschild (1989) for respiratory RADs for workers (mean coefficient = 0.0158) were selected for the low concentration-response estimate because the study excludes some nonrespiratory RADs that might be related to pollution exposures and is based on a healthier than average sample (i.e., workers). The selected high concentration-response parameter is the mean of the two highest coefficients in the 6 year analysis (mean coefficient = 0.0076) by Ostro (1987). The Ostro (1987) and Ostro and Rothschild (1989) coefficients give percentage changes in RAD or RRAD for a 1 g/m^3 change in $PM_{2.5}$. Daily average estimates from the studies based on HIS data of 0.052 RAD and 0.0083 RRAD per person are used to determine the relationship between number of RAD and $PM_{2.5}$. The following functions were obtained by converting the selected coefficients from $PM_{2.5}$ to PM_{10} and are applied to the adult population 20 years and over.[8] Based on information reported by Ostro (1987), the assumed ratio of $PM_{2.5}$ to PM_{10} is 0.625. The central concentration-response estimate is thus:

$$0.0048 \times 0.052 \times 0.625 = 1.6 \times 10^{-4} . \quad (4\text{-}28)$$

8. The relevant starting age for adult RADs is somewhat ambiguous. The HIS includes 18 and over in the adult sample. The population data available were in 5-year age groups, so for this purpose we defined adults as 20 years and older.

Following the same procedure for the low and high parameters, using the average daily RRAD in the low estimate calculation, we obtain:

$$\text{Low daily RAD} = 0.8 \times 10^{-4} \times PM_j \times POP_{20j} \quad (4\text{-}29a)$$
$$\text{Central daily RAD} = 1.6 \times 10^{-4} \times PM_j \times POP_{20j} \quad (4\text{-}29b)$$
$$\text{High daily RAD} = 2.5 \times 10^{-4} \times PM_j \times POP_{20j}, \quad (4\text{-}29c)$$

where:

POP_{20j} = nonasthmatic population in location j 20 years of age and older
PM_j = change in daily average PM_{10} in area j.

The low, central, and high PM_{10}-related RAD concentration-response parameters are applied to the population age 20 and older, and are given equal probability weights. Because daily symptom concentration-response functions for asthmatics are available based on studies focussed specifically on those with diagnosed asthma (see Section 4.4.4), we exclude the asthmatic population from the calculations of restricted activity days. Although asthmatics were not specifically excluded from the RAD studies, nonasthmatics are more representative of the response of the general population because only a small fraction of the general public has diagnosed asthma. We therefore apply the RAD concentration-response function to the nonasthmatic portion (94%) of the adult population.

4.4.6 Minor Restricted Activity Days: Ozone

Ostro and Rothschild (1989) and Portney and Mullahy (1986) both used the U.S. HIS to examine the relationship between ambient ozone levels and restrictions in activity. The survey asks respondents to estimate the number of days on which they have experienced various categories of illness or symptoms in the two-week period preceding the interview. Both studies used count models to estimate a relationship between the number of illness days for each individual and the ozone levels near the respondent s residence over the two-week recall period of the HIS. Ostro and Rothschild (1989) present separate results for each of the six years, 1976 to 1981, they analysed, while the results Portney and Mullahy (1986) reflect an analysis of only the data from 1979.

The studies focussed on minor restricted activity days (MRADs), which are days on which some but not all activities are restricted because of illness (as defined by the HIS). The ozone measure used in both studies was the 14-day average of the daily high-hour ozone measurement. Both studies included measures of particulate matter as well as ozone in the statistical analysis. Although ozone and particulate levels were not highly correlated in these studies, we select ozone results from estimations that included particulate matter as well as ozone to reduce the chance that the measured ozone effect is overstated because of any correlation with particulate levels. In both of these studies the functional form used for the statistical analysis gives an estimated coefficient that is the percentage change in illness-day frequency per unit of ozone.

While there are some differences in the analyses conducted in the two studies, a comparison of the results is still instructive. Portney and Mullahy (1986) studied respiratory RADs for all adults, while Ostro and Rothschild (1989) studied respiratory RADs and MRADs for working adults. Portney and Mullahy (1986) found a statistically significant relationship between ozone and respiratory RADs. They interpret this ozone effect as causing minor restrictions because they did not find an effect between ozone and more severe types of RADs: work-loss days or days spent in bed because of illness. Ostro and Rothschild did not find a significant positive effect between ozone and respiratory RADs in any of the 6 years, but they did find a significant effect in three of the 6 years for MRADs. It is important to note that the sample of working adults used by Ostro and Rothschild is different from the all-adults sample used by Portney and Mullahy, which could result in different findings.

The estimated coefficients from these studies represent the following relationship:

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(4-30)

where:

ΔPr	=	change in probability of an illness day per unit of ozone
Pr	=	baseline probability of an illness day due to all causes
b	=	estimated coefficient
ΔO_3	=	change in ozone.

Table 4-6 presents a comparison of the 1979 results from the two studies. Both of the coefficients give the percentage change in illness days per unit of ozone. When we multiply by the average illness-day rate in each of the samples for the illness-day definition used in each analysis, the resulting concentration-response parameters for the daily risk of illness per unit of ozone are very similar. Thus, if we multiply the daily baseline probability of an illness day for an individual by the coefficient, we obtain the change in the daily probability of an illness day for an individual per unit of ozone:

(4-31)

Table 4-6
Comparison of 1979 Restricted Activity Day Results from Two Studies[a]

Study	Estimated Coefficient (standard error)	Illness-Day Definition	Baseline Average Daily Illness Rate[b] (Pr)	Daily Risk of Illness/ppb (Pr)
Ostro and Rothschild (1989)	$5.66^{c} \times 10^{-3}$ (1.56)	MRAD	0.0214	1.21×10^{-4}
Portney and Mullahy (1986)	$9.32^{d} \times 10^{-3}$ (3.87)	Respiratory RAD	0.0115	1.07×10^{-4}

a. Both studies use 1979 HIS survey data.
b. The authors report average annual MRADs are 7.8 per person and average annual respiratory RADs are 4.2 per person in the study samples.
c. The authors report coefficients based on g/m^3 units of ozone. We converted to ppb using 1950 g/m^3 = 1000 ppb for ozone.
d. The authors used several different functional forms. This coefficient is taken from their Equation (3-3), which is a functional form similar to that used by Ostro and Rothschild and includes sulphates in the specification.

Although the results of the two studies for 1979 show considerable consistency, the Ostro and Rothschild results for all 6 years show considerable variability. For three of the years there is a statistically significant effect of ozone on MRAD frequency, ranging from 0.7×10^{-4} additional MRAD per day for each ppb increase in daily high-hour ozone to 2.3×10^{-4} additional MRAD per day for each ppb increase in ozone. However, two years show a statistically significant coefficient with a negative sign, and one year shows a statistically insignificant coefficient. This variability in results across different years requires that some way to summarise the overall findings be developed to use the results for quantitative assessment purposes. Alternative approaches that we considered include the following:

Calculate the simple arithmetic mean of the results for different years, which gives equal weight to all estimates regardless of their statistical significance.

Use information from other studies about the effects of ozone on symptoms to help evaluated the different findings. Because there is clinical evidence of adverse effects of ozone exposure on acute respiratory symptoms and no evidence of a beneficial health effect of ozone exposure, an argument could be made that based on prior information, only the positive coefficients should be interpreted as credible.

Calculate the variance weighted mean of the results for different years, which assumes that the results for different years are estimates of the same true but unknown relationship.

Relax the assumption that the estimates for different years are for the same true underlying relationship between ozone and MRADs, and allow that the relationship may differ from year to year for reasons unknown and unspecified in the model. This is essentially the random-effects model proposed by Harrison et al. (1993).

Each of these alternative approaches has some merit and some limitations. Choosing among them based on available information is a matter of judgement, because available information is not sufficient at this time to answer the questions that these alternatives raise. The implications of the alternatives are, however, significant. Table 4-7 shows the MRAD concentration-response parameter for each ppb of daily high-hour ozone calculated from the Ostro and Rothschild results using alternative averaging assumptions. The results vary by more than a factor of 10.

Table 4-7
Ostro and Rothschild Results Using Alternative Averaging Approaches

Approach	Daily Individual Risk of MRAD/ppb High-Hour Ozone
Simple Arithmetic Mean	0.7×10^{-5}
Simple Mean of Positive Coefficients	11.1×10^{-5}
Mean Weighted by Inverse of the Variance	4.7×10^{-5}
Random Effects Model Mean	0.6×10^{-5}

For quantitative use in this analysis we choose the estimate close to the middle of the four alternatives presented in Table 4-7. The selected central parameter is thus the average of the findings for each of the 6 years weighted by the inverse of its statistical variance. For low and high parameters we use minus and plus one standard error of the weighted mean. The central parameter calculated in this way is about one-half the estimates for 1979 obtained by Ostro and Rothschild and by Portney and Mullahy (Table 4-7). The following daily MRAD concentration-response parameters for each ppb of daily high-hour ozone are used in this analysis.

$$\text{Low daily MRADs} = 1.93 \times 10^{-5} \times O_3 \quad (4\text{-}32a)$$
$$\text{Central daily MRADs} = 4.67 \times 10^{-5} \times O_3 \quad (4\text{-}32b)$$
$$\text{High daily MRADs} = 7.40 \times 10^{-5} \times O_3 . \quad (4\text{-}32c)$$

The high parameter is thus equivalent to the lower of the findings for the 3 years for which Ostro and Rothschild found a statistically significant effect between ozone and MRAD frequency. The central and low parameters give more weight to the negative findings. We apply a 50% probability weight to the central estimate, and equal weights of 25% each to the low and high estimates.

Because daily symptom concentration-response functions for asthmatics are available based on studies focussed specifically on those with diagnosed asthma (see Section 4.4.4), we exclude the asthmatic population from the calculations of minor restricted activity days. Although asthmatics were not specifically excluded from the MRAD studies, nonasthmatics are more representative of the response of the general population because only a small fraction of the general public has diagnosed asthma. We therefore apply the MRAD concentration-response function to the nonasthmatic portion (94%) of the population. We apply the MRAD function to all ages. Although the MRAD coefficients reported in these two studies were based on adults, earlier work by Portney and Mullahy (1993) included children and found some statistically significant relationships between restricted activity days and ozone. We therefore include children in the MRAD calculation.

4.4.7 Acute Respiratory Symptoms: PM_{10} and Ozone

Krupnick et al. (1990) estimated a relationship between the daily occurrence of acute upper and lower respiratory symptoms among a panel of adults and children in Southern California and daily levels of air pollution. Krupnick et al. (1990) used pooled cross-sectional and time-series data based on a health survey conducted in 1978-1979 of families living in Glendora, Covina, and Azusa, California. Health diaries were maintained for 182 days, and 290 families participated. ARS is a binary variable reflecting the presence or absence of any of 19 respiratory-related symptoms, including chest discomfort, coughing, wheezing, sore throat, head cold, chest cold, sinus trouble, hay fever, headache, and doctor diagnosed flu. This health endpoint includes some days with symptoms bothersome enough to result in a restricted activity day, but also includes days when noticeable symptoms are present but no change in activities occurs.

Krupnick et al. (1990) applied a Markov process model to determine the relationship between air pollution and respiratory symptoms. The model incorporated the probability of illness on the prior day and controlled for autocorrelation. Air pollution variables for COH (a measure of visibility impairing particles in the air), ozone, and sulphur dioxide were included in the model as well as independent variables for socioeconomic measures, presence of a chronic condition, and smoking habits. The initial results with multiple pollutants in separate equations for adults and children showed statistically significant coefficients of roughly similar magnitudes for COH in the adult and children equations.

The COH coefficient from Krupnick et al. s (1990) Equation 3 specification is 0.0088, with a standard error of 0.0046. We apply this coefficient to both adults and children because this specification was not estimated for children, but other specifications did show an association between COH and symptoms in children.

Data provided to us by the authors show a ratio of COH (units/100 ft) to TSP for the study period of 0.116. Using the PM_{10}/TSP ratio of 0.55, this gives a COH-to-PM_{10} ratio of 0.211. The marginal effect of COH was calculated by incorporating the stationary probabilities as described

in the paper.[9] Because the study did find symptom effects for children in some specifications, we apply these calculations to the entire population. The central concentration-response parameter is based on the regression coefficient from the Krupnick et al. Equation 3. The high and low parameters are based on plus and minus one standard error of the regression coefficient respectively.

$$\text{Low daily ARS} = 2.2 \times 10^{-4} \times (\ \Delta PM_j) \times POP_j \quad (4\text{-}33a)$$
$$\text{Central daily ARS} = 4.6 \times 10^{-4} \times (\ \Delta PM_j) \times POP_j \quad (4\text{-}33b)$$
$$\text{High daily ARS} = 7.0 \times 10^{-4} \times (\ \Delta PM_j) \times POP_j\ , \quad (4\text{-}33c)$$

Because the definition of ARSs includes days that fall into the category of restricted activity days, we subtract RADs to obtain net ARS parameter estimates. The RADs parameters apply only to the population age 20 and older, so we multiply the RADs parameters by 0.728 (the share of the Canadian population age 20 and older from the 1996 census) and then subtract these from the ARS parameters. The resulting net ARS concentration-response parameters are as follows:

$$\text{Low net daily ARS} = 1.62 \times 10^{-4} \times (\ \Delta PM_j) \times POP_j \quad (4\text{-}33d)$$
$$\text{Central net daily ARS} = 3.44 \times 10^{-4} \times (\ \Delta PM_j) \times POP_j \quad (4\text{-}33e)$$
$$\text{High net daily ARS} = 5.18 \times 10^{-4} \times (\ \Delta PM_j) \times POP_j\ , \quad (4\text{-}33f)$$

where:

POP_j = population in location j
ΔPM_j = change in daily average PM_{10} in area j.

The Krupnick et al. (1990) Equation 3 results for ozone are also selected for quantitative use in this analysis. These results are for adults and are from a regression that includes coefficient of haze and sulphur dioxide, thus minimizing the chance of an overstated ozone coefficient due to correlation with other pollutants. As with the PM_{10} concentration-response parameters, because the study did find symptom effects for children in some specifications, we apply these calculations to the entire population. The incremental effect per unit of ozone was calculated by incorporating the stationary probabilities as described above. The low and high parameters are based on minus and plus one standard error of the regression coefficient. The individual daily ozone-related ARS concentration-response parameters are as follows:

$$\text{Low daily ARS} = 0.70 \times 10^{-4} \times \Delta O_3 \times POP_j \quad (4\text{-}34a)$$
$$\text{Central daily ARS} = 1.37 \times 10^{-4} \times \Delta O_3 \times POP_j \quad (4\text{-}34b)$$
$$\text{High daily ARS} = 2.04 \times 10^{-4} \times \Delta O_3 \times POP_j\ . \quad (4\text{-}34c)$$

9. The authors provided mean estimates of the transitional probabilities to us for the all-adults sample, which were not reported in the paper. For all adults, p_1 averages 0.7775 and p_0 averages 0.0468; p_1 is the probability of reporting symptoms given that symptoms were present on the previous day, and p_0 is the probability of reporting symptoms given that no symptoms were present on the previous day.

Because for ozone the definition of ARSs includes days that also fall into the category of minor restricted activity days, we subtract the concentration-response parameters for MRADs to obtain the following net ozone ARS concentration-response parameters:

$$\text{Low net daily ARS} = 5.07 \times 10^{-5} \times O_3 \times POP_j \quad \text{(4-34d)}$$
$$\text{Central net daily ARS} = 9.03 \times 10^{-5} \times O_3 \times POP_j \quad \text{(4-34e)}$$
$$\text{High net daily ARS} = 13.0 \times 10^{-5} \times O_3 \times POP_j \; . \quad \text{(4-34f)}$$

Additional evidence supporting the relationship between ozone and acute respiratory symptoms is provided by Schwartz et al. (1988). They found a significant correlation between the frequency of several different acute respiratory symptoms and daily ozone levels in Los Angeles. The results are not used quantitatively here because the study sample comprised young female nursing students and was therefore less representative of the general population than the Krupnick et al. sample.

Probability weights for both pollutants are 50% for the central parameter and 25% each for the low and high parameters. Because daily symptom concentration-response functions for asthmatics are available based on studies focussed specifically on those with diagnosed asthma (see Section 4.4.4), we exclude the asthmatic population from the calculations of acute respiratory symptom days for both PM_{10} and ozone. Although asthmatics were not specifically excluded from the ARS study, nonasthmatics are more representative of the response of the general population because only a small fraction of the general public has diagnosed asthma. We therefore apply the ARS concentration-response function to the nonasthmatic portion (94%) of the population.

4.4.8 Children with Acute Bronchitis: PM_{10}

Dockery et al. (1989) studied the relationship between lower respiratory illness in children and particulate matter concentrations in six cities in the United States. The study related annual concentrations of TSP, PM_{15}, $PM_{2.5}$, sulphate, and sulfur dioxide to the presence of chronic cough, acute bronchitis, chest illness, persistent wheeze, and asthma. These illnesses were noted during a health examination and intake questionnaire taken for the sampled children in each city. A condition of asthma or acute bronchitis was based on a physician s diagnosis in the previous year. Chronic cough was defined as a cough being present for at least three months in the past year. The results of a logistic regression analysis show a statistically significant relationship between annual average particulate matter levels and the probability of the child having bronchitis or chronic cough in the past year.

A recent study (Dockery et al., 1996) replicates the above study using 18 U.S. and 6 Canadian cities. Children ages 8 to 12 were assessed via questionnaire between 1988 and 1991. Among the cities, the annual incidence rates for acute bronchitis in children (B) in the past year ranged from

3% to 10%, with an average of 6.5%. The logistic regression analysis controlled for sex, history of allergies, parental asthma, parental education, and current smoking in the home. Particulate matter measures used in the analysis included sulphates, $PM_{2.1}$, and PM_{10}.

The study included children in the range of 8 to 12 years old. However, we apply the concentration-response function to the entire under 20 population on the assumption that the response is similar across the broader age group. We prefer the potential error of this assumption to the alternative of assuming no effect for young children and older teens.

The change in the probability of the health outcome can be calculated from the partial derivative of the logistic function with respect to PM_{15}. The partial derivative is:

$$B = b \times Pr \times (1 - Pr) \times \quad PM_{15} , \qquad (4\text{-}35)$$

where:

B = change in probability of the health outcome
b = estimated regression coefficient
Pr = the baseline probability of the health outcome.

Statistically, the strongest results were for incidence of prevalence of acute bronchitis in children (within the past year) and the particulate matter measures (the impact of gaseous air pollutants such as ozone was also evaluated in the study). Specifically, a 6.8 g/m^3 increase in annual sulphate was associated with an relative risk of 1.65 (95% CI = 1.12, 2.42), and a 17.3 g/m^3 increase in PM_{10} was associated with an relative risk of 1.50 (95% CI = 0.93, 2.43). The associated PM_{10} regression parameter (b) is calculated as follows: ln(1.50)/17.3 = 0.0234 with a standard error of 0.014. Even though the PM_{10} results are less statistically robust, we selected the PM_{10} results as the basis for the B risk factor estimates because they are based on the PM measure of interest in this analysis. These estimates are slightly lower than, but very similar in magnitude to, what we would obtain if we adjusted the sulphate results for the average sulphate share of PM_{10} based on the reported study data. Based on the PM_{10} results, a 1 g/m^3 change in PM_{10} generates a central B concentration-response parameter of $0.0234 \times [0.065 \times (1 - 0.065)] = 1.42 \times 10^{-3}$. For the low and high parameters we use the central estimate plus or minus one standard error, respectively. Therefore the annual B concentration-response parameters are as follows:

$$\text{Low annual cases of Child B} = 0.57 \times 10^{-3} \times (\quad PM_j) \times POP_{<20j} \qquad (4\text{-}36a)$$
$$\text{Central annual cases of Child B} = 1.42 \times 10^{-3} \times (\quad PM_j) \times POP_{<20j} \qquad (4\text{-}36b)$$
$$\text{High annual cases of Child B} = 2.27 \times 10^{-3} \times (\quad PM_j) \times POP_{<20j} , \qquad (4\text{-}36c)$$

where:

B = annual cases of bronchitis in children

$POP_{<20j}$ = population under age 20 years in area j
PM_j = change in annual average PM_{10} in area j.

The selected probability weights are 50% for the central estimate, and 25% each for the low and high estimates.

4.5 AIR TOXICS AND CANCER RISK

AQVM 3.0 includes toxic air pollutants that are emitted by point sources as well as by conventional gasoline and diesel vehicles (as evaporative emissions, tailpipe emissions, and during vehicle refuelling). The air toxics considered in AQVM 3.0 are: acetaldehyde, benzene, 1,3 butadiene, and formaldehyde. These air toxics are known (benzene) or probable (acetaldehyde, 1,3 butadiene, and formaldehyde) human carcinogens for which quantitative estimates of the risk of developing cancer as a result of exposure have been developed (U.S. EPA, 1994; 1998). While exposures to these pollutants have been associated with systemic effects other than the development of cancers, quantitative estimates of these are unavailable so only the cancer risks are incorporated in AQVM 3.0.[10]

The primary route of exposure to these air toxics is inhalation with subsequent absorption into the body through the lungs. While these air toxics may be deposited on soil, plants, and surface water through wet and dry deposition, they do not accumulate because of rapid volatilization. Therefore, significant exposure through other pathways (e.g., ingestion, absorption) is not expected and a methodology is developed that estimates the number of avoided new cancer cases based on changes in inhaled ambient concentrations.

The inhalation cancer risks associated with these air toxics are developed based on reviews and assessments of cancer studies in animals and/or humans for the chemical. From these reviews, inhalation unit risk estimates are developed which represent the increased probability of a person developing cancer from breathing air containing a specified concentration of the chemical over a lifetime (70 years) (U.S. EPA, 1994). According to the U.S. EPA, as a result of the methods used to derive the inhalation unit risk estimates, the values should be interpreted as upper bound estimates where the true risk associated with the chemical is unlikely to exceed this value, and may be much lower (U.S. EPA, 1994). As a result of these conservative risk estimates, it is possible that the number of cancers estimated in AQVM 3.0 for a change in the ambient concentration of any of these air toxics could significantly overstate the actual number of new cancer cases that would be avoided (assuming an improvement in concentration levels).

10. Other air toxics from point source emitters such as power plants are also of concern. Similar damage assessment methods for nine metals and organics not covered in AQVM 3.0 (arsenic, beryllium, cadmium, chromium, nickel, dioxin, furans, lead, mercury, PCBs, POMs) were developed in Rowe et al. (1995) and will be considered for inclusion in future revisions to the AQVM.

However, without improved epidemiologic information there is little that can be done to incorporate this uncertainty outside of specifying alternative risk parameters in sensitivity tests.

Inhalation unit risk estimates for acetaldehyde, 1,3 butadiene, and formaldehyde for AQVM 3.0 are taken from the U.S. EPA s Integrated Risk Information System (IRIS) database (U.S. EPA, 1994) and are presented in Table 4-8 along with information on the chemical s carcinogen classification and the type of cancer usually associated with exposure to the chemical.

Table 4-8
Carcinogenic Risk Factors for Air Toxics

Toxic	U.S. EPA Carcinogen Classification	Type of Cancer	Inhalation Unit Risk Estimate (g/m^3)
Acetaldehyde	B2	Nasal	2.2×10^{-6}
1,3 Butadiene	B2	Incidence in multiple sites	2.8×10^{-4}
Formaldehyde	B1	Nasal	1.3×10^{-5}

Notes: Carcinogen Classification
A = Human carcinogen, sufficient human evidence.
B1 = Probable human carcinogen, limited human evidence.
B2 = Probable human carcinogen, sufficient animal evidence, inadequate or no human evidence in humans.
C = Possible human carcinogen.
D = Not classifiable as to human carcinogenicity.
E = Evidence of noncarcinogenicity for humans.

Source: (U.S. EPA, 1994).

The number of avoided new cancer cases associated with a decrease in the annual ambient concentration of acetaldehyde, 1,3 butadiene, or formaldehyde is estimated as follows:

$$\text{Annual new cancer cases} = \text{CONC}_j \times \text{Inhalation Unit Risk}_i \times 0.014 \times \text{POP}_j , \quad (4\text{-}37)$$

where:

CONC_j = change in average annual concentration for toxic in location j
Inhalation Unit Risk_i = cancer risk factor for air toxic i (see Table 4-8)
POP_j = population for location j.

Because the inhalation unit risks reflect the lifetime increase in cancer risk they are divided by 70 (equivalent to multiplying by 0.014), the average lifespan used to develop the risk factors, to estimate annual cancer risk. In addition, because these chemicals have only a single point estimate for their risk, the risks are all treated as central parameter estimates and are assigned probability weights of 100%.

Benzene is a known carcinogen (class A from the list presented in Table 4-8, U.S. EPA 1998). Benzene exposures are most strongly associated with increasing the risk of developing acute myeloid leukemia (AML) in those exposed (U.S. EPA, 1998). The U.S. EPA recently reassessed its inhalation unit risk estimate for benzene based on studies that have estimated the relationship between occupational benzene exposures for a clearly identified set of workers, the Pliofilm cohort, and the development of leukemias (U.S. EPA, 1998). The Pliofilm cohort consists of rubber workers exposed to benzene in the workplace from 1940 to 1965 and for whom follow-up medical data through 1987 were collected. This cohort was used in the development of the benzene cancer risk estimates in the EPA s reassessment, as well as in its original work, because it is one of the only groups that received the necessary exposure and medical follow-up needed to support the development of inhalation unit risk estimates. In addition, the Pliofilm cohort is appropriate for this use because of the limited potential this group had to have experienced confounding exposures to other potentially carcinogenic chemicals in the workplace.

However, despite these attractive features, a range of benzene inhalation unit risk estimates has been obtained from different analyses of the data from the Pliofilm cohort. This range of estimates reflects differing assumptions about the level of exposure experienced by the workers, because benzene monitoring during the study years was infrequent, limited, and imprecise, leaving the available results open to considerable interpretation.

In its review of studies evaluating the Pliofilm cohort, the U.S. EPA ultimately decided to replace its previous point estimate of the inhalation unit risk of 8.0×10^{-6} per $\mu g/m^3$ of benzene with a range of 2.2×10^{-6} to 7.8×10^{-6} per $\mu g/m^3$ of benzene (U.S. EPA, 1999). The reassessment further concluded that any risk estimate within this range would have equal scientific validity (U.S. EPA, 1998).

AQVM 3.0 incorporates these results by specifying a three-point distribution for the inhalation unit risk of benzene. The low and high parameters correspond to the respective endpoints of the range provided by the U.S. EPA, and the central parameter estimate represents the midpoint of the low and high values. Because the inhalation unit risks express the lifetime (70 years) risk of developing cancer as a result of continuous exposure to 1 $\mu g/m^3$ of benzene, AQVM 3.0 makes an adjustment to annualize the risks in its benefit calculations by multiplying by 0.014 (this is consistent with the treatment of the other air toxic cancer risks). The resulting annual concentration-response functions for benzene-related leukemia cases in AQVM 3.0 are as follows:

$$\text{Low annual cases of Benz Canc} = 2.2 \times 10^{-6} \times 0.014 \times POP_j \times Benz_j \quad (4\text{-}38a)$$

$$\text{Central annual cases of Benz Canc} = 5.0 \times 10^{-6} \times 0.014 \times POP_j \times Benz_j \quad (4\text{-}38b)$$

$$\text{High annual cases of Benz Canc} = 7.8 \times 10^{-6} \times 0.014 \times POP_j \times Benz_j, \quad (4\text{-}38c)$$

where:

Benz Canc = benzene-related leukemia

POP_j = population in area j
$Benz_j$ = change in annual average benzene in area j.

Given the equal validity of these risk estimates, equal probability weights are assigned to the low (33%), central (34%), and high (33%) parameter estimates.

4.6 CARDIAC HOSPITAL ADMISSIONS AND CARBON MONOXIDE

Recent research suggests that ambient carbon monoxide (CO) contributes to risks of hospital admissions for cardiac illnesses in the elderly, including admissions for congestive heart failure and possibly for coronary artery disease. These findings are plausibly consistent with earlier findings in clinical studies that CO exposures shorten the time to onset of angina pain for heart disease patients who are exercising moderately (e.g., Allred et al., 1991). The clinical findings have, however, been difficult to extrapolate to changes in ambient outdoor CO concentrations and normal everyday exposures in the population.

Two recent epidemiology studies on cardiac hospital admissions provide quantitative evidence of a relationship between day-to-day fluctuations in ambient outdoor CO concentrations and cardiac hospital admissions for the elderly (Schwartz and Morris, 1995; Burnett et al., 1996). Both studies controlled for particulate matter concentrations, which are sometimes correlated with CO concentrations, and for daily weather and seasonal variations. Even after controlling for particulate matter concentrations, both studies found a statistically significant relationship between some categories of cardiac hospital admissions and CO concentrations. In both study areas, the CO concentrations were well below the current Canadian 1-hour objective of 30 ppm. In the 10 Canadian cities included in the Burnett et al. (1996) study (Montreal, Ottawa, Toronto, Hamilton, London, Windsor, Winnipeg, Edmonton, Calgary, and Vancouver), the mean daily high-hour CO concentration ranged from 1.6 ppm to 3.3 ppm, and the 95th percentile ranged from 3.5 ppm to 8.7 ppm. Comparable concentrations occurred in Detroit (Schwartz and Morris, 1995) with a mean of the daily high-hour CO concentrations for the study period of 2.4 ppm and a 90th percentile of 3.8 ppm.

Schwartz and Morris (1995) examined hospital admissions for coronary artery disease, congestive heart failure, and cardiac dysrhythmias for patients 65 years old and older in all hospitals in the Detroit metropolitan area from 1986 to 1989. Total population in the study area was approximately 4.4 million. After controlling for daily weather, season, and daily PM_{10} concentrations, a statistically significant effect of CO on admissions for congestive heart failure remained. The estimated relative risk for a 1.28 ppm increment in daily high-hour CO was 1.022 (95% CI = 1.010 to 1.034).

When Burnett et al. (1996) analysed hospital admissions in the 10 Canadian cities (total study area population approximately 11 million) for 1981 through 1991, they found a statistically significant relationship between daily admissions for elderly patients with congestive heart

failure or coronary artery disease and daily CO concentrations, after controlling for daily fluctuations in other air pollutants and weather as well as seasonal trends. Data on PM_{10} concentrations were not available for the study period. COH was used as a proxy for daily particulate matter concentrations. The estimated relative risk for both categories of admissions combined was 1.039 (p = 0.0104) for a 1.9 increment in daily high-hour CO. Average daily hospital admission rates appear to be considerably lower in the Canadian cities as compared to the Detroit data for these categories of admissions.

Limitations in these studies need to be acknowledged. Most important is the possibility of collinearity with fine particulate concentrations that might be confounding the observed relationship with CO. These studies have used the best available data and analysis approaches for addressing this issue, but limitations remain. Still, the observed relationship between cardiac admissions and CO seems plausible and robust. We use the following approach for quantification of cardiac hospital admissions associated with changes in ambient CO concentrations (CHA_{CO}). For the low concentration-response parameter, we select zero effect because of the limited amount of published evidence on CO and this health endpoint.

For a central concentration-response parameter we select the results from Detroit for congestive heart failure, which are quite consistent with the Canadian results for congestive heart failure admissions. For the high parameter we use the Canadian results for congestive heart failure and coronary artery disease combined. For all the calculations we applied the estimated relative risk per ppm to the average daily hospital admission rate in Canada. The 1992 average daily hospital admission rate in Canada for congestive heart failure was 4.4 per 100,000 elderly population. The 1992 average daily hospital admission rate for ischemic heart disease was 4.6 per 100,000 elderly population. For example, the central daily CHA concentration-response parameter per ppm CO is calculated as

$$(0.022 \div 1.28) \times (4.4 \times 10^{-5}) = 7.56 \times 10^{-7} . \quad (4\text{-}39)$$

We multiply by 365 to calculate the annual concentration-response parameter per unit change in the annual average of the CO measure. The selected low, central, and high concentration-response parameters are therefore:

$$\text{Low annual cases of } CHA_{CO} = 0 \quad (4\text{-}40a)$$

$$\text{Central annual cases of } CHA_{CO} = 2.76 \times 10^{-4} \times CO \times POP_{65+\,j} \quad (4\text{-}40b)$$

$$\text{High annual cases of } CHA_{CO} = 6.74 \times 10^{-4} \times CO \times POP_{65+\,j} , \quad (4\text{-}40c)$$

where:

CHA_{CO} = annual cases of cardiac hospital admissions due to CO
CO = change in annual average of daily high-hour CO in ppm
$POP_{65+\,j}$ = elderly population (65 years and over) in area j.

Given the formative state of this research, we assign equal (33%) probability weights for the low, central, and high parameter estimates.

CHAPTER 5
ECONOMIC VALUATION

This chapter presents the methods and monetary estimates used to value human health impacts and nonhealth environmental impacts, including visibility aesthetics, materials damage and soiling, agricultural crop loss, recreational fishing damages, and environmental impacts, that may result from climate change. The human health valuation in AQVM 3.0 is based on the methods developed for the Canadian Council of Ministers of the Environment Task Force on Cleaner Vehicles and Fuels in the report entitled *Environmental and Health Benefits of Cleaner Vehicles and Fuels, Supplemental Report 3: Selected Economic Evidence of Monetary Valuation of Human Health Effects* (Chestnut, 1995b). These methods and values are in line with a large body of literature on the valuation of health effects. Similar interpretations and applications of the available literature include National Economic Research Associates (1993), Lee et al. (1995), Triangle Economic Research (1995), Rowe et al. (1995), European Commission (1995), and U.S. EPA (1997a, b). The AQVM 3.0 methodology for monetary valuation of changes in visibility, materials, and crop loss damages is based on Rowe et al. (1995), updated where possible with Canadian data.[1]

The approach to the economics literature review and study selection is specific to each category of health or environmental effects. For many categories there are only a few valuation estimates available in the literature. In these cases the study selection process is simply a matter of evaluating all of the available studies, determining whether the quality of the research is acceptable for these purposes, and selecting the specific valuation estimates or functions for use in this assessment. The approach of selecting a range of values reflecting the range in the literature is used in each case, but the specific bases for the selections vary and are explained in each section of this chapter. For a few valuation categories, such as mortality risk and visibility aesthetics, there are many studies that give relevant empirical estimates. Fortunately there have also been several extensive literature reviews and compilations of these empirical studies. We draw upon the insight and recommendations of these previous reviews, as well as our own evaluation of the literature in these areas.

Many of the monetary values used in AQVM 3.0 are based on U.S. studies that report values in U.S. dollars. When a comparable Canadian value was available, for example, the daily wage rate and medical costs, we used the Canadian value. When a comparable value was not available, we used the purchasing power parity (PPP) index, which measures the relative value of currency

1. The economic valuation of the endpoints in this chapter is the same for the TSP/PM_{10}, $PM_{2.5}$, and SO_4 AQVM 3.0 model options.

based on the purchasing power of the currencies to convert U.S. values to their Canadian equivalent. The PPP index is preferred to the currency exchange rates because it translates U.S. values to Canadian values according to the Canadian price system. To convert U.S. values to Canadian values, we used the following process:

1. For studies with a base year of 1983 or later, U.S. values were converted to their Canadian equivalent using the PPP index value for the base year. For studies with a base year before 1983, the first year for which we had PPP index values, the U.S. values were first inflated to their 1983 U.S. dollar equivalent using the appropriate U.S. inflation index (e.g., consumer price index or medical cost index) at which point they were converted to their Canadian dollar equivalent using the 1983 PPP index value of 1.24.[2]

2. The converted values were inflated to 1996 Canadian dollars with the appropriate Canadian inflation index (e.g., CPI or medical cost index).[3]

As an example, consider a study that estimated a willingness-to-pay value for a health endpoint of $100 in 1990 U.S. dollars. First, the estimate is converted to its equivalent of $122 1990 Canadian dollars by multiplying by the purchasing power parity index value of 1.22 between U.S. and Canadian gross domestic product (GDP) for 1990. Second, the 1990 Canadian value is inflated to $139 1996 Canadian dollars using the Canadian CPI, which had a value of 119.5 in 1993 and 135.7 in 1996.

Alternatively, if the study had estimated a willingness-to-pay value for the health endpoint of $100 in 1975 U.S. dollars the value would first be inflated to its 1983 U.S. equivalent value of $185, based on a 1975 U.S. CPI value of 53.8 and a 1983 CPI value of 99.6. This value would be converted to an equivalent 1983 Canadian value of $229 by multiplying by the 1983 PPP of 1.24 and would subsequently be converted to a 1996 Canadian value of $351 based on a Canadian CPI values of 88.5 in 1983 and 135.7 in 1996.

Monetary value estimates based on medical care costs were inflated using the medical care price index. Other monetary values (usually willingness-to-pay values) were inflated using the consumer price index.

2. All PPP values used in this chapter are from Statistics Canada (1993b; 1996b). This conversion method was recommended in personal communication from Paul De Civita of Environment Canada.

3. All Canadian consumer price index and medical cost index information in this chapter comes from Statistics Canada (1996a). U.S. price indices are from U.S. Bureau of Census (1994).

5.1 CONCEPTS AND ISSUES FOR VALUING HEALTH EFFECTS

5.1.1 What Is Meant by Value

To economists, the term *value* has a specific meaning that we refer to as *economic value*. The most important, but often overlooked, features of economic value are that it is a theoretical construct and that monetary measures of it are inferred by analysts from the actions that people make or state in accordance with their preferences. Economic value is reflected by *choices* whereby something is given up and something gained.

The study of choices allows economic values to be defined and quantified. Choice implies that a person is confronted with a selection of alternatives and that the consideration of the alternatives defines a tradeoff. Contemporary economic theory of individual behaviour, based on the assumption of preference satisfaction, suggests that when a person is confronted by choices, the alternative that is chosen must be at least as desirable, from the perspective of that person, as the alternatives that were not chosen. The theory implies that the alternative chosen is at least as good or as valuable as the alternatives that were not chosen; the value of the alternative chosen is thus defined in terms of the alternatives foregone. For example, if a person chooses to relinquish three apples to gain a peach, an analyst can state that under the circumstances of the choice (perhaps known in their entirety only to the person), the economic value of the peach to the person is at least equal to the economic value of three apples. If the choice were to give up $1 for the peach and the person chose the peach, the analyst would conclude that the value of the peach to that person was at least $1.

Willingness to Pay

Economists use the term willingness to pay (WTP) to refer to a monetary measure of individual welfare. This term has an analogue in the term willingness to accept (WTA). WTP refers to the maximum amount an individual would pay to obtain a change that reflects an improvement in his or her state or circumstance. Maximum WTP is defined as the payment amount at which a person is indifferent between having the change at that price or not having the change.[4] Assume, for example, a person s maximum WTP for a peach were $1. If, in fact, the peach sells for only $0.75, the individual receives a benefit of $0.25, but the WTP is still $1. WTA refers to the minimum amount that an individual would need to receive to be indifferent to a change in circumstance. For example, if I have a peach that you want and I would be willing to part with the peach for no less than $1, then my WTA is $1. These terms are sometimes inappropriately equated with the contingent valuation estimation method that is sometimes used to estimate WTP values. Contingent valuation is a stated preference method of eliciting measures of individual WTP or WTA using questionnaires or interviews. WTP and WTA estimates can be estimated

4. WTP is also defined for a change that reflects a decrement in welfare as the amount at which a person is indifferent between preventing the change at that price or having the change occur.

using any number of preference elicitation methods, including those depending on revealed preferences (such as those observed in labour, commodity, or housing market decisions) and on stated preferences (such as those elicited in contingent valuation or conjoint analysis studies).

Can the Economic Value of Some Things Not Be Measured?

It is argued by some that there are things that humans cannot put a price tag on. Aspects of the environment often fall into this category. That might well be true, but it does not imply that individuals cannot determine how important aspects of the environment are to them. As noted above, economic values are inferred from the choices made by individuals. It would be wrong to think of economic values as dollar-denominated numbers in one s brain to be down loaded when a person is asked the worth of a beautiful ocean sunset; rather, such a value might be inferred from the things that one is willing to give up to see the sunset (e.g., the cost of travel time and money to visit the ocean). To economists, the importance of things (tangible or intangible) is revealed by what a person will give up to obtain them. The lower bound on the value of the item obtained is equated to what was given up. If the thing given up was money, the value can be expressed in monetary units; otherwise, it is expressed in the natural units of the thing given up.

5.1.2 Health Valuation Concepts

There are many potential economic and social consequences associated with adverse health effects that result from air pollution, including:

1. ***Medical costs.*** These include personal out-of-pocket expenses of the affected individual (or family), plus costs paid by public health care or private insurance, for example.

2. ***Work loss.*** This is the monetary value of lost productivity, usually measured as lost income whether the individual is compensated for the time off work due to illness or not. For example, some individuals may perceive no income loss because they received sick pay, but sick pay is a cost of business and reflects lost productivity.

3. ***Increased costs for chores and care giving.*** These include special care giving and services that are not reflected in medical costs. These costs may occur because some health effects reduce the affected individual s ability to undertake some or all normal chores, and because he or she may require care giving.

4. ***Other social and economic costs.*** These include restrictions on or reduced enjoyment of leisure activities, discomfort or inconvenience (pain and suffering), anxiety about the future, and concern and inconvenience to family members and others.

The appropriate economic measure of the benefits of preventing adverse health effects is the total social value of reducing all of the above consequences. This includes the cost and no-cost consequences to the affected individual plus the costs and no-cost consequences to the rest of society. For example, medical costs or work loss costs may be shared by the affected individual and by others in society through personal, employer, and government insurance and assistance programs.

Two economic measures of the value of health effects are typically used in the literature, cost-of-illness (COI) and willingness to pay (WTP). Both types of measures are used in this study. COI measures include only medical costs and lost income as a proxy for work loss (consequences 1 and 2 above), and thus do not reflect the total welfare impact of an adverse health effect. Therefore, using COI measures in a quantitative assessment results in a clear downward bias in the valuation of adverse health effects. COI measures, however, have the practical advantages of being easily understood and often readily available because they are based on available market and expenditure data.

Total social WTP is the summation of (1) the affected individual s WTP to avoid the adverse consequences, (2) the WTP of friends and family who expend time, effort, and monies caring for the affected individual, and (3) the medical and work loss costs paid by society rather than by the affected individual, family, and friends. Because there are few literature values for (2), these values are not fully reflected in AQVM 3.0 and result in a potential understatement of air pollution control benefits.

WTP is typically measured by analysing prices that are paid for goods and services. The maximum price that an individual is willing to pay for a good or service is a measure of how much they value that good or service. Prices cannot be directly observed for preventing health risks because prevention of health risks is seldom directly purchased in the market. However, there are instances when the monetary tradeoffs that people are willing to make between income and health risks can be observed or measured. There are two general economic approaches for measuring WTP for nonmarket goods such as health risk prevention. The first is to analyse actual situations in which WTP for health risks may be indirectly revealed; the second is to have subjects respond to a hypothetical situation designed to have them reveal their WTP.

An example of the first approach is a wage-risk study, which estimates wage premiums for increased risks of work-related deaths. This is done by analysing factors that determine differences in actual wages between jobs, including on-the-job risks of death. The amount of additional wages that people are paid per unit of additional risk of fatal injury is a measure of the monetary value of that risk to the individual who voluntarily accepts that risk in exchange for a given wage increment. The primary advantage of this type of study is that it is based on actual behaviour. The primary limitations are that it is difficult to find situations in which there is a clear tradeoff between money and risk, and it is difficult to statistically isolate WTP for a risk increment from other factors involved in the specific behaviour.

An example of the second approach is a contingent valuation method (CVM) study, in which subjects are presented with a situation that involves a tradeoff between income or expenditures and a specific health risk or health effect. The subjects are then asked to estimate what they would be willing to pay to change that risk by a specific amount. It is important that the situation presented to study subjects be realistic and easy to understand. The primary concern with this type of study is whether subjects are able to give accurate responses to the CVM questions.

5.1.3 Health Valuation Issues

Although WTP for changes in health risks is the conceptually correct monetary value measure for this assessment, there are some limitations in available estimates. These limitations result from uncertainties in the available estimates, inexact matches between the health risks for which WTP estimates are available and the health risks of interest in this assessment, and the lack of available WTP estimates for some of the health risks of interest.

WTP estimates are available for risks of death, but there are some differences between the types of fatal risks for which WTP estimates are available and those of interest in this assessment. WTP estimates are available for some but not all types of morbidity of concern in this assessment.

Issues in Applying Available WTP Estimates for Fatal Risks

There are several uncertainties in applying the available WTP estimates for changes in risks of death in this analysis. The justification for using the available estimates is that they provide estimates of what people are willing to pay to reduce their risks of death by small amounts. The risks involved in this analysis are also small, but there are some differences with regard to who is at risk and what the risk is. Two particular aspects of potential significance are the cause of death and the health status of the people at risk. There is very little available empirical evidence about how these factors might affect the value of reducing risks of death. There is, therefore, some unresolved uncertainty in applying available WTP estimates in this analysis.

Cause of death. It is possible that people are more concerned about avoiding some kinds of death than others. For example, the Jones-Lee et al. (1985) results suggest that some people are more afraid of death from cancer than of death from automobile accidents. This may be related to the perceived pain, suffering, and expense associated with the illness that precedes death in the case of cancer. Some studies also suggest that people find involuntary risks, such as pollution exposure, less acceptable than voluntary risks, such as traffic accidents (Violette and Chestnut, 1983). Studies have not been able to separate these different aspects of the different risks of death in terms of the potential effect on WTP. The most reliable WTP studies to date have focused on accidental deaths, primarily on-the-job and vehicle accidents. The types of death of interest for this analysis are related to various illnesses, both chronic and acute. Based on the limited evidence available about how people respond to different types of risks, it is likely that if there is

any error in applying available WTP estimates in this analysis it will be to understate the WTP to avoid the types of risks of interest in this analysis.

For this analysis, available WTP estimates for changes in risks of death are applied to all estimated mortality risks regardless of the cause of death. Although arguments could be made for small adjustments in some cases, any such adjustment is overshadowed by the level of uncertainty in using these estimates, which cannot be reduced at this time. For example, WTP estimates based on accidental death probably do not reflect the medical costs typically associated with treatment of the chronic or acute illness that may precede premature death due to air pollutant exposure. However, COI estimates suggest that average lifetime medical costs per chronic respiratory disease patient are under $100,000 (Krupnick and Cropper, 1989). This omission is not very significant relative to a selected range of WTP estimates found throughout the literature of $2 to $10 million per fatality.

Health status. The available WTP estimates for changes in risks of death are based on results from study samples of individuals with average levels of health. Although it cannot be determined from available epidemiologic studies, it is possible that those individuals at greatest risk of premature death due to exposure to air pollutants are those who are already in poor health. Some instances may involve chronic illnesses, because of which the individual may already have a reduced life expectancy even in the absence of pollution exposure. For example, Schwartz and Dockery (1992a) found increased rates of death due to chronic respiratory disease, pneumonia, and cardiovascular disease associated with higher levels of particulate matter. Some of these individuals apparently suffered from a preexisting chronic disease. There is not sufficient evidence available to say how having a chronic illness might affect WTP for changes in risks of death, but it is possible that the reduced life expectancy and reduced enjoyment of life associated with many chronic illnesses may result in lower WTP to reduce risks of death. On the other hand, facing serious illness and reduced life expectancy may result in higher value placed on protecting the remaining time. The incidence of chronic illnesses is also likely to be correlated with age. Evidence of the effect of age on WTP for changes in risks of death is discussed in Section 5.2.3.

WTP to COI Ratios

WTP estimates are not available for some of the nonfatal health effects considered in this analysis. In these cases, COI estimates are used and are adjusted upward to compensate for the expected ratio of WTP to COI estimates for any given health effect. This adjustment is based on limited available evidence on WTP/COI ratios, but we believe the resulting adjusted health valuation estimates are probably less biased than would occur if only unadjusted COI estimates were used. Because this ratio is likely to be specific to each health effect, any such ratio based on existing studies must be seen as an approximation to improve valuation and reduce known bias that would occur if unadjusted COI estimates were used to value health effects.

Three studies provide evidence on WTP/COI ratios for the same study population addressing the same change in the same health effect. In each study, the participants were individuals diagnosed

with the health effect. These studies addressed changes in incidence of asthma symptoms (Rowe et al., 1984; Rowe and Chestnut, 1986), increased frequency of angina symptoms (Chestnut et al., 1988), and risks of cataracts (Rowe and Neithercut, 1987). In each study, participants rated the importance of each of the components of WTP (listed in Section 5.1.1), and provided WTP estimates for reducing or preventing these health effects. The participants rated some non-COI consequences as more important to avoid than the COI consequences. This again suggests that WTP significantly exceeds COI.

The dollar ratio results listed in Table 5-1 are based on estimated individual and social COI in dollars, and on individual WTP in dollars. Individual COI is less than social COI because society incurs some costs the individual does not (because of insurance coverage, sick pay, and other types of compensation). Because social COI exceeds individual COI, the WTP/COI ratio for individuals exceeds the ratio for individual WTP/society COI. Also available from the asthma and cataract studies are respondent ratings of their COI as a share of their perceived total damages. From these ratings, the individual and society WTP/COI ratios are computed and reported in Table 5-1.

Table 5-1
WTP/COI Ratios

Health Effect		WTP/COI Affected Individual	WTP/COI Society
Asthma Symptoms	Dollar ratio	1.6 to 2.3	1.3 to 1.7
Cataracts	Dollar ratio	4.25	2.4
	Respondent rated share of total damages ratio	5.3	2.1
Angina Symptoms	Respondent rated share of total damages ratio	2.5 to 4	NA

Sources: Asthma: Rowe et al. (1984), Rowe and Chestnut (1986).
Cataracts: Rowe and Neithercut (1987).
Angina: Chestnut et al. (1988).

Across the three studies, the individual WTP/society COI ratios range from 1.3 to 2.4. The COI in these studies range from a few dollars to $7,000 per episode of cataracts. Based on these results, a WTP/COI ratio of 2.0 is selected for morbidity effects, with the exception of nonfatal cancers. Because cancer treatment costs are relatively high, we use a WTP/COI ratio of 1.5 for

nonfatal cancers. Thus, when WTP estimates are not available, available COI estimates are multiplied by 2.0 or 1.5 to approximate WTP. We refer to this as adjusted COI.

Basing a WTP/COI adjustment on these study results is admittedly uncertain. The study samples are small and the range of health effects is limited. However, making no adjustment in COI estimates for valuation purposes would result in a clear downward bias. The selected adjustment factor of 2.0 is fairly conservative, based on available evidence, to minimize the chance of over adjusting. Additional evidence that these adjustment factors are conservative exists in the WTP estimates for risks of death. Average COI estimates for fatalities are typically in the middle hundreds of thousands. WTP estimates per fatality are in the millions, a difference of an order of magnitude.

Valuing Health Effects versus Health Risks

Sometimes in this discussion of monetary valuation for health effects we distinguish between health effects and health risks. A health effect refers to an illness or symptom, including death, that is experienced by someone. A health risk is the quantitative probability that any one individual might experience a given health effect. Changes in air quality cause changes in the number of health effects in the exposed population, but from the point of view of the individual what changes is the risk of experiencing a given health effect. This is because it is unknown exactly which individuals might be affected. WTP estimation techniques for more serious health effects such as mortality or chronic illness tend to focus on changes in the risks of such health effects that an individual might experience. For example, WTP studies for mortality do not estimate what individuals would be willing to pay to prevent a certain death, but rather estimate what they are willing to pay for small changes in risks of death.

5.2 MONETARY VALUATION ESTIMATES FOR MORTALITY RISKS

5.2.1 Introduction

Attempts at estimating the monetary value of reductions in mortality that may be achieved by public policy actions often evoke the comment that you can t put a value on a life. A lead article in the *Washington Post Magazine* titled What s a Life Worth? criticized the Reagan Administration for even considering the topic. This criticism of estimating the monetary value of reductions in mortality risks misses the boat because lives are not being valued; the values are for reductions *in the risks* of premature death. Behaviour that helps to reveal such values is not rare; it is observable every time someone takes a higher wage in exchange for a somewhat riskier job, or raises driving speed to save a few minutes. Hundreds of journal articles have been written on the subject, creating a rich body of values for death-risk reductions, and hundreds of cost-benefit analyses of regulations that affect death risks have relied on this literature.

Several economic studies have estimated average WTP in the United States, Canada, Australia, and the United Kingdom for small changes in risks of accidental death.[5] Reviews of this literature include Fisher et al., 1989; Miller, 1989; Cropper and Freeman, 1991; and Viscusi, 1992. These WTP estimates have been widely used in benefit analysis of public policy options that would result in changes in risks of death for the public (Viscusi, 1992). They are sometimes referred to as value of life estimates because they are expressed on a per life basis. But it is important to note that they are based on WTP of the individual for reducing his or her risk of death by a small amount, not on the total value of a human life under all circumstances.

The estimates provided by these studies are average dollar amounts that individuals are willing to pay for small reductions in risks of death. For example, one study might find an average WTP of $300 for an annual reduction in risk of death of 1 in 10,000. These estimates are extrapolated to a per life basis by summing individuals WTP over enough people that a value per life saved is obtained. In this example, this value would be $3 million per life, the result of $300 multiplied by 10,000 people. The term used for this estimate in much of the economics literature is value of a statistical life (VSL), to denote that it is a summation of WTP for small changes in risks of death.

Available estimates of WTP to prevent small changes in risks of death are based on situations where individuals are observed making tradeoffs between probabilities of death and some benefit, such as income. Most of these studies have estimated wage premiums associated with different levels of on-the-job risks. Additionally, some contingent valuation studies have been conducted in which subjects have been asked what they would be willing to pay to reduce, for example, their risks of fatal accidents at work or in traffic accidents. A few averting behaviour studies have also been conducted that estimate costs associated with observed behaviours that reduce risks, such as smoke detector usage in the home or seat belt usage in automobiles.

For the most part, available WTP estimates are for risks of accidental death in circumstances where individuals are voluntarily exposed to risks (e.g., choosing a job or driving in a car). The estimates are also drawn largely from studies of working-age adults. Some potentially important differences exist between the contexts of these available estimates and the environmental health risks being evaluated in AQVM 3.0. Environmental health risks are related to illness rather than accidents and may in some cases fall disproportionately on the elderly and those with already compromised health. The potential implications of these differences and effect of age on WTP are discussed in Section 5.2.3. Section 5.2.4 describes the specific WTP estimates selected as the default AQVM 3.0 values for changes in mortality risks. Section 5.2.5 provides a brief discussion of emerging alternatives to the VSL based approach for valuing mortality risks. Refinements to the current approach may soon be developed based on these emerging alternatives, but there is not sufficient empirical basis for changing the default approach at this time. AQVM 3.0 allows the user to explore the implications of alternative assumptions on this issue.

5. Many of these studies, namely the wage-risk studies, provide estimates of WTA rather than WTP. We use WTP here as a shorthand reference to both measures.

5.2.2 Summary of Available WTP Estimates for Mortality Risks

Four recent reviews of this literature evaluated and summarised available WTP estimates for small changes in risks of death for potential use in analyses of public policy decisions (Fisher et al., 1989; Miller, 1989; Cropper and Freeman, 1991; Viscusi, 1992). Each review concludes with a list or range of "best" estimates that the authors judged as most appropriate for use in evaluating public policy decisions that result in small changes in risks of death for the public. All of these reviews covered basically the same body of literature, but the most recent review (Viscusi, 1992) included a few additional studies that were not completed when the earlier reviews were done. These reviews are consistent in many of their conclusions regarding which of the available estimates are most appropriate for use in policy analysis, but there are also differences. The conclusions, and their basis, of each of these four reviews are taken into consideration in selecting a central, low, and high estimate of WTP for changes in risks of death for use in this analysis. The selected estimates for this analysis are discussed in Section 5.2.4. Low, mean, and high VSL estimates recommended by the authors of each of the four reviews as best for policy analysis are listed in Table 5-2.

Table 5-2
Recommended VSL Estimates Selected by Various Reviewers

Review	Selected VSL Rounded to Millions (1996 C$)		
	Low	Mean	High
Fisher et al. (1989)	$3	$8	$14
Miller (1989)	$2	$3	$5
Cropper and Freeman (1991)	$3	$5	$9
Viscusi (1992)[a]	$4	N/A	$10

a. An overall mean VSL was not recommended by Viscusi (1992), just a range.

Fisher et al. (1989) list 21 studies in their Table 1 that each give a VSL estimate. The authors reject three studies listed as "early low-range wage-risk estimates," primarily because of problems in the risk data used. The authors also reject the "consumer market studies," which fall into the category of averting behaviour studies, because they argue that each of the estimates is clearly downward biased because of study design problems or data limitations. They also reject one of the "new wage-risk studies" that examined wages for police officers in the United States, because of the limited scope of the study sample and potential problems with the on-the-job mortality rate data used. This leaves 13 VSL estimates judged by these authors as most

appropriate for use in policy analysis. These estimates range from $3 million to $14 million, and have an arithmetic mean of about $8 million (1996 Canadian dollars). All but two of the 13 studies are wage-risk studies. The remaining two studies are contingent valuation studies, which both obtained results of about $5 million (1996 Canadian dollars). These results fall in the lower half of the overall range. Fisher et al. caution that all the estimates above $9 million are based on wage-risk studies using U.S. Bureau of Labor Statistics data for on-the-job risks. These data are limited in that they give risk information by industry, but not by occupation. There is no specific reason why these data would cause any upward bias in VSL results, but results that are not verified by similar conclusions using different data sources are somewhat less robust. The authors therefore conclude that the $3 million to $9 million (1996 Canadian dollars) range is the strongest because it has been verified by different studies using varying data sources, but they do not rule out the possibility that the higher estimates might be correct.

Cropper and Freeman (1991) present an adapted version of Table 1 from Fisher et al. They deleted four of the 21 studies. The authors do not explain these exclusions, but presumably they found them to be less appropriate for policy analysis than the remaining 17. Two of the deleted studies were in categories that were rejected by both sets of reviewers, so their exclusion causes no change in the conclusions. The primary difference in the conclusions of these two reviews is that Cropper and Freeman make a stronger statement that using the U.S. Bureau of Labor Statistics on-the-job risk data apparently causes upward bias in the VSL estimates, based on comparisons of results using different types of data. Excluding the estimates based on U.S. Bureau of Labor Statistics data leaves six VSL estimates judged as "best" for use in policy analysis. These are from four wage-risk studies and two contingent valuation studies. The wage-risk estimates selected by Cropper and Freeman range from $3 million to $9 million (1996 Canadian dollars), and the contingent valuation estimates selected range from $4 million to $5 million (1996 Canadian dollars). The arithmetic mean of all six selected VSL estimates is about $5 million (1996 Canadian dollars).

Viscusi (1992) provides separate discussions and summaries of averting behaviour, wage-risk, and contingent valuation studies. His overall conclusion is that the most appropriate range of VSL estimates for use in policy analysis is $4 million to $10 million (1996 Canadian dollars). He also rejects the available averting behaviour study results for use in policy analysis because of clear downward biases in the study designs and data. Viscusi lists 27 VSL estimates from 22 wage-risk studies and eight estimates from six contingent valuation studies. Similar to the conclusions of the previous reviewers, Viscusi raises questions about some of the earlier wage-risk studies that used inappropriate risk data and obtained relatively low VSL results. He also raises some questions about some of the wage-risk studies that obtained results above $9 million. Viscusi concludes that the best VSL results from wage-risk studies are between $4 million and $10 million. Viscusi suggests that the two earliest contingent valuation studies were exploratory and that less weight be given to these two estimates (one is very low, the other is very high). The arithmetic mean of the remaining four contingent valuation estimates is either $4 million or $6 million, depending on whether the median or the mean estimate is selected from one of the studies. The range of the contingent valuation estimates is $2 million to $5 million or

$13 million, depending on whether the median or the mean value is selected from one of the studies.

Miller (1989) uses a different approach than that used in the other three reviews and reaches some different conclusions. He selects a larger number of available VSL estimates as potentially appropriate for use in policy analysis, but makes several adjustments in the estimates to reconcile differences in study design or limitations in data. Miller includes 29 VSL estimates as of reasonably good quality. Included in these 29 estimates are most of the estimates selected in the other reviews as most appropriate for policy analysis. An important difference is that Miller includes results from eight averting behaviour studies, which are rejected by the other reviewers as likely to be biased downward. An additional four are from contingent valuation studies, and the remaining 17 are wage-risk estimates. Miller made several adjustments to the estimates, most of which resulted in lowering the estimates, especially for some of the wage-risk studies with the highest results. The adjustments Miller made included (1) converting the wage-risk results to after-tax dollars, (2) adjusting for differences in labour risk data sources, (3) adjusting for failure to include nonfatal injury risks in the analysis, (4) adjusting to a uniform value of time or discount rate if used, and (5) adjusting for differences in perceived versus actual risks. The conceptual arguments for some of these adjustments may be valid, but the reliability of the data used to determine the exact adjustment to make is in many cases questionable. Miller concludes by choosing a mean VSL estimate of $3 million (1996 Canadian dollars), and a range of $2 million to $5 million.

Four wage-risk studies for Canadian labour markets (Meng, 1989; Meng and Smith, 1990; Martinello and Meng, 1992; and Vodden et al., 1994) report comparable WTP values. Table 5-3 summarises low, high, and mean WTP estimates from the studies.

Table 5-3
Ranges of VSL Estimates from Canadian Wage-Risk Studies

Study	VSL (1996 C$ millions)		
	Low	Mean	High
Meng (1989)	$4.5	$4.9	$5.3
Meng and Smith (1990)	$1.3	$7.9	$11.2
Martinello and Meng (1992)	$6.4	$7.2	$8.1
Vodden et al. (1994)		$6.1	

Martinello and Meng (1992) studied the effect of worker injury and mortality rates on wages in logging, mining and manufacturing. The study included data from a 1986 labour market survey

for blue-collar workers aged 20 to 64, which was merged with injury risk data (by industry) and mortality risk data (by industry and occupation). Risk data for Quebec was not comparable, so Quebec was excluded from the study. Martinello and Meng reported estimation results for several wage-risk specifications, and recommended that the VSL estimates from their specifications containing nonlinear relationships between wages and risk be used. The range of VSL estimates from the equations having nonlinear wage-risk relationships has a low value of $6.4 million, a high value of $8.1 million, and a mean value of $7.2 million (1996 Canadian dollars). The study also evaluates the effect of unionization on the wage-risk relationship, finding that compensation for risk is roughly comparable between union and nonunion jobs.

A similar study by Vodden et al. (1994) of Ontario workers reported a mean VSL $6.1 million (1996 Canadian dollars). This study included worker compensation for on-the-job injuries in the wage-risk relationship, as well as fatality and injury risks. The main result was that worker compensation programs tended to decrease the risk premium in wages.

Meng and Smith (1992) used a 1984 national election survey that contained detailed employment information, and merged it with occupational fatality data. They did not incorporate information on injury risks. This may have introduced an upward bias in their VSL estimates if some of the wage differential attributable to higher injury risks had been attributed to higher fatality risks, provided the two are positively correlated. The range of resulting VSL estimates has a low value of $1.3 million, a high value of $11.2 million, and a mean of $7.9 million (1996 Canadian dollars).

In an earlier wage-risk study, Meng (1989) included other occupational characteristics that may affect wage, such as repetitive tasks or ability to control work hours. He merged labour market data for males aged 21 to 64 from a 1981 national survey with occupational fatality data and other occupational characteristics. Various specifications generated a range of VSL estimates from a low of $4.5 million to a high of $5.3 million (1996 Canadian dollars). The mean value was $4.9 million.

The VSL ranges shown in Table 5-3 from the Canadian studies are comparable to the ranges presented the literature reviews discussed above, which are based on predominantly U.S. studies but also include studies conducted in Europe, Australia, Canada, and New Zealand. The mean VSL values selected by the broader literature reviews average about $5 million, while the means from the Canadian studies average about $6 million. Given the size of the range from low to high in these studies as a whole, these average VSL values are quite comparable. With the exception of the low value from Meng and Smith (1990), all the Canadian estimates fall within the range of values in Table 5-2.

5.2.3 The Potential Effect of Age on WTP for Changes in Mortality Risks

Although it has been suspected that age may be a factor in risk of death due to air pollution exposure, until recently there has been little quantitative evidence in the available epidemiologic literature. Schwartz and Dockery (1992a) report evidence that the measured association between daily mortality rates and daily levels of ambient particulate matter is greater for people over the age of 65. They provide sufficient information to estimate the change in the number of deaths expected for people over 65 and under 65 for a given change in ambient particulate matter.

This raises the question of whether WTP for changes in risks of death in the current time period is different for people over 65 than for the average adult. There is limited empirical evidence regarding this question, but some information is available. The expectation is that WTP will be lower for a 65-year-old than for the average adult, because expected remaining years of life are fewer. This expectation is based on the presumption that WTP for one s own safety is derived from the utility one receives from one s own life, and that this utility is to some extent a function of the amount of time one expects to remain alive.

Some analysts have suggested that effects of age might be introduced by dividing average WTP per statistical life by average expected years of life remaining (either discounted or not) to obtain WTP per year of life (Miller, 1989; Harrison and Nichols, 1990). Such a calculation implies very strong assumptions about the relationship between life expectancy and the utility a person derives from life, namely, that utility is a linear function of life expectancy. Although this might be correct, it is also plausible that this calculation will result in significant understatement of WTP for the elderly. An understatement could result for a number of reasons. One is that there may be a value to being alive that is independent of the amount of time one expects to live. Another is that as one ages, the remaining time may be more highly valued than it was in midlife.

We have identified one study that provides unconstrained empirical evidence concerning how WTP for small changes in risks of death varies with age. Jones-Lee et al. (1985) conducted a contingent valuation study concerning motor vehicle accidents and report an estimated WTP function for characteristics of the respondents, including age.[6] (There are some other studies that provide some suggestive evidence regarding how WTP for reducing risks may change with age, but each of these studies imposes some constraints on the conclusions in the form of unverified model assumptions.)

Jones-Lee et al. conducted a general population survey in the United Kingdom in which about 1,000 respondents were asked how much additional money they would be willing to pay for transportation with a bus company with a better safety record than an alternative company. All relevant risk information was quantitatively specified and the survey appears to have been well designed and executed. Implied WTP per life (VSL) was calculated for each response. For

6. This summary and proposed adjustment in the monetary values based on available empirical evidence of the effect of age on WTP for changes in mortality risks is drawn from Rowe et al. (1995).

example, the VSL is $6 million when the WTP response is $240 for a reduction in risk of death of 4 in 100,000. Variations in the implied VSL estimates across respondents were then examined as a function of age and other characteristics of the respondents. An appropriate functional form was used that allowed WTP to be a nonlinear function of age (age + age^2).

The results reveal a statistically significant relationship between age and VSL, which was statistically strongest for the responses to the first bus safety questions. The results indicate gradually increasing VSL until about age 45, then gradually declining VSL. The results for both the bus safety questions imply that the VSL for a person aged 65, all other things being equal, is about 90% of the VSL for a person aged 40.

The Jones-Lee et al. (1985) results with respect to age, based on the responses to the first bus safety question, are:

$$\text{VSL} = \text{Constant} + 12{,}489 \times (\text{Age} - \text{Mean Age}) - 660 \times (\text{Age} - \text{Mean Age})^2 + B_iX_i \,, \qquad (5\text{-}1)$$

where:

VSL = the implicit VSL given by the respondent
B_iX_i = the other independent variables in the WTP regression.

The authors do not report mean age for the sample, but describe the sample as nationally representative. For purposes of interpreting the regression results, we use 40 years as an average age, which is close to the average age of adults in the United States. The average VSL is reported as 1.6 million British pounds. We then calculated illustrative VSL estimates at selected ages using the following formula:

$$\text{VSL} = 1{,}600{,}000 + 12{,}489 \times (\text{Age} - 40) - 660 \times (\text{Age} - 40)^2 \,. \qquad (5\text{-}2)$$

This calculation assumes that other factors that influence VSL do not change with age. The risk of error due to this assumption seems small because only the age variables were statistically significant in this regression.

To allow for simple comparison to the results of other studies, we calculated VSL at each age using Equation 5-2. We then calculated VSL at each age as a percentage of VSL at age 40. These percentages are plotted in Figure 5-1.

Moore and Viscusi (1988) estimated a wage-risk premium for a sample of workers in the United States. They defined risk on the job as the probability of a fatal accident multiplied by the discounted remaining life years of the individual. They used a nonlinear estimation technique to

Figure 5-1
Value of a Statistical Life as a Function of Age

estimate both the risk coefficient and the implicit discount rate for time. They also included an expected annual annuity variable to account for the possibility that a wage-risk premium might not be as high if available insurance covers some of the risk to dependents. The results showed a significant relationship between wages and risks of fatal accidents and implied a value per statistical life of about $11.0 million (1996 Canadian dollars). The finding of a significant (negative) relationship between wages and expected annual annuity suggests that estimates that ignore potential death benefits may understate WTP to reduce risks of death. The estimated discount rate was 10% to 12%.

The Moore and Viscusi model assumes a constant value per year of life, and future years are discounted at rate *r*. The model, therefore, does not provide an unconstrained test of how VSL varies with age. VSL at different ages is simply a function of the discount rate, according to this model, and is therefore proportional to discounted remaining life years. The model implies that WTP for small changes in current risks decreases with age throughout a person s lifetime. How fast it declines depends on the discount rate. Moore and Viscusi define discounted remaining life years as:

$$DRLY = 1/r \times [1 - \exp(-r \times R)] , \quad (5\text{-}3)$$

where:

DRLY = discounted remaining life years
R = expected life years remaining.

The implications of different discount rates on WTP for changes in risks of death can be illustrated as follows. VSL will be proportional to the discounted remaining life years (DRLY). This means that the ratio of VSL at age 40 to VSL at age 65 will be the same as the ratio of DRLY at age 40 and DRLY at age 65. The implications of Moore and Viscusi s results from their linear wage function (r = 9.6%) with respect to the age of the worker are shown in Figure 5-1. It should be noted that the estimates are based on a sample of 317 working adults, which included few individuals over age 60 (62 is two standard deviations above the mean age). Also, life expectancies do not actually decline linearly with age, as is assumed in the calculations that underlie Figure 5-1. Average life expectancy in 1983 was 75 years at birth in the United States, but it was 17 years for 65-year-olds.

Cropper and Freeman (1991) provide a summary of the life-cycle consumption-saving model that can be used to derive a theoretical definition of WTP for changes in the probability of death. This model is based on the premise that utility is a function of consumption. The authors note that if there is additional utility derived from survival per se, then the life-cycle model provides a lower bound estimate of WTP. Of interest is what the model predicts in terms of how WTP for changes in risks of death in the current time period changes as a function of age. For a quantitative example, this depends on assumptions regarding a lifetime pattern of earnings, endowed wealth, the rate of individual time preference, and other parameters of the model. These will all vary for different individuals, and uncertainty exists empirically about population averages for many of these factors. However, using reasonable values to calibrate the model is illustrative.

Cropper and Freeman (1991) note that if consumption is constrained by income early in life, the model predicts that VSL increases with age until age 40 to 45 and declines thereafter. Shepard and Zeckhauser (1982) illustrate this point with numerical examples for the life-cycle model. When they estimate the model with reasonably realistic parameters and assume no ability to borrow against future earnings or to purchase insurance, they find a distinct hump in the VSL function that has a peak at about 40 years and drops to about 50% of the peak by 60 years. When they allow more ability to borrow against future earnings and to purchase insurance, the function flattens and at age 60 drops only to 72% of the VSL at age 40.

For comparison purposes, all of the estimates discussed above are plotted in Figure 5-1 along with the relationship between VSL and age implied by a simple linear decline with age. This linear decline implies that VSL at age 65 is about 30% of VSL at age 40. This is a much larger decline in VSL as a function of age than implied by the available empirical results reported above. The strongest weight should be given to the Jones-Lee et al. results because they are based

on a representative general population survey and were not unduly constrained by an imposed functional form. However, survey results can be highly variable and need to be interpreted cautiously until verifying results from multiple studies are obtained.

The life-cycle model results are quite variable depending on assumptions used to quantify the model. These assumptions have not been verified empirically. Because the model defines utility as a function of consumption, and consumption is a function of time, it is expected that if the life-cycle estimates err, it is on the side of overstating the effect of age on VSL (in other words, reducing VSL too much at age 65 relative to age 40). The error would result if there is some value to just being alive independent of consumption. At consumption levels above subsistence, this is quite plausible. Therefore, these estimates should be interpreted as representing the maximum plausible reductions in VSL as a function of age.

5.2.4 Mortality Risk Valuation Estimates Selected for AQVM 3.0

Obviously, there is some judgment involved in selecting central, high, and low values for the WTP for changes in risks of death. Because of the comparability between the U.S. and Canadian evidence, we will adopt VSL value midpoint and range estimates that are based on values used in Rowe et al. (1995) which are similar to the values selected by Cropper and Freeman (1991) based on their review of this literature. These values are slightly lower than might be selected if only the Canadian studies were considered, but because of the broad comparability between the results of the Canadian studies and those of the broader international literature it seems appropriate to select values based on the broader literature rather than on the Canadian studies alone.

The selected VSL estimates based on the available literature are, in 1996 Canadian dollars, $3.1 million for the low, $5.2 million for the central, and $10.4 million for the high (double the central estimate, and less than several of the highest reported values). The VSL estimates available from the literature are based primarily on samples of working-age adults. A few of the contingent valuation studies in this literature included individuals of retirement age, but this age is not well represented in the mean VSL values. These selected VSL estimates are therefore applied only to the under 65-year-old population.

Available evidence suggests that WTP for small changes in risks of death for people over age 65 can be expected to be lower than WTP for the same change in risk at age 40; however, there is considerable uncertainty about how much lower. The most relevant direct evidence suggests that the decline in VSL with age may be relatively small (e.g., 90% of the age 40 WTP at age 65). The evidence strongly suggests that a linear decline in VSL with age significantly understates actual VSL over age 65. Based on our evaluation of the evidence described above regarding VSL and age, we utilize the Jones-Lee et al. (1985) results to calculate a weighted average VSL based on the approximate age distribution for the U.S. population 65 and older. This produces an adjustment to VSL for those 65 and older of about 75% of the average VSL for adults under 65.

An age-weighted average VSL for this analysis is then calculated on the assumption that 85% of the particulate-related deaths are experienced among people 65 and over (see Chapter 4). The results are shown in Table 5-4. These are the default VSL estimates applied to the predicted changes in premature deaths in this assessment for mortality risk changes associated with particulate matter (including sulphates) and ozone air pollution changes.

Table 5-4
Selected Monetary Values for Mortality Risks in AQVM 3.0

Population Group	Selected VSL Estimates (1996 C$ million)		
	Low	Central	High
65 years old	$2.3	$3.9	$7.8
<65 years old	$3.1	$5.2	$10.4
Age-weighted average VSL[1]	$2.4	$4.1	$8.2
Probability associated with the estimates for uncertainty analysis	33%	50%	17%

1. Assuming 85% of deaths are individuals aged 65 and over.

The selection of probability weights for the low, central, and high estimates is somewhat arbitrary because there are several uncertainties in using these estimates in this analysis for which no quantitative information is available. The selected weights therefore reflect the uncertainty in the underlying WTP estimates for small changes in risks of accidental death for working-age adults, but do not fully reflect the uncertainty in applying these estimates in this analysis. The weight selected for the central estimate is 50%, because the underlying WTP estimates are predominately in the $3 to $6 million range. A weight of 33% is given to the low estimate and a weight of 17% is given to the high. This reflects that the high estimate is represented by fewer studies and a somewhat skewed distribution in the available WTP estimates. These weights result in a weighted mean value that approximates the selected central estimate.

5.2.5 Emerging Alternatives for Mortality Risk Valuation

A commonly proposed revision to the VSL approach would be to count and value not lives saved but life-years saved. A measure of life-years saved is a potentially more accurate measure of the quantity of life saving that is being accomplished by a given program. Life-years saved is estimated by subtracting the age at death, for the deaths prevented, from the life expectancy for the person whose premature death was prevented. This calculation is not done on a person by person basis, but rather on an age group basis. What is needed to make this calculation is the number of premature deaths prevented in each of the age groups exposed to the environmental

change. Life-years saved is then calculated as the number of lives saved in each age group multiplied by the average remaining life expectancy in each age group.

Estimates of life-years saved provide a scaling of the mortality risk change relative to the remaining life expectancy of those affected by the environmental change. Counting life-years saved will effectively differ from counting lives saved only if the change in risk varies by age, or by some other identifiable population characteristic, relative to the distribution of overall mortality risks in the population.[7]

If reductions in mortality risks change are quantified as life-years saved rather than numbers of premature deaths avoided, then what is needed for monetary valuation is the value of a statistical life-year (VSLY) saved rather than the value of a statistical life (VSL). The relationship between VSL and VSLY is frequently presumed to take the following form (Moore and Viscusi, 1988):

$$VSL_{ageX} = (1/r) \times [1 - \exp(-r \times R_{ageX})] \times VSLY , \quad (5\text{-}4)$$

where:

VSL_{ageX} = value of statistical life at age X
R_{ageX} = expected life-years remaining at age X
VSLY = value of current statistical life-year
r = discount rate for future years.

This expression presumes that the VSLY for the current time period remains a constant value and that future years are discounted at some rate of time preference (r). Thus, VSL is the same as the present value of the sum of the values for each remaining expected life-year, discounted at the rate r. The relationship between VSL and VSLY, according to this expression, is a function of the remaining life expectancy of the individual and the discount rate.

Moore and Viscusi (1988; 1990) have used labour market data and a two-stage estimation approach to infer a discount rate from the estimated parameters of a wage-risk model in which risk is defined as expected life-years lost. They find discount rates vary with characteristics of the individual and that they are generally in the same range as market discount rates.

When calculating a VSLY from an estimate of VSL, the discount rate for future years makes a big difference. For example, if we have a VSL of $5 million for a person with a remaining life expectancy of 40 years, the VSLY will be $125,000 if the discount rate is 0%, $214,592 if the

7. Age is not necessarily the only factor to consider. It may be known, for example, that those at risk of premature deaths are those in already poor health for reasons unrelated to pollution exposures. The already lower life expectancy of this poor health cohort would be the appropriate basis for calculating life-years saved for exposure reductions for this group. Seldom, however, is information this specific available.

discount rate is 3%, $329,815 if the discount rate is 6%, and $462,963 if the discount rate is 9%. The higher the discount rate, the higher the implicit VSLY for the current year, for a given VSL.

Practical Limitations of VSLY Approach

Important limitations remain in our ability to successfully implement the VSLY approach. Empirical estimates to date based on labour market data have been based on models that presume a constant VSLY for the current year through a person s remaining lifetime. There is no reason to expect that this is necessarily the case. In fact, there is some limited evidence to the contrary. If VSLY were constant, then VSL would be a consistently declining function of age, with the rate of decline being a function of the discount rate. In a contingent valuation study of transportation safety, Jones-Lee et al. (1985) found VSL to be increasing with age to about age 40 and then decreasing with age.[8] This suggests that VSLY could be changing over time with changing patterns of income and other factors through person s life. As life expectancy declines, limited remaining years might each be more highly valued. Changes in health that adversely affect a person s enjoyment of life could have the opposite effect.

Changes in Life Expectancy

Another approach for defining a change in mortality risk that is related to life-years saved is to define the change in risk in terms of a change in life expectancy. It may be possible in some cases to estimate the average change in life expectancy for a population whose exposure to a harmful pollutant is changed. Such an estimation requires information similar to what is needed to estimate life-years saved. Proponents of this approach suggest that as with life-years saved, this is a more accurate way of characterizing the magnitude of the change in mortality risk. Fundamental empirical questions remain, however, regarding how people value a change in life expectancy.

A few recent stated preference studies (e.g., Johannesson and Johansson, 1996) have attempted to determine WTP values for changes in life expectancy, but these efforts remain exploratory and subject to difficulties in communicating life expectancy concepts to general population subjects. Results cannot yet be considered adequate for use in cost-benefit analyses. Johannesson and Johansson (1996) asked a random sample of Swedes to estimate their WTP now for a medical treatment that if given at 75 years of age would increase their remaining life expectancy from 10 years to 11 years, assuming that they survive to age 75 in the first place. This approach has been criticized as ambiguous, since many different paths of conditional probabilities of survival at different ages are consistent with an extension of life expectancy. Also, the way this change in life expectancy is presented, it focuses the respondent on a year of life tacked on at the end which makes it seem as though nothing is changed until the last year is reached. Changes in

8. Based on the Jones-Lee et al. results, the VSL applied in AQVM 3.0 for mortality risk reductions was given a different value for premature deaths prevented in the population over age 65 versus under age 65. This is in effect a limited adjustment for differences in life-years saved across the two age groups.

life expectancy are probably more accurately depicted as a shift in survival probabilities for every remaining year of life. Fabian et al. (1994) illustrate this with a bar chart showing survival probabilities over five-year increments throughout a cancer patient s possible remaining life, with and without a specific cancer treatment under consideration. Fabian et al. note that Cropper has suggested (in personal communication) that it would be more accurate for respondents to be asked to value a change in the conditional probability of death at various ages, conditional on reaching each age. These remain difficult concepts to communicate.

5.3 MONETARY VALUATION OF CANCER RISK

There are no adequate WTP estimates for reducing cancer risks or for reducing mortality risks specifically related to cancer. The approach used here is to combine WTP estimates for mortality risks in general with adjusted COI estimates for nonfatal cancers according to the average cancer survival rate. WTP estimates for reducing the risk of developing a new cancer case are, thus calculated by combining cancer survival rate information with VSL estimates and estimates of the value of nonfatal cancer cases using the following equation:

Cancer WTP =
[(1 - survival rate) × fatal case value] + (survival rate × nonfatal case value). (5-5)

Cancer survival rates depend on the type of cancer, its location, the stage at which the cancer is diagnosed, and the number of years from diagnosis under consideration. For AQVM 3.0 we use five-year survival rates to measure a cancer s lethality and as an input into equation 5-5. In AQVM 3.0 exposure to acetaldehyde, 1,3 butadienne, and formaldehyde is not associated with the development of a specific type of cancer. As a result, we use the average five-year survival rate of 40% observed across all cancers for all patients in Quebec (National Cancer Institute of Canada, 1995), assuming this rate is similar in other provinces. As previously discussed, (see Section 4.5 on cancer risks) benzene exposures are specifically associated with the development of acute myelogenous leukemia (AML). The observed 5-year relative survival rate for AML cases from 1980-1997 in Saskatchewan of 12.2% (R. Semenciw and C. Waters, Cancer Bureau, Health Canada, personal communication, 1999) is incorporated in the estimates of WTP to reduce the risk of developing AML, again assuming that this provincial rate accurately reflects the national average.

5.3.1 Monetary Valuation of Fatal Cancers

The valuation of new cancer cases that result in a fatality incorporates VSL estimates because there are no adequate empirical estimates of WTP for changes in risks of death from cancer. As with the VSL estimates for mortality risks, an age-weighted average value is calculated that accounts for the distribution of cancer-related mortalities between the age < 65 and age 65 groups. For new nonspecific cancer cases associated with exposures to acetaldehyde, 1,3

butadienne, and formaldehyde, the age-weighted value reflects the fact that 70% of all nonspecific cancer-related deaths in Canada occur among those 65 and older (National Cancer Institute of Canada, 1993). For new AML cases the age-weighted average mortality risk value reflects that 56% of all deaths attributed to AML in Canada from 1993-1994 occurred among those 65 and older (C. Waters, Cancer Bureau, Health Canada, personal communication, 1999). Table 5-5 presents the impact of age-weighting the VSL estimates on the final values for new fatal cases of nonspecific cancer and AML.

Table 5-5
Monetary Values for Fatal Cancer Cases ($1996 Canadian)

	Age < 65 (millions)	age 65 (millions)	% of Fatal Cases where Age 65	Age-Weighted Value (millions)
Fatal Nonspecific Cancer Cases (Acetaldehyde, 1,3 Butadienne, and Formaldehyde)				
Low	$3.1	$2.3	70%	$2.6
Central	$5.2	$3.9	70%	$4.3
High	$10.4	$7.8	70%	$8.6
Fatal AML Cases (Benzene)				
Low	$3.1	$2.3	56%	$2.7
Central	$5.2	$3.9	56%	$4.5
High	$10.4	$7.8	56%	$8.9

5.3.2 Monetary Valuation of Nonfatal Cancers

COI estimates provide the starting point to value cancer morbidity because WTP measures are unavailable. The COI measure best suited for estimating the costs associated with a new nonfatal cancer case is an *incidence-based* measure of the present value of the stream of costs a patient can expect to incur over the course of the illness. This measure includes expected future health care costs and anticipated productivity losses, starting from the point of diagnosis.

The alternative COI measure is a *prevalence-based* estimate based on the health care costs and productivity losses that accrue to all individuals with cancer in a given time period. As a result, prevalence-based estimates incorporate expenditures for both new cases and cases that were previously diagnosed. The prevalence-based measure is commonly used in estimating COI values because the required data (i.e., actual hospital expenditures for a given diagnosis) are fairly readily available, and because they provide useful estimates of the financial burden of an illness

as a whole. However, prevalence-based estimates are not very useful for assessing the financial benefits of preventing new cases.

A major incidence-based cost of cancer study was performed by Hartunian et al. (1981) using data from the Third National Cancer Survey. Costs were estimated in two parts: the direct costs and the indirect costs. The direct costs reflect hospitalization, physical, medication, treatment, and administrative costs. The direct costs depend on the site and extent of the cancer. The indirect costs capture the difference in the expected earnings of people with and without cancer (for homemakers lost productivity was estimated using information on the market value of household services). The indirect costs depend on the age of the cancer patient, the site and extent of the cancer, sex of the patient, and the survivability of the cancer. While the results of this study are a bit dated (estimates are expressed in 1975 U.S. dollars) they are the most recent incidence-based estimates available at this level of detail for cancer.

Hartunian et al. report average COI estimates per cancer case for nine major types of cancer, by gender of the patient, and by eight patient age groups. We use the midpoint COI estimate across the full range of cancer types and patient characteristics as the starting point for new nonfatal cancer cases associated with exposures to acetaldehyde, 1,3 butadienne, and formaldehyde because the type of cancer associated with these pollutants is not further specified. For the new nonfatal AML cases associated with benzene we select the midpoint COI estimate presented for leukemia.

In the following sections the steps used to estimate the average direct and indirect costs per nonfatal cancer case for AQVM 3.0 starting from the Hartunian et al results are presented in detail for the unspecified cancers and then summarised for the AML cases.

Direct (medical) Costs

1. The estimated midpoint from the range of average direct costs per cancer patient by cancer type from Hartunian et al.(see their Table 5-15) was used as the starting point for developing the direct costs of an unspecified cancer case. The values from Hartunian et al. ranged from $5,172 to $19,524 resulting in a estimated midpoint of $12,348 (all values in 1975 U.S. dollars).

2. This original midpoint estimate for direct costs is inflated to its equivalent value in 1983 U.S. dollars using the U.S. medical care price index values of 47.5 in 1975 and 100.6 in 1983 (U.S. Bureau of the Census, 1994). The estimate is then converted to its equivalent in 1983 Canadian dollars by multiplying by the 1983 purchasing power parity index value of 1.24. Finally, the estimate is inflated to $54,000 1996 Canadian dollars (rounded to the nearest thousand) using the Canadian health care price index values of 85.1 for 1983 and 142.1 for 1996.

Following the same procedure for leukemia cases results in a direct cost estimate of $42,000 (1996 Canadian dollars rounded to the nearest thousand) from a starting midpoint estimate of $9,529 (1975 U.S. dollars) for leukemia cases (see Hartunian et al. s Table 5-15).

Indirect (productivity) Costs

3. The estimated midpoint from the range of Hartunian et al. s estimates of average foregone earnings per cancer patient by cancer type was selected as the starting point for developing the indirect costs of an unspecified cancer. The values ranged from $136 to $225,631 (see their Table 5-19) resulting in an estimated midpoint of $112,884 (all values in 1975 U.S. dollars). This value includes the foregone earnings of all cancer patients, including both fatal and nonfatal cancers.

4. This estimate is inflated to 1983 U.S. dollars using the U.S. consumer price index values of 53.8 for 1975 and 99.6 for 1983 (U.S. Bureau of the Census, 1994) and is then converted to its equivalent in 1983 Canadian dollars using the 1983 PPP index value of 1.24. The estimate is then inflated to $397,000 (1996 Canadian dollars, rounded to the nearest thousand) using the Canadian consumer price index values of 88.5 for 1983 and 135.7 for 1996.

5. The indirect cost figure from Hartunian et al. is a present value of lifetime foregone earnings per cancer case, including both fatal and nonfatal cases. What we want, however, is an estimate of the typical foregone earnings per nonfatal case. Rice et al. (1985) report total annual indirect costs for nonfatal cancers are about 15% of total annual indirect cancer costs for all cancers, but they do not report average costs per cancer case. We use the 40% five year survival rate for Quebec (National Cancer Institute of Canada, 1995) to estimate the share of cancers that are nonfatal assuming that the values from Quebec are representative of the Canada-wide value. Thus, $397,000 is multiplied by 15% and divided by 40% to approximate the average indirect costs per nonfatal cancer case.[9] This results in a value of $149,000 (1996 Canadian dollars, rounded to the nearest thousand), as an estimate of present value of lifetime foregone earnings for the average nonfatal cancer case.

Steps 3 through 5 above were followed for leukemia cases with only one adjustment; because the relative 5-year survival rate for AML cases is 12.2% we adjusted the share of total annual indirect costs for nonfatal AML cases from 15% to 4.6% to maintain the same ratio between the survival rate and the indirect cost share for AML cases as was used for the unspecified cancer

9. If total indirect costs for all cancers is X and total cancer cases is N, the Hartunian et al. estimate approximates the average indirect cost for all cancer cases, $X \div N$. However, what we want is an estimate of the average indirect cost for only nonfatal cancer cases, $X_n \div N_n$, where X_n equals total indirect costs for nonfatal cancers and N_n equals the number of nonfatal cancer cases. We know from Rice et al. (1985) that X_n equals $0.15 \times X$. We can also estimate that N_n equals $0.40 \times N$. Thus, $X_n \div N_n$ can be estimated from the Hartunian et al. estimate of $X \div N$ as follows: $X_n \div N_n = (0.15 \times X) \div (0.40 \times N) = (0.15 \div 0.40) \times (X \div N)$.

cases. With this adjustment, and using the midpoint from the range of Hartunian et al. s estimates of average foregone earnings per leukemia patient of $112,970 (1975 U.S. dollars) an value of indirect costs of $149,000 (1996 Canadian dollars, rounded to the nearest thousand) is obtained.

Total COI per Nonfatal Cancer Case

6. The total incidence-based COI value for nonfatal cancers is estimated by summing the indirect and direct costs. This value is $203,000 for the unspecified cancer cases associated with acetaldehyde, 1,3 butadienne, and formaldehyde, and $191,000 for the AML cases associated with benzene.

The procedures described above consist primarily of selecting midpoint values from the Hartunian et al. report and adjusting to current Canadian dollars. The fifth step is taken to estimate average indirect costs per nonfatal cancer because Hartunian et al. do not report separate estimates for fatal and nonfatal cancers. Indirect costs for fatal cancers are expected to significantly exceed indirect costs for nonfatal cancers because the total time lost from work would be much higher for fatalities (i.e., all expected remaining work life) than for nonfatal cancers from which many individuals would be able to return to work after treatment. We therefore rely on available prevalence-based COI estimates for annual indirect costs associated with morbidity and mortality for all cancers in the United States (Rice et al. 1985) to provide an estimate of the percentage of total indirect costs due to cancer that are attributable to nonfatal rather than fatal cancers. This presumes fatalities due to cancer are a similar proportion of the total cancer cases whether measured as a prevalence or an incidence. This is not an entirely satisfactory assumption, but it appears preferable to the alternative of no adjustment in the indirect cost estimates for fatal versus nonfatal cancers.

There are additional limitations of these estimates for use in this analysis that should be acknowledged:

Changes in the direct costs of cancer since 1975 may not be fully reflected in the U.S. or Canadian medical consumer price index.

The range of costs used to develop the midpoint estimate for an unspecified cancer case incorporates costs from several different types of cancer, some of which may not be relevant to exposures to acetaldehyde, 1,3 butadienne, and formaldehyde.

We also know that the WTP for an avoided nonfatal cancer case will be greater than the estimated COI values because WTP estimates incorporate values for the avoided pain and suffering and restriction of nonwork activities that are not captured within the COI-based estimates. Thus, the sum of the COI values for direct and indirect costs needs to be adjusted upward. We multiply the summed COI value for cancer cases, regardless of the type of cancer, by a WTP/COI ratio of 1.5 to estimate the associated WTP value for an avoided cancer case. See Section 5.1.3 for a discussion of the empirical basis for this type of adjustment to COI estimates

when the desired value is WTP. This adjustment results in a central dollar value per nonfatal unspecified cancer of $305,000 and a central dollar value for a nonfatal AML case of $287,000 (1996 Canadian dollars). For low and high values, we use the same ratio as was determined for WTP for other morbidity risks. The low is thus one-half the central and the high is twice the central. The resulting low and high estimates are $153,000 and $610,000 for the unspecified nonfatal cancer cases and $144,000 and $574,000 for nonfatal AML cases (rounded to the nearest thousand) respectively.

5.3.3 Cancer Case Valuation Estimates Selected for AQVM 3.0

Table 5-6 presents the results of the combining the values per nonfatal cancer case with the age-adjusted values per fatal cancer case according to equation 5-5 to produce age and outcome weighted values per cancer case according the to the pollutant in question.

Because uncertainty in the WTP estimates per fatal cancer case contributes a significant portion of the uncertainty in the WTP estimates per cancer case, and because the fatal cancer case values dominate the age and outcome weighted estimates, we use the same probability weights for the cancer case values as those selected for the mortality values reported in Table 5-4.

5.4 MONETARY VALUATION ESTIMATES FOR MORBIDITY

WTP estimates of value are available for about half of the nonfatal health effects identified in Chapter 4, and primarily for the least serious health effects. However, most of the WTP studies completed to date have limitations because of small sample sizes and limited variation in the health effect studied, and few of these studies have been replicated. Some interpretations and adjustments in the results of the WTP studies were necessary in applying them for this analysis. These studies have been reviewed and synthesised in previous air quality benefits studies (Rowe et al., 1986; Krupnick and Kopp, 1988; Hall et al., 1989). This analysis relies to a large extent on these previous reviews for specific interpretations.

When WTP estimates are not available, the monetary estimates are based on COI information, and the COI values are inflated to WTP estimates with a WTP/COI factor of 2 (the derivation of this factor is discussed in Section 5.1.2). The COI information used in this analysis reflects medical costs and lost productivity due to illness. The average daily Canadian wage for 1996 is used as a measure of lost productivity for days when all normal activities are prevented because of illness. Such days include days spent in the hospital, one day for each emergency room visit, and days spent in bed because of illness.

Table 5-6
Monetary Values for Cancer Effects in AQVM 3.0 (1996 C$)

Values for Cancers Associated with Acetaldehyde, 1,3 Butadienne, and Formaldehyde

	Value per Nonfatal Cancer Case	Value per Fatal Cancer Case[a]	Average Value for All Cancer Cases[b]	Probability Weights
Low	$153,000	$2.6 million	$1.6 million	33%
Central	$305,000	$4.3 million	$2.7 million	50%
High	$610,000	$8.6 million	$5.4 million	17%

a. Reflecting that approximately 70% of cancer deaths in Canada are experienced by those over age 65.
b. Based on the average 5-year survival rate of 40% for all cancers in Quebec.

Values for Cases of AML Associated with Benzene

	Value per Nonfatal Cancer Case	Value per Fatal Cancer Case[a]	Average Value for All Cancer Cases[b]	Probability Weights
Low	$144,000	$2.7 million	$2.4 million	33%
Central	$287,000	$4.5 million	$4.0 million	50%
High	$574,000	$8.9 million	$7.9 million	17%

a. Reflecting that approximately 56% of AML deaths in Canada from 1993-1994 are experienced by those over age 65.
b. Based on the average 5-year relative survival rate of 12.2% for AML in Saskatchewan for cases from 1980-1997.

The average daily wage is used as a measure of the average opportunity cost of time for employed and not-employed individuals, on the presumption that those who are not employed value their leisure or household services at a level equal to the wage they forego in choosing not to pursue paid employment. This approach may somewhat overstate foregone wages for the elderly and women, who make up a large share of the not-employed group and may have less than average earning power in the labour market. On the other hand, this approach does not reflect any productivity losses beyond the average work-day hours, thereby understating productivity losses for employed and not-employed individuals who perform household, childcare, and community service work beyond the usual work-day hours. This omission,

however, is offset by the adjustment used to proxy WTP when using the COI estimates. For these calculations, the 1996 average daily wage of C$117 for all workers in Canada is used.[10]

The available WTP studies provide some information on the relationship between the central estimate and the range of WTP estimates within and across studies. In general, these ranges are from minus 50% to plus 50% to 100% of the central estimate. Therefore, unless otherwise noted, a range of ±50% is applied to the central estimate of WTP for a health effect in this analysis to derive the low and high estimates.[11] The monetary values used for the health endpoints, other than mortality and cancer, in this assessment are summarised in Table 5-7. The specific derivations of these estimates are explained below.

5.4.1 Adult Chronic Bronchitis

Viscusi et al. (1991) and Krupnick and Cropper (1992) conducted a set of survey exercises to estimate WTP for reducing risks of developing chronic respiratory disease. In both studies, respondents were presented with trade-off options for risks of developing chronic bronchitis (or chronic respiratory disease in general) versus cost of living. Respondents were presented with hypothetical residence location options where in some locations risks of developing chronic respiratory disease are lower but cost of living is higher. An additional trade-off question was for risks of developing chronic bronchitis versus risks of death in an auto accident. An interactive computer program was used to adjust the trade-off until the respondent reached a point of indifference between the two options. At this point, a maximum WTP to prevent developing chronic bronchitis is revealed.

The health endpoint defined in these studies does not exactly match that defined in the Abbey et al. (1993) study, upon which the estimates of new cases of chronic bronchitis are based (see Chapter 4). The primary difference is the level of severity. The WTP studies defined a severe case of chronic bronchitis. The Abbey et al. results reflect a more average case. In this section we present the results of these WTP studies and a procedure for adjusting the results to better reflect the level of severity of interest for this analysis.[12]

10. Statistics Canada reports average weekly earnings of $586 for 1996. This is approximately $117 per day.

11. Throughout the development of the morbidity monetary estimates, values are rounded only as a last step. Rounding occurs only after conversion from U.S. values to their Canadian equivalent, inflating values to the appropriate year, and, where appropriate, applying the ±50% adjustment to the central estimate to derive the low and high estimates.

12. This adjustment procedure is based on information reported by Krupnick and Cropper (1992) and was suggested by Alan Krupnick in personal communication.

Table 5-7
Selected Monetary Values for Morbidity Effects in AQVM 3.0

Morbidity Effect	Estimate per Incident (1996 C$)			Primary Source	Type of Estimate[a]
	Low	Central	High		
Adult chronic bronchitis	$175,000	$266,000	$465,000	Viscusi et al. (1991) Krupnick and Cropper (1992)	WTP
Respiratory hospital admission	$3,300	$6,600	$9,800	Canadian Institute for Health Information (1994)	Adjusted COI
Cardiac hospital admission	$4,200	$8,400	$12,600	Canadian Institute for Health Information (1994)	Adjusted COI
Emergency room visit	$290	$570	$860	Rowe et al. (1986)	Adjusted COI
Child bronchitis	$150	$310	$460	Krupnick and Cropper (1989)	Adjusted COI
Restricted activity day	$37	$73	$110	Loehman et al. (1979)	WTP & Adjusted COI
Asthma symptom day	$17	$46	$75	Rowe and Chestnut (1986)	WTP
Minor restricted activity day	$20	$33	$57	Krupnick and Kopp (1988)	WTP
Acute respiratory symptom day	$7	$15	$22	Loehman et al. (1979) Tolley et al. (1986a)	WTP
Probability weights for all morbidity values	33%	34%	33%		

a. WTP = Contingent valuation WTP estimate. Adjusted COI = COI × 2 to approximate WTP.

The samples for the two studies differ. Viscusi et al. selected a representative sample of about 390 respondents. Krupnick and Cropper selected a sample of individuals who had a relative with a chronic respiratory disease. The Krupnick and Cropper sample was smaller (about 190 respondents) and less representative of the general population (lower average age and higher average income), reflecting a large percentage of respondents taken from the University of Maryland staff and students. The intent of the Krupnick and Cropper study was to test for the effect of familiarity with the disease on WTP responses.

Both studies used a definition of chronic bronchitis that reflects a severe case. The description of the disease included persistent symptoms of cough and phlegm, limits in physical activities, and ongoing medical care. Krupnick and Cropper used this definition in one version, and asked respondents to consider the risk of developing a case of chronic respiratory disease like your relative s in a second version. The relatives had chronic bronchitis, asthma, or emphysema. Respondents provided information on the severity of the relative s disease based on the number of symptoms present. This ranged from 0 to 13, where 13 reflects the severe chronic bronchitis case defined in the earlier questions. The analysis of WTP responses included the effect of the severity of the relative s case on the WTP response. At the mean of the variables, the estimated elasticity of WTP with respect to severity was 1.16. This means that WTP increased by 1.16% for every 1% increase in the 0 to 13 symptoms scale.

The WTP results from Viscusi et al. are more appropriate for this assessment because they are from a study sample that is more representative of the general population. The responses reflect the maximum amount the respondents revealed they would be willing to pay to reduce their annual risk of developing chronic bronchitis by a specified amount. The authors then calculated the implicit WTP per statistical case avoided. The median response in the study for the cost of living trade-off was approximately $457,000, and the arithmetic mean was about $883,000 (both values 1990 U.S. dollars).

The authors caution that the mean is affected by a small number of fairly high estimates and recommend that the median is more representative of the sample. We cautiously accept this recommendation and use the reported median value as the basis for our central estimate until the accuracy of the high estimates can be further verified in repeated studies and analyses. For a low estimate for a severe case of chronic bronchitis we select the 20th percentile value of $300,000 and for a high estimate we select the 80th percentile value of $800,000 (1990 U.S. dollars). These values were converted to their 1996 Canadian dollar equivalents by first multiplying by the 1990 PPP index value of 1.22 and then inflating using the Canadian CPI values of 119.5 for 1990 and 135.7 for 1996.

We use the elasticity estimate to adjust the WTP estimate from the value for a severe case to the value for a more average case of chronic bronchitis. The elasticity estimate is calculated from results reported by Krupnick and Cropper for a combined analysis of chronic bronchitis, asthma, and emphysema. Using this estimate for chronic bronchitis assumes that the elasticity of WTP with respect to severity is similar for chronic bronchitis to that for all three diseases combined.

The mean severity rating reported for the Krupnick and Cropper sample is 6.5, based on the 0 to 13 scale. Using the elasticity at the mean of 1.16, this suggests that WTP for an average case is 58% lower than for a case at 13 on the scale. Using this to adjust the Viscusi et al. estimates, we get a central WTP estimate of $266,000, a low of $175,000, and a high of $465,000 (1996 Canadian dollars), rounded to the nearest thousand, for an average case of chronic bronchitis.

It is important to note that these WTP estimates for preventing a new case of chronic bronchitis reflect the perceived welfare effects of living with chronic bronchitis over the entire course of the illness, which can span many years. It is a measure of the present value of the welfare effect that occurs over a multiple-year period. This is somewhat different than the other morbidity effects considered in this analysis which are short-term effects. In using the WTP values for chronic bronchitis we are assigning the full welfare effect for the new chronic bronchitis case in the year in which the clinical onset of the disease occurs. We do the same with the acute morbidity effects, but in those cases the illness typically begins and ends in the same year.

5.4.2 Respiratory Hospital Admissions

WTP estimates for respiratory hospital admissions (RHA) are not available. We, therefore, use the adjusted COI approach, which requires data on hospitalization costs and foregone wages. Average hospitalization cost for a given illness is derived by multiplying the resource intensity weight (RIW) for that illness, which is an index of relative demand of hospital resources, by the average cost of a unit of RIW, which was $2,500 in 1992.[13] For example, the RIW for a case involving respiratory infection is 1.1597, which implies an average hospitalization cost of approximately $2,900 (1992 Canadian dollars) (Canadian Institute for Health Information, 1994). For overall respiratory hospital admissions, we took an average across hospitalization costs for several respiratory illnesses-related to PM_{10} and ozone exposure using admission rates reported in Burnett et al. (1994; 1995) as weights. We calculated a similar average across lengths of hospital stay that were reported for the same illnesses. Hospital length of stay is used in the estimation of foregone wages.

The resulting estimated cost of a hospital stay for treatment of respiratory disease in Canada is $2,505 (1992 Canadian dollars), and the average length of stay is 5.7 days. The cost is inflated to $2,608 (1996 Canadian dollars) using the Canadian medical care price index values of 136.5 for 1992 and 142.1 for 1996. This estimate of hospital expenditures will tend to understate true costs because it does not include fees for physician services. The length of stay is multiplied by the average daily wage (W) to approximate the value of lost productivity for employed and not-employed individuals on the presumption that it is a measure of average opportunity costs for all individuals. The medical cost and lost productivity estimates are summed and multiplied by the

13. Personal communication from David Stieb, Health Canada.

WTP/COI ratio of 2 to account for additional potential pain and suffering and activity losses not reflected in the COI numbers. The central estimate is thus calculated as follows:

$$\text{Central \$/RHA} = [(5.7 \times W) + \$2608] \times \text{WTP/COI} \qquad (5\text{-}6)$$
$$W = \$117 \text{ (1996 C\$)} .$$

Therefore, the central estimate is $6,600. Applying a plus or minus 50% adjustment results in a low estimate of $3,300 and a high estimate of $9,800 (all values rounded to the nearest $100).

5.4.3 Cardiac Hospital Admissions

In the absence of WTP values for cardiac hospital admissions, we used the same method described for respiratory hospital admissions to calculate a COI-based estimate of value. RIWs for heart diseases related to PM_{10} and ozone were also multiplied by the unit value of $2,500 to derive illness specific costs. We calculated weighted averages of the hospitalization costs and lengths of stay based on admission rates for various cardiac diagnoses as reported by Burnett et al. (1994; 1995) and supplemented by Canadian hospital admissions data.[14] The weighted average hospitalization cost per case is $3,394 (1992 Canadian dollars) and length of stay is 5.6 days. The cost is inflated, using the Canadian medical care price index values of 136.5 for 1992 and 142.1 for 1996, to $3,533 (1996 Canadian dollars). Lost productivity is measured as the average daily wage multiplied by the length of stay. Summing hospital and lost productivity costs and multiplying by the WTP/COI ratio of 2 gives an approximation to value. The central estimate is calculated as follows:

$$\text{Central \$/CHA} = [(5.6 \times W) + \$3533] \times \text{WTP/COI} \qquad (5\text{-}7)$$
$$W = \$117 \text{ (1996 C\$)} .$$

The central estimate is $8,400, and applying a plus or minus 50% adjustment results in a low estimate of $4,200 and a high estimate of $12,600 (all values rounded to the nearest $100).

5.4.4 Emergency Room Visits

WTP estimates for emergency room visits (ERV) are not available. We therefore use the adjusted COI approach. This approach is applied to an average ERV fee of $85 in 1984 U.S. dollars (U.S. EPA, 1988). This becomes $168 (1996 Canadian dollars) after converting using the 1984 PPP index value of 1.25 and inflating using the Canadian medical care price index value of 89.9 for 1984 and 142.1 for 1996. This value is added to the average daily wage as a measure of the lost productivity associated with the visit, on the presumption that an ERV is associated with

14. Personal communication from David Stieb, Health Canada.

an average of one work-loss day. The resulting COI estimate is multiplied by the WTP/COI ratio of 2 to account for additional potential pain and suffering and activity losses not reflected in the COI numbers. The central estimate is thus calculated as follows:

$$\text{Central \$/ERV} = [W + \$168] \times \text{WTP/COI} \qquad (5\text{-}8)$$
$$W = \$117 \text{ (1996 C\$)} .$$

Therefore, the central estimate is $570. Applying a plus or minus 50% adjustment results in a low estimate of $290 and a high estimate of $860 (all values rounded to the nearest ten).

5.4.5 Child Bronchitis

WTP estimates for bronchitis in children are not available. We therefore use the adjusted COI approach. Krupnick and Cropper (1989) report average annual medical treatment costs of $42 in 1977 U.S. dollars for a child with bronchitis. This estimate is first inflated to its 1983 U.S. dollar equivalent using the U.S. medical consumer price index values of 57.0 for 1977 and 100.6 for 1983. The 1983 U.S. dollars value is then multiplied by the 1983 PPP index value of 1.24 and inflated using the Canadian medical care price index values of 85.1 for 1983 and 142.1 for 1996 to convert to the 1996 Canadian dollar equivalent of $153. This treatment estimate is multiplied by the WTP/COI ratio of 2 to account for additional potential pain and suffering and activity losses. The central estimate is thus calculated as follows:

$$\text{Central \$/B/year} = \$153 \times \text{WTP/COI} . \qquad (5\text{-}9)$$

Therefore, the central estimate is $310. Applying a plus or minus 50% adjustment results in a low estimate of $150 and a high estimate of $460 (all values rounded to the nearest ten). These estimates are probably too low because they do not reflect any value for lost productivity during the time the children are ill. Monetary estimates for lost productivity because of illness for children are not readily available.

5.4.6 Restricted Activity Days

A restricted activity day (RAD) is a measure of illness defined by the Health Interview Survey (HIS) as a day on which illness prevents an individual from engaging in some or all of his or her usual activities. This includes days spent in bed, days missed from work, and days with minor activity restrictions because of illness. WTP estimates for preventing a RAD are not available. We therefore approximate WTP for an average RAD using available COI data and WTP estimates for days with symptoms.

RADs reflect a combination of complete activity restrictions and minor activity restrictions. It is unknown what proportion of RADs attributable to air pollution exposure is minor rather than

severe. Recent data from the HIS indicate that about 40% of all RADs are bed-disability days. The results of Ostro (1987) suggest that RADs associated with air pollution exposure may be less severe on average than all RADs. We therefore presume a lower proportion of bed-disability days for this analysis than the national average for all RADs. We select an assumption that 20% of RADs due to air pollution exposure are bed-disability days.

Productivity losses associated with more serious RADs (bed-disability days) are estimated as equivalent to the daily wage rate for employed individuals. We apply the same measure of lost productivity for not-employed individuals on the presumption that it is a measure of average opportunity costs for all individuals. This lost productivity estimate is multiplied by the WTP/COI ratio of 2 to account for additional potential pain and suffering, additional leisure activity losses, and potential medical costs that are not reflected in the lost productivity estimates. Taking a weighted average of the value for bed-disability days and more minor RADs (valued as minor restricted activity days see Section 5.4.8) gives the average value for an air pollution induced RAD as follows:

$$\text{Central \$/RAD} = [0.20 \times W \times \text{WTP/COI}] + [0.80 \times \$33] \qquad (5\text{-}10)$$
$$W = \$117 \text{ (1996 C\$)} .$$

Therefore, the central estimate is $73 (1996 Canadian dollars). Applying a plus or minus 50% adjustment results in a low estimate of $37 and a high estimate of $110.

5.4.7 Asthma Symptom Days

Krupnick and Kopp (1988) review two studies that provide monetary value estimates for asthma symptom days. The first is a study by Krupnick (1986), which presents the medical expenditures associated with ozone-induced asthma symptoms. The expenses vary by the baseline frequency of symptoms and by the assumed prices for medical services. Krupnick and Kopp use these figures as a benchmark for calibrating estimates of WTP.

The second study (Rowe and Chestnut, 1986) is a WTP survey study that obtained asthmatics estimates of WTP to prevent an increase in bad asthma days (BADs). Each respondent defined for himself a BAD on a 1 to 7 severity scale for asthma symptoms. After analysing the WTP responses, Rowe and Chestnut found WTP estimates that are about 1.8 times greater than the medical costs found by Krupnick. Krupnick and Kopp point out that this finding is consistent with economic logic and lends credibility to both studies. Thus, for WTP values to prevent an asthma symptom day, Krupnick and Kopp rely on the Rowe and Chestnut estimates.

Rowe and Chestnut found that the WTP responses were positively associated with the baseline frequency of asthma symptoms. The values also varied by how an asthmatic defined a BAD. For example, when a BAD was defined as a day with any symptoms, the WTP estimate was $9 (1984 U.S. dollars). At the higher end of the scale, when a BAD was defined as a day with

more than moderate symptoms, the WTP was $41 (1984 U.S. dollars). A central estimate is $25 (1984 U.S. dollars). We follow Krupnick and Kopp and adopt these WTP estimates converting them to their 1996 Canadian dollars equivalents of $17 for the low estimate, $46 for the central, and $75 for the high by multiplying the original values by the 1984 PPP index value of 1.25 and then inflating using the Canadian consumer price index values of 92.4 for 1984 and 135.7 for 1996.

5.4.8 Minor Restricted Activity Day

There are no studies specifically addressing the WTP to avoid a minor restricted activity day (MRAD). For WTP estimates, we generally follow the approach taken by Krupnick and Kopp (1988). They relied primarily on WTP estimates obtained in three studies of symptoms: Loehman et al. (1979), Tolley et al. (1986a), and Berger et al. (1987). In each of these studies, survey respondents were asked how much they would be willing to pay to avoid a day with various specified symptoms such as serious or minor coughing. The focus of these studies was on respiratory symptoms that might be related to air pollution levels. The results from these studies are difficult to interpret for this analysis because there is a fairly wide variability in the responses and the definitions of symptoms. However, Krupnick and Kopp note that an MRAD must be more severe than a single symptom day (congestion, cough, etc.); hence, they concentrate on the WTP estimates for severe symptoms in Loehman and symptom combinations in Tolley. Moreover, Krupnick and Kopp argue, convincingly, that a MRAD must be valued less than a work-loss day where one is entirely unable to work due to illness.

Krupnick and Kopp selected a low estimate of $11 (1984 U.S. dollars), which is based on the median estimate of Loehman s severe symptom day. For a central estimate, they select $18 (1984 U.S. dollars), which is Loehman s high value for a severe symptom day. Krupnick and Kopp s high value, $31 (1984 U.S. dollars), is based on Tolley s median estimate for a symptom combination. These values are incorporated into AQVM 3.0 after converting to the equivalent 1996 Canadian dollars by first multiplying by the PPP index value of 1.25 for 1984 and then inflating using the Canadian consumer price index values of 92.4 for 1984 and 135.7 for 1996. The low, central, and high values are thus $20, $33, and $57, respectively.

5.4.9 Acute Respiratory Symptom Days

Krupnick et al. (1990) estimated the number of study subjects who reported any respiratory symptoms on a given day as a function of air pollutant levels on that day. These included 19 specific symptoms such as coughing, congestion, and throat irritation. The symptoms were noticeable to the subjects, but did not necessarily result in any changes in the person s activities on that day. This health effect therefore includes but is not limited to restricted activity days. In the procedures used to add the health effects cases, restricted activity days are subtracted from acute respiratory symptom days because of the overlap in the definitions of these health effects.

The monetary valuation required for acute respiratory days is therefore a value for the days on which symptoms are noticeable but do not restrict normal activities for that day.

Loehman et al. (1979) and Tolley et al. (1986a) obtained estimates of WTP to avoid a day with a single minor respiratory symptom such as head congestion or coughing. Their median results per day range from $4 to $12 (1984 U.S. dollars). We prefer the median results from these studies because neither study did any adjusting for potentially inaccurate high WTP responses, resulting in reported mean WTP estimates that far exceed the median values. The medians may be too low relative to the average WTP that we would prefer to use in this analysis, but there is less risk of significant upward bias in the median estimates from these studies. We prefer to err in this direction. We select $8 (1984 U.S. dollars) as typical of the range of estimates obtained in these two studies for minor respiratory symptoms. We select these three values $4, $8, and $12 (1984 U.S. dollars) to serve as the starting point for the low, central, and high values used in this study. The equivalent 1996 Canadian dollars were determined by first multiplying the U.S. values by the 1984 PPP index value of 1.25 and then inflating using the Canadian consumer price index values of 92.4 for 1984 and 135.7 for 1996. The low, central, and high values were thus $7, $15, and $22 respectively.

5.5 VALUATION FUNCTIONS FOR NONHEALTH ENVIRONMENTAL BENEFITS

The scientific data available to derive the concentration-response function for environmental benefits, including visibility, materials soiling, materials damage, agricultural damage, and recreational fishing, are limited in comparison to data available for functions derived above for health benefits. Given these limited data, the criteria for study selection must be relaxed, and fewer studies are used to derive these functions. Furthermore, concentration-response and economic valuation are usually melded into a single valuation function. As the first implementation of nonhealth, or environmental, benefits in a policy tool in Canada, further refinement of the functions included in AQVM 3.0 is expected as more data become available from upcoming scientific assessments on ozone and acid deposition.

5.5.1 Visibility Aesthetics Damages

Society has long recognized that there is a value to preserving visibility. In the United States, section 169A of the Clean Air Act, added in 1977, establishes a national goal of both remedying and preventing in major national parks and wilderness areas visibility impairment caused by human activity. Visibility is also considered a welfare effect in setting secondary national ambient air quality standards for urban and rural areas. In Canada, however, pollution standards have been based solely on point-of-impingement concentrations, not on supplemental air quality parameters such as visibility (Stuart and Hoff, 1993). Because Canadian law does not recognize visibility as an air quality parameter, we must rely on scientific and economic data for the United States to develop a value for visibility in AQVM 3.0.

Visibility impairment is caused by light scattering and absorption by particulate matter (aerosols) and nitrogen dioxide (NO_2) gas. Fine particles less than 2.5 m in diameter are the most effective at scattering light. Fine particles such as sulphate and nitrate aerosols that are hygroscopic (have liquid water associated with them) are especially effective at scattering light. This means in eastern Canada, for example, there is lower visibility during periods of high relative humidity (Stuart and Hoff, 1993). In this analysis, visibility is measured in terms of visual range, which is defined as the farthest distance at which a large black object is perceptible through haze. Visual range is inversely proportional to light extinction of particles (Chapter 4 of Rowe et al., 1995).

Visual range can be related to changes in PM_{10} concentrations by determining the fraction of PM_{10} below 2.5 m in diameter and applying the appropriate light extinction coefficients (see Rowe et al., 1995, for an evaluation for power plant emissions). Because of the great range of visibility levels and particle types in Canada, a relationship between changes in PM_{10} concentrations and visual range has not been included in AQVM 3.0. Users must enter the actual percentage change in visual range or the percentage change from baseline visual range as estimated by Stuart and Hoff (1993). Policies that change PM_{10} concentrations in given regions will probably change visual range in the same regions. Therefore, AQVM 3.0 users are advised to link visual range and PM_{10} and input an appropriate change in visual range when running AQVM 3.0.

Visibility benefits assessments to date indicate that visibility aesthetic benefits of air pollution control can be substantial. This section draws on and updates the literature review for the United States National Acid Precipitation Assessment Program (NAPAP) in Chestnut and Rowe (1990a) on the economic valuation of changes in visibility. Based on the methods in the NAPAP review, general visibility value functions are developed for use values for visibility in residential settings where people live, work, and recreate. Values for visibility changes in Canadian national parks are not considered because of a lack of literature for a benefits transfer. In the United States, such values have been estimated to be significant in some locations (Chestnut and Rowe, 1990b).

Although the method and estimates are based on a substantial amount of previous research, there are still important uncertainties in the visibility damage function method. The method and selected values are primarily based on results from contingent valuation studies. There are important issues and uncertainties about the validity of those results, and about extrapolating the results to small changes in visibility and applying United States values to Canada. However, based on information available at this time, there is no clear direction of bias introduced through these uncertainties. The omission of potential visibility values for special locations such as national parks and wilderness areas represents a potential downward bias in AQVM 3.0.

Visibility Benefit Categories

Visibility has a value to people primarily through its effect on the viewing activities of consumers. Consumer values for changes in regional haze can be divided into active use and passive (or nonuse) values. Active use values are related to the direct effect of experiencing

various visibility conditions on the individual s well-being. Passive values are the values an individual holds for protecting visibility for use by others (bequest value), for indirect use such as viewing in pictures and movies, and for knowing that it is being protected regardless of current or future use (existence value).

For this analysis, we further separate visibility effects in terms of residential and recreational settings. Residential settings include urban, suburban, and rural areas where people live, work, and participate in everyday recreation such as ball games, walking, and picnics. We define recreational settings as major state and federal recreational sites such as state and national parks and wilderness areas. Therefore, for the purposes of reviewing existing literature, we address the following categories of benefits:

> ***residential active use values*** related to effects on individuals at work, home, and recreation near their home
>
> ***residential passive values*** related to effects on other individuals, or purely for the sake of improved visibility
>
> ***recreational active use values*** related to expected effects when one visits a major recreational site such as a national park or wilderness area
>
> ***recreational passive values*** related to bequest and existence values for visibility conditions at major recreational sites.

Based on available empirical literature, Chestnut and Rowe (1990a) indicate that residential active use values probably account for more than half of all values for changes in visibility due to regional haze in the eastern United States. This is because most people spend most of their work and recreation time near their homes and because of the substantial numbers of individuals affected by visibility changes in residential settings (as defined above).

Chestnut and Rowe (1990a) also indicate that recreational passive values tied to bequest and existence value motives are likely to exceed recreational active use values (as defined above). If a large number of individuals hold even small passive values for visibility at these sites, such values can exceed on-site use values when summed across the total affected population. However, these values are based on surveys for U.S. national parks and there is not enough evidence to transfer these survey results to Canada at this time. Therefore, recreational active use and passive values are not included in AQVM 3.0.

Finally, there is little evidence to suggest that residential passive values are significant, or to estimate such values. Therefore, residential passive values are omitted from the model.

Perception Threshold Issues

A change in visibility must be perceptible to the affected individual if he or she is to place some value on that change. Current estimates suggest that a change in visual range must be at least 10% to 20% to be perceptible to the human observer (Trijonis et al., 1990). However, it is not so obvious how small changes in visibility conditions should be treated in a benefits analysis. Some changes, especially when measured in seasonal or annual averages, may not exceed perception thresholds, and it may therefore be asserted that they have no value. This conclusion has two problems.

The first problem is whether or not a change is interpreted as perceptible may depend on the averaging time used to measure the change. It is possible that a given change in emissions could result in a perceptible change in visibility on some days and affect well-being on those days, but when these changes are averaged over a season or a year, the change appears to be below the perception threshold and may be incorrectly treated as having no value. Carson et al. (1990) found that a share of respondents to a visibility valuation survey gave positive, nonzero WTP responses for perceptible visibility improvements that would occur on only three days a year in a residential area.

The second problem is that although emissions from one facility may not cause perceptible changes in visibility on any day, they may still contribute to perceptible visibility degradation when combined with emissions from other sources in the vicinity. The danger here is that by examining the question of visibility source by source we may find that no one source creates a perceptible change, but when all sources are combined the effect may be quite perceptible.

For AQVM 3.0, no visibility perception threshold is incorporated into the model. The primary reason for this decision is that each source that contributes some additional amount of fine particles in Canada is contributing some incremental amount to the overall visibility degradation that exists. This is the case even if the incremental emissions from that source alone are not sufficient to cause a perceptible change.

Available information indicates that significant and perceptible visibility degradation due to anthropogenic air pollutants occurs throughout Canada and the United States. Current summer daytime visibility for three locations in eastern Canada ranges from 19 to 43 km (Stuart and Hoff, 1993). Trijonis et al. estimate that if the only visibility degrading particles in the atmosphere in the eastern United States were those that would be expected from natural sources, average visual range on days with typical humidity, but without precipitation or fog, would be 90 km, plus or minus 40 km. This suggests a very substantial level of visibility degradation due to anthropogenic emissions under current conditions in the eastern United States. Because of long-range transport of the particles responsible for visibility degradation, such degradation is a widespread regional phenomenon in the eastern United States, and presumably throughout Canada (Trijonis et al., 1990).

Contingent Valuation Method Issues

Most quantitative information about visibility values available at this time is based on results obtained using the contingent valuation method (CVM). The CVM involves using surveys to ask respondents how much they would be willing to pay for specified changes in visibility conditions. It is a technique designed to obtain estimates of WTP for goods and services such as visibility for which direct prices are not revealed in the market. Without this technique, many such nonmarket goods and services remain unvalued in quantitative analyses. This technique has been widely reviewed and debated (e.g., Cummings et al., 1986; Mitchell and Carson, 1989), and many application and interpretation issues remain unresolved. There are analyses of the importance of air quality, including visibility, to property value that found results consistent with the CVM studies (Chestnut and Rowe, 1990a).

Residential Active Use Values

Three CVM studies have estimated use values for changes in visibility in residential urban areas in the United States. McClelland et al. (1991) estimated values for Atlanta and Chicago; Tolley et al. (1986b) estimated values for Chicago, Boston, Washington, DC, Atlanta, Cincinnati, Mobile, and Miami. Rae (1983) estimated values for Cincinnati, and several studies in California cities are also discussed.

The Two Cities Study by McClelland et al. (1991) makes some important contributions to the literature in the area of CVM and visibility valuation by addressing some of the criticisms of the previous research. We therefore place considerable emphasis on the results of this study, particularly given that the results are generally consistent with or lower than results from previous studies.

A mail survey was conducted in 1990 for the Two Cities Study in Chicago and Atlanta with about 500 completed responses. Respondents were shown photographs illustrating three different air quality levels in their area and were told how many days per year each level currently occurs on average. Respondents were asked what their household would be willing to pay per year to have air quality on 25 of the worst days improve to the best air quality level shown. This amounted to about a 14% improvement in average annual visual range. Respondents were asked to say what percentage of their response was attributable to concern about health effects, soiling, visibility, or other air quality impact. The average response was about $300 (1996 Canadian dollars, rounded to the nearest $5) per year, with about 18% attributed by respondents to visibility.

The authors conducted two analyses and adjustments on the responses. One was to estimate and eliminate the potential selection bias in WTP estimates due to nonresponse to the WTP questions by some respondents (including what has been called protest responses). The other was to account for the potential skewed distribution of errors due to the skewed distribution of responses (a long tail at the high end). Both of these adjustments caused the mean WTP estimate to

decrease. The annual average household value for visibility was $54 before the adjustments. The adjustment for the potentially skewed distribution of errors brought the mean WTP to about $35. The adjustment for nonresponse to the WTP question reduced the mean WTP further from $35 to $25 (all values in 1996 Canadian dollars). The authors interpreted the adjustments as providing a lower bound on the "true" WTP value. The analysis of the WTP responses also found that income, education, and age were significant in predicting WTP responses. No statistically significant differences were found between the two cities, although different scenes (specific to each city) were used in the photographs.

The Two Cities Study makes an important contribution to addressing the potential for upward bias that may result if respondents mix health and other benefits of pollution reduction with values for visibility improvements when they answer the WTP questions. This has been a significant criticism of the ambitious Six Cities Study (Tolley et al., 1986b), which undermined the credibility of the Six Cities results and led some analysts to the conclusion that the Six Cities results are probably too high (Chestnut and Rowe, 1990a). Previous work by some of the same researchers who conducted the Two Cities Study (Irwin et al., 1990) has demonstrated that some respondents are unable to abstract from the other effects of air pollution when answering questions about visibility. The approach taken in the Two Cities Study was therefore to ask for a total value for changes in air quality and then ask the respondent to allocate this value among the different air pollution effects that might concern him or her, including visibility. This is similar to an approach recommended by Carson et al. (1990) in which respondents are asked to value changes in air quality that represent various combinations of health and visibility impacts. Although the mechanics of the Carson et al. approach are different, the thought process required by the respondent is similar. In both cases, the degree to which respondents are embedding values for health and other nonvisibility concerns in their responses for visibility is greatly reduced.

It is difficult to compare the results for the different valuation studies because they are for different changes in visibility. In an effort to examine for consistent values and patterns across studies, Chestnut and Rowe (1990a) used the following function to put the mean WTP results from the different studies into a common framework:

$$HHWTP_i/year = b \times \ln(VR2_i/VR1_i) \quad (5\text{-}11a)$$

$$TVISD_i/year = b \times \ln(VR2_i/VR1_i) \times HH_i\ , \quad (5\text{-}11b)$$

where:

$HHWTP_i$/year	=	annual WTP per household in area i for visibility changes in that year [For $VR_2 > VR_1$, HHWTP/year is positive, or there are benefits. For $VR_1 > VR_2$ (visibility degradation), HHWTP/year is negative, or there are damages.]
$TVISD_i$/year	=	total annual WTP for visibility changes in area i for residents of area i
$VR1_i$	=	starting annual average visual range
$VR2_i$	=	annual average visual range after the change in emissions

ln	=	natural log
b	=	estimated coefficient
HH_i	=	households in the affected area i.

This function implies that WTP is constant for a given percentage change in visual range and that WTP is zero when there is no change in visual range. Chestnut and Rowe (1990a) selected this function because it is simple and consistent with results of perceptions studies that suggest percentage changes in visibility measures are a good way to characterize an individual s perceptions of visual air quality, but other functional forms are also plausible. This function takes into account differences in starting and ending levels of visual range and is also consistent with the economic assumption of diminishing marginal utility for visibility enhancement.

Fitting all available estimates of WTP for changes in visibility conditions in residential areas into Equation 5-11a requires putting the changes in visibility conditions valued in each study into the same numeric terms. Chestnut and Rowe (1990a) selected the change in annual average visual range because most of the studies presented respondents with changes in annual conditions, defined either as a change in the annual average or a change in the distribution of good, fair, and poor visibility days. Chestnut and Rowe report that there is no definitive evidence that the presentation of a distribution rather than an average level consistently results in higher or lower WTP responses. Evidence on this is limited, however, because of the relatively small number of studies completed to date.

Verification of the validity of this functional form is difficult given the limited number of observations available from studies conducted to date. Most studies have estimated WTP for one or two different scenarios of change in visibility conditions. Comparisons of results across studies are limited in that there are usually several significant differences in study design, and each difference has the potential to cause differences in the results. The area of greatest uncertainty is for small changes in visibility as the function approaches the intercept. Most studies have estimated values for fairly substantial changes in visibility, and few have considered both very small and very large changes within the same study.

One study, by Carson et al. (1990), in Cincinnati found positive but decreasing WTP values as the visibility change decreased, but specific dollar results were not reported. Another pilot study considered both small and large changes in annual average visual range; Balson et al. (1990) estimated WTP for changes in visibility at the Grand Canyon, including scenarios for changes on many days throughout the year and for changes on just a few days a year. In terms of the implicit changes in annual average visual range for each scenario, they span changes of close to 70% to changes of only a few percent.

Table 5-8 summarises the results of visibility valuation studies that are relevant to estimating residential use values for changes in visibility. This is an update of a similar table provided by Chestnut and Rowe (1990a), who also provide a detailed discussion of all of these studies, which is not repeated here. What is most critical for our current purposes is to assess how the

Table 5-8. Comparison of Residential Visibility Valuation Study Results

Study	City	Mean WTP (1996 $, Adjusted)	Starting VR (miles)	Ending VR (miles)	b Coefficient	WTP for 20% VR
[illegible]	Atlanta	[illegible]	17.6	20	[illegible]	[illegible]
[illegible]	Chicago	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	Chicago	[illegible]	[illegible]	[illegible]	508	$93
[illegible]	Atlanta	[illegible]	[illegible]	[illegible]	574	$105
[illegible]	Boston	[illegible]	[illegible]	[illegible]	515	$94
[illegible]	Mobile	[illegible]	[illegible]	[illegible]	381	$69
[illegible]	Washington, DC	[illegible]	[illegible]	[illegible]	776	$141
[illegible]	Cincinnati	[illegible]	[illegible]	[illegible]	147	$26
[illegible]	Miami	[illegible]	[illegible]	[illegible]	313	$57

Comparison of Residential Visibility Valuation Study Results

Study	City	Mean WTP (1990$)	Starting VR (miles)[b]	Ending VR (miles)[b]	b Coefficient	WTP for 20% Change in VR[c]
	Cincinnati	$269	11.47575	16.45025	740	$135
	Farmington	-$180 / $125		12	277	$51
	Los Angeles	$159	2	2828	145	$26
	San Francisco	$407 / $223 / $253	2 12			
	Los Angeles		18.6	16.3	1624	$295
	San Francisco	$351	16.3	18.6		$299 / $600 / $675

McClelland et al. (1991) results compare to results of previous studies. A comparison of the b coefficients (as defined in Equation 5-11a) reported in Table 5-8 shows how the fully adjusted b coefficient from the Two Cities Study is at the low end of the Six Cities results, which actually varied considerably across the different cities. This result is somewhat higher than the Brookshire et al. (1979) result for Los Angeles.

The last study in Table 5-8, Trijonis et al. (1984), is a property value study conducted in Los Angeles and San Francisco that provides estimates of WTP for better air quality at the residence location, based on differences in property values statistically attributed to differences in visual air quality for the residence locations in the sample. The implied results for a 20% improvement in visual range substantially exceed the CVM results in most cases. This is consistent with the expectation that the property value results will reflect values for all aspects of air quality, including concerns about health as well as visibility. These results, based on a different estimation technique, using real market data, are reasonably consistent with the Two Cities results. The Two Cities results found that, on average, respondents attributed about 18% of their total WTP for changes in air quality to visibility which suggest a household annual WTP for a 20% change in visual range, based on the fully adjusted estimate, of about $200 (1996 Canadian dollars, rounded to the nearest $5) when all air quality concerns are included. This limited evidence suggests that there is no substantial upward bias in the CVM results.

Selected Quantification Approach for Residential Active Use Values in AQVM 3.0

In AQVM 3.0, we must use residential active use values estimated for the United States because similar values are not available for Canada. The original U.S. WTP values were converted to their Canadian equivalents from 1990 U.S. dollar baselines by multiplying by the 1990 PPP index value of 1.22 and then inflating using the Canadian CPI values of 119.5 for 1990 and 135.7 for 1996. Equation 5-11a was then used to calculate a corresponding beta, b, value. We select the b coefficient value of 195 (rounded to the nearest 5) based on the fully adjusted McClelland et al. WTP result of $25 (1996 Canadian dollars), for calculating the central estimate of residential use values for Canada. This study was well-designed and addressed many of the criticisms raised about previous contingent valuation studies for residential use values related to visibility. Less weight should be given to the Tolley et al. results, which have been widely criticized for several significant study design flaws, most specifically that health effects values may be embedded in the visibility values, leading to potential upward bias in the results.

We select a low estimate of b equal to 145 (rounded to the nearest 5) based on a pooling of the Brookshire et al. Los Angeles study results which has the lowest average WTP estimate obtained in any of the studies to date in residential areas with baseline visibility conditions at all comparable to New York. We select a high estimate of b equal to 275 (rounded to the nearest 5) based on the partially adjusted McClelland et al. WTP results of $35 (1996 Canadian dollars). This partial adjustment accounts for the skewed distribution of responses that has troubled some analysts in terms of questioning the credibility of the high responses at the tail of the

distribution, but does not include the estimated values for those who were not willing to give a WTP response.

Table 5-9 illustrates what these selected estimates imply about the average annual residential active use values per household for 5% and 10% improvements in average visual range. These values are illustrative. The value function (Equations 5-11a, and 5-11b) can be used to compute values for any percentage improvement in average visual range. We give equal probability weights the central, low, and high estimates reflecting the uncertainties in visibility valuation.

Table 5-9
Selected Residential Active Use Value Estimates (C$1996)

	Low	Central	High
b coefficient	145	195	275
Probability weights	33%	34%	33%
Implied Annual Average Household WTP:			
5% improvement	$7	$10	$13
10% improvement	$14	$19	$26

5.5.2 Materials Damages from Particulate Matter and Sulphur Dioxide

Materials damage caused by air pollution is recognized as a potential source of economic loss (Horst et al., 1983; Rowe et al., 1986; Baedecker et al., 1990; Brown and Callaway, 1990). However, defensible quantitative estimates of economic effects are available for only a limited number of the types of materials damages that are suspected, and the bulk of these studies are located in the United States. Therefore, the fundamental assumption for materials damages is that Canadian households have similar materials in them and similar cleaning habits as U.S. households. This assumption may over- or under-state actual damages in Canada; however, there is not sufficient evidence to determine a bias in either direction.

Because much of the economics literature on this topic is somewhat dated the materials damage estimates are best interpreted as providing an indication of the potential order of magnitude of damages. Chamber and field studies have demonstrated that materials damage caused by air pollution can take a number of forms, including soiling of exposed surfaces; surface erosion, blistering, and discolouration of paint; corrosion and tarnishing of structural metals and electronic components; fading, soiling, and reduction of the tensile strength of fabrics; and soiling and spalling of stone building materials and monuments. All of these effects occur to some extent in unpolluted atmospheres from natural environmental conditions such as moisture, temperature, and wind fluctuations; atmospheric oxygen concentrations; sunlight; and the activity

of microorganisms. But the presence of air pollutants, especially particulate matter (PM), SO_2, and acidic deposition, appears to aggravate or accelerate these processes.

Chamber and field data on physical damage are not sufficient for estimating economic losses. Information is also needed on the amount and location of such material and on the monetary value individuals place on preventing such damage. Monetary estimates of value are sometimes based on the additional maintenance such as more frequent painting that would be needed to offset the damage from air pollutants. An important question regarding economic estimates of losses developed in this way is the extent to which maintenance schedules really are influenced by damage due to air pollution. For example, anecdotal evidence has been reported that suggests that chain link fences are sometimes replaced for reasons unrelated to corrosion and at shorter intervals than would be required by significant corrosion due to air pollution (Brown and Callaway, 1990).

The estimates of economic effects due to materials damage from air pollutants presented here include household cleaning and maintenance expenditures associated with PM and SO_2, and maintenance cost estimates for galvanized steel based on SO_2 damage functions. Potentially important categories of materials damage that are not quantified, because adequate quantitative information is not available, include PM soiling in the commercial and industrial sectors, soiling and damage to stone buildings and monuments, and damage to exposed painted surfaces outside the household sector.

Particulate Matter and Materials Soiling

Anthropogenic PM, both emitted directly and formed in the atmosphere from gaseous precursors, gradually settles on all exposed surfaces and causes soiling. Available studies providing estimates of the economic effects of such soiling are limited to the household sector. Such soiling in the commercial and industrial sectors may also be associated with significant economic impact, but the information necessary to quantify damages other than in the household sector is not available. This damage category applies to any source that directly emits particulate matter or that emits gases that contribute to the formation of particles in the atmosphere (SO_2, NO_x, VOC).

Five studies have provided estimates of economic effects to households from PM soiling (Cummings et al., 1981; Manuel et al., 1982; Watson and Jaksch, 1982; Gilbert 1985; McClelland et al., 1991). Each of these studies has important limitations, but as a whole they provide a useful range of estimates for quantifying this effect.

Four of the studies are based on analyses of household cleaning costs associated with different ambient levels of PM near the residence. This summary relies, in part, on a review and comparison of the results of three of these studies provided by Horst et al. (1983). The fifth is a contingent valuation study that asks respondents for willingness to pay estimates for reductions in various air pollution effects (McClelland et al., 1991).

Of the four studies of household cleaning costs, the most sophisticated analyses were conducted by Manuel et al. (1982) and by Gilbert (1985). These studies developed soiling damage estimates for households based on an analysis of household expenditures as a function of differences in ambient PM levels. The analysis relied on a household production function approach in which the household combines market goods and household technology to produce desired services such as cleanliness. PM enters the model by shifting the cost functions the household faces in producing desired services. The model was estimated with data from the 1972-1973 Bureau of Labor Statistics Consumer Expenditure Survey for more than 20 metropolitan areas across the United States. The strength of this analysis is that it is based on actual expenditures made by households, and therefore reflects actual maintenance and cleaning practices as well as implicitly accounting for the physical effects of the pollutants through the cost functions.

Manuel et al. (1982) included 13 categories of household expenditures in the model. The PM measure, total suspended particulates (TSP), was found to be statistically significant in two expenditure categories: laundry and cleaning, and utilities (e.g., electric). The authors hypothesized that utility expenditures may be related to electric appliance use for cleaning. The household expenditure categories found to be related to TSP or SO_2 were statistically significant in different expenditure functions, suggesting that the effects of each pollutant are separable. An important limitation of the Manuel et al. study is that the model did not include the time cost incurred for do-it-yourselfers, because the Consumer Expenditure Survey did not include these data. This may be a significant omission when it comes to household cleaning and suggests a potentially important downward bias in this study s estimates of household soiling effects due to PM. The results of this study have been used in several benefits analyses in the United States concerning alternative PM standards, including the 1983 analysis of mobile source diesel particulate standards (Horst et al. 1983), and the 1988 Regulatory Impact Analysis conducted by the U.S. EPA for SO_2 (1988).

The original 1982 Mathtech (Manuel et al. 1982) report gives results in terms of changes in the second high 24-hour TSP measure in each location. Subsequent modifications of the Mathtech model changed the pollution measure to the annual average TSP measure. Results based on the revised Mathtech model reported by the U.S. EPA (1988) that include human morbidity and PM soiling damages averaged $3.21 (1984 U.S. dollars) per person per year for a 1 g/m^3 change in annual average TSP. The soiling component of this was not reported separately. We contacted Robert Horst, the primary author of this analysis, and asked if the soiling component was available separately. He provided estimates based on an analysis of alternative coke plant emissions standards being conducted in 1992 for the U.S. EPA. The comparable estimate of morbidity and soiling effects was $3.70 (1990 U.S. dollars), which has an equivalent 1996 Canadian dollar equivalent of $5.13 after multiplying by the 1990 PPP index value of 1.22 and inflating using the Canadian CPI values of 119.5 for 1990 and 135.7 for 1996. The soiling portion of this total value was reported to be $0.48 1990 U.S. dollars, equivalent to $0.67 1996 Canadian dollars following the above procedure, or about 13% of the total reported value. This is an average annual benefit per person for a 1 g/m^3 change in annual average TSP. Presuming an average household size of 2.63 individuals (Statistics Canada, 1995) this translates to $1.75 1996

Canadian dollars (rounded to the nearest $0.05) per household annually per g/m^3 change in annual average TSP.

Gilbert (1985) examined both short-term and long-term adjustments by the household to differences in air pollution levels. The short-term analysis was similar to the Manuel et al. study in its theoretical approach and data, although the specifics of the model were different. The long-term adjustments considered the possibility that households might eventually change residences in response to changes in pollution levels and relied on results of previous hedonic property value studies. The benefits of a reduction in TSP levels were much larger when based on the long-term adjustment model, although the short-term benefits were much smaller than the Manuel et al. results. The results are reported in terms of changes in annual second high 24-hour TSP levels. Assuming that these are roughly three times the annual average levels (based on the ratios of the standards for these different averaging times) and adjusting the 1992 dollars, the Gilbert results suggest annual household benefits per g/m^3 annual average TSP of US$0.01 to US$0.02 in the short term and US$1.00 to US$30.00 in the long term. Neither of these estimates includes the value of household time. This very wide range of results is a bit difficult to interpret. They do highlight the uncertainty in the estimates for this benefits category. Even though the household production approach is based on actual behaviour and expenditures, the model is complex and difficult to estimate and the results are apparently quite variable depending on the exact specification of the model.

Cummings et al. (1981) and Watson and Jaksch (1982) conducted separate analyses using the same underlying database. The data were from a 1970 survey of households in the Philadelphia area concerning the frequency of different household cleaning tasks. Cummings et al. added household labour cost for do-it-yourselfers and estimated a fairly simplistic model of cleaning costs as a function of annual average TSP levels. The results indicate annual cleaning costs per household of $6.63 (1980 U.S. dollars) per g/m^3 change in annual average TSP. This is equivalent to $15.24 (1996 Canadian dollars) after first inflating to 1983 U.S. dollars using the U.S. CPI values of 82.4 for 1980 and 99.6 for 1983, second converting to the equivalent Canadian value with the 1983 PPP index value of 1.24, and finally inflating using the Canadian CPI values of 88.5 for 1983 and 135.7 for 1996. This is about eight and a half times the results from the Mathtech model. The model estimated by Watson and Jaksch (1982) was more like the Mathtech model in that it focused on the value households place on certain activities rather than direct pollution damage functions. Horst et al. (1983) compared the results of all three models applied to the same change in TSP levels and found that the Watson and Jaksch model predicted household soiling damages closer to, but smaller than that predicted by the Cummings et al. model. Results using the Watson and Jaksch model were about five times the results using the Mathtech model.

Horst et al. (1983) note some concerns about the models estimated by Cummings et al. and by Watson and Jaksch, including the limited geographic variability of the data and the not entirely satisfactory characterization of the household cleaning behaviour process. Nonetheless, the results of all three studies suggest significant economic effects to households from PM soiling.

The Mathtech results are probably downwardly biased because of the exclusion of the value of time for do-it-yourselfers, but it is not clear that the bias is as much as a factor of five to eight and a half, as suggested by the results of the other two studies.

A fifth study of a different type was reviewed to see if the results suggest where between the previous estimates a realistic central estimate might fall. The Two Cities study (McClelland et al., 1991) was discussed in Section 5.6 concerning visibility. This study obtained WTP estimates by households for changes in air quality in Chicago and Atlanta. Respondents were asked to allocate their annual WTP for air quality improvements across three categories of potential air pollution effects: human health, visibility, and soiling and other damages to materials. Respondents were asked to give WTP for about a 15% change in visual air quality, and the WTP component for the soiling and materials damages category amounted to about $21 (1990 U.S. dollars), equivalent to $29.09 (1996 Canadian dollars) using the same adjustment procedure described for the Mathtech results, per household per year. If we make some assumptions in interpreting this estimate, we can derive a rough dollars per $\mu g/m^3$ estimate for household soiling. Typical annual average TSP levels in eastern United States cities are in the range of 50 to 60 $\mu g/m^3$. A 15% change is therefore roughly 8 $\mu g/m^3$. If we apply the $29.09 entirely to soiling concerns we get an annual average figure of about $3.64 (1996 Canadian dollars) per $\mu g/m^3$, or about twice the Mathtech estimate. Because of the uncertainties in the underlying assumptions, this estimate is not precise, but it gives an idea of the order of magnitude implied by the McClelland et al. results regarding potential household soiling damages for TSP, and provides supporting evidence for the use of the expenditure study results in this assessment.

It is reasonable to assume that soiling damage is proportional to the mass of the particles, regardless of the size of the particles. Thus, we apply the per $\mu g/m^3$ estimates for TSP to the estimated change in PM_{10} in this analysis without any adjustment. This is in contrast to the assumption used in the health effects section, which was that all the impact was caused by the PM_{10} portion, an assumption that required the adjustment of health effects coefficients estimated for changes in TSP to obtain a coefficient applicable to PM_{10}. This means that the resulting estimates of soiling associated with PM_{10} are caused by the PM_{10} fraction only and do not reflect any soiling damage that may result if changes in the ambient levels of larger particles also occur.

The Mathtech annual estimate of $1.75 (1996 Canadian dollars) per household per $\mu g/m^3$ is selected as the low estimate. It is plausible that at least half of the costs of household cleaning are for the time value of do-it-yourselfers, which was not included in the Mathtech analysis. A central value of $3.50 (1996 Canadian dollars) per household per $\mu g/m^3$ is therefore selected. This central estimate is consistent with the McClelland et al. (1991) results. An upper estimate $8.75 (1996 Canadian dollars) is selected as five times the Mathtech estimate based on the Watson and Jaksch (1982) results. The central, low, and high estimates are all assigned equal probability weights for the uncertainty analysis. The calculation procedure is as follows:

$$S_i = a \times HH_i \times \quad PM_{10i}\ , \qquad (5\text{-}12)$$

where:

S_i = annual soiling damage at location i
HH_i = number of households (population/2.6) in location i
PM_{10i} = change in annual average PM_{10} in g/m^3 at location i
a = low = \$1.75 33%
central = \$3.50 34%
high = \$8.75 33%.

SO_2 and Household Materials Damage

SO_2 gas is a byproduct of fossil fuel combustion. Dry deposition of SO_2 can be adsorbed directly to a damp or wet material surface, where it converts to sulphuric acid. Direct absorption of dry deposition to material surfaces is a more complicated and less well-documented process. Baedecker et al. (1990) cite numerous studies in which SO_2 exposure is correlated with corrosion of exposed metal surfaces, specifically galvanized steel. Baedecker et al. also cite evidence that paint coatings degrade more rapidly in the presence of SO_2. Rowe et al. (1995) cite evidence that SO_2 is associated with increased rates of degradation in textiles and leather. This section provides quantitative economic effects estimates of general materials damage for households and increased rates of corrosion for galvanized steel materials due to SO_2.

Mathtech (Manuel et al., 1982) found statistically significant relationships between SO_2 levels and household expenditures for materials repair, textiles, and fuel for transportation. The authors note that the first two associations are consistent with evidence regarding the kinds of SO_2 damage observed in chamber and field studies, but that the association with transportation expenses is less intuitive. It is plausible, however, that higher transportation costs are associated with higher levels of household maintenance expenditures. One limitation of the household expenditures model is that the causal linkages are implicit and not directly revealed in the results. As a whole, however, the Mathtech results are reasonably consistent with *a priori* expectations in terms of the types of expenditures associated with each pollutant in the analysis.

Mathtech does not report average household damages per unit of SO_2, but Harrison et al. (1993) report damage estimates per unit of SO_2 that were calculated by the authors of the Mathtech study using the household expenditures model for use in the Harrison et al. externalities analysis. The estimates were calculated at various levels of SO_2, and were found to be relatively linear with respect to a reasonable range of pollutant levels. The results suggest an average annual materials damage of \$0.90 (1992 U.S. dollars) per household for each g/m^3 of SO_2. We select this as an estimate for use in this analysis, inflated to \$1.17 (1996 Canadian dollars) using the 1992 PPP index value of 1.23 and the Canadian CPI values of 128.1 for 1992 and 135.7 for 1996, per household per g/m^3 of ambient SO_2 in the low, central, and high estimates of materials damage due to SO_2.

SO_2 Damage to Galvanized Steel Materials

Concentration-response functions for corrosion rates of galvanized steel when exposed to SO_2 are fairly well established (Baedecker et al., 1990). This section presents estimates of economic damage associated with corrosion of galvanized steel, based on studies that have estimated galvanized steel inventories in the United States and estimated increased maintenance costs expected as a result of higher corrosion rates. An important uncertainty in these economic estimates is that actual changes in maintenance costs as a function of corrosion rates have not been empirically verified. We interpret available economic damage estimates as upper bound estimates because they may overstate the responses actually made to higher rates of corrosion.

Galvanized steel has many different uses. Table 5-10 lists major categories of galvanized steel use and the available studies that provide estimates of economic damages due to SO_2-caused corrosion. For use in this analysis, we select estimates provided by Mathtech (1983), because they cover four important uses of galvanized steel and the results are reported in such a way that average per capita damages per g/m^3 of ambient SO_2 can be derived. A second Mathtech study (1985) estimated damage to several types of building materials, including galvanized steel, but the report does not provide damage estimates for individual materials in a way that allows these results to be extrapolated.

Table 5-10
Studies Providing Estimates of Damages of Ambient SO_2 on Galvanized Steel Materials, by Inventory Component

Galvanized Steel Inventory Component	Source for Estimates of Damage
Transmission towers	Mathtech (1983; 1985)
Bridges	Mathtech (1983)
Galvanized wire	Mathtech (1983)
Chain link fencing	Mathtech (1983)
Building uses in urban areas	Horst et al. (1986)
Metal structures (rural)	None
Highway guard rails (rural)	None

Mathtech (1983) developed estimates of the benefits from expected SO_2 emissions reductions for urban areas in six states: Indiana, Michigan, New York, Ohio, Pennsylvania, and West Virginia. Benefits were calculated based on the controls expected to be placed on the sources of SO_2 emissions in these states, using the assumption that the percentage reduction in SO_2 emissions would lead to the same percentage improvement in the ambient level of SO_2 in each state. The estimated reductions in SO_2 emissions and ambient concentrations ranged from 13% in New York to 60% in Indiana. Benefits were calculated based on an inventory of steel bridges (highway and railroad), transmission towers, galvanized wire, and chain-link fences. A damage function approach was used, in which benefits are presumed to derive from reduced maintenance requirements as a result of the reduced pollutant levels.

Mathtech combined a concentration-response function for corrosion with inventories of galvanized steel components. The effects of different levels of SO_2 exposures on the galvanized steel surfaces of the different components were simulated using a corrosion concentration-response function for galvanized steel. A critical maintenance threshold for mitigating corrosion damage for each inventory component was determined. Estimates of reduced corrosion rates and longer maintenance cycles between critical maintenance efforts were then developed for simulated SO_2 exposures. This information was then used to estimate the difference in the present value of the maintenance costs (i.e, economic benefits) at different levels of pollution for each component.

A limitation of this study approach is that it assumes that actual maintenance practices for galvanized steel materials are sensitive to pollution damage. It is possible, however, that other factors dominate maintenance practices. A small survey of users indicated that chain link fence is sometimes replaced before it reaches a critical maintenance threshold due to corrosion for a variety of reasons unrelated to corrosion (Brown and Callaway, 1990). This is an interesting result that should be investigated further, but the small sample of users for a single component of the galvanized steel inventory does not provide an adequate basis for generalizing about the importance of this issue.

The Mathtech study results are reported in Table 5-11 on a per unit of inventory basis. We use New York as a proxy for Canada; the total annual benefit estimate for New York reported in the study was US$36.48 million (1983 dollars). The reduction in average ambient SO_2 for New York was 4.53 g/m^3 (from 34.84 g/m^3 to 30.31 g/m^3 on an annual average basis). Thus, the estimate implies an average of US$8.05 million per g/m^3 per year. If one assumes that galvanized steel materials are distributed spatially in proportion to the population, then one can use an estimate of urban households in 1980 in New York (roughly 16.016 million urban population divided by 2.7 persons per household) to calculate an average value per household per g/m^3 of ambient SO_2. Accordingly, the estimated average annual value is US$1.36 per g/m^3 per household (1983 dollars). That is, for each 1 g/m^3 increase in ambient SO_2, anywhere in the state of New York, the resulting value of the damages to galvanized steel is US$1.36 per household per year (1983 dollars), or $2.60 per household per year in 1996 Canadian dollars after

adjusting by respective the 1983 PPP index value of 1.24, inflating with the Canadian CPI values of 88.5 for 1983 and 135.7 for 1996, and rounding to the nearest $0.05.

Table 5-11
Annual Benefits of Reduced Materials Damage due to Local Reductions in SO_2 Concentrations in Six States

State	Percentage Reduction in SO_2	Benefits (1983 U.S. dollars)			
		Towers ($/tower)	Wire ($/ton)	Fence ($/ton)	Total (10^6)
Indiana	60%	$11.59	$20.55	$19.72	$40.82
Michigan	20	3.38	6.26	6	22.98
New York	13	2.43	4.38	4.21	36.48
Ohio	58	12.31	21.14	20.27	97.88
Pennsylvania	38	7.89	13.7	13.13	69.93
West Virginia	56	12.33	20.92	20.06	9.07

Source: Mathtech, 1983.

We expect that the galvanized steel estimate for the materials components included in the Mathtech study represents an upper bound because of the possibility that maintenance practices are not fully sensitive to pollution damage. We include the C$2.60 household estimate of damages to galvanized steel materials in the high estimate for SO_2 materials damage, we include half that amount, $1.30, in the central estimate, and we include zero damages in the low estimate.

SO_2 Materials Damage Estimates for AQVM 3.0

As a result of the SO_2-related household expenditures and galvanized steel material damages being mutually exclusive endpoints the low, central, and high estimates from each endpoint are combined and then rounded to the nearest $0.05. As a result, the final low household SO_2 materials damage estimate is $1.20 ($1.17 + $0.00) with central and high estimates of $2.50 ($1.17 + 1.30) and $3.80 ($1.17 + $2.60) (all values in 1996 Canadian dollars) respectively.

In AQVM 3.0, SO_2 materials damages are calculated as follows:

$$M_i = b \times HH_i \times \quad SO_{2i}\,, \tag{5-13}$$

where:

M_i = annual SO_2 materials damage at location i
b = annual SO_2 materials damage per household per g/m^3 SO_2:
low = \$1.20 33%
central = \$2.50 34%
high = \$3.80 33%
HH_i = number of households (population/2.6) in location i
SO_{2i} = change in annual average SO_2 in g/m^3 at location i.

5.5.3 Agriculture Damages from Exposure to Ozone

This section reports the methods used for valuing benefits of reduced impacts of ozone to commercial crops in Canada during the summer season. The method is based on well established crop loss literature for the United States and data on crop production and pricing from Canada. However, several important crops have been omitted (canola and potatoes) because of the lack of data on ozone sensitivity for these crops. These omissions should be remedied in the next version of the AQVM, when more information on these crops will be available from upcoming ozone studies. The general method used here should be applicable to those crops.

The most important agricultural products in Canada are shown below with their average annual value in parentheses (Agriculture Canada, 1996):

grains and oilseeds (C\$7.5 billion)
beef (C\$3.6 billion)
dairy (C\$3.5 billion)
forages (C\$2.2 billion)
hogs (C\$1.9 billion)
poultry (C\$1.8 billion)
horticulture (C\$1.3 billion)
others (C\$1 billion).

Of these important agricultural products, we focus this analysis on the plant derived products (grain and oilseeds, forages, and horticulture) whose sensitivity to exposure to ambient ozone is documented. There is a large amount of literature documenting reduced crop yields as a result of exposure to elevated ambient ozone levels, and one of the most extensive studies was completed as part of U.S. EPA s National Crop Loss Analysis Network (NCLAN) program. Exposure-response experiments were carried out on cultivars of 14 crops by NCLAN scientists over a 7-year period. The NCLAN experiments involved fumigating crops in field chambers 7 or 12 hours per day at different ozone concentrations (from ambient to three times ambient levels). The ozone levels were then measured and transformed into seasonal average exposures for each

experimental treatment. The functional correspondence between seasonal doses and yields was then estimated and reported in Heagle et al. (1988).

To apply these results to Canada, we first examined the most important crops in Canada. Canada s 10 most important horticulture crops based on farming operation receipts in 1994 were:

wheat excluding durum
canola
floriculture and nursery
vegetables
potatoes
corn for grain
soybeans
barley
durum wheat
tobacco.

The most important crops vary widely by province, but these crops comprise almost 70% of the total horticulture crops products in Canada. Of these crops, the following were included in the NCLAN study: wheat, corn, soybeans, and tobacco. Forage products including hay and alfalfa were also included in the NCLAN study, however, insufficient data were available to include baseline production and price data for the products in AQVM 3.0.

Estimates of crop yield losses at various ozone levels for these six crops are shown in Table 5-12. The actual NCLAN concentration-response functions are in many cases complex and nonlinear. To allow implementation of these functions in AQVM 3.0, we assume simple linear concentration-response coefficients (for a 1 ppb change in ozone) between the range of 30 and 50 ppb that approximate the more complex nonlinear concentration-response functions. This assumption probably will overstate benefits in the range of 30 ppb and understate benefits in the range of 50 ppb. However, this assumption is consistent with the limited data accessible at this time and the initial version of the AQVM. We expect that this assumption and set of data will be improved in subsequent versions.

Table 5-12
Estimates of Yield Losses for Six Crops from Various Ozone Levels (%)[a]

Crop	Mean Ozone Concentration ppb			
	30	40	50	60
Corn	0	1.7	3.7	6.7
Soybean	3	5.5	10	15.3
Wheat	3	9	15	20.8
Hay (alfalfa)		5	8	11.5

Hay (other hay)		6.7	12.7	20
Tobacco			5	9

a. Ozone is measured as June-September, 9 a.m.-9 p.m. hourly average. Yield losses are measured against an assumed background ozone level of 25 ppb.

Source: Heagle et al., 1988.

The coefficients for the concentration-response functions for the six crops included in AQVM 3.0 are shown in Table 5-13. The valuation process in AQVM 3.0 and application of these concentration-response coefficients is outlined in the following three steps:

Table 5-13
Concentration-Response Coefficients for AQVM 3.0 Agriculture Endpoints (percentage reduction in yield for 1 ppb increase in ozone)[a]

Crop	Low	Central	High
Corn	0.00	0.15	0.2
Soybean	0.1	0.3	0.45
Wheat	0.2	0.3	0.6
Hay (alfalfa)	0.2	0.3	0.6
Hay (other hay)	0.3	0.6	1.2
Tobacco	0.2	0.4	0.8
Weight	33%	34%	33%

a. 1 ppb change in June-September, 9 a.m -9 p.m. ozone. Linearised dose response relationships over 30-50 ppb.

Source: Heagle et al., 1988.

Step 1: Estimate Change in Yield for Crop

For a given average seasonal ozone change in a province (as input into AQVM 3.0), the percentage change in yield is calculated by multiplying the change in ambient ozone by the concentration-response coefficient as shown in Equation 5-14.

$$\%\ \text{Yield}_{i,j} = a \times \text{S12-O}_{3j}\ , \qquad (5\text{-}14)$$

where:

$\text{Yield}_{i,j}$ = % change in yield of crop i in province j
a = concentration-response coefficient from Table 5-12
S12-O_{3j} = change in the seasonal 12-hour (9 am-9 pm) average ozone levels in site j.

Step 2: Estimate Change in Production

The change in production for each crop is estimated by multiplying the change in yield calculated in Equation 5-14 by the average production of that crop in each province as shown in Equation 5-15. Baseline crop production by province is provided in Table 3-4 of Chapter 3. Because there were not sufficient data for forage products (hay and alfalfa) these benefits are zero.

$$\text{Yield}_{i,j} = \%\quad \text{Yield}_{i,j} \times \text{Average Production}_{i,j}\ , \tag{5-15}$$

where:

$\text{Yield}_{i,j}$ = change in production of crop i in province j
$\text{Average Production}_{i,j}$ = average production of crop i in province j.

Step 3: Estimate Economic Value

The economic value of the change in production (or benefit) is estimated by multiplying the change in production calculated in Equation 5-15 by the average price of the crop in that province as shown in Equation 5-16. Price data by province is found in Table 3-4 of Chapter 3.

$$\text{Benefits}_{i,j} = \quad \text{Yield}_{i,j} \times \text{Average Price}_{i,j}\ , \tag{5-16}$$

where:

$\text{Benefits}_{i,j}$ = $ for crop i in province j
$\text{Average Price}_{i,j}$ = average price for crop i in province j.

The benefits are then aggregated across the four crops within each province (some may be zero) and totalled nationally to estimate total benefit. The central estimate of benefits reported for crops is the result using the central estimate for the concentration-response function shown in Table 5-11. The high and low estimate for benefits is equal to the 20th and 80th percentile of a distribution using the low, central, and high estimates and the corresponding weights for the concentration-response functions in the statistical uncertainty analysis (see Chapter 6).

Issues

There are several issues associated with the method employed here. First and most important, the assessment omits commercially important crops that may be sensitive to changes in ambient ozone concentrations. This should be an important area for further investigation in future revisions to the AQVM.

Second, changes in ozone that change crop yields may result in farm and market responses. For example, with reduced ozone, increased crop production may result in reduced market prices to balance increased supply with demand. Farmers may react by switching among crops to reduce the impact on profits. The reverse holds for the impacts of increased ozone. Thus, the method used in this assessment of assuming no change in cropping patterns and no changes in market prices will overstate benefits (damages) of ozone reductions (increases). More elaborate models have been developed that model farm and market responses, and measure the appropriate economic surplus measures of damage associated with changes in ambient ozone (for example, see Adams et al., 1989 and Rowe and Chestnut, 1985). For large changes in ozone in important agricultural regions such as the San Joaquin Valley of California, using the simplified approach here can overstate damages by as much as 50% (Rowe and Chestnut, 1985). However, for small ozone changes and thus small production changes, or for localized ozone changes that only affect a subset of the agricultural production regions, the overestimate of damages is much less likely to result in significant changes in cropping patterns or market prices and the bias introduced by the simplified approach used is expected to be small.

A third issue is that the method used omits any consideration to agricultural subsidies. Changes in yields could affect subsidies, or other farm support programs, if and to the degree they exist for the impacted crops. However, unless the change in ozone is so large as to cause substantive changes in production, the bias introduced by omitting these considerations may be small.

Fourth, AQVM 3.0 estimates crop production changes for an entire province, rather than portions of a province, and are based on monitors that are often located in urban areas. This approach is used because production data is not readily available on a sub-province level. This feature of the analysis is likely to introduce error in the computations because of increased inaccuracies by assigning average baseline ozone and average changes in ozone to the entire province.

5.5.4 Recreational Fishing and Acid Deposition

In this section we develop and apply a method to compute recreational fishing damages resulting from changes in acid deposition in the rivers and lakes of Ontario and Quebec. The results presented here should be interpreted as providing a preliminary indication of the potential magnitude of damages because of limitations in the literature available for this task. In the following paragraphs we first present background information, describe the study area relevant for the assessment, and introduce the method used. Second, we compute economic damages in terms of a dollar per affected angler-day per 1% change in acid deposition. We then apply these figures to estimates of affected angler-days to develop an economic damage function for use in AQVM 3.0.

Assessment Method

Figure 5-2
Simplified Linkages between Acid Deposition Precursor Emissions and Recreational Fishing Damages

The linkages between changes in acid deposition precursor emissions and any resulting changes in recreational fishing damages are illustrated in Figure 5-2. Acid deposition is the result of precursor emissions of SO_2, NO_x, and VOCs, and chemical reactions that occur in the environment. Acid deposition precursors can be transported long distances from a source location and distributed over a large area. AQVM 3.0 does not model emission transport and acid deposition; it requires the model user to provide the percentage changes in acid deposition as an input variable to the assessment. Therefore, it is the relationship between deposition and fishing damages that we address here.

Acid deposition can alter lake chemistry (e.g., cause changes in pH levels and the concentration of metals); the degree to which depends on many factors that can vary from lake to lake. Changes in water chemistry can alter the suitability of the water body for fisheries (either through direct effects on the fish or indirect changes in habitat and food quality). These changes can result in the reduction or elimination of some species at some locations.

Fewer numbers of fish and fish species at fishing locations can result in reductions in the catch per unit effort at a site (CPUE). This in turn can result in less enjoyment at a site or additional time and travel costs to travel to alternative, and perhaps less desirable, sites (i.e., sites that would not otherwise have been selected if the target site had not been affected by acid deposition). These damages to recreational anglers are measured through changes in consumer surplus (CS), a standard economic measure of value.

A number of Canadian studies have examined the scientific linkages between acid deposition, changes in water chemistry, and changes in aquatic biota. Many of these studies were undertaken in the late 1980s and were prompted by government initiatives to better understand the effects of acid deposition.

Important studies included Kelso et al. (1986), Matuszek and Beggs (1988), Kelso et al. (1990), Matuszek, Goodier, and Wales (1990), RMCC (1990), and Kelso and Johnson (1991). The Kelso and Johnson (1991) study also provided a useful summary of some of the Canadian research that had taken place to that time.

In the United States, a significant amount of scientific information was compiled in the late 1980s and early 1990s for the National Acid Precipitation Assessment Program (NAPAP). Information of potential relevance to fisheries was reported in the NAPAP (1990) volume on aquatic processes and effects. Much of the information reported through NAPAP focused on effects in the Adirondacks in the northeastern United States. However, in some cases the reports included in NAPAP also compiled information from scientific studies performed in other geographic areas, including Canada. Additional analyses of acid deposition impacts to fisheries and recreational fishing in the northeastern United States have been presented in Englin and Kealy (1990), Morey and Shaw (1990), NAPAP (1991), and Rowe et al. (1995, Chapter 14).

We defined the AQVM 3.0 study area based on information regarding estimated baseline deposition loads and threshold (or target) loads at which adverse impacts may be expected to occur. RMCC (1990) presents estimates of median wet SO_4^{2-} deposition for each of the Canadian tertiary watershed aggregates (AGs) as defined by LWG (1989). Only seven AGs, in Ontario and Quebec, have an estimated median deposition load close to or above the target load of 20 kg/ha/yr wet SO_4^{2-} deposition. Changes in deposition from a baseline that is below the target load are not expected to yield incremental impacts on recreational fishing (RMCC 1990). Because there are no other AGs in Canada that have estimated median deposition loads close to or above the target load, these seven AGs define the areas in which small changes in deposition may lead to recreational fishing impacts.

Our assessment method is to develop three alternative sets of estimates, based on available scientific and economic studies, of the economic damages related to changes in acid deposition in Ontario and Quebec. While each of the alternatives has relative strengths and limitations, combined they provide a relatively consistent picture of damages.

Our first alternative is to estimate the relationship between acid deposition and economic damages using the results of a study by Talhelm et al. (1987). This is the only identified study that directly relates changes in acid deposition to changes in recreational fishing damages in Ontario and Quebec. This direct relationship eliminates the need for, and the compounding uncertainties inherent in, estimating each of the scientific and economic linkages in Figure 5-2. However, the Talhelm study is relatively old and does not use methods entirely consistent with the current state of the art for recreation valuation. Use of this study also requires the assignment of economic values to the change in fishing activity.

In our second alternative, we estimate the relationship between acid deposition and economic damages using the results of Englin et al. (1991). This study was performed for NAPAP; it directly relates changes in acid deposition to changes in recreational fishing damages in the northeastern United States (New York, New Hampshire, Maine, and Vermont). Using this study also eliminates the need to address each of the linkages in Figure 5-2. However, differences between northeastern U.S. deposition, water bodies, fish species, and recreational

patterns and values add uncertainty when applying the results of this study to Ontario and Quebec.

Our third alternative is to combine available data from Canadian scientific studies and Canadian economic studies for each of the steps in Figure 5-2 between deposition and economic damages. The advantage of this alternative is that it relies on information specific to Canada. However, many of these studies were not designed to perform the linkages required for this analysis, and we are required to make many simplifying assumptions. The combination of these studies therefore results in potentially significant compounded uncertainty in the estimates.

Alternative 1: Estimates of Change in Consumer Surplus per Angler-Day from a Change in Acid Deposition Based on Direct Relationship between Acid Deposition and Economic Damages Using Talhelm et al.

Talhelm et al. (1987) estimated the percentage change in consumer surplus from recreational fishing that would result from a percentage change in acid deposition. The study area consisted of 232 lakes in the Haliburton-Muskoka region of Ontario, about 200 km north of Toronto. This area is appropriate since it lies within the group of AGs with estimated median deposition loads close to or greater than the target load. These AGs are therefore subject to incremental impacts.

This study used the product travel cost approach, which estimates changes in welfare from changes in the costs of purchasing alternative recreational fishing products (i.e., fishing activities of various kinds). Changes in the costs of recreational fishing may result from changes in environmental quality or resource management regimes. The findings of the study suggested that a 5% increase in annual hydrogen loads from acid deposition would lead to a 0.2% decrease in aggregate annual consumer surplus from recreational fishing.

To apply the results of the Talhelm study, we assume that the relationship between change in acid deposition and change in consumer surplus is symmetric and linear in the range around the magnitude the authors examined. Specifically, we assume that the study findings (a 0.2% decrease in annual consumer surplus as a result of a 5% increase in annual acid deposition) can be scaled in linear fashion for use in the smaller percentage change scenarios that may be of interest to AQVM 3.0 users. A 1% increase (decrease) in annual acid deposition in watershed AGs close to or above the target load is accordingly assumed to result in a 0.04% (0.2%/5) decrease (increase) in annual consumer surplus from recreational fishing.

Significant uncertainty is associated with the estimate of change in consumer surplus produced by the Talhelm study. This is due largely to uncertainties in modelling the effect of acid rain on freshwater lakes and hence the attributes of lakes (including fisheries productivity) that are important to anglers. In discussing the acid rain simulation model used in their study, the authors rightly acknowledge that as is well known, there is a high degree of uncertainty about rate of lake acidification (Talhelm et al., 1987; p. 428). To reflect such uncertainty, we characterize the Talhelm result as running from 50% to 150% of their point estimate, that is, indicating that a 1%

change in annual acid deposition yields a percentage change in annual consumer surplus of between 0.02% and 0.06%. This provides a better indication of the uncertainty associated with the scientific and economic linkages.

To translate the estimated change in consumer surplus from percentage terms to dollars per angling occasion (day or trip), it is necessary to estimate baseline consumer surplus per occasion. The *1990 Survey of Recreational Fishing in Canada* (Department of Fisheries and Oceans, 1994) presents, by province, the averages of the additional costs that active anglers indicated they were willing to pay per day for fishing (Table 36). This provides one measure, based on the particular survey questions used and the specific samples of anglers surveyed, of mean consumer surplus per angler-day. For Quebec and Ontario, the averages were approximately $14 (1990 Canadian dollars), or approximately $16 (1996 Canadian dollars) after inflating using the Canadian CPI values of 119.5 for 1990 and 135.7 for 1996 and rounding to the nearest dollar. This value, however, appears low relative to the results of several studies on the value of cold water recreational fishing. For example, Walsh et al. (1990) report a substantially higher median value of approximately US$28 per day (1987 dollars), or about $45 per day (1996 Canadian dollars) after converting using the 1987 PPP index value of 1.25 and inflating with the Canadian CPI values of 104.4 for 1987 and 135.7 for 1996 then rounding to the nearest dollar, for cold water fishing.[15]

We use the value suggested by the data in the *1990 Survey of Recreational Fishing in Canada* ($16 per angler-day) as a low estimate of consumer surplus per day. For the central estimate, we use the approximate average, $31, of the value from the survey ($16) and the (converted) median value reported by Walsh et al., $45. For the high estimate, we use the median value of $45 from Walsh et al. Combining these per-day values with the above low, central, and high estimates for percentage change in consumer surplus results in an Alternative 1 estimate ranging from $0.003 to $0.03 change (all values in 1996 Canadian dollars) in CS per angler-day for a 1% change in acid deposition.[16]

Alternative 2: Estimates of Change in Consumer Surplus per Angler-Day from a Change in Acid Deposition Based on Direct Relationship between Acid Deposition and Economic Damages Using Englin et al.

Englin et al. (1991) report estimates of recreational fishing damages (and benefits) from changes in acid deposition in the northeastern United States (New York, New Hampshire, Maine, and

15. This median value from Walsh et al. was based on 39 estimates from the literature on the value of cold water recreational fishing.

16. Note that the low (high) estimate of change in per-day consumer surplus was derived by combining the low (high) estimates of both baseline daily consumer surplus and the linkage between change in acid deposition and recreational fishing values. This results in relatively large differences between the low, central, and high estimates. This method was used deliberately to reflect the substantial uncertainty associated with the estimation of changes in per-day values resulting from changes in acid deposition.

Vermont) that provide a point of comparison to the estimates developed under Alternative 1 above. They estimated changes for two NAPAP deposition reduction scenarios as well as for two sensitivity analysis scenarios, and used two different models (a hedonic travel cost model and a random utility model) to develop two sets of estimates for each scenario. For our purposes, the two sensitivity analysis scenarios, a 50% reduction and a 30% increase in acid deposition loads, are most relevant. We adapt the Englin et al. results based on their random utility model, which uses more defensible methods than those used in the hedonic travel cost model (see Rowe et al., 1995).

For a 1% change in acid deposition, the Englin et al. results indicate a mean change in consumer s surplus per trip of $0.03/per trip (1996 Canadian dollars). To compare this result to those of Alternative 1, we assume an average trip length ranging from one to two days. Using Alternative 2, the estimates range from $0.015 to $0.03 (1996 Canadian dollars) change in CS per angler-day for a 1% change in acid deposition.[17]

Alternative 3: Estimates of Change in Consumer Surplus per Angler-Day from a Change in Acid Deposition Based on Combining Canadian Scientific Studies with Canadian Economic Studies

For this alternative, we first must specify the change in CPUE that is expected to result from a 1% change in acid deposition. This involves specifying (1) an expected change in water chemistry as a result of a change in acid deposition, and (2) then the expected change in CPUE or fish abundance resulting from that change in water chemistry. Both of these linkages are uncertain and may vary substantially across different lakes.

To develop a relationship between acid deposition and water chemistry, we refer to results from the application of a dynamic water chemistry model in the Turkey Lakes Watershed of central Ontario (RMCC 1990, p. 4-105). This model was used to assess the long-term trends under various deposition scenarios. The application compared, among other simulations, estimated pH levels for a 27.8 SO_4^{2-} kg/ha/yr scenario with those for a 15.8 kg/ha/yr scenario. The results indicated that the latter scenario would yield pH levels 0.8 higher (less acidic) than the former scenario. In the terms relevant to our application, this means an increase of 0.8 pH for a 43% decline in acid deposition. Assuming for simplicity that pH is a linear function of acid deposition in the range covered by the above model, the results imply an increase of 0.019 pH for a 1% decline in acid deposition (0.8/43 = 0.019).

Very little information exists to allow for the estimation of the *incremental* effects, as opposed to threshold effects, of a change in mean pH on sportfish abundance or CPUE. Scientific studies have focused more on the relationship between pH and aquatic species richness than on the

17. Englin et al. also report total damages for all anglers for a 50% reduction in acid deposition in the northeastern United States of $20.7 million (1994 Canadian dollars), whereas the central estimate derived from the Talhelm et al. analysis is about $13.4 million (1994 Canadian dollars).

relationship between pH and either recreational catch rates or measures of species abundance. A number of studies have also estimated the critical pH levels at which particular aquatic species experience adverse effects. Experimental formats include bioassays and field surveys, as well as whole lake experiments in which lakes are intentionally acidified so that the resulting fish population losses can be assessed. Summaries of such studies are provided in RMCC (1990) for Canada and NAPAP (1990) for the United States and elsewhere.

To develop a relationship between incremental changes in mean pH and changes in CPUE, we consider the results of an analysis performed by Kelso et al. (1986). The authors regressed catch per sampling effort on pH levels in headwater lakes in eastern Canada.[18] The results of that analysis for Ontario lakes indicated that an increase of 1 pH was associated with a 74% increase in sampling catch per effort. This result can be combined with the findings of the dynamic water chemistry model (described above) to yield an estimate of a 1.4% increase in sampling catch per effort as a result of a 1% decline in acid deposition [(0.019 in pH per 1% in load) × (74% in CPUE per pH)]. Lacking better information, we assume that this would also result in a 1.4% increase in recreational angler CPUE. That is, we assume that CPUE is proportional to measured abundance of fish.

The final linkage in the chain of effects involves the influence of CPUE on CS. Adamowicz et al. (1994) estimate that a 10% increase in fishing catch rate results in an average benefit ranging from $0.11 to $1.80 per trip (1994 Canadian dollars), equivalent to $0.11 to $1.87 (1996 Canadian dollars) adjusting using the 1994 CPI of 130.7 and the 1996 CPI of 135.7, depending on what type of model is used. Their preferred model, a joint model that uses both stated and revealed preference information, results in an estimate of $0.47 per trip following the adjustment to 1996 Canadian dollars. Peters et al. (1995) use a standard random utility model to estimate that a 10% change in CPUE would yield a change in welfare of $0.11 per trip (adjusted to 1996 Canadian dollars with the CPI from original 1994 value). If we use the joint model estimate from Adamowicz et al. and assume that CS is linear in CPUE, then a 1% decrease in acid deposition would lead to an increased CS equal to ($0.47/trip per 10% increase in CPUE) × (1.4% increase in CPUE per 1% decline in deposition), which equals $0.07 per trip for a 1% decline in acid deposition. The same calculation using the Peters et al. result yields approximately $0.02 per trip for a 1% decline in acid deposition. Thus a range of $0.02 to $0.07 (1996 Canadian dollars) per trip is indicated by applying the results of these studies.

To convert the per-trip values to per-day values, we use data in Department of Fisheries and Oceans (1994) that suggests the average trip length for freshwater angling in Ontario and Quebec ranges from one to two days. Therefore, the Alternative 3 estimates range from $0.01

18. Note that the measure used was catch per scientific sampling effort, rather than catch per unit time by recreational anglers. Catch was defined as total number of fish caught per 24 hours, for all species.

($0.02/2 days) to $0.07 ($0.07/1 day)[19] (1996 Canadian dollars) change in CS per angler-day for a 1% change in acid deposition.

Comparison of Results from Alternatives 1, 2, and 3

To summarise, the alternatives above have resulted in the following estimates for change in CS per angler-day from a 1% change in acid deposition:

Alternative 1: $0.003 to $0.03 per angler-day
Alternative 2: $0.015 to $0.03 per angler-day
Alternative 3: $0.01 to $0.07 per angler-day (all values in 1996 Canadian dollars).

To reflect the substantial amount of uncertainty and intrinsic variation in the scientific and economic linkages between acid deposition and recreational fishing values, we incorporate the entire range of values above in AQVM 3.0. This results in low, central, and high estimates for the effect on consumer surplus from a 1% change in acid deposition of $0.003, $0.03, and $0.07 (1996 Canadian dollars) per angler-day, respectively.

Estimated Number of Annual Freshwater Angler-Days

The above computations apply to all freshwater angler-days in the affected region. To estimate *total* changes in annual CS due to changes in acid deposition loads, it is necessary to estimate the number of affected freshwater angler-days per year in the areas (AGs) of interest. This estimation is discussed below.

The number of freshwater angler-days in the relevant area is a subset of the total number of freshwater angler-days fished by active anglers in Ontario and Quebec, and specifically excludes Great Lakes fishing. There is uncertainty associated with this quantity given the different levels of spatial aggregation at which acid rain deposition data and angler participation data are collected. We have used data from the *1990 Survey of Recreational Fishing in Canada* in combination with additional information supplied by staff of the Department of Fisheries and Oceans. Our low, central, and high estimates of the number of annual angler-days at rivers and lakes in the Ontario and Quebec AGs of interest are, respectively, 11.19 million, 22.38 million, and 33.58 million angler-days.

Load-Response Function

The AQVM 3.0 procedure for calculating the change in aggregate annual recreational fishing values due to incremental changes in acid deposition follows from the information and assumptions presented above. The equation is as follows:

19. Note that the high estimate of $0.07 (1996 Canadian dollars) per angler-day results from using the extreme high estimate of CS per trip and the low estimate of days per trip.

$$CS_{RF} = b \times AD \times \%\ \ LOAD\ , \qquad (5\text{-}17)$$

where:

CS_{RF} = change in aggregate annual consumer surplus from recreational fishing in those watershed aggregates at or above the target load of 20 kg/ha/yr (Ontario and Quebec)

b = change in per-day consumer surplus from recreational fishing due to a 1% change in acid deposition:

low	=	$0.003 (C$1996)	33%
central	=	$0.03	34%
high	=	$0.07	33%

AD = number of freshwater angler-days in those watershed aggregates at or above the target load of 20 kg/ha/yr:

low	=	11.19 million	33%
central	=	22.38 million	34%
high	=	33.58 million	33%

% LOAD = *percentage* change in annual acid deposition in those watershed aggregates at or above the target load of 20 kg/ha/yr. (A 1% change is entered in AQVM 3.0 as 0.01, not 1.0.)

5.5.5 Global Climate Change and Greenhouse Gases

Changes in the emission of greenhouse gases (GHGs) may result in global climate change and impacts to the ecology and society. Some of the impacts associated with climate change include (Watson et al., 1996):

coastal defence from sea level rise
dryland loss
wetland loss
species loss
agricultural loss
forestry loss
fishery losses
energy (hydroelectric) losses
water resource shifts
amenity

life/morbidity
air pollution
migration
natural hazards.

While there are case studies of potential damages for some of these endpoints, and other literature on the potential overall damages possible for varying climate change scenarios (as well as mitigation cost estimates), we conclude that at this time there is insufficient literature to develop damage function estimates for GHGs that should be relied on as the default value in AQVM 3.0. Therefore, we do not propose or endorse any specific values for GHGs and the AQVM 3.0 defaults to a $0 value per ton of GHG emissions.

To investigate the sensitivity of the benefits analysis to the incorporation of GHGs, AQVM 3.0 allows users to input the change in GHGs emissions (in tonnes) and to input alternative values per equivalent tonne of carbon ($/tC). For example, Rowe et al. (1996a) show that when using GHG values on the order of $10/ton, as suggested in some literature (and by some policy makers), GHG emissions can be one of the more significant benefit categories for some electric power plant air emission control scenarios. Therefore, the ability to conduct sensitivity analysis for GHGs provides potentially important computational support in air quality benefit analyses.

While we do not endorse any GHG value per tonne, a recent paper by Fankhauser and Pearce (1993) provides a synthesis of recent estimates of the social costs of CO_2 emissions and reflects the types of values used by some planning agencies. These values may be a useful starting point for sensitivity analyses on the potential significance of GHGs in air quality benefits analyses.

The bulk of the quantitative and scientific assessments on the effects of global climate change use a scenario in which the atmospheric concentration of carbon dioxide is doubled ($2\times CO_2$). The consensus among scientists now is that $2\times CO_2$ will cause an increase in the global mean temperature of 1.5°C to 4.5°C (Fankhauser and Pearce, 1993). Regional and seasonal deviations from these global averages are likely to be considerable, but little is known about these yet. The economic studies that have estimated expected damages due to doubling of CO_2 emissions roughly agree on the overall result of 1% to 2% of gross national product (GNP) for developed countries. The results are expected to be twice that for developing countries (Fankhauser and Pearce, 1993). In translating the results of the economic studies into a damage per ton of emissions value, Fankhauser and Pearce considered the damages of GHG emissions through time because GHG are stock pollutants; that is, damages are not caused by the flow of emissions but by their accumulation in the atmosphere. The results of their analysis are shown in Table 5-14.

The benchmark estimated by Fankhauser and Pearce is that CO_2 emissions impose social costs of $20/tC for emissions in 1990, a value that rises over time to about $28/tC by 2030. For other GHGs such as methane (CH_4) and nitrous oxide (N_2O), the social costs are related to the costs for CO_2 by the global warming potential index and other parameters by Fankhauser and Pearce (1993). Their estimates of the social costs for these other GHGs are shown in Table 5-15.

Table 5-14
The Social Costs of CO_2 Emissions ($/tC)
as Calculated by Fankhauser and Pearce (1993)

Original Study Author(s)	1991-2000	2001-2010	2011-2020	2021-2030
Nordhaus (first study)	$7.3 ($0.3-$65.9)			
Ayres and Walter	$30-$35			
Nordhaus (second study)	$5.3	$6.8	$8.6	$10.0
Peck and Teisberg	$10-$12	$12-$14	$14-$18	$18-$22 ($3.4-$57.6)
CSERGE-Fankhauser	$20.4 ($6.3-$47.7)	$22.9 ($7.2-$53.8)	$25.4 ($8.1-$60.3)	$27.8 ($8.8-$66.2)

Note: Figures in parentheses denote 90% confidence intervals [except for Nordhaus (first study) which denote upper and lower brackets].

Table 5-15
The Social Costs of Methane and Nitrous Oxide
as Estimated by Fankhauser and Pearce (1993)

Gas	1991-2000	2001-2010	2011-2020	2021-2030
Methane ($/t$CH_4$)	$110 ($49-$213)	$132 ($59-$259)	$155 ($70-$297)	$179 ($79-$359)
Nitrous Oxide ($/t$N_2O$)	$2,940 ($800-$7,465)	$3,433 ($948-$8,749)	$3,925 ($1,090-$9,652)	$4,571 ($1,241-$11,419)

Note: Figures in parentheses denote 90% confidence intervals.

CHAPTER 6
UNCERTAINTY ANALYSIS IN AQVM 3.0

There are many uncertainties involved in developing benefit estimates for alternative air quality scenarios. For example, uncertainties arise from limitations in the scientific literature, and from variations in the results of studies that address the same air pollution effects or economic values. As a result, simply developing a "best estimate" of benefits fails to provide policy makers with important information about the uncertainties and limitations of the estimate.

Several approaches were used to qualitatively and quantitatively address these issues for AQVM 3.0. These approaches have evolved from methods developed by Rowe et al. (1995) and reflect the Sulphur in Gasoline Panel's input (Thurston et al., 1997b).

1. Qualitative summary tables of "Key Omissions, Biases, and Uncertainties" are provided for major components of the methods used in AQVM 3.0. These tables list the key sources of omissions, biases, and uncertainties and comment on the potential direction and magnitude of the error that may exist as a result of these in the final estimates. The tables are presented in Section 6.1.

2. Where possible, the uncertainties associated with selected components of the assessment are quantified. Distributions for the coefficients of most concentration-response and monetary valuation functions in AQVM 3.0 were selected from the literature. These distributions are described by low, central, and high values and associated probability weights. AQVM 3.0 uses Monte Carlo techniques to combine these parameter distributions and calculate the distribution of the resulting aggregate benefits estimates. Section 6.2 discusses the quantitative treatment of uncertainty in AQVM 3.0.

3. AQVM 3.0 is designed to support sensitivity analyses that explicitly examine the impact of changes in key parameter values and model assumptions. Suggestions and examples of sensitivity analyses to use with AQVM 3.0 are described in Section 6.3.

6.1 QUALITATIVE ANALYSIS OF UNCERTAINTY

For those components of the benefits analysis that do not lend themselves to quantitative uncertainty analysis, we list the key omissions, biases, and uncertainties (OBUs) in Tables 6-1 through 6-4. The primary goal of these tables is to identify and evaluate uncertainties that may affect the total benefits estimates, but that are not quantified in the benefits estimates produced by

Table 6-1
Key Omissions, Biases, and Uncertainties
Health Effects from PM_{10} and Ozone

Omissions/Biases/ Uncertainties	Effects on Estimates	Comments
Potential health effects not quantified	-	There is literature to suggest chronic and acute effects may occur in addition to those included in AQVM 3.0.
Concentration-response relationships	?	Uncertainty in the results of the original studies for many reasons, including (1) statistical association in epidemiology studies does not prove causation, (2) measurement error and averting behaviour could cause downward bias, (3) omitted confounding variables could cause upward bias.
Transfer of concentration-response relationships	?	Estimates are based on transfers across time and location. Possible unaccounted-for differences add uncertainty.
Adjustments of parameters for different pollution measures used in original studies	?	Many studies have been performed with air pollutant measures other than 24-hour PM_{10} or daily high-hour ozone. Conversions to these measures are approximate given the limited comonitoring or reporting of alternative measures in original studies.
Presumed linearity of concentration-response	?	The effect of assuming a constant risk per unit of PM_{10} is difficult to assess with available information. Error could occur in either direction.
Background threshold assumptions	+	Some evidence indicates that health effects occur below the current air quality objectives, but if impacts do not occur down to background pollution levels, benefits will be overstated. AQVM 3.0 allows some sensitivity analyses on this question.
Overall impact	?/-	No clear directional bias is dominant.

Note: + means the potential error is positive, - means it is negative, and ? means the direction of the potential error is not known.

Table 6-2
Key Omissions, Biases, and Uncertainties
Monetary Valuation of Human Health

Omissions/Biases/ Uncertainties	Effects on Estimates	Comments
Uncertainties in underlying WTP literature	?	Economic literature and methods to estimate WTP for changes in health risks are not complete in addressing the questions raised in valuing pollution-related health risks.
Conversion from U.S. currency to Canadian currency with purchasing power parity	?	Measures of value for health effects for U.S. citizens may not be the same as for Canadians because of cultural, social, or other reasons. Purchasing power parity does not capture these differences.
WTP/COI ratios for morbidity effects	?	COI does not capture all of the factors considered in WTP estimates for morbidity effects. Impact is minor here since not widely used in this analysis.
Using age adjusted VSL for acute exposure mortality risks	?	Direction of bias is not entirely clear: some biases would be downward (e.g., involuntary nature of exposure) while others would be upward (e.g., immediate vs. future risk). Limited evidence exists for age adjustment.
Acute and chronic disease values	?	Limited literature on the valuation of these endpoints provides for uncertainties about the values. Directional bias is not apparent.
Using age adjusted VSP for chronic exposure mortality risks	+/?	The nature of the risk is uncertain. But, because it is in the future, and may be of a few months or years, use of the VSL measures is uncertain or could overstate benefits.
Overall impact	?	No clear directional bias is identified.

Note: + means the potential error is positive, - means it is negative, and ? means the direction of the potential error is not known.

Table 6-3
Key Omissions, Biases, and Uncertainties Environmental Effects

Omissions/Biases/Uncertainties	Effects on Estimates	Comments
Omitted impacts	-	Forests, wildlife, water quality, and other resource impacts are omitted.
Visibility omission of recreation use and nonuse values and nonresident use values for residential	-	Some data are available from U.S. studies on recreational nonuse values, but could not be transferred to Canada.
Materials soiling omission of do-it-yourself labour time in low estimate	-	Low estimate most likely understates damages.
Crops and O_3 omissions of ozone sensitivity for canola and potatoes	-	Omitting these major crops could greatly understate benefits for ozone mitigation policies.
Materials damages omission of important parts of the materials inventory	-	This omission may be significant.
Visibility use of CVM studies for values	?	There is no clear direction of bias, if any.
Visibility functional form of value equations and no threshold assumption	?	Alternative assumptions may be used, but there is no established direction of bias.
Visibility use of visual range as the appropriate visibility indicator variable	?	There is no known direction of bias or magnitude of error. Colour and other variables may also be important.
Materials damages application of U.S. study results to Canada	?	No evidence exists to support direction or magnitude of bias.
Recreational fishing limited data on value of changes in recreational fishing in these areas	?	The direction of bias is unknown until more relevant Canadian studies are available.
Recreational fishing sensitivity of consumer surplus to lake acidification from one study	?	Only one U.S. study is available. Canadian research does not provide quantitative information at this time.
Materials soiling highly variable results from household production analysis	?	Estimates are uncertain.
Materials damages damage function assumed to be linear	?	Potential error is small for small changes in pollutant levels.
Crops and O_3 use of 1994 prices to estimate economic value of yield loss	?	For large changes in ozone across all of Canada, market prices could be affected.

Table 6-3 (cont.)
Key Omissions, Biases, and Uncertainties Environmental Effects

Omissions/Biases/Uncertainties	Effects on Estimates	Comments
Crops and O_3 use of seasonal average change in ozone to proxy 7-hour and 12-hour change	?/-	Summer season 7-hour and 12-hour change may exceed average. Probably small with seasonal average.
Crops and O_3 use of crop loss estimates for entire province rather than smaller area	?/+	Ozone will vary across province and some crops will be exposed to greater changes than others.
Materials damage SO_2 methodology for galvanized steel may not treat consumer behaviour realistically	+	Methodology omits substitution by consumers and that some replacement may be for reasons other than corrosion.
Crops and O_3 use of linear concentration-response function across range of 20-50 ppb	+/-	Use overstates benefits for low range of ambient levels and understates damages for high range.
Overall impact	-	No overriding bias is clear in the included benefits. Forest, vegetation, wildlife, water quality, and other resource impacts may exist but are omitted.
Note: + means the potential error is positive, - means it is negative, and ? means the direction of the potential error is not known.		

AQVM 3.0. Many of these uncertainties are related to assumptions required in the concentration-response and valuation methods. Each OBU listed is accompanied by an assessment of its potential effect on the quantitative estimates produced with AQVM 3.0, and comments on the analysis. The effect on estimates is indicated by a (+) for those OBUs that tend to overstate or increase the estimates, a (-) for those OBUs that tend to understate or decrease the estimate, or a (?) if the direction effect is unknown. We use the term bias here in the sense that it is sometimes clear that available literature overstates or understates the model parameters that we are trying to quantify. In this case, we can say something more than that the estimates are uncertain: we can say what the likely direction of error is.

Tables 6-1 and 6-2 include the key OBUs for the quantitative analysis and monetary valuation of health effects from PM_{10} and ozone. Points raised regarding PM_{10} generally apply to sulphates and $PM_{2.5}$ as well. Table 6-3 includes the OBUs for environmental effects. Table 6-4 includes additional OBUs related to quantitative analysis of health effects from carbon monoxide and air toxics. Few, if any, of the OBUs are so dominant and well established that they clearly lead to an overstatement or understatement of the benefits of air pollution. However, the omission of some

Table 6-4
Key Omissions, Biases, and Uncertainties
Health Effects of Air Toxics and Carbon Monoxide

Omissions/Biases/ Uncertainties	Effects on Estimates	Comments
Air toxics: noncarcinogenic effects are omitted	-	Noncarcinogenic effects are probably small when compared to cancer and may be partially captured in the particulate matter morbidity analysis.
Air toxics: unit risk factors for cancer overstate risks	+	Risk factors have a margin of safety that varies by chemical. Ingestion risk factors are assumed to equal inhalation risk factors except for benzene. All species of a chemical are assumed to have the same risk factors.
Air toxics: no consideration of cancer latency	+	Ignores discounting of future costs.
Air toxics: limited monitors	+/?	Monitor may be located in proximity to point or area sources and may not provide data that are representative of general concentrations and population exposures.
Air toxics overall impact	+	Upper bound risk factors are believed to be an overriding upward bias in the estimates.
Carbon monoxide: health effects overall impact	-	Estimates are based on limited number of epidemiology studies. Dollar estimates probably understate WTP.

Note: + means the potential error is positive, - means it is negative, and ? means the direction of the potential error is not known.

health and welfare impacts may cause AQVM 3.0 to understate both the potential benefits of air quality improvements and the costs of air quality degradations.

Here we identify and briefly discuss the identified omissions, biases, and uncertainties that are believed to be among the most significant in terms of potential error for the results of AQVM. More detailed identification and discussion of omissions, biases, and uncertainties are found in Chapters 4 and 5, in the Sulphur Study expert panel report (Thurston et al., 1997b), and Rowe et al. (1995).

6.1.1 Health Risks

For the health risk quantification, the most important issues concern the potential omission of health endpoints and pollutants, the measurement of mortality risks, potential confounding between air pollutants (and potentially other variables that influence health), and uncertainties regarding potential health effect thresholds. Because reduced health risks generally account for

an overwhelming majority of the benefits from reduced air pollution, potential errrors in the quantification of changes in health risk as a result of changes in air pollution concentration are key to assessing the accuracy of the benefits assessment.

It is likely that there are some additional health risks from air pollution that have not been quantified because the literature does not provide sufficient information to do so. For example, similar air quality benefits studies conducted before 1993 did not include long-term exposure mortality risks because the mortality risk studies had not been published. Similarly, few studies exist quantifying other health risks from long-term exposure. AQVM 3.0 includes estimates of the effect of particulate matter on risks of developing chronic bronchitis, but literature cited in the EPA ozone criteria document (EPA, 1996a) suggests that there may be some long-term exposure risks to respiratory health from ozone as well. However, the literature does not yet provide a basis for developing quantitative estimates of this risk. The significance of potential omitted health risks cannot be ascertained other than to indicate that the health benefits of reduced air pollution are most likely understated.

The omission of quantified health risks for some air pollutants will also understate health risk changes for some types of pollution control policies. For example, quantfied health risks are included for only four air toxics. However, based on available literature to date, the air pollutants that exist at levels sufficient to cause the most health risks to the population are included in AQVM 3.0, either directly or as a component of other measures such as particulate matter.

In selecting health risk parameters for AQVM 3.0 the intent has been to determine from the available literature the specific health risks associated with individual pollutant measures. However, the available information from the literature is limited by the fact that it is often difficult to isolate the effect of one pollutant when others are also present and correlated with each other in terms of day-to-day or location-to-location concentration fluctuations. That is, it is not always clear which pollutant, or mix of pollutants, is causing a particular health impact. Thus, for example, our assigning a health impact to one pollutant such as particulate matter may in fact be capturing the impacts of other omitted pollutants (e.g., air toxics), included (ozone, CO) pollutants, or constituents of the measured pollutant (e.g., sulphates as a constituent of particulate matter). The direction of potential error because of this difficulty is uncertain: for some pollutants the risks may be overstated, for others they may be understated.

The measurement of mortality risks perhaps presents one of the consequential uncertainties in the analysis. Decisions about whether these risks should be quantified as statistical lives, life years lost, or changes in survival probabilities, and how much weight to give to long-term exposure studies versus the short-term exposure studies, can all have significant impact on the computed mortality risks (see Section 4.3).

Finally, alternative assumptions regarding potential health effect thresholds can dramatically alter the final estimates of benefits for reductions in air pollution. If, for example, the baseline air pollution levels in a location are below an assumed threshold level, further reductions in air

pollution would have no benefits. Because the literature does not at this time provide sufficient information to determine whether there are thresholds and at what levels, the default setting in AQVM is no threshold, but the user can select alternative threshold levels for particulate matter and ozone to test the implications of this assumption. The significance of alternative threshold assumptions will vary considerably, depending on the level of the threshold selected, pollutants, locations, and changes in ambient concentrations considered in any one assessment, but the direction of the change in the benefits estimates is to lower the health risk changes for a given change in air pollution concentrations.

There are considerable issues with the measurement of air toxic damages and carbon monoxide damages. Many air toxics are currently omitted in AQVM 3.0. But, for those that are included, the risk factors are likely to lead to overstated benefit measures because of the way the cancer risk factors are developed. For carbon monoxide we have very limited literature from which to estimate health risks and the benefits of reducing CO are probably understated.

6.1.2 Health Effect Valuation

The dominant uncertainty in the health effects valuation is the selection of monetary values to assign to the mortality risks, both because this endpoint is the largest in most AQVM 3.0 assessments in terms of monetary value and because there are uncertainties in the interpretation of the mortality health risks. Issues of whether to interpret the values as VSL type risks, but predominately for older individuals, or as lost life-years, or as changes in survival rates for all individuals can result in significantly different economic valuation (see Section 5.2 of this report).

6.1.3 Environmental Effects and Benefits

Measured environmental effects typically account for a much smaller share of the measured total benefits of air pollution control that do measured health benefits. Thus, omissions, biases, and uncertainties in measuring these benefits are typically less consequential to the uncertainty of the total benefits measures. However, the errors and biases in the measurement of environmental effects and benefits may be large relative to the measured effects and values. Of most concern is the unknown significance of omitted benefits to forests, commercial crops and noncrop vegetation, and terrestrial and aquatic wildlife and ecosystems. Also omitted are potential visibility impacts in special recreation areas and potential damage and soiling to many materials. Although it is sometimes assumed that omitted environmental effects of air pollution changes may be consequential because the list of potential effects is quite long, there is little concrete evidence to say whether those effects might be large or small. In addition, there are also significant measurement uncertainties in the quantified benefits for recreational fishing, visibility, and materials damage owing to uncertainties in the underlying studies and in the benefits transfer processes used to apply the studies to general application in AQVM 3.0.

Finally, the valuation of health, welfare, and ecologic impacts of climate change associated with greenhouse gases (GHGs) may also be significant, or inconsequential. Assuming values proposed by some analysts, GHGs may be one of the more significant benefit categories when an air pollution control scenario includes substantive reductions in GHGs (see Section 5.5.5).

6.2 QUANTITATIVE ANALYSIS OF UNCERTAINTY

A three-step approach is used to statistically specify and address uncertainty in the quantitative benefits estimates produced by AQVM 3.0. The process is illustrated in Figure 6-1 and discussed below.

Step 1. Specify uncertainty by selecting low, central, and high values for the parameters in the concentration-response and monetary valuation functions. Probabilities are assigned to the low, central, and high values to reflect the relative confidence in each estimate.

Step 2. Calculate the uncertainty in benefits for each individual endpoint, such as mortality from particulate matter, for each location.

Step 3. Estimate the cumulative uncertainty for the total benefits estimate across all endpoints for each location and for the entire assessment area.

6.2.1 Step 1: Specify Low, Central, and High Estimates

Uncertainty in the literature for concentration-response and economic value functions is reflected in selection of low, central, and high concentration-response parameters and economic values, and in a probability weight that we have assigned to those model parameters. The procedure to select low, central, and high values for key model components varies across the individual endpoints but uses one or more of the following methods:

Evaluation of results across studies. Low, central, and high values are selected based on an evaluation of the range of results from the best available studies.

Expert judgment. Expert judgment of the analyst is nearly always required, based on review of the literature and results of past analyses.

Figure 6-1
Quantitative Uncertainty Analysis

***Standard error estimates*.** If only one well-executed study is available, the standard errors of the study results are sometimes used to provide a basis for evaluating the range of likely values and the probabilities to assign to the low, central, and high values.

Central estimates generally reflect our best estimate, but in some cases may simply represent the central value across a range of alternatives that may all be subject to an upward or downward bias. Typically, the high and low values do not represent the absolute highest or lowest values reported in the literature or that might be plausible under some circumstances. Rather, the high and low values are selected to be reasonably plausible alternatives to the central estimate based on the relevant literature and analyst judgment.

The selection of probability weights assigned to the low, central, and high estimates (which sum to 1) reflects the relative confidence that should be placed on these estimates. The weights are used to cumulate uncertainty in the aggregate benefits estimates. The procedure to select these probability assignments relies on the same types of information used to select the low, central, and high values. The probability assignment represents that share of the probability distribution function that the low, central, and high values are assumed to represent (rather than the percentile point of the distribution). The three examples below illustrate the types of assignments made and the logic applied.

1. ***Low 33%, Central 34%, High 33%.*** This type of assignment is used when the alternative estimates appear equally likely to be correct.

2. ***Low 25%, Central 50%, High 25%.*** This type of assignment is often used when estimates are all drawn from a single study. The low and high values then typically represent a one standard error spread around a mean value in a *t* or normal distribution. Such values can be thought of as point estimates approximating the lower and upper 25% to 30% of the probability distribution function for that parameter.

3. ***Low 33%, Central 50%, High 17%.*** This type of assignment is used when the central value gets a fairly strong weight, but the low estimate is given twice the weight of the high estimate because there is reason to place less confidence in the high estimate.

The selection of the most appropriate values for characterizing uncertainty in the quantitative assessment is determined by the judgment of the authors developing the quantitative methods. As demonstrated by Morgan et al. (1990), expert judgment may vary significantly, depending on the background, experience, and discipline of the expert. We expect there will be divergent opinions over the selection of the low, central, and high values and even more widely divergent opinions concerning the assignment of probabilities because these are based on more subjective judgment than the parameter estimates. Experts throughout Canada and the Scientific Authorities were consulted during selection of the low, central, and high values and the corresponding probabilities (including the Sulphur in Gasoline Expert Panel). AQVM 3.0 users can evaluate alternative judgments by substituting in alternative low, central, and high values and/or probability assignments (see *Report 1: User s Guide*).

6.2.2 Step 2: Calculate Distributions for Individual Benefit Endpoints for Each Location

Uncertainty accumulates as the individual components of the benefit computation are processed, often multiplicatively, to obtain a benefit estimate for individual endpoints (Step 2 in Figure 6-1). For example, when considering human health impacts due to particulate matter, uncertainty in the concentration-response function (CRF) is propagated into the valuation function.

For each endpoint, the cumulative uncertainty associated with the CRF and economic valuation is computed by combining the three step (low, central, high) distributions for the CRFs and valuation assumptions to form nine combinations, each with a value and probability assignment. These nine combinations are all the possible combinations and include low CRF with low valuation, low CRF with central valuation, low CRF with high valuation, and so forth, each multiplied by the population group and air quality change for each census division. The resulting set of nine values is arranged by order of magnitude and, with the probability assignments, form a nine-step damage distribution. From these data, estimates of damages for each individual endpoint are developed for each location in the analysis based on the location-specific air quality and population data. The values at the 10th and 90th percentiles of these nine-step endpoint

damage distributions are the low and high values, and the mean of this distribution is the mean value. The combination of the CRF central value and the valuation central value is the endpoint central value (which may or may not equal the mean or median of the nine-step combined distribution).[1]

6.2.3 Step 3: Compute the Distribution of Total Benefits across All Benefit Endpoints and Locations

AQVM 3.0 uses Monte-Carlo methods to compute the total benefits distribution for the total of all the individual endpoints for each province and for the Canadian totals. This involves 5,000 iterations of the following process. For each iteration, a CRF parameter and a dollar valuation parameter are randomly selected for each endpoint based on the various CRF and dollar value distributions. Using the selected parameters, the total damages are computed for each model location and aggregated to provincial totals and total Canadian damage estimates. The 5,000 iterations are used to create the distribution of total damages for each province and for the Canadian total. The approximate 10th and 90th percentile points on these provincial total and Canadian total damage distributions are reported as the low and high values. Again, the central value is computed using the central value for all CRF and valuation distributions for all endpoints, and represents our "best estimate" of the benefits of air quality improvements. It should be noted that the low (or high) total damages across the endpoints does not equal the summation of the low (or high) values for each endpoint for each province or for the Canadian total because we are combining distributions with different means and variances.

6.2.4 Incorporating Uncertainty from Atmospheric Modelling

AQVM 3.0 was developed to use best estimates, rather than distributions, for the air quality inputs. Incorporating air quality uncertainty into the analysis will increase the spread of the 10th percentile and 90th percentile benefit estimates. Future versions of the AQVM are expected to address this issue. In the meantime, users can preliminarily address this added uncertainty on the side by combining the air quality distribution with the benefits distribution information provided by AQVM 3.0 (for an illustration of this procedure, see Section 7.2.4 of the Sulphur in Gasoline Health and Welfare Expert Panel report, Thurston et al., 1997b).

1. It is important to note that the nine step endpoint damage distributions sometimes have significant step intervals. For example, as discussed in the Sulphur in Gasoline Expert Panel report (Thurston et al., 1997b), for the sulphur mortality risk endpoint (before multiplying by population and air quality changes), the value 0.408 represents the 0 percentile to 7.3 percentile, the value 0.68 represents the 7.3 percentile to 18 percentile (and is selected as the "low" value), and so forth. As a result, there is no unique 10th percentile distribution point.

6.2.5 Comparison with Alternative Uncertainty Approaches

The approach used in AQVM 3.0 to statistically address uncertainty in the selection of parameter values has advantages and disadvantages over other, similar approaches. One alternative is to use what we will call a full Monte Carlo approach such as was done in Banzhaf et al. (1996) and Deck et al. (1996). In this alternative, as in the AQVM 3.0 analysis, one first selects studies to consider. In Step 1, one develops a distribution for a parameter of interest by repeatedly and randomly selecting among the various studies, and then randomly selecting a value representing the study based on the mean value and variance of the parameter as reported in the study. A repeated process of selecting studies and parameter values from the study develops a mother distribution for the parameter of interest that reflects the information from all considered studies. This mother distribution would be used instead of the low, central, and high value distribution described in the AQVM 3.0 Step 1. Step 2 and Step 3 proceed in a similar manner as described for AQVM 3.0 by again using Monte Carlo methods to select values from each concentration-response and economic parameter to compute the benefit distribution for each individual effect and for the total of all effects and locations.

There are several key differences between the approach to Step 1 used in AQVM 3.0 and that in the full Monte Carlo alternative described above, including (1) judgments about how uncertainties within and across studies, and in the literature as a whole, are evaluated; (2) the statistical representation of the uncertainties; and (3) the implementation of the alternatives by a model user. We discuss these items in turn.

Turning to the first issue, the full Monte Carlo approach is appealing as a means to synthesize the results across various studies to aid in developing analyst judgment. The full Monte Carlo approach is also appealing when one has a limited basis to make judgments across studies one can either weight the studies equally, or use statistically derived weights, for example based on coefficient standard errors across the results from the studies. However, this may be as much a limitation as a strength of the full Monte Carlo approach. For example, in the PM_{10} mortality risk studies, there are many time-series studies measuring acute exposure risks in very different locations. Some of these studies are better suited for use as the basis of a quantitative assessment either, for example, because they cover longer periods of time, or because they more thoroughly address the impact that potentially confounding factors could have on the estimated PM_{10} effect. In this case, applying equal, or statistically derived, weights may or may not be an appropriate basis for parameter selection. Second, continuing with PM_{10} mortality risk studies, there are only two prospective studies, one of which may have more merit than many of the time-series studies combined. Surely the individual prospective and time-series studies should not be weighted equally, both on merit, and because there are actually two different kinds of measures of PM_{10} mortality risks being considered. Similarly, the PM_{10} mortality risk studies should not be weighted based on the similarity of results because, again, the prospective studies are for a different risk measure for which it is reasonable to assume the value should be higher than in the time-series studies.

In the AQVM 3.0 approach, the distribution of a CRF parameter or valuation parameter (the low, central, and high values and probabilities) is explicitly defined based on analyst judgment of individual studies and of the literature as a whole. Just as in the full Monte Carlo approach, this judgment can rely on meta-analysis or other format statistical procedures addressing the variance across studies and within individual studies; i.e., the AQVM 3.0 first step can use the full Monte Carlo first step, and then convert the results to a three-step distribution (AQVM is designed to allow more steps in future versions of the model). However, where there are few studies (or even one study), or when there are studies that seriously question the existence of nonzero coefficients, but do not provide analytic results to include in the Monte Carlo results, the variance within and across the quantitative studies may not appropriately represent the uncertainties in the estimated parameter values based on other factors used to evaluate the literature. For example, the cost-of-illness numbers typically have no variance, but we still believe there is variability and uncertainty in these estimates. The AQVM 3.0 approach readily and explicitly allows other factors to be considered and incorporated into the uncertainty modelling. If these types of factors are considered in the full Monte Carlo approach, then this approach becomes quite similar to the AQVM 3.0 approach (although the researcher then needs to specify a continuous distribution of the uncertainty, rather than a three-point distribution).

Second, the full Monte Carlo approach represents uncertainty as a continuous distribution of the parameter under consideration. This generally will be a benefit vis-a-vis the AQVM 3.0 approach because the combination of parameter distributions (e.g., CRF and valuation distributions) will be more smooth and there will fewer issues of discontinuities when evaluating results for selected distribution percentiles (see footnote #1). Further, the full Monte Carlo approach may include some nonzero probability of a zero value for many or all parameters, whereas the AQVM 3.0 three-point distribution will include a zero parameter only if it is specified as the low (and perhaps central and high) value.

Turning to the third issue, because the AQVM 3.0 approach simplifies the parameter distribution, it has two benefits to model users vis-a-vis the full Monte Carlo approach. First, the AQVM 3.0 approach reduces run times because it reduces the need to conduct the Step 1 Monte Carlo simulations (we have a specified three-point distribution rather than generating the distribution in each model run) and reduces the number of iterations required to conduct the Monte Carlo simulations in Steps 2 and 3 (we select from three-point distributions instead of from continuous distributions). As a result, AQVM 3.0 run times are reduced from hours (depending on the application) to tens of minutes. However, this benefit may become less significant in the future as the performance of PCs continues to improve. Second, the AQVM 3.0 approach allows model users to readily input their own judgments into the analysis to evaluate the sensitivity of the results to these judgments. This type of user-friendly evaluation of alternative judgments is generally more complicated for users of the full Monte Carlo approach.

In summary, the AQVM 3.0 approach is consistent with the objective of having a fast, user-friendly model that readily allows users to conduct sensitivity analyses incorporating alternative literature and judgment. To achieve these benefits, the AQVM 3.0 generates less smooth benefit

distributions that are potentially less robust in the tails of the distribution compared to the full Monte Carlo approach. As more health effects and economic valuations studies become available, we expect that AQVM will integrate finer distributions in Step 1 for some endpoints, reflecting Monte Carlo analyses of results of mutliple studies for a given endpoint.

6.2.6 Threshold Modelling Options for Human Health Endpoints

As discussed in Chapter 4, there is remaining uncertainty as to whether there are health effect thresholds for PM_{10}, SO_4, or ozone below which further reductions in ambient concentrations will not yield additional health benefits. The model is designed to allow the user to select alternative thresholds for these pollutants to test the effect of alternative assumptions on the final results.

For PM_{10} and SO_4, there are options for acute and chronic exposure thresholds. The acute exposure threshold applies to all the health effects linked to daily pollution concentrations; these include acute exposure mortality, hospital admissions, emergency room visits, asthma symptom days, restricted activity days, and respiratory symptom days. The chronic exposure threshold applies to those endpoints associated with annual (or longer) exposures; these include chronic exposure mortality, chronic respiratory disease, and acute bronchitis in children. For ozone, there is only an acute exposure threshold for all the health endpoints included in the model.

The threshold option for acute exposure uses pollution concentration data in the baseline air quality data files and the pollution concentration changes entered by the user. In the baseline air quality data files there are annual geometric mean concentrations (GM) and annual geometric standard deviations (GSD) for each location. These together are used to estimate a (log-normal) distribution of daily concentrations at each location, as shown by the baseline (B) in Figure 6-2. The change in annual average pollution concentration (AQ) is entered by the user (as either an absolute change or as a percentage change from the baseline, which the model converts to an absolute change). The baseline plus the AQ, assuming the same change on each day of the year, determines the new daily concentration distribution. This is illustrated for a negative AQ (i.e., an air quality improvement) in Figure 6-2. The acute threshold selected by the user (T_a) is then applied by calculating the number of days in a year that the baseline and new pollution concentrations are above the threshold in each location. This is 365 - D_1 in Figure 6-2.[2] The health endpoint is then calculated for those days for the full AQ. For those days for which B and B + AQ straddle the selected threshold (D_1 - D_0 in Figure 6-2), AQVM 3.0 estimates the difference in the new air quality and the threshold and applies it to the appropriate number of

2. For ozone data, the relevant time period is the 153-day ozone season. Because the computation in the model proportionally adjusts the air quality change for the fraction of the year or season that has either a total [(365 - D_1) ÷ 365] or partial [(D_1 - D_0) ÷ 365] health effect, it automatically adjusts to the relevant length of time.

Figure 6-2
Acute Exposure Threshold

days. For those days on which both B and B + AQ are below the selected threshold (D_0 in Figure 6-2), the change in health is zero.

The chronic exposure threshold (T_c) is selected by the user to represent the annual average concentration below which no further changes in chronic exposure health effects are presumed to occur. For each location, if $B > T_c$ and $(B + AQ) > T_c$, then the full AQ is applied in that location for the chronic exposure health endpoints. If $B < T_c$ and $(B + AQ) < T_c$ the change in chronic exposure health endpoints is zero for that location. If B and (B + AQ) straddle T_c, then the AQ is adjusted to reflect only the change above the threshold. For air quality improvements, this would be $B - T_c$; for air quality decrements, this would be $(B + AQ) - T_c$.

6.3 SENSITIVITY ANALYSIS WITH AQVM 3.0

Sensitivity analysis is an important tool for evaluating the impact associated with the uncertainty in key variables. Within AQVM 3.0, there are many assumptions that can significantly affect the benefits estimates and therefore should be examined through sensitivity analyses. Sensitivity analyses with AQVM 3.0 are conducted by running the model with alternative assumptions for the variable(s) in question (users can change the baseline assumptions in AQVM 3.0 by following the directions provided in the AQVM *Report 1: User s Guide*).

Key assumptions for which sensitivity analyses can be readily conducted include:

Endpoints. Health and welfare endpoints can be eliminated from the model.

Coefficients. Low, central, and high values for the CRF and economic valuation coefficients can be readily changed.

Probabilities. The probability assignments to the low, central, and high CRF and economic valuation coefficients can be readily changed.

Thresholds. The model default is that the CRFs for human health effects of PM_{10}, SO_4, and ozone apply to the lowest observable ambient concentrations. The user can readily substitute other threshold assumptions, by pollutant.

Air inputs. The user can run AQVM 3.0 with alternative air quality runs to test the sensitivity of results to this input.

Geographic coverage. The user can run the model for individual provinces, or for Canada as a whole, and for all census divisions or just those with monitors.

CHAPTER 7
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APPENDIX A
CONCENTRATION-RESPONSE FUNCTIONS FOR SULFATE AEROSOL HEALTH EFFECTS

This appendix explains the derivations of the human health concentration-response functions for sulphate aerosols that are included in AQVM 3.0. Sulphate aerosols (SO_4) make up a substantial share of airborne particulate matter, and there is some evidence in the health effects literature that distinguishes the health effects of sulphates from the health effects of particulate matter as a whole. The sulphate concentration-response functions give the AQVM 3.0 user the choice to calculate health effects based on changes in ambient sulphate concentrations instead of changes in PM_{10} or $PM_{2.5}$. The sulphate option is intended for analysis of policies that are likely to effect primarily sulphate aerosol concentrations; these are primarily policies related to SO_2 emissions. When several components of particulate matter, including sulphates, are likely to be affected by a given policy, the concentration-response functions for PM_{10} should be used.

The material in this appendix is drawn from the report by the Health and Environment Impact Assessment Panel, Joint Industry/Government Study on Sulphur in Gasoline and Diesel Fuels (Thurston et al., 1997b). For this effort, a panel of air pollution health effects experts, including some of the original authors of the AQVM, reviewed the literature and selected concentration-response functions for sulphate aerosols for use in assessment of sulphur dioxide emissions control policy options in Canada.

Concentration-response functions are used to specify the relationship between ambient air pollution concentrations and human health responses. The methods used here are similar to those developed for other efforts in the area such as the Canadian Council of Ministers of the Environment Task Force on Cleaner Vehicles and Fuels in the report entitled *Environmental and Health Benefits of Cleaner Vehicles and Fuels, Supplemental Report 2: Selected Concentration-Response Functions* (Chestnut and Ostro, 1995) and *Human Health Benefits from Sulfate Reductions under Title IV of the 1990 Clean Air Act Amendments* (Chestnut, 1995a).

The study selection criteria for sulphate concentration-responses functions are the same as those presented in Section 4.1.1. The discussion below describes how potential double counting in the health effects estimates is accounted for. In Sections A.3 and A.4 we discuss and specify concentration-response functions for sulphate-related mortality and morbidity.

A.1 SUMMARY OF SELECTED CONCENTRATION-RESPONSE FUNCTIONS

Table A-1 lists the selected concentration-response functions for each of the sulphate health effects categories. Sections A.2 and A.3 discuss the studies underlying the functions and detail the development of the functions.

Some of the sulphate health effects categories reported in the literature overlap. For example, acute respiratory symptoms days (ARSs) include some days that are also restricted activity days (RADs). To avoid double counting, we make adjustments in the concentration-response parameters. We assume that all pollution-related respiratory and cardiac hospital admissions (RHAs and CHAs respectively) involve an initial emergency room visit (ERV). We also assume that all RADs are also acute respiratory symptom days (ARSs). Ostro et al. (1993) report that 28% of the acute respiratory symptom days include a lower respiratory symptom. We therefore assume that RADs are split between upper and lower respiratory symptoms in the same proportions. We subtract only the lower respiratory RADs from ARSs because the latter only include days with lower respiratory symptoms. As a result, the following subtractions are made to calculate net sulphate concentration-response functions for each of these categories:

$$\text{Net ERVs} = \text{Total ERVs} \quad (\text{RHAs} + \text{CHAs}) \tag{A-1}$$

$$\text{Net ARSs} = \text{Total ARSs} \quad (0.28 \times \text{RADs}) \, . \tag{A-2}$$

A.2 PREMATURE MORTALITY

Over the last few decades, many epidemiologic studies have found statistically significant associations between concentrations of particulate matter and mortality among the general population. The earliest studies focused on relatively rare episodes of extremely high pollution concentrations in the 1940s and 1950s in the United States and in the United Kingdom (U.S. EPA, 1982a). More recent studies have found an association at concentration levels typical of most metropolitan areas in North America (e.g., Dockery and Pope (1994) review this literature).

The earliest studies of this type were cross-sectional studies examining annual mortality rates across U.S. cities with different average particulate matter levels, often including 100 or more cities (e.g., Lave and Seskin, 1977; Evans et al., 1984; Ozkaynak and Thurston, 1987, Lipfert, 1993). More recently, many time-series studies have found statistically significant associations between daily mortality and daily fluctuations in particulate matter concentrations in a wide ranges of cities (e.g., Pope et al., 1992; Schwartz and Dockery, 1992a, b). Very recently, two prospective studies using individual-specific data and tracking mortality for a study sample in multiple cities over multiple years, also found associations between premature mortality and particulate matter concentrations (Dockery et al., 1993; Pope et al., 1995).

Table A-1
Estimated Human Health Responses for a 1 g/m^3 Change in Annual Average Sulphate Concentration (annual individual risk factors)

Health Effect Category	Concentration-Response Parameter (weights)
Premature mortality Sources: Pope et al. (1995); Schwartz et al. (1996)	Low 1.14×10^{-5} (22%) Central 2.55×10^{-5} (67%) High 5.70×10^{-5} (11%)
Chronic respiratory disease (CRD) Source: Abbey et al. (1995a)	For population 25 years and over: Low 0.71×10^{-4} (25%) Central 1.35×10^{-4} (50%) High 2.00×10^{-4} (25%)
Respiratory hospital admissions (RHAs) Source: Burnett et al. (1995)	Low 1.3×10^{-5} (25%) Central 1.6×10^{-5} (50%) High 1.8×10^{-5} (25%)
Cardiac hospital admissions (CHAs) Source: Burnett et al. (1995)	Low 1.0×10^{-5} (25%) Central 1.3×10^{-5} (50%) High 1.7×10^{-5} (25%)
Net emergency room visits (ERVs) Source: Stieb et al. (1995)	Low 6.00×10^{-5} (25%) Central 7.40×10^{-5} (50%) High 8.40×10^{-5} (25%)
Asthma symptom days (ASDs) Source: Ostro et al. (1991)	For population with asthma (6%): Low 3.3×10^{-1} (25%) Central 6.6×10^{-1} (50%) High 9.9×10^{-1} (25%)
Restricted activity days (RADs) Source: Ostro (1990)	For nonasthmatic population (94%) 20 years and older: Low 1.55×10^{-2} (25%) Central 2.68×10^{-2} (50%) High 3.81×10^{-2} (25%)
Net days with acute respiratory symptoms (ARSs) Source: Ostro et al. (1993)	For nonasthmatic population (94%): Low 4.28×10^{-2} (25%) Central 13.6×10^{-2} (50%) High 22.4×10^{-2} (25%)
Acute bronchitis in children (B) Source: Dockery et al. (1996)	For population under age 20: Low 2.7×10^{-3} (25%) Central 4.4×10^{-3} (50%) High 6.2×10^{-3} (25%)

Some skepticism remains about whether these studies reflect a true causal relationship primarily because a specific biological mechanism to fully explain and verify this relationship has not been demonstrated in clinical or laboratory research (Utell and Samet, 1993). However, the epidemiologic studies are consistently finding a statistically significant association between air

pollution and mortality, using different study designs and locations, and over a wide range of particulate matter concentrations, including levels well below the current Canadian objectives or U.S. standards. In addition, recent controlled animal exposure studies have demonstrated plausible mechanisms by which severe effects, including death, may occur after concentrated ambient air pollution exposure (e.g., Godleski et al., 1996). Godleski et al. s work, it should be noted, was conducted at higher than ambient concentrations (up to 30x), for very short periods (3 days). It is therefore a reasonable exercise to estimate the reductions in premature mortality that might occur if air pollution concentrations were reduced.

A.2.1 Summary of Selected Quantitative Evidence

This section does not provide a detailed review of all available literature, but focuses on available results in the literature that are best suited for the purposes of this analysis. The study selection process incorporates results from prospective cohort and time-series studies. From both perspectives the results show an association between mortality and air pollution concentrations (including particulate matter and sulphate), and results from both types of studies are used to develop concentration-response parameters for this analysis.

An important question that has been raised with regard to the time-series mortality results is whether they may simply represent an increase in mortality in a population subgroup that is already very ill and close to death. A few researchers have attempted to address this question by examining the data to determine if there is a measurable decline in mortality after higher pollution days. This is a challenging statistical question and the evidence remains inconclusive at this time. If the only effect of particulate matter exposure were to accelerate oncoming death by a few days, average mortality rates would not differ between higher and lower pollution locations. However, cross-sectional studies do find differences in average mortality rates. Therefore, it is unlikely that the time-series results represent a shortening of life that is insignificant in all cases.

Long-Term Exposure Studies

Two types of long-term exposure studies have found statistically significant associations between mortality rates and air pollution levels in the United States. The first type is an ecologic cross-sectional study design in which mortality rates for various locations are analysed to determine if there is a statistical correlation with average air pollutant levels in each location. Such studies have consistently found measurably higher mortality rates in metropolitan areas with higher average levels of particulate matter. However, concern persists about whether these studies have adequately controlled for potential confounding factors. Lipfert (1993), Ozkaynak and Thurston (1987), and Evans et al. (1984) provide examples of ecologic cross-sectional studies. These studies each conducted a thorough examination of data, including average TSP or sulphate concentrations, for 100 or more U.S. metropolitan areas and emphasized controlling for potential confounding factors such as occupations, education, or migration. The findings of these studies varied in terms of the pollutants found to have a statistically significant relationship with mortality rates and the implied magnitudes of the effects.

A second type of long-term exposure study is a prospective cohort study in which a sample population is selected and followed over time in each location. In 1993, Dockery et al. published results for a 15-year prospective study based on samples of individuals in 6 cities. In 1995, Pope et al. published results of a 7-year prospective study based on samples of individuals in 151 cities in the United States. These studies are similar in some respects to the ecologic cross-sectional studies because the variation in pollution exposure is measured across locations rather than over time. These studies rely on the same type of pollutant exposure data, average pollutant levels measured at stationary outdoor monitors, as that used in the ecologic studies. However, the mortality data are for identified individuals, which enables much better characterization of the study population and other health risks than when area-wide mortality data are used. With the individual-specific data, the authors of the prospective studies were able to control for mortality risks associated with differences in body mass, occupational exposures, smoking (present and past), alcohol use, age, and gender.

Dockery et al. (1993) found a mortality-rate ratio of 1.26 over the 15-year study period from the most polluted to least polluted city; this ratio applied to several measures of particulate matter, SO_2, and NO_2. Pope et al. (1995) found a mortality-rate ratio of 1.15 and 1.17 for sulphates and fine particles respectively over the 7-year study period from the most polluted to least polluted city. All findings were statistically significant. Abbey et al. (1991) did not find any evidence of premature mortality associations with air pollution in a smaller, nonsmoking cohort in California.

The Pope at al. study in particular represents a very important contribution to the study of mortality and particulate matter because of the prospective design and the very large number of study locations included. The findings of a significant association between mortality and particulate matter in this study are very supportive of some of the findings in previous single-year cross-sectional studies.

The prospective cohort studies also speak to the question raised about time-series results regarding the degree to which the time-series results really represent a meaningful shortening of life. The prospective and cross-sectional studies would not reveal a statistically significant relationship if particulate exposures were associated only with deaths that would have occurred anyway within a very short time period (days or weeks). It remains uncertain, however, how many of those at risk have an already significantly diminished quality of life due to debilitating chronic illness unrelated to air pollution exposure.

The prospective studies provide strong evidence that long-term exposures to higher average particulate matter concentrations are associated with statistically significantly higher risks of premature mortality. However, the prospective studies have been criticized by some (Lipfert, 1995; Vedal, 1997) for their inability to account for potentially different historical levels of air pollution than indicated by the data available for the published analyses. These studies were also criticized for not more directly or fully accounting for potential confounding by differences in diet, physical activity, and socioeconomic status. Nevertheless, these studies still support an association between life expectancy and annual average particulate levels.

The results of the two prospective studies are summarised in Table A-2, along with the results from four selected annual cross-sectional studies, for comparison purposes. To facilitate some quantitative comparison, elasticities giving the percentage change in mortality for every one percent change in the pollutant measure, evaluated at the mean of the mortality rates and pollution measures in each study, were calculated. This controls to some extent for the differences in particulate matter measures and length of study periods. The elasticity estimates were further converted to percent change in mortality per unit of sulphate. The elasticities and mortality effects vary by an order of magnitude among these studies, though most of the variability is introduced by one study (Dockery et al., 1993). The result the panel elected to emphasize (Pope et al., 1995) was close to the median for the entire group.

Table A-2
Comparison of Long-Term Exposure Mortality Study Results for Sulphates

Study	Time Period	Number of Localities	Sulphate Mean (g/m^3)	Estimated Elasticity[a]	Mortality Effect[b] per g/m^3 Sulphate
Pope et al. (1995)	1982-1989	151	11.0	0.077	0.007
Dockery et al. (1993)	1974-1989	6	7.6	0.220	0.029
Lipfert (1993)	1980	149	9.3	0.019	0.002
Ozkaynak and Thurston (1987)	1980	98	11.1	0.086	0.0077
Evans et al. (1984)	1960	98	10.3	0.038	0.0037
Plagiannakos and Parker (1988)	1976-1982	9 Ontario counties	10	0.05	0.005

a. Elasticity is the percentage change in mortality for each one percent change in the pollution measure, estimated at the mean pollution measure. This is calculated from the reported relative risk results for the prospective studies using the formulas: Relative Risk = exp(b × PM); change in probability of death per unit PM (M ÷ PM) = b × Pr × (1 Pr), where Pr is the probability of death in the study; and elasticity = (M ÷ PM) × (mean PM ÷ mean M).
b. This is the percentage change in mortality per 1 g/m^3 sulphate at the mean sulphate concentration in each study.

Some of these cross-sectional studies also considered mortality relationships with gaseous pollutants. In the study of 6 cities by Dockery et al. (1993), Steubenville, Ohio, had the highest values for SO_4^{-2}, SO_2 and NO_x, making it difficult to assign the effect to any one of them with certainty. Lipfert et al. (1988) found essentially the same elasticity for all three pollutants, as computed by a long-range transport model based on emissions and weather data. Plagiannakos and Parker (1988) also found about the same elasticity for SO_2 and SO_4^{-2}. Pope et al. (1995) did not evaluate SO_2 or NO_x. Thus these long-term mortality responses should not be considered as only attributable to particulate matter or to sulphate, supporting the panel s use of sulphate as an index of total pollution effects.

Time-Series Studies of Acute Exposure

In recent years, numerous studies of air pollution and human mortality have indicated effects of acute air pollution exposures on daily mortality using time-series methods. The primary strength of time-series studies is that health and pollution variations in the same population (e.g., for a single city) are followed over time, so that the study population acts as its own control eliminating the need to statistically adjust for differences across population characteristics (e.g., race, income, education, etc.). Time-series statistical models use these respective day-to-day variations in exposure and effects data to determine whether mortality or morbidity counts rise and fall as air pollution concentrations rise and fall from day to day in the study area. A limitation of time-series studies though is that they focus only on short-term effects, and do not reflect any potential adverse consequences of chronic exposures to air pollution.

A.2.2 Selection of Premature Mortality Response Parameters

In this analysis, we consider both time-series epidemiologic studies of daily mortality counts and cross-sectional studies of annual mortality rates. Results from a time-series study are used to develop the low sulphate-related premature mortality concentration-response parameter, while a cross-sectional study is used to develop the high parameter. The central parameter is based on a weighted mean of the low and high parameters. The committee decided to give a two-thirds to one-third relative weighting of the time-series (low parameter) and cross-sectional (high parameter) studies, respectively, in the development of the central parameter. The concentration-response relationships are provided below as percentage changes (rather than as absolute population count-based estimates) so that the impacts could potentially be adjusted from region to region, depending upon the baseline mortality rate of each locale, should that information be obtained and the model adjusted to consider it. For the purposes of this study, however, the health benefits are calculated in every city by applying these percentages to the prevailing annual Canadian baseline nonaccidental death rate (6,700/million persons/year).

The time-series study used to develop the low sulphate-related mortality concentration-response parameter for this analysis was chosen because it directly considered sulphate air pollution, finding it to be significantly correlated with daily mortality in the six North American cities considered (Schwartz et al., 1996). The low parameter was therefore derived from Schwartz et al. (1996) 6-city time-series study from the mean mortality effect size (for a 5th to 95th percentile increase in sulphate) minus one standard deviation (to give the study s low estimate of the sulphate effect size), divided by the mean 5th to 95th percentile change in sulphate concentration during the study, as follows: (3.8% 0.8%) ÷ 17.0 g/m^3 SO_4 = 0.17% change in mortality per g/m^3 SO_4.

Numerous cross-sectional studies in the literature, as described above, have indicated that, after controlling for other confounders, places with higher sulphate concentrations have higher annual mortality rates than areas with lower concentrations. In this analysis, we employ the recent Pope et al. (1995) study in order to derive our high sulphate-related mortality concentration-response parameter. This study, while confirmatory of past such cross-sectional studies, is chosen because it analysed individuals, and could therefore better control for potential confounders (such as

smoking) on an individual level (rather than at the aggregate city level, as in prior studies). Also, the Pope study directly considered sulphates in a large number of North American cities.

The high sulphate-related premature mortality concentration-response parameter was therefore derived from the Pope et al. (1995) study by calculating the mean mortality effect size (for the most polluted areas vs. the least polluted) and adding one standard deviation (SE) (to give the study s high estimate of the sulphate effect size), divided by the mean sulphate concentration difference between most and least polluted areas during the study, as follows: Pope (+1 SE) = (1.15 + 0.035) relative risk per 19.9 g/m^3 SO_4 = ln(1.185) ÷ 19.9 × 100 = 0.85% change in total mortality per g/m^3 SO_4.

The central estimate of the premature mortality effect coefficient was then derived from the Schwartz et al. (1996) time-series study and the Pope et al. (1995) cross-sectional study discussed above by giving a two-thirds weight to the time-series result, and one-third weight to the cross-sectional study. High and low estimates are developed as stated above with the weightings determined by assigning the standard deviation of the relevant study one-half the weight of the central estimate and combining with the two-thirds weighting for the time-series studies and one-third weighting for the cross-sectional studies. The weighting distribution employed was based on a consensus of the panel, and provides an expected value for the distribution which equals the central estimate.

Thus, the selected percentage change in mortality responses per g/m^3 annual average SO_4 (with selected probability weights in parentheses) are:

Low	=	0.17% (22%)
Central	=	0.38% (67%)
High	=	0.85% (11%).

A.3 ACUTE AND CHRONIC MORBIDITY

In this section, we describe the derivations of concentration-response-functions for selected morbidity effects. Epidemiologic studies have found effects ranging from elevated rates of hospital admissions to small differences in lung function measurements. The studies selected as the basis for quantitative estimates for this report provide evidence with clear clinical significance. This means symptoms that are noticeable to the subject and can be expected to have some impact on the individual s well-being. For this reason, studies that look only at effects on lung function have not been included. Although this may be a medically relevant health endpoint, it cannot at this time be translated into changes in symptoms or illness that can be readily valued.

A.3.1 Chronic Respiratory Disease

For at least two decades, there has been some evidence suggesting that higher ambient particulate matter exposures are associated with higher rates of chronic respiratory disease (CRD). Much of this evidence, however, has been based on cross-sectional analyses, comparing disease or symptom prevalence rates in different communities with different average pollution levels (e.g., Ferris et al., 1973; 1976; Hodgkin et al., 1984; Portney and Mullahy, 1990). These studies are able to suggest a possible association, but are difficult to use for quantitative estimates of specific concentration-response functions. This difficulty stems primarily from uncertainty about how to characterise the relevant exposure units, in particular the time aspects of exposure. Chronic symptoms presumably occur as a result of long-term exposures, but cross-sectional analyses are not very enlightening about whether, for example, it is the five-year average, the twenty-year average, or the number of times a given concentration is exceeded that is the relevant exposure measure. Without this information, it is difficult to predict quantitatively how risks change when exposures change.

Recently published articles (Abbey et al., 1991; 1993; 1995a, b) have reported results of a 10-year cohort study conducted at Loma Linda University in California with a large sample of nonsmoking adults. The follow-up in this study allowed for measures of exposure over the 10-year period and for obtaining information on changes in chronic respiratory disease incidence over time. Thus, new cases of disease were analysed in relation to pollution exposure for a matching time period. This study provides, for the first time, a somewhat more definitive concentration-response function for chronic respiratory disease. However, uncertainties about the potential effect of exposures that preceded the study period, and lag times between exposure and illness onset still exist with these findings.

The Loma Linda University Study

In the first stage of the Loma Linda University study, a large sample (approximately 7,000) of Seventh Day Adventists (selected because they do not smoke), was interviewed in 1977. Health histories, current respiratory symptoms, past smoking and passive smoking exposure, and residence location histories were obtained. Hodgkin et al. (1984) compared the chronic respiratory disease status of respondents who had lived for at least 11 years in either a high or a low pollution area in Southern California. After adjusting for sex, race, age, education, occupational exposure, and past smoking history, residents of the higher pollution area had a rate of chronic obstructive pulmonary disease (COPD) (including chronic bronchitis, asthma and emphysema) that was 15% higher than for residents in the low pollution area. Using the same 1977 Loma Linda sample, Euler et al. (1987) also report results showing a statistically significant association between past TSP exposure, based on residence ZIP-code history, and the prevalence of chronic respiratory disease.

Abbey et al. (1991; 1993) performed a cohort study with the Seventh Day Adventist sample in 1987, which provides better quantitative concentration-response information. Nearly 4,000 subjects who had been interviewed in 1977 were interviewed in 1987. All were 25 years old or older in 1977. Estimates of air pollutant exposures were developed based on subjects

reported residence locations over the 10-year period and pollutant measures from stationary outdoor monitors at each location over the 10-year period.

Several different health outcomes were examined including new cases of emphysema, chronic bronchitis, or asthma, in 1987 for those not reporting any definite symptoms of these diseases in 1977. Disease definition was based on self-reported symptoms using the standardised respiratory symptoms questionnaire developed by the National Heart and Lung Institute for the United States. Respondents were classified as having definite symptoms of emphysema, chronic bronchitis or asthma if they met specific criteria for the disease diagnosis. Having definite symptoms of any one of these three was defined as definite airway obstructive disease (AOD). Having definite chronic bronchitis was defined as having symptoms of cough and/or sputum production on most days for at least 3 months/year, for 2 years or more. Emphysema and asthma required physician s diagnosis as well as associated symptoms. Respondents with some respiratory symptoms, but who did not meet the full criteria for that disease were classified as possible.

Abbey et al. (1995a) extends the earlier studies by analysing associations between these chronic respiratory disease outcomes and both fine particles and sulphates. Logistic models were estimated using the mean concentration of these two pollutants, along with PM_{10}, ozone, and other pollutants. Fine particles were estimated from empirical estimates related to airport visibility. Regarding sulphates, a statistically significant association was observed with AOD ($p < 0.05$). Sulphates were also associated with changes in the severity of AOD and chronic bronchitis over the ten year study period. Abbey et al. found no association with either SO_2 or NO_x. These findings are for a subsample of 1,600 respondents living within a certain distance of a local airport (visual range observations from these airports were used to estimate $PM_{2.5}$ in the study) and appear sensitive to the specific cohort under consideration. For example, using the full cohort ($n = 3,900$) (Abbey et al., 1995b), the association between sulphate and AOD was not statistically significant (at $p < 0.05$). The actual p value was not reported so it is unclear how strong the association was. It should be noted that unlike most of the other pollutant measures used in this study, for which data began in 1973, sulphate data begin in 1977.

Threshold Evidence for Chronic Respiratory Disease

The same uncertainty exists regarding the potential existence and level of a threshold for chronic effects of long-term air pollution exposure as for health effects associated with short-term exposures, but some additional comments are warranted. There is no clear a priori reason to expect that a threshold for short-term exposures would necessarily be the same, higher, or lower than a threshold for long-term exposures.

While several studies (Abbey et al., 1991, 1993; Chestnut et al., 1991) suggest thresholds for this endpoint and TSP, their relevancy to this analysis (i.e., reduced sulphate and pollutant gases) is questionable. Abbey et al. (1995b) do report some evidence of a threshold for chronic respiratory disease effects with $PM_{2.5}$ however, as discussed earlier, the issue of thresholds as a source of error in benefit estimates, is not regarded as significant.

Chronic Respiratory Concentration-Response Parameters from Abbey et al. (1995a)

The chronic respiratory results from Abbey et al. (1995a) are selected for quantification in this analysis. The estimates of AOD effects may be conservative in that the estimates do not reflect any mortality due to chronic respiratory disease that may have occurred during the 10-year period. Subjects are in the sample only if they were alive in 1987.

Two uncertainties in the quantitative estimates based on Abbey et al. (1995a, b) should be noted. One is that the authors have reported that a few subjects who describe symptoms that are subsequently classified as chronic bronchitis do not continue to report these symptoms in follow-up studies. This suggests that these were not true chronic bronchitis cases. The second uncertainty is how long a change in particulate exposure must exist before a change in chronic bronchitis incidence occurs. The estimates are annualised here, but are probably going to overstate the change in new chronic bronchitis cases in the first few years after a reduction in sulphate concentrations.

Abbey et al. (1995a), Table 6 reports a statistically significant association between sulphates and AOD. The estimated logistic regression coefficient (obtained from the author) was 0.0174, with a standard error of 0.0083. The partial effect of sulphates can then be calculated as the product of

$$AOD = b(p)(1 \quad p) \quad SO_4 ,$$

where:

b	=	estimated regression coefficient
p	=	baseline prevalence of AOD.

The baseline prevalence is 135 ÷ 1588 = 8.5%. Therefore, the increase in AOD over a ten-year period per unit sulphate is 0.00135. The effect per year per one g/m^3 change in sulphate would be 1.35×10^{-4}. The low and high parameters, based on minus and plus one standard error, are 0.71×10^{-4} and 2.00×10^{-4}, respectively. Therefore the low, central and high sulphate-related chronic respiratory disease concentration-response parameters, calculated in terms of expected annual increase in new cases of chronic respiratory disease, including chronic bronchitis, are:

Low	=	0.71×10^{-4}
Central	=	1.35×10^{-4}
High	=	2.00×10^{-4}.

The probability weights selected for the central parameter is 50%, with the low and high parameters each given 25% weights.

A.3.2 Hospital Admissions

Recent evidence indicates an association between ambient air pollution, including sulphates, and both respiratory hospital admissions (RHAs) and cardiac hospital admissions (CHAs) (e.g., Burnett et al., 1995; Thurston et al., 1994; Burnett et al., 1997). Evidence of a relationship between RHAs and CHAs and sulphates, controlling for collinear ozone concentrations, is provided by Burnett et al. (1995) for Ontario, Canada. Additional evidence of a relationship between RHAs and sulphates is provided by Thurston et al. (1994) for Toronto, and by Thurston et al. (1992) for selected cities in New York. For this analysis, specific quantitative estimates are derived from the Burnett et al. (1995) Ontario study because they are for both RHAs and CHAs. The Thurston et al. studies are examined for supporting evidence, but are not used quantitatively for several reasons. First, they did not examined hospital admissions for the entire year (only summertime admissions considered). Second, the Thurston et al. (1992) study did not simultaneously control the sulphate effect for ozone, thus possibly overestimating the risk attributable to sulphates alone. Third, the Thurston et al. (1994) study only examined six weeks of admissions in July and August for the three year period 1986 to 1988. The standard error of the sulphate coefficient was much higher for this study than the Burnett et al. (1995) investigation. The combined risk among the two studies, weighted by the inverse of the variance of the estimated log relative risk, was similar to that of the Burnett et al. (1995) study. Supporting evidence for an effect of particles on cardiac hospital admissions is provided by Schwartz and Morris (1995).

Burnett et al. (1995) studied the relationship between hospital admissions for respiratory and cardiac disease and both sulphate and ozone from 1983 through 1988 in Ontario, Canada. Air pollution data were obtained from a large network of monitors existing throughout Ontario. Admissions data from 168 acute care hospitals in Ontario below the 47th parallel were used. After elective admissions were excluded, counts of daily admissions for all ages and for age-specific and disease-specific categories were created. A time-series regression model was used that removed the influences of day-of-week effects, slow moving serial correlations due to seasonal patterns, and differences between hospitals. Ultimately, the effects of air pollution on deviations in the expected number of admissions to each hospital on any given day were estimated. Regression models included temperature effects and were specified with ozone and sulphate considered alone and together as explanatory variables. The results indicated that one-day lags of both ozone and sulphates were associated with respiratory admissions, and that sulphates, but not ozone, were associated with cardiac admissions. The sulphate effects were observed in both the summer and winter quarters, both males and females, and across all age groups (Burnett et al., 1995).

In more recent analyses of summer respiratory and cardiac hospital admissions in Toronto, Burnett et al. (1997) found that sulphate, NO_2 and SO_2 were each significant predictors of Toronto hospital admissions as single pollutants, but that sulphate was not significant when regressed jointly with either CO, SO_2 or NO_2, nor with O_3, SO_2 and NO_2 together. As was the case with daily mortality in Toronto, it is thus important to consider mixtures rather than just single pollutants. In this case, the use of sulphate alone could severely under predict the apparent total air pollution effect on hospital admissions.

Thurston et al. (1992, 1994) provide supporting evidence of an association between RHA during summer months and either sulphate or ozone concentrations, or both. Their work reports results for models that include both ozone and sulphate, so their results for both pollutants are relevant for comparison to the Burnett et al. results. Burnett et al. (1994) found that the mean sulphate concentration was associated with a 2.2% increase in daily summer RHAs when only sulphate was included in the model, and that the mean ozone concentration was associated with a 6.0% increase in daily summer RHAs when only ozone was included in the model. The single pollutant results are similar to results obtained by Thurston et al. (1992) for New York City, which were 3.5% for mean sulphate and 5.3% for mean ozone. These estimates are also reasonably consistent with the findings obtained in the Toronto study (Thurston et al., 1994).

Bates and Sizto (1989) provide some additional evidence on the issue. They estimated a stepwise regression for respiratory hospital admissions during the summer months in Ontario. First they included temperature, which explained 0.89% of the variance in RHA. Then they added sulphate, which increase the explained variance to 3.3%. When ozone was then added, the explained variance increased to 5.6%. This suggests that adding ozone to the regression explains about as much of the variance as that explained by the sulphate variable.

Low, central, and high sulphate-related RHA concentration-response parameters are selected based on the results of Burnett et al. (1995) from a model that included both sulphates and ozone in the regression, to reduce the chance of overstating the sulphate effect because of the collinearity between sulphates and ozone in the study area. We apply a 50% probability to the central parameter, and 25% each to the low and high estimates, which are the central minus and plus one standard error. Specifically, Burnett et al. (1995) report a 3.5% increase in RHAs for a 13 g/m^3 increase in sulphate when ozone was included in the model. The average daily RHA for the study period was 16.0 per million population. Thus, 3.5% of the 16.0 daily RHA are attributed to 13 g/m^3 sulphate. Therefore, the daily RHA per 1 g/m^3 sulphate is: $0.035 \times (16.0 \times 10^{-6}) \div 13 = 4.31 \times 10^{-8}$. We multiply by 365 to obtain the estimated annual number of RHAs for a change in annual average sulphate concentrations. The central concentration-response parameter of changes in annual RHA incidence is thus as follows, with the low and high parameters selected as the central minus and plus one standard error:

Low	=	1.3×10^{-5}
Central	=	1.6×10^{-5}
High	=	1.8×10^{-5}.

Burnett et al. (1995) also reported a statistically significant association between sulphates and cardiac hospital admissions (CHA) throughout the year, while no association was found for ozone. Burnett et al. (1995) report a 3.3% increase in CHAs for a 13 g/m^3 increase in sulphate when ozone was included in the model. Thus, 3.3% of the average daily CHAs per million population (14.4) in the study area gives the number of additional daily CHAs per 13 g/m^3 sulphate. Dividing by 13 gives the daily CHAs per g/m^3 sulphate [$0.033 \times (14.4 \times 10^{-6}) \div 13 = 3.66 \times 10^{-8}$]. We multiply by 365 to obtain the estimate annual number of RHAs for a change in annual average sulphate concentration. We apply a 50% probability to the central parameter, and

25% each to the low and high. The central concentration-response parameter for changes in annual CHAs is thus as follows, with the low and high parameters selected as minus and plus one standard error of the central parameter:

Low = 1.0×10^{-5}
Central = 1.3×10^{-5}
High = 1.7×10^{-5}.

A.3.3 Emergency Room Visits

For the purpose of quantifying the effects of reduced sulphur-in-fuels on emergency room visits (ERVs), and in the absence of a sulphate-specific study which addresses this, the panel agreed that the Saint John Particle Health Effects Study (Stieb et al., 1995) provided data which formed a basis for the best approach. This study indicates that, for each respiratory disease hospital admission in Saint John, NB, there are 5.3 emergency department visits for respiratory diseases and 1.4 emergency department visits per admission for cardiac diseases. Assuming these emergency department visits to hospital admission ratios apply elsewhere in Canada, and using the above derived risk coefficients for hospital admissions in each category, this yields the following ERV concentration-response parameters per annual average g/m^3 SO_4:

Low: $(5.3 \times 1.3 \times 10^{-5}) + (1.4 \times 1.0 \times 10^{-5}) = 0.83 \times 10^{-4}$
Central: $(5.3 \times 1.6 \times 10^{-5}) + (1.4 \times 1.3 \times 10^{-5}) = 1.03 \times 10^{-4}$
High: $(5.3 \times 1.8 \times 10^{-5}) + (1.4 \times 1.7 \times 10^{-5}) = 1.19 \times 10^{-4}$.

To estimate the net ERV concentration-response parameters the corresponding low, central, and high concentration-response parameter estimates for the RHA and CHA endpoints are subtracted from the ERV estimates above. This adjustment results in the following net ERV concentration-response parameters:

Low net: 6.00×10^{-5}
Central net: 7.40×10^{-5}
High net: 8.40×10^{-5}.

The selected probability weights are the same as those for hospital admissions: 50% for the central, and 25% each for the low and high parameters. Additional evidence supporting relationships between emergency room visits and air pollution was provided by Samet et al. (1981) for TSP and SO_2 in Steubenville, Ohio, and by Schwartz et al. (1993) for PM_{10} and asthma-related emergency room visits in Seattle.

A.3.4 Aggravation of Asthma Symptoms

Several studies have related air pollutant concentrations to exacerbation of asthma symptoms in individuals with diagnosed asthma. Several epidemiologic studies with currently diagnosed asthmatics provide quantitative information to allow estimates of the frequency of elevated asthma symptoms (ASDs) as a function of ambient particulate matter concentrations (Whittemore and Korn, 1980; Ostro et al., 1991; Ostro et al., 1995; Thurston et al., 1997a). The latter three studies include sulphate as a measure of exposure. No information was considered on possible effects of SO_2 or NO_2 on asthma attacks.

All of these studies had subjects (diagnosed asthmatics) record daily asthma symptoms during the duration of the study. An elevation of asthma symptoms was defined for each subject based on each individual s manifestation of asthma symptoms. This typically meant a notable increase in symptoms, such as shortness of breath or wheezing, and/or in use of medication relative to what was normal for that individual. Daily particulate matter and ozone levels were then examined for correlations with day-to-day fluctuations in asthma symptom frequency, controlling for other factors such as weather and previous-day symptoms.

Ostro et al. (1991) examined the association between several different air pollutants, including sulphates, $PM_{2.5}$, and acidic aerosols, and increases in asthma symptom days among adults during winter months in Denver. A significant association was found between the probability of moderate or severe asthma symptom days (measured as shortness of breath) and sulphate particulate levels, after controlling for temperature, day of week, previous-day illness, and use of a gas stove. Ozone levels were very low, near background levels, and did not create a confounding influence.

Specifically, the above study reports that an increase of one log unit of sulphate is associated with a 0.0077 increase in symptoms. The prevalence of daily shortness of breath during the study was 18% and the mean of sulphate was 2.11 g/m^3. Linearising, each unit change in sulphate is associated with a 0.0077 ÷ 2.11 = 0.0036 change in the probability of symptoms.[1] This amounts to a 2% change in symptoms per one g/m^3 change in sulphate. There may be an upward bias in these results if used year-round, as the data were collected only during the winter months in Denver. A subsequent study, Ostro et al. (1994) indicated that many of these asthma attacks were likely precipitated by prior respiratory infections.

Ostro et al. (1995) involved a panel of 85 African-American children, aged 7 to 12, with asthma living in Los Angeles. The sample included a wide range of asthma severity and socioeconomic levels. The daily prevalence of shortness of breath (daily mean = 5.6% was recorded by each child with help from his or her childcare provider. After controlling for weather, respiratory infections and serial correlation, an association was reported between PM_{10} and the probability of a symptom. The mean concentration of PM_{10} of 56 g/m^3 was associated with a relative risk of 1.6 (95% CI 1.10 to 2.32). Assuming sulphates are as toxic per unit as PM_{10}, a one unit

1. Symptom frequencies were modelled as a function of the log of daily sulphate concentration. The derivative of symptom frequency relative to changes in daily sulphate concentrations is therefore ÷ daily sulphate concentration. We simplify this function by substituting the mean daily sulphate concentration.

change in sulphate is associated with an increase in symptoms of 0.84%. The estimated beta coefficient for this study is 0.0084 with a standard error of 0.0033. Therefore the effect per day per g/m^3 of sulphate is (0.0084 × 0.056 × 0.944) or 4.44 × 10^{-4}. The upper and lower estimates are obtained by applying one standard error. Therefore, the low and high estimates per day are 9.00 × 10^{-5} and 7.9 × 10^{-4} respectively.

Finally, Thurston et al. (1997a) examined asthma attacks among a sample of moderate to severe asthma children at an American Lung Association asthma camp. In order to investigate associations between summertime haze air pollution and asthma at an individual level, 52, 58, and 56 children (ages 7-13) attending a summer asthma camp were followed during the last week of June in 1991, 1992, and 1993, respectively. Daily records were kept of the environmental conditions, as well as of subject medication use, lung function, and medical symptoms. Air pollution was found to be significantly and consistently correlated with acute asthma exacerbations, chest symptoms, and lung function decrements. Ozone levels were also quite high on the high sulphate days in this study. The results suggest that a day experiencing a one unit change in sulphate is associated with a 2.2% increase in the number of asthma exacerbations.

Therefore, there are three studies that indicate that the per unit effect of sulphate on asthma symptoms are in the range of 1% to 2%. The Ostro et al. (1991) and Thurston (1997a) studies give similar results, despite very different levels of ozone. For the purposes of this study, the estimates from Ostro et al. (1991) are used to provide quantitative estimates. Specifically, the low, central and high sulphate-related ASD concentration-response parameters are derived as follows: 0.0077 (±0.0038) ÷ 2.11 g/m^3 SO_4, or 3.6 × 10^{-3} per g/m^3 SO_4, for the central parameter. The high and low estimates are plus and minus one standard error. Assuming, as per Chestnut (1995), that this is applicable only in the cold months, the annual numbers are 182.5 times these daily numbers. Thus, the concentration-response parameters per annual average g/m^3 SO_4 for the portion of the population with asthma (6%) are:

Low	=	3.3 × 10^{-1} (25%)
Central	=	6.6 × 10^{-1} (50%)
High	=	9.9 × 10^{-1} (25%).

As noted above, the ASDs estimates in this analysis are calculated assuming that 6% of the population has asthma. This is an estimate of the percentage of the Canadian population with diagnosed asthma (Statistics Canada, 1994).

A.3.5 Restricted Activity Days

Ostro (1990) used similar health data to explore the association between restricted activity days (RADs) for respiratory conditions and several measures of particulate matter including sulphates, taken from U.S. EPA s Inhalable Particle Monitoring Network over a 3-year period. A statistically significant association was reported between RADs and sulphate. The reported

Poisson regression coefficient was 0.0083 with a standard error of 0.0035. These estimates are generally similar to those reported earlier for $PM_{2.5}$ and are used for our quantitative estimates. The central parameter implies that a one g/m^3 change in sulphate increase RAD by 0.83%. The study sample experienced 3.23 RADs for respiratory conditions per year per individual. Therefore, the central concentration-response parameter (with a 50% weight) is 0.0083 × 3.23 or 0.0268, while the low and high parameters are ± one standard error. Therefore, the concentration-response parameters are:

Low	=	0.0155
Central	=	0.0268
High	=	0.0381.

As qualitative support for this relationship, the Panel noted that Ostro (1987) found a significant relationship between RADs and $PM_{2.5}$ as estimated from airport visual range, a parameter strongly influenced by fine particles, such as sulphates (Ozkaynak et al., 1985).

Because daily symptom concentration-response functions for asthmatics are available based on studies focused specifically on those with diagnosed asthma, we exclude the asthmatic population from the calculations of respiratory-related restricted activity days. Although asthmatics were not specifically excluded from the RAD studies, the latter are more representative of the response of the general population because only a small fraction of the general public has diagnosed asthma. We therefore apply the RAD concentration-response function to the nonasthmatic portion (94%) of the population 20 years and older.

A.3.6 Acute Respiratory Symptoms

Ostro et al. (1993) examined the association between air pollutants, including sulphate, and lower and upper respiratory symptoms (ARS). Using a logistic regression model and controlling for weather, gas stove use, time, gender and the existence of chronic disease, a statistically significant association was reported between sulphate and lower respiratory symptoms, defined as dry cough, cough with phlegm, shortness of breath, chest cold, croup, asthma, bronchitis, flu or pneumonia. The prevalence of lower respiratory symptoms was 1.5%. The results indicated that a 10 g/m^3 change in sulphate was associated with an odds ratio of 1.30 (95% CI = 1.09 to 1.54). Therefore, the associated regression coefficient is 0.0262 (ln 1.3) per g/m^3 with a standard error of 0.009. For quantification, we use the central estimate ± one standard error. The annual effect of sulphates can then be calculated as the product of:

$$b(p)(1 \quad p)365 = (0.0262)(0.015)(0.985)(365) = 0.141 ,$$

where:

b	=	the estimated response coefficient
p	=	the baseline prevalence of lower respiratory symptoms.

Therefore the low, central and high annual sulphate-related ARS concentration-response parameters are:

Low	=	0.046
Central	=	0.141
High	=	0.232.

Because the definition of ARSs includes days that also fall into the category of restricted activity days, we subtract RADs to obtain net ARS parameters. The RADs parameters apply only to the population age 20 and older, so we multiply the RADs parameters by 0.728 (the share of the Canadian population in the 1996 census that is age 20 and over). In addition, because the ARS definition used for sulphates includes only lower respiratory symptoms it is necessary to further adjust the RADs parameters multiplying them by 0.28 which reflects the share of the respiratory symptoms in the Ostro et al. (1993) study that were lower respiratory symptoms. The adjusted RADs parameters are then subtracted from ARS values above resulting in the following net ARS concentration-response parameters:

Low net	=	4.28×10^{-2}
Central net	=	13.6×10^{-2}
High net	=	22.4×10^{-2}.

Using the same data set as Ostro et al. (1993), Krupnick et al. (1990) found a relationship between acute respiratory symptoms and/or COH (a measure of carbonaceous particles) and ozone. The two studies are not directly comparable since the two studies used somewhat different endpoints to characterize acute respiratory symptoms. Ostro et al. are used since they incorporated sulphates as one of their air pollutant measures.

Because daily symptom concentration-response functions for asthmatics are available based on studies focused specifically on those with diagnosed asthma, we exclude the asthmatic population from the calculations of acute respiratory symptom days. Although asthmatics were not specifically excluded from the ARS studies, the latter are more representative of the response of the general population because only a small fraction of the general public has diagnosed asthma. We therefore apply the ARS concentration-response function to the nonasthmatic portion (94%) of the population.

A.3.7 Acute Bronchitis in Children

Dockery et al. (1989) studied the relationship between lower respiratory illness in children and particulate matter concentrations in six cities in the United States. The study related annual concentrations of TSP, PM_{15}, $PM_{2.5}$, sulphate, and sulphur dioxide to the presence of chronic cough, bronchitis, chest illness, persistent wheeze, and asthma. These illnesses were noted during a health examination and intake questionnaire taken for the sampled children in each city. A condition of asthma or bronchitis was based on a physician s diagnosis in the previous year.

Chronic cough was defined as a cough being present for at least 3 months in the past year. A logistic regression reveal a statistically significant relationship between annual average PM_{15} levels and the probability of the child having bronchitis or chronic cough in the past year.

A recent study (Dockery et al., 1996) replicates the above study using 24 cities, including several in Canada (Leamington, Egbert, Pembroke, and Dunnville, ON; Yorkton, SK; and Penticton, BC). Children ages 8 to 12 were assessed via questionnaire between 1988 and 1991 about chronic cough. This was defined as cough first thing in the morning for as long as 3 months in a row and/or cough at other times during the day or night for as long as 3 months in a row. Among the cities, the prevalence rates for bronchitis ranged from 3% to 10%. The logistic regression analysis controlled for sex, history of allergies, parental asthma, parental education, and current smoking in the home. The study reported a statistically significant association between sulphate and acute bronchitis (no significant relationship with SO_2; NO_2 not considered). Specifically, a 6.8 g/m^3 increase in annual sulphate was associated with an odds ratio of 1.65. The associated regression coefficient would be $\ln(1.65) \div 6.8 = 0.0736$ with a standard error of 0.029. Therefore, a one g/m^3 change in sulphate generates a central concentration-response parameter of $0.073 \times 0.065 \times 0.935$ or 0.0044. For quantification, we use the central estimate plus and minus one standard error. Therefore, the selected low, central and high concentration-response parameters measured in terms of expected annual increase in incidence of acute bronchitis in children (B) per one g/m^3 change in annual average SO_4 are:

Low	=	0.0027
Central	=	0.0044
High	=	0.0062.

APPENDIX B
REVISIONS TO AQVM AND RESPONSE TO COMMENTS

B.1 INTRODUCTION

The initial version of the Air Quality Valuation Model (AQVM 1.0) was developed in the summer of 1996. In the summer and fall of 1996, Environment Canada and Health Canada commissioned peer review comments, and comments were also submitted by other interested parties.[1] The comments are provided in Appendix C. Overall, the comments were favourable, especially as to the usefulness of the AQVM for helping to address policy questions related to air pollution control.

In Section B.2 of this appendix we summarize the most significant revisions made to the AQVM since the initial version was written in 1996. Revisions were made to the model in response to the peer review comments, to reflect new literature, and to incorporate a sulphate module based on the Joint Industry/Government Study of Sulphur in Gasoline and Diesel Fuels (hereafter Sulphur Study, Thurston et al., 1997b). In Sections B.3 through B.7, we provide brief responses to the more substantive peer review comments. For minor comments, we simply made the corrections as warranted. The intent of this appendix is to provide additional perspective on the selection of methods, literature, and assumptions in AQVM, not to provide point-by-point debate of the literature, nor to provide detailed literature summaries.

For many of the issues raised, there is extensive debate in the literature and a variety of perspectives and uncertainties that are valid to recognize. As discussed in the review comments by Alan Krupnick, many of the choices made in developing the AQVM methods are necessarily based on professional judgment and interpretation of the literature. It is therefore important to note that the literature, methods, and specific assumptions selected, and the results obtained, for the updated AQVM (AQVM 2.0) are highly consistent with the comparable elements of other recent large scale air quality benefit studies that have undergone extensive peer review, including:

> The New York Environmental Externalities Cost Study (Rowe et al., 1995), which included a review board retained by industry, governments, and interest groups.

1. Peer reviewers included Alan Krupnick of Resources for the Future, Svere Vedal of the University of British Columbia, A. Myrick Freeman of Bowdoin College, Bill Desvousges of Triangle Economic Research, Wiktor Adamowicz of the University of Alberta, and Jonathan Samet of Johns Hopkins University. Additional comments were provided by Clare Cottinham of the UK Department of Environment, and in a report prepared by Industry Canada.

The Canadian Joint Industry/Government Study, Sulphur in Gasoline and Diesel Fuels (Thurston et al., 1997b), and its expert panel (see below).

The U.S. EPA s Clean Air Act Retrospective Benefit-Cost Study (U.S. EPA, 1997a), which was reviewed extensively by expert panels for the U.S. EPA Science Advisory Board.

The U.S. EPA s Regulatory Impact Analysis for the Particulate Matter and Ozone National Ambient Air Quality Standards (U.S. EPA, 1997b), which received U.S. EPA Science Advisory Board review.

The U.S. EPA s analysis of Human Health Benefits from Sulfate Reductions under Title IV of the Clean Air Act (Chestnut, 1995a).

The European Commission s ExternE: Externalities of Energy study (European Commission, 1995), which was a collaborative study by experts from many European countries of the environmental externalities of the common forms of electric energy production, with extensive international expert review.

Consistency with methods selected by analysts for these studies supports the choices made for the AQVM, but does not ensure validity. New literature will emerge in the future that may result in different conclusions and judgments regarding the impacts of air pollutants on human health and the environment. For these reasons the AQVM has been revised to incorporate more flexibility, allowing users to conduct sensitivity analyses with alternative model assumptions.

Because many reviewers provided similar and related comments, the responses are organized by topic rather than by reviewer. In Section B.3 we address general modelling issues. In Sections B.4 and B.5 we address the health and welfare effects assessment issues. In Section B.6 we address economic valuation issues. Other remaining issues are briefly addressed in Section B.7.

B.2 REVISIONS TO THE AQVM

In this section we summarize the most significant revisions made for version 2.0 of the AQVM, and cite the AQVM report sections where more information can be found.

1. ***Sulphate model version.*** As part of the Joint Industry/Government Sulphur Study, a panel of health, environment and economics experts (Sulphur Study expert panel)[2] developed methods to assess health and welfare benefits from reduced ambient concentrations of sulphates and sulphur dioxides, which were included in a report issued in the summer of 1997 (Thurston et al., 1997b). AQVM was revised to include the Sulphur Study expert panel methods and assumptions as a separate module, which was used to compute benefits estimates for the pollution control scenarios considered for the Sulphur Study. Original portions of AQVM were also revised to be consistent with the updated methods and judgment in the Sulphur Study expert panel report. See the *AQVM Report 1: User s Guide* for how to access the sulphate module, Appendix A of this report for the sulphate health effects concentration-response functions, and Chapter 5 for the economic values used. The Sulphur Study expert panel report includes responses to comments on the health, welfare, and economic valuation methods, and those comments are consistent with the responses provided below for AQVM.

2. ***Sensitivity analyses for health, welfare, and economic assumptions.*** AQVM 2.0 provides the user with the ability to readily change many of the modelling assumptions, including the concentration-response-function coefficients, the economic value coefficients, and the probability weights assigned to each of the coefficients. The background on the selected AQVM 2.0 default parameters is discussed in *Report 2: Methodology*. Some revisions were made to the AQVM default parameters since the initial version was written in 1996, based on reviewer comments, the Sulphur Study expert panel findings, and other updates to reflect newly available literature. While it is our judgment that the AQVM provides an appropriate selection of model parameters based on the available literature at this time, AQVM 2.0 provides flexibility for users to readily conduct sensitivity analyses using alternative values for many of the parameters and quantification methods used in the model to reflect new literature or alternative professional judgment of the literature. The user is cautioned to maintain correspondence between the assumptions used and the model outputs through careful labelling of the scenarios. Note that AQVM 2.0 does not allow the user to change the functional form of the concentration-response functions or many other computational procedures that are embedded in the quantification methods used in the AQVM.

3. ***User-friendly model enhancements.*** The AQVM has been significantly revised to improve its flexibility, user interfaces have been streamlined, and users may now save their scenario input data on air quality changes and retrieve these files later to rerun the

2. This panel was chaired by Dr. George Thurston of New York University School of Medicine, and included David Bates of the University of British Columbia Department of Health Care and Epidemiology; Dr. Richard Burnett of Health Canada, Environmental Health Directorate; Dr. Fred Lipfert, independent consultant; Dr. Bart Ostro of the California Environmental Protection Agency, Dr. Beverly Hale of the University of Guelph, Horticultural Science Department; Dr. Alan Krupnick of Resources for the Future, Dr. Robert Rowe of Hagler Bailly (now of Stratus Consulting), and Derek Ireland of Industry Canada.

scenarios with alternative assumptions or to make changes in the air quality scenario data. (See *Report 1: User s Guide*).

4. ***Threshold assessment.*** In response to reviewer comments, and reflecting the importance that alternative assumptions about health effects thresholds can have on computed benefits of air quality changes, AQVM has been modified to allow users to select concentration-response thresholds for PM_{10}, SO_4, and ozone health endpoints. Because the AQVM is intended to be a streamlined model, thresholds cannot be specified for individual health endpoints. Rather, a threshold can be specified for groups of health endpoints. The selection and application of this model option is discussed in *Report 1: User s Guide*.

5. ***Updated agricultural data.*** Baseline agricultural price and production data have been updated to be based on multiple years rather than just 1994. See Chapter 3 of *Report 2: Methodology* for the sources and description of the data used.

6. ***Revised recreational fishing assessment.*** Based on peer reviewer comments, we revised the method to compute recreational fishing damages resulting from acid deposition. See *Report 2: Methodology,* Section 5.5.4.

7. ***Census divisions with monitors switch.*** The AQVM computes changes in health and welfare effects for changes in air pollutant concentrations in every census division. A database of baseline average air pollution concentrations is included in the model. This database was developed from available ambient monitoring data for 1990 to 1994. The user can specify air pollution concentration changes expected for a given control strategy as absolute or percentage changes from the baseline. However, the baseline air pollution database is probably more accurate for some locations than others, because there were not monitors for every pollutant in every census division. When there was not a monitor for a given pollutant in a given census division, the concentration was estimated based on the closest available monitors, which may in some cases be considerable distances away. The user, therefore, has the option to limit the computations of changes in health and welfare effects to those census divisions in which an ambient monitor was located. This is a pollutant specific switch. Selecting this option in AQVM runs the model, for each pollutant, only for those census divisions where monitors are located for that pollutant. This will most likely understate the impacts and benefits for a given scenario by omitting census divisions without monitors (unless there are no air quality improvements in these locations), but the results will not be subject to the potential error that results from the extrapolation of baseline air quality concentrations to locations without monitors. See *Report 1: User s Guide*.

8. ***Uncertainty routines.*** The uncertainty routines have been simplified to run more quickly and accurately. First, the user has the option to simply and quickly compute the central estimates, without the statistical uncertainty analysis. With this approach, users can

quickly conduct sensitivity analyses to see how central estimates change with alternative parameter assumptions. Second, for individual endpoints, the model simply computes the 10th and 90th percentile on the combined health impact and economic value distribution for each health endpoint from the nine point distribution (representing the combined states of the low, central, and high values and parameters for the health coefficients and for the corresponding economic coefficients). Third, for the aggregation of benefits across endpoints and locations, the program @Risk is used to conduct a Monte Carlo simulation that uses the uncertainty specifications for health and welfare impacts, and economic values, for all endpoints (see Chapter 6).

9. ***Other revisions.*** Other revisions have been made reflecting new literature and updating computations used in the model. These include:

   - Significant revisions to the ozone mortality risk section to reflect new literature. The revisions are consistent with U.S. EPA (1997b).

   - Revisions in the particulate matter mortality risk section to give greater emphasis on the prospective studies vis-a-vis the time-series studies, which is consistent with the Sulphur Study expert panel recommendations and recent U.S. EPA air quality benefit analyses (U.S. EPA 1997a; 1997b).

   - All 1994 economic values were recomputed from U.S. values or from other years with updated Purchasing Power Parity (PPP), Canadian Consumer Price Index (CPI), and Canadian Medical/Health Care Price Index information. A consistent conversion methodology was applied, converting to Canadian values in the year of the original study and then updating to 1994 values using the appropriate Canadian price indices.

B.3 GENERAL MODELLING ISSUES

B.3.1 Approach and Documentation

Several comments requested more extensive documentation on and discussion of the overall approach used, and more extensive discussion of the full set of literature as well as of the individual studies relied on (see especially the comments from Samet). However, the AQVM reports were designed to be accessible to a broad set of readers, to provide the summary basis for the approach and literature used, and to document the detailed assumptions used. The AQVM effort was not designed to provide extensive reviews of the methods and the large body of literature available. More extensive documentation of the methods and the literature is provided in several of the reports identified in the introduction to this appendix. Also, detailed reviews of the available scientific literature regarding the health and welfare effects of air pollutants are

available in the Canadian Environmental Protection Act, Federal Provincial Advisory Committee, Working Group on Air Quality Objectives and Guidelines Science Assessment Documents and in the U.S. EPA criteria documents.

B.3.2 Omissions, Biases, and Uncertainties

Industry Canada and Krupnick suggested that more effort be given to identify, rank, and quantify, the importance of unquantified omissions, uncertainties, and biases. Similarly, Samet suggested that more be done to address uncertainty, and to separate omissions, biases, and uncertainties. We agree more effort would be useful. We have added more discussion and evaluation as to which of the omissions, uncertainties, and biases are likely to be the most significant. Further quantification of uncertainties and evaluation of the significance of omissions and biases are desirable and may be considered in future versions of AQVM.

Adamowicz suggested that more caveats be added to the report concerning the limitations of benefits transfer and that the selected uncertainty quantification approach may understate the variance of benefits and may even overstate damages by not including zero in the benefits distribution. Krupnick identified similar issues. These issues are now briefly identified in the text. Desvousges expressed concern with how the meta analysis approach to uncertainty quantification is presented in the AQVM report, and we reviewed and edited this section of the report to better present the comparison of statistical uncertainty approaches.

B.3.3 Other Modelling Issues

Monitor Location and Population Exposure

Vedal stated that monitors are often placed for strategic reasons and may overstate typical population exposure. Samet also noted that the exposure data are limited, and that concentrations may not equal exposure, depending on individual activity patterns and location. These points are true. However, the epidemiology studies used to estimate changes in health risks (other than for air toxics) are also based on stationary outdoor monitor data. Typical individual activity patterns are therefore reflected in the epidemiology coefficients. However, significant inaccuracies may occur when extrapolating baseline air pollution concentrations to locations without monitors. To address this issue, we created an AQVM option to limit the calculations for each pollutant to only those census divisions in which monitors are located. It is important to note that this option will generally understate benefits of air quality improvements by omitting populations for whom benefits may occur.

Conversion Factors

Krupnick raised a question about the applicability to a Canadian assessment of conversion factors for different measures of particulate matter based on California studies. The factors for converting epidemiology study results based on particulate matter measures other than PM_{10} (e.g., TSP, SO_4, $PM_{2.5}$) to equivalent health effects estimates per unit change in PM_{10} are ideally based on data for the location(s) where the study was conducted, not where the study results are being applied. This topic is discussed in general in Section 4.1 of the *Report 2: Methodology*. Sources for specific conversion factors are listed when they are used in interpreting a specific study, and are matched to the study s original location as much as possible. The 0.55 PM_{10} to TSP ratio is based on data from throughout the United States and is applied when interpreting epidemiology studies done in the United States using TSP data. Conversion factors based on California data were used for converting results from studies conducted in California.

Latency

Industry Canada suggested that benefits occur later than costs do, and need to be discounted. This is an issue separate from the AQVM computation. The AQVM computes annual benefits expected to occur in the same year that air quality changes occur. Once the stream of annual costs and benefits is identified, those costs and benefits then need to be appropriately discounted to present values if one wants to do a comparison in present value terms. This computation is conducted outside of the AQVM and thus is not relevant to the AQVM documentation.

High Level of Aggregation

Industry Canada noted that the AQVM is not well suited to examine policies that reduce pollution on a handful of days because it uses annual average pollutant measures. This is generally true, although a user could apply the average change for the few days as if it were the annual average, then adjust the annual benefits downward to reflect the percentage of the year actually affected.

Weighting of Evidence across Endpoints

Industry Canada suggested consideration be given to developing a weighting of evidence across endpoints: for example, identify that the evidence for endpoint 1 is strong and for endpoint 2 is weak, and perhaps value the impacts reflecting the strength of evidence. This is an interesting suggestion, but beyond the scope of the AQVM at this time. Currently, one can infer the quality of evidence from the coefficient spread and probability weights given to various endpoint concentration-response coefficients and economic values (less confidence results in larger variances), and from discussions about the individual endpoints. Weighting the values generated (e.g., some endpoints get a weight of 1, and others get weights of less than 1) is yet another level of complication (what weight to give) and may lead to misleading results. For example, instead of using the best (although uncertain) estimate, this approach would result in scaling downward the more uncertain estimates. Rather than weighting endpoints according to the amount of uncertainty in the estimates, we recommend that users conduct sensitivity analyses to understand

how results are affected by changes in the model assumptions, including elimination of endpoints.

Selection of Population Age Groups

Samet requested information regarding the basis for selecting age groups to be included in the model. The age categories were selected to be consistent with the information in the epidemiology and valuation studies used in the AQVM. Although other age groupings may, or may not, better reflect what are believed to be the most sensitive population groups, we define the risk groups to be consistent with the available literature results used for specific endpoints.

B.4 HEALTH ASSESSMENTS

B.4.1 Thresholds

Several reviewers noted the importance of the assumptions made with regard to health effects thresholds. We agree that this is an important question and that alternative assumptions can make a big difference in the magnitude of the benefits estimates. AQVM 2.0 makes a default assumption of no threshold, and gives the user the option to select daily and annual thresholds for PM health effects and a daily (high-hour) threshold for ozone health effects. Different reviewers argued different points of view on this question, and some suggested that the default assumption should be something other than no threshold. The bottom line is that there is not sufficient evidence at this time from either the epidemiologic or the toxicologic literature to provide a defensible basis for selecting a specific health effects threshold level for PM or ozone, but there is substantial epidemiologic evidence of some health risk below current air quality standards in Canada and the United States. Any default assumption made, therefore, could be debated. We urge users to examine alternative assumptions regarding health effects thresholds, and note that the default assumption in the model of no threshold gives the upper limit on health benefits for the current AQVM 2.0 endpoints and specifications.

B.4.2 PM Mortality Risk Estimates Based on a Combination of Short-Term and Long-Term Exposure Studies

Two reviewers (Krupnick and Vedal) raised some questions about combining evidence from two different types of mortality studies. Short-term exposure studies look at fluctuations in PM concentrations over time in a given location and long-term exposure studies look at differences in concentrations across locations. Because both types of studies show a statistically significant relationship between mortality and ambient PM concentrations, the conclusion that there is a causal relationship between PM and premature mortality is strengthened. These two types of studies have different strengths and weaknesses, so we chose to use both to develop estimates of

changes in mortality associated with changes in PM concentrations. Short-term exposure studies are not able to capture the effects of long-term exposures, but to the extent that locations with higher average exposures also have more frequent or higher short-term increases in PM concentrations, the effects of short-term exposures could be reflected in the long-term exposure studies. Also, there is some uncertainty about the significance of the shortening of life reflected in the short-term exposure studies it is difficult to say whether those who died prematurely would have otherwise lived additional days, weeks, or years. The long-term exposure studies necessarily reflect a substantial (i.e., years, not days) shortening of life otherwise the results would not be statistically significant. For both of these reasons, if we had to choose one type of mortality study for the model, it would be the long-term exposure studies. Because they show a larger mortality risk than the short-term exposure studies, relying exclusively on the long-term exposure studies would have increased the mortality risk estimates used in the model. We are therefore potentially understating the mortality risks by using a combination of the two types of studies.

B.4.3 Collinearity of Air Pollution Constituents

Industry Canada noted that there are uncertainties in the underlying health effects literature regarding the exact causal constituents of the observed health effects associated with particulate matter. The Industry Canada comments are correct in that an assumption in the calculation of the PM_{10} health effects is that every g/m^3 change in concentration has the same health effect regardless of its chemical composition. (The only exception to this assumption is for the sulphate version of AQVM, which includes sulphate specific concentration-response functions. The sulphate version is intended for use when the policy being evaluated is expected to affect primarily sulphur related emissions rather than all types of particulate matter and its precursors.) The Industry Canada comments correctly note that there may be more error in the PM health benefits estimates if a policy is targeting a single PM constituent rather than a broad range of PM constituents, however, they imply that the error is likely to be in the direction of overstating the health benefits of a PM reduction. Because the health benefits are based on the average health effect of all PM constituents, applying these to any single PM constituent is as likely to understate as overstate the benefit of a reduction in ambient concentrations.

Vedal noted that there are also uncertainties in the underlying health effects literature regarding the separation of health effects associated with particulate matter from those associated with ozone in locations where these pollutants fluctuate collinearly, and questioned whether ozone health effects can be assumed to be at the same level (per unit change) in locations where PM concentrations are not also simultaneously high. The second question applies specifically to the Ontario respiratory hospital admissions study and the California acute respiratory symptoms study. It is correct that it is difficult in epidemiologic studies to statistically isolate the independent effects of two pollutants that are highly correlated. However, we have taken several steps to minimize the chance of overstating the ozone health benefits because of collinearity with PM. First, all of the ozone health effects estimates are based on analyses that included a measure

of PM in the models, which reduces the chance that the ozone coefficients are upwardly biased because of collinearity with PM. Second, the most economically significant ozone health effect, premature mortality, is drawn from studies in many different locations across which the degree of collinearity between ozone and PM concentrations varies. Collinearity between ozone and PM is therefore less likely to be confounding the estimated effects of ozone on premature mortality. Finally, there is little evidence in the literature of significant synergisms between ozone and PM, such that the effect of ozone is enhanced when PM concentrations are higher, although this is a difficult question to assess and remains an area of uncertainty.

B.4.4 Other Issues

Ozone Mortality

Krupnick referred to premature mortality associated with ozone as a highly controversial endpoint, citing the U.S. EPA criteria document (U.S. EPA, 1996a). Studies published since the literature review for the criteria document was completed (1995 to 1997) provide increasingly strong evidence of a statistically significant ozone effect on mortality, although some studies continue to find no statistically significant effect. Based on this new literature, and the review of this literature for the U.S. EPA assessment of proposed changes to the ozone ambient air quality standards (U.S. EPA, 1997b), the mortality estimates for ozone in AQVM have been revised and reflect a stronger mortality effect than in the previous version. A low value of zero effect is still retained.

Region Specific Outcome Rates

Vedal suggested that the model would be improved by using region specific baseline outcome rates for mortality and hospitalization, because these health effects are calculated as a percentage change from baseline incidence rates. Currently, AQVM uses national average incidence baselines for these calculations and is not designed to compute impacts based on region specific baseline incidence data. This may be a useful future modification if there is meaningful variation (relative to the uncertainty in the concentration-response parameters) in regional rates for these variables.

Acute Morbidity Effects Estimates

Several specific issues were raised regarding a few of the acute morbidity health effects estimates. Vedal mentioned two additional studies to consider for acute respiratory symptoms for PM and ozone. With limited resources and a perpetually evolving literature, we focused our updates on premature mortality, the most significant endpoint from a valuation point of view, and on newly available Canadian studies. Users have the flexibility to change concentration-response functions to reflect results of new studies for any of the selected endpoints.

Krupnick mentioned "controversy" over the series of Ostro (and others) studies on restricted activity days. Issues raised in the citation he gives for this comment are primarily about the relatively high variability in the estimated ozone effects; this issue is discussed in detail in Chapter 4 of *Report 2*, and is accounted for in the selection of the range of estimates for ozone related, minor restricted activity days.

Samet questioned why the exacerbation of asthma by SO_2 was not considered. For the SO_2 standard, the U.S. EPA reviewed this issue and concluded that there were very few hot spots sufficient to potentially cause asthma aggravation for exercising adults. Because of the limited locations of concern and small population at risk (exercising asthmatics), we can expect this damage category to be de minimis relative to other damages for the intended applications of AQVM, which are large scale rather than for small point source control evaluations. See also Rowe et al. (1995) for additional discussion on this issue.

Air Toxics

Freeman and Samet mentioned a number of uncertainties and issues with the selection of air toxics to include, and with the measure of air toxics baselines and risk factors. These uncertainties are now identified in Section 4.5 of *Report 2: Methodology*. Industry Canada also reiterated the issues associated with the IRIS risk factors as overstating the central tendency. As noted in the methodology report, we concur, but at this time we have no other basis for developing cancer risk estimates for air toxics, or to adjust the IRIS risk factors to "central tendencies" in a manner that clearly reduces bias. That is, arbitrary reductions in the IRIS risk factors, or the use of zero, could overadjust and result in more bias than would occur from the use of the unadjusted risk factors. Because of the significant uncertainties and limitations in the air toxics literature, we conclude in Section 4.5 of *Report 2: Methodology* that the air toxics analysis is best viewed as indicative of the potential order of magnitude of damages. Future versions of the AQVM can incorporate, where appropriate, revised inhalation unit risk values for the air toxics, reflecting accepted changes in these values or results from studies that have become available since the previous version. In the meantime, the impact of alternative judgments of the appropriate risks can be evaluated through sensitivity analyses.

Adamowicz expressed concern that increased cancer risks today may, for those who survive the cancer, also lead to increased cancer risks in the future, and ignoring this could lead to understated damages. The damages we assign to cancer are based on observed five-year survival rates because subsequent cancers beyond five years are often considered in the medical community as new cases. Because the same cancer could again occur after five years, and could entail more costs or loss of life, the damages may be understated, but we expect this omission to be small relative to the overstatements in the IRIS risk factors. Beyond this, the link between cancer now and another unrelated cancer in the future is interesting speculation, but we have no literature to support the existence or quantification of this linkage.

Hospital Admissions

Vedal questioned the primary reliance on the Ontario study for hospital admissions because of concerns about whether the results can be generalized to settings that do not have coexisting particles and acid aerosols, as well as ozone. We relied primarily on this study for the estimates of hospital admissions because throughout the major population centres of the Windsor-Quebec corridor air quality conditions are reasonably similar, and because of our decision to emphasise Canadian studies. This seemed particularly important for hospital admissions because this morbidity endpoint might be significantly affected by the health care system. For example, we found that the reported hospital admission rates for respiratory and cardiovascular diagnoses were substantially lower in the Canadian studies than in the United States.

Samet also questioned why the Thurston et al. (1992, 1994) studies were not used for hospital admissions. As identified in the text, these studies are used as supporting evidence but not as a basis for quantification because either they do not separately identify impacts associated with particulates and ozone in a model that controls for both pollutants (1992 study) or they cover a limited period of time during what is expected to be the high pollution season (1994 study). Thus, these studies were judged to be less useful than other available studies that conducted year-round analyses and from which ozone and particulate concentration-response functions can be developed based on models in which both pollutants were controlled for (see also the discussion of pollutant collinearity in Section B.4.3 of this appendix).

B.5 NONHEALTH ASSESSMENT

B.5.1 Visibility Damages

Adamowicz raised several questions about the visibility valuation methods and assumptions, including that health and visibility benefits may have double counting, whether alternative functional forms (to the linear in percentage changes form used) might be appropriate, and that visibility values may differ between urban and other areas such as national parks. In response, as noted in Section 5.5.1 of *Report 2: Methodology*, the possibility exists for health values being included in the visibility values. However, several studies, and specifically the McClelland et al. (1991) study most heavily relied on, take specific steps to reduce this potential double counting. Next, alternative functional forms could be used, but the selected functional form is consistent with both the economics of diminishing marginal utility and the visibility perceptions research, which also supports a scale based on percentage changes in visibility conditions. Finally, the difference between visibility benefits for urban/residential locations and other locations is discussed in the AQVM report.

Desvousges noted that considerable emphasis is placed on the McClelland Two-Cities study and suggested that more discussion on the limitations of the study should be included. The AQVM

report is intended to be a summary report on methods. Additional detailed review of this and other visibility studies is provided in other literature, including Rowe et al. (1995) and Chestnut and Rowe (1990a), and is now identified in the text.

Industry Canada (in its appendix) made some comments and speculations regarding visibility valuation. These issues were already addressed in Section 5.5 of *Report 2: Methodology* and no further response is needed.

B.5.2 Agriculture Damages

Industry Canada, Desvousges, Krupnick, and Freeman noted that there are simplicities in the agricultural assessment that need a bit more identification, specifically that there is no production response, price response, or market distortion assumed in the assessment. We concur, and this is now briefly addressed in the text.

Adamowicz expressed concern about using solely 1994 for the baseline data. The revised AQVM now uses price and production data for 1993-1995, except for tobacco, which uses 1990-1995. Adamowicz further noted there is no concentration-response function for canola, the largest cash crop. The omission of canola and other crops results in understated benefits, so additional crops would be desirable to incorporate in future AQVM revisions. However, without a concentration-response function for the omitted crops, we do not have a basis for their inclusion. Concentration-response-function values for other crops could be selected by users in a what if sensitivity analysis, but cannot be incorporated into AQVM in a manner that clearly reduces bias (e.g., if the selected coefficient is too high, benefits could be overstated by more than they are currently understated). In addition, we were advised by agricultural experts familiar with the NCLAN study (John Lawrence at Boyce Thompson Institute at Cornell University) that it would be inappropriate to apply to canola any of the concentration-response functions for the other crops in AQVM. Further, as noted to us by Beverly Hale at the University of Guelph, canola is primarily a western Canada crops in areas where ozone levels are relatively low. Thus the omission of canola is not likely to be as significant as might be thought by just considering the production numbers.

B.5.3 Forests and Livestock

Adamowicz suggested that SO_2 forest and livestock damages could be considered for inclusion in the AQVM. These impacts are not in AQVM, but are an area of potential interest for future revisions if the literature will support benefit estimation. Based on the usefulness of the literature and on the likely relative magnitude of forest impacts vis-a-vis human health impacts, the Sulphur Study expert panel also concluded that it was not appropriate to use study resources to estimate economic impacts of SO_2 and sulphates on forests.

B.5.4 Acid Deposition and Recreational Fishing

Adamowicz and Desvousges raised a number of issues and provide recommendations to improve the recreational fishing assessment. Most of these recommendations have been implemented and are discussed in Section 5.5.4 of this report.

B.5.5 Materials

Krupnick noted that the quality of the literature used for materials damage estimates is relatively weak, an assessment with which we concur and identify in the report. Otherwise, only minor issues about materials were raised, which are generally already discussed in the AQVM report or are about methods that are routinely used in the air pollution control benefit studies identified in the introduction to this appendix.

B.6 ECONOMIC VALUATION

B.6.1 Use of Contingent Valuation Method Results

Industry Canada discussed at length many issues with the contingent valuation method (CVM), which uses surveys to determine environmental values. There is an extensive literature on this debate. Although we disagree with many of the specific points and literature interpretations presented in the comments, we do not discuss them in detail because they are largely irrelevant for the AQVM. This is because the focus of the CVM debate centres on the ability to accurately measure nonuse (or passive use) values, usually for environmental goods for which respondents may be somewhat to very unfamiliar. In contrast, the CVM literature used in the AQVM addresses active use values for human health impacts, recreational fishing impacts, and visibility impacts in residential settings. These three environmental goods are goods that respondents are familiar with. While there is imprecision in these use value estimates (and all economic value estimates), the measured use values are expected to provide reasonable indicators of true values, and not necessarily biased upward or downward. The reasonableness of the CVM values for health impacts is readily observed by comparing the health values to the associated health costs that individuals experience for the same endpoints (see Table 5-1 in *Report 2: Methodology* and discussion in Section B.6.2 below) and the respondents indication that health costs are generally not the sole, or often even the dominate, benefit of avoiding adverse health impacts. The acceptability of the CVM studies used is also reflected by the selection of the same or similar studies and values in each of the peer reviewed studies identified in the introduction to this appendix.

B.6.2 Valuation of Mortality Risks

Many of the reviewers offered comments or raised questions on the monetary valuation of mortality risks. Interpretation decisions regarding the available literature related to valuation of changes in mortality risks can have a substantial effect on the final benefits estimates for any air quality change scenario that results in a change in mortality risk. Given the importance of this issue and that there remain some significant uncertainties in the underlying epidemiologic and economic literatures, the discussion of this issue has been expanded in *Report 2: Methodology*.

One of the key issues noted by several of the reviewers and discussed in *Report 2: Methodology* is that the available economics literature focuses almost entirely on risks of accidental deaths for working age adults. Differences between risks of accidental death and the mortality risks associated with air pollution could cause differences in how much the individuals at risk are willing to pay to reduce the risk, but the currently available economics and epidemiologic literatures do not provide sufficient basis for quantitative adjustments for all the differences. We are therefore left with making some adjustments that have a reasonable empirical basis (i.e., the age adjustment), and simply listing the remaining uncertainties as important caveats to the AQVM estimates.

One assertion made by several of the reviewers (Industry Canada, Vedal, Krupnick, and Desvousges) is that individuals at risk from air pollution exposure are mostly elderly and in poor health. These comments presuppose that reducing air pollution extends life only for those whose quality of life is quite poor. The evidence is not sufficient at this time to exclude the possibility that reducing air pollution may extend periods of wellness prior to onset of chronic illness, as well as to reduce the severity of acute illnesses from which some individuals fully recover. For example, the significant increase in life expectancy in this century has not been to simply increase the period of poor health at the end of life. For example, life expectancy at birth in the United States has increased by about 6 years over the past 35 years, and by over 25 years over the past 70 years. Recent vital statistics demonstrate that remaining life expectancy has been increasing for individuals of all age groups.

There are some data from the short-term exposure studies that show there is indeed a different age distribution for those who experience the most risk from air pollution compared to those most at risk of accidental deaths. However, it does not appear to be only the elderly and the sick who experience air pollution risks. Many studies have not estimated risks by age group, but several have found statistically significant, although smaller, risks for those under age 65. Regarding the assertion that those at risk are likely to be in chronically poor health, we simply do not have any data on the health status of those at risk from air pollution versus the general population. This may be a plausible presumption regarding some of the individuals at risk of premature mortality from short-term fluctuations in air pollution exposures; however, some of those individuals may have a serious acute illness such as pneumonia, from which they might have fully recovered. With the long-term exposure studies, there is no reason to presume that reducing air pollution necessarily extends lives only for those who are already chronically ill. In

fact, for PM at least, it appears that air pollution exposure also contributes to the onset of chronic respiratory disease.

We have made some adjustments in the mortality valuation for the differences in the age distribution. The reviewers are correct in noting that this adjustment does not account for potential differences in health status, but it is not clear whether or how an adjustment for health status should be made.

Industry Canada misinterpreted the illustration of the Jones-Lee et al. (1985) results that provide the basis for the age weighted adjustment to the mortality risk WTP values. They say that the results are an outlier compared to the other studies presented. However, the Jones-Lee et al. study is the only one that does not impose constraining theoretical assumptions on the relationship between WTP and age. Most of the other results presented are based on theoretical, not empirical, analyses of the question of how age affects WTP for reducing immediate risk of mortality.

Industry Canada asserted that because of the likely age distribution of those at risk from air pollution exposure, we should be including WTP for younger people to have their risks reduced some years hence as well as WTP for the elderly for reductions in contemporaneous risks. This is not necessarily correct. With short-term exposure fluctuations for PM and ozone, the risk is immediate. What we want therefore is the WTP for immediate reductions in risk, weighted for the age distribution relevant to this risk. This is what the value selected for mortality risk reflects. For long-term exposure risks and other mortality risks with some latency such as cancers, what we want to know is the WTP for a shift in future survival probabilities that may extend many years into the future as a result of an exposure change that happens today. We have neither satisfactory epidemiologic data nor economic data necessary to make these calculations in this case. The epidemiology study for long-term PM exposures (Pope et al., 1995), for example, does not provide the information necessary to compute the length of time that an exposure must be elevated before increased mortality risks are observed.

Vedal raised the question of whether increases in public health care costs that may result if lives are extended because of reductions in air pollution might offset the benefits to the individual of being able to live longer. The economic interpretation of such expenditures is that they are undertaken because the value to society of the health care obtained is worth the cost of the health care expenditure. Therefore, if we extend lives by reducing air pollution exposure, we do not subtract the additional consumption expenditures for any goods and services, including health care, made as a result. This comment again presumes that reducing air pollution extends life only for those who are already old and sick.

B.6.3 WTP/COI Ratios

Both Krupnick and Adamowicz raised concern with the adjustment of COI measures of damage to obtain the desired WTP measure of damage (see Section 5.1.3 in *Report 2: Methodology*). Adamowicz suggests that WTP/COI may be greater than 2, but also highly variable across illness (we concur), and Krupnick would drop the correction altogether (we did not). On the other hand, the Industry Canada reviewer accepted the correction as reasonable given the existing literature.

First, we should put the issue in perspective. For a typical PM or sulphate application of AQVM, the mortality risk benefits dominate, followed by chronic bronchitis and acute respiratory symptoms, none of which are affected by the WTP/COI correction. For these applications, the WTP/COI adjustment will typically be about 2-3% of measured benefits. Second, using COI alone will result in a clear downward bias in benefit estimates for the affected endpoints. Using the proposed WTP/COI correction is expected to result in less bias for each endpoint, although bias may remain and may vary across endpoints. Specifically, where a ratio of 2 is used, bias will be increased only if the true ratio is between 1.0 and 1.5, and where a ratio of 1.5 is used, bias will be increased only if the true ratio is between 1.0 and 1.25. Because the literature suggests the WTP/COI ratio is in excess of 1.5 for the health effects considered, we have a consistent expectation that the correction factors used will reduce bias and are a reasonable, if not conservative, correction.

Finally, available evidence in addition to what is cited in Section 5.1.3 further supports that the WTP/COI ratios selected may be conservative and will reduce bias. Studies by Berger et al. (1987) report WTP and individual COI for cough, headache, and other minor symptoms with WTP/COI ratios exceeding those in Table 5-1. The same result is found in Dickie et al. (1991). Turning to more significant endpoints, Agee and Crocker (1996) report an individual WTP/COI for reducing lead in children of 3.1, which is consistent with the evidence in Table 5-1, and the U.S. EPA (1997b) estimates the individual s WTP to reduce moderate chronic bronchitis ($260,000 in $1996 US, as in AQVM) to be 3.4 to 6.3 times the individuals COI. Again, these results are consistent with the results in Table 5-1.

B.6.4 Chronic Bronchitis Valuation

Vedal expressed concern regarding the procedure to adjust the values for severe chronic bronchitis in the valuation study to assign a value to the level of severity in the Abbey et al. health study. This issue is already addressed in detail in Section 5.4.1 of *Report 2: Methodology*. Although this is a less than ideal procedure, the acceptance of the procedure is reflected in that it has been adopted, and peer reviewed, in each of the studies identified in the introduction to this appendix.

B.6.5 Other Economic Issues

Adamowicz noted issues with the use of a purchasing power parity index for converting nonmarket values as opposed to market values for which it was designed, and suggests that damages may be understated. He raises an interesting issue for continued investigation, but for which we do not believe an alternative strategy is ripe for inclusion in AQVM.

The potential for double counting, specifically for emergency room visits and hospital admissions was identified by several reviewers and (Industry Canada, Desvousges, and Krupnick) has been explicitly addressed in Section 4.2 of *Report 2: Methodology*.

Adamowicz noted that an average wage rate is used rather than regional wage rates. At present, AQVM is not set up to include regional differences in damages per health impact. Ideally we prefer to have WTP values, which too may vary by region. Wage rates are used where WTP estimates are not available. Wage rates are used to compute a work loss component of a COI estimates, which is then adjusted to WTP. Given the proxy nature of the work loss component of WTP, and the other approximations in this computation, we did not feel that the added precision of computing regional wage rates was merited.

B.7 OTHER ISSUES

Adamowicz, Desvousges, and Industry Canada (in its appendix) expressed concern about the ability to quantify impacts and damages for GHGs, and we concur. As noted in Section 5.5.5 of *Report 2: Methodology*, we do not endorse or propose a value per ton of GHGs. However, because of the potential significance of GHG emissions and benefits in some air pollution control scenarios, the model is designed to assist the user in investigating how computed benefits would change if one assumed alternative values for GHG emissions. For example, Rowe et al. (1995) show that when using the GHG values suggested by some regulators and in some literature, GHG emissions can be one of the more significant benefit categories for some electric power plant air emission control scenarios. Thus, the ability to investigate such issues may be beneficial for AQVM users. Recognizing the limitations in assigning benefits to GHG emission reductions, AQVM defaults to a $0/tonne value, which the user must replace to conduct sensitivity analysis on this issue. Industry Canada suggested that including the endpoint and allowing the user to input values may lead to users producing results "with the credibility of the AQVM behind them." This is clearly not the intent of incorporating the GHG options in the model, as has been clarified in the report discussions.

Industry Canada identified an issue of the "chilling effect of regulation" where there are other costs that are unmeasured and others that may be understated, and identifies other cost side issues. AQVM is a benefits model and we consider these issues no further.

APPENDIX C
COMMENTS ON DRAFT AQVM METHODOLOGY REPORT

APPENDIX D
CONCENTRATION-RESPONSE FUNCTIONS FOR $PM_{2.5}$ HEALTH EFFECTS

This appendix describes selected concentration-response functions for morbidity and mortality effects associated with airborne particulate matter 2.5 microns in diameter and smaller ($PM_{2.5}$). The $PM_{2.5}$ concentration-response functions are intended for analyses of policies or scenarios where $PM_{2.5}$ is the primary component of particulate air pollution affected. Concentration-response functions for other measures of particulate air pollution including sulphate aerosols (Appendix A) and PM_{10} (Chapter 4) are also available in AQVM 3.0. These concentration-response functions are meant to be mutually exclusive alternatives and are not intended to be used in combination with one another. The user should select the measure of airborne particulate matter that best reflects the expected change in air quality resulting from the scenario under consideration.

The concentration-response functions for $PM_{2.5}$ are similar to, and in some cases drawn from the same studies as, those for PM_{10} (see Chapter 4). This is consistent with the fact that $PM_{2.5}$ is a substantial component of PM_{10}. On average in Canada, $PM_{2.5}$ represents about 40% to 60% of PM_{10}. Recent research suggests that $PM_{2.5}$ is at least equally, and perhaps more, harmful than the larger size aerosols that make up the remainder of PM_{10} (U.S. EPA, 1997b). However, it is, difficult to determine the relative harmfulness of these two categories of particulates because they are typically highly correlated from day to day and from location to location. The concentration-response functions selected here for $PM_{2.5}$ show a somewhat more harmful effect relative to the concentration-response functions for PM_{10}.

Section D.1 summarises the selected $PM_{2.5}$ concentration-response functions. Sections D.2 and D.3 describe how each concentration-response function was developed from the available literature for $PM_{2.5}$ associated mortality and morbidity, respectively.

D.1 SELECTED CONCENTRATION-RESPONSE FUNCTIONS

Table D-1 lists the selected concentration-response functions for each of the $PM_{2.5}$ health effects categories. The selection criteria for the studies from which these concentration-response functions were drawn are described in Section 4.1.1. Preference was given to results in the literature obtained using $PM_{2.5}$ measures of particulate matter, but results based on other measures were used for health effects that have not been analysed using $PM_{2.5}$ measures.

Table D-1
Selected $PM_{2.5}$ Concentration-Response Functions for Human Health Effects

Health Effect Category	Concentration-Response Parameter (probability weights)
Annual mortality risk per 1 g/m^3 change in annual average $PM_{2.5}$ concentration. Sources: Pope et al. (1995); Schwartz et al. (1996a)	Low 0.87×10^{-5} (22%) Central 2.14×10^{-5} (67%) High 4.82×10^{-5} (11%)
Chronic bronchitis (CB) annual risk per 1 g/m^3 change in annual average $PM_{2.5}$ concentration. Source: Abbey et al. (1995b)	For population 25 years and older: Low 4.13×10^{-5} (25%) Central 8.27×10^{-5} (50%) High 12.4×10^{-5} (25%)
Respiratory hospital admissions (RHA) daily risk factors per 1 g/m^3 change in daily average $PM_{2.5}$ concentration. Source: Burnett et al. (1995)	Low 1.00×10^{-8} (25%) Central 1.21×10^{-8} (50%) High 1.42×10^{-8} (25%)
Cardiac hospital admissions (CHA) daily risk per 1 g/m^3 change in daily average $PM_{2.5}$ concentration. Source: Burnett et al. (1995)	Low 0.79×10^{-8} (25%) Central 1.02×10^{-8} (50%) High 1.26×10^{-8} (25%)
Net emergency room visits (ERV) daily risk factors per 1 g/m^3 change in daily average $PM_{2.5}$ concentration. Source: Stieb et al. (1995)	Low 4.62×10^{-8} (25%) Central 5.61×10^{-8} (50%) High 6.61×10^{-8} (25%)
Asthma symptom day (ASD) daily risk factors given a 1 g/m^3 change in daily average $PM_{2.5}$ concentration. Sources: Whittemore and Korn (1980); Ostro et al. (1991)	For population with asthma (6%): Low 1.62×10^{-4} (33%) Central 2.64×10^{-4} (34%) High 3.65×10^{-4} (33%)
Restricted activity day (RAD) daily risk factors given a 1 g/m^3 change in daily average $PM_{2.5}$ concentration. Sources: Ostro (1987); Ostro and Rothschild (1989)	For nonasthmatic population (94%) 20 years and older: Low 1.31×10^{-4} (25%) Central 2.50×10^{-4} (50%) High 3.95×10^{-4} (25%)
Net day with acute respiratory symptom (ARS) daily risk factors given a 1 g/m^3 change in daily average $PM_{2.5}$ concentration. Source: Krupnick et al. (1990)	For nonasthmatic population (94%): Low 1.25×10^{-4} (25%) Central 2.79×10^{-4} (50%) High 4.14×10^{-4} (25%)
Child acute bronchitis (B) annual risk factors given a 1 g/m^3 change in annual average $PM_{2.5}$ concentration. Source: Dockery et al. (1996)	For population under age 20: Low 0.62×10^{-3} (25%) Central 1.65×10^{-3} (50%) High 2.69×10^{-3} (25%)

For each health effect category, low, central and high concentration-response parameters are developed and assigned probability weights that are used in the uncertainty analysis. The general rationale and procedures for selecting and weighting the low, central, and high parameters are described in Section 4.1.6. The uncertainty analysis in AQVM 3.0 is explained in Chapter 6.

Some of the health effects categories reported in the literature may overlap. For example, acute respiratory symptoms days (ARSs) probably include some days that are also restricted activity days (RADs). To avoid double counting health effects, we make some adjustments in the concentration-response parameters. We assume that all pollution-related respiratory and cardiac hospital admissions (RHAs and CHAs, respectively) involve an initial emergency room visit (ERV). We also assume that all pollution-related restricted activity days (RADs) are also acute respiratory symptom days (ARSs). As a result, the following subtractions are made to calculate net $PM_{2.5}$ health effect parameters for each of these categories:

$$\text{Net ERVs} = \text{Total ERVs} - (\text{RHAs} + \text{CHAs}) \quad \text{(D-1)}$$

$$\text{Net ARSs} = \text{Total ARSs} - \text{RADs}. \quad \text{(D-2)}$$

D.2 PREMATURE MORTALITY

Over the last few decades, many epidemiologic studies have found statistically significant associations between ambient airborne concentrations of particulate matter and mortality among the general population. The earliest of these mortality studies focused on relatively rare episodes of extremely high pollution concentrations in the 1940s and 1950s in the United States and the United Kingdom (U.S. EPA, 1982a). More recent studies have found an association at concentration levels typical of most metropolitan areas in North America [e.g., Dockery and Pope (1994) review this literature].

The earliest studies of this type were cross-sectional studies examining annual mortality rates across U.S. cities with different average particulate matter levels, often including 100 or more cities (e.g., Lave and Seskin, 1977; Evans et al., 1984; Ozkaynak and Thurston, 1987, Lipfert, 1994). More recently, many time-series studies have found statistically significant associations between daily mortality and daily fluctuations in particulate matter concentrations in a wide range of cities (e.g., Pope et al., 1992; Schwartz and Dockery, 1992a,b). Very recently, two prospective studies using individual-specific data and tracking mortality for a study sample in multiple cities over multiple years, found associations between survival rates and particulate matter concentrations (Dockery et al., 1993; Pope et al., 1995).

Some skepticism remains about whether these studies reflect a true causal relationship primarily because a specific biological mechanism to fully explain and verify this relationship has not been demonstrated in clinical or laboratory research (Utell and Samet, 1993). However, epidemiologic

studies are consistently finding a statistically significant association between air pollution and mortality, using different study designs and locations, and over a wide range of particulate matter concentrations, including levels well below the current Canadian objectives or U.S. standards. In addition, recent controlled animal exposure studies have begun to suggest plausible mechanisms by which severe effects, including death, may occur after concentrated ambient air pollution exposure (e.g., Godleski et al., 1996). Godleski et al. s work, it should be noted, was conducted at higher than ambient concentrations (up to 30 times), for very short periods (3 days). Future research may shed more light on the mechanism by which particulate matter increases mortality risk and such findings might suggest necessary revisions in the way we are estimating particulate matter health effects in AQVM 3.0. In the meantime, we are using results from available studies in a way that represents a reasonable interpretation of the available evidence.

D.2.1 Summary of Selected Quantitative Evidence

This section does not provide a detailed review of all available literature, but focuses on the available results that are best suited for the purposes of this analysis. The study selection process relied on the study selection criteria previously discussed in Section 4.1.1. The selected studies incorporate results from prospective cohort and time-series studies. From both perspectives the results show an association between mortality and particulate matter, and results from both types of studies are used in developing concentration-response parameters for use in AQVM 3.0.

Long-Term Exposure Studies

Two types of long-term exposure studies have found statistically significant associations between mortality rates and air pollution levels in the United States. The first type is an ecologic cross-sectional study design in which mortality rates for various locations are analysed to determine if there is a statistically significant correlation between the rates and average air pollutant levels in each location. Such studies have consistently found measurably higher mortality rates in metropolitan areas with higher average levels of particulate matter. However, concern persists about whether these studies have adequately controlled for potential confounding factors. Lipfert (1994), Ozkaynak and Thurston (1987), and Evans et al. (1984) provide examples of ecologic cross-sectional studies. These studies each conducted a thorough examination of data, including average particulate matter (PM) or sulphate concentrations, from 100 or more U.S. metropolitan areas with special emphasis place on controlling for the effects of potential confounding factors such as occupation, education, or migration. The findings of these studies vary in terms of pollutants found to be significant and the estimated magnitudes of the PM effects.

A second type of long-term exposure study is a prospective cohort study in which a sample population is selected and followed over time. In 1993, Dockery et al. published results for a 15-year prospective study based on samples of individuals in six cities in the United States. In 1995, Pope et al. published results of a seven-year prospective study conducted in collaboration

with the American Cancer Society based on samples of individuals in 151 cities in the United States. These studies are similar in some respects to the ecologic cross-sectional studies because the variation in pollution exposure is measured across locations rather than over time using average pollutant levels measured at nearby stationary outdoor monitors. However, the mortality data are for identified individuals, which enables much better characterization of the study population and other health risks than when area-wide mortality data are used. With the individual-specific data, the authors of the prospective studies were able to control for mortality risks associated with differences in body mass, occupational exposures, smoking (present and past), alcohol use, age, and gender.

Dockery et al. (1993) found a mortality-rate ratio of 1.26 over the 15-year study period from the most polluted to least polluted cities. Pope et al. (1995) found a statistically significant mortality-rate ratio of 1.17 for highest to lowest median $PM_{2.5}$ concentrations over 50 U.S. cities during a seven-year study period. Abbey et al. (1991) did not find any evidence of premature mortality associations with air pollution in a smaller, nonsmoking cohort in California in a 10-year prospective analysis.

The Pope at al. study in particular is a very important contribution to the study of mortality and particulate matter because of its prospective design and the very large number of study locations and subjects involved. The findings of a statistically significant association between mortality and particulate matter in this study are very supportive of similar findings in some of the previous single-year cross-sectional studies. The Pope et al. (1995) $PM_{2.5}$ results reflect the analysis of data for over 295,000 subjects and 50 metropolitan areas over a seven-year period from 1982 to 1989. By comparison, the Dockery et al. (1993) prospective cohort study analyses data for 8,111 adults from six eastern U.S. cities over a 14-year period. The Pope et al. (1995) study developed risk ratios from Cox proportional hazard models in which the median fine particulate concentration for a metropolitan area from 1979 to 1983 was entered as an independent variable along with socioeconomic variables accounting for, among other factors, a subject s education, smoking status, and alcohol consumption. In addition, meteorological controls were included to account for relatively hot or cold conditions.

The prospective cohort studies also address the question regarding the degree to which the time-series results really represent a meaningful shortening of life. The prospective and cross-sectional studies would not reveal a statistically significant relationship if particulate exposures were associated only with deaths that would have occurred anyway within a very short time period (days or weeks).

The prospective studies have been criticized by some (Lipfert, 1995; Vedal, 1997) for their inability to account for potentially different historical levels of air pollution than indicated by the data available for the published analyses. These studies were also criticized for not more directly or fully accounting for potential confounding by differences in diet, physical activity, and

socioeconomic status. Nevertheless, these studies support an association between life expectancy and annual average particulate levels.

The results of the two prospective studies are summarised in Table D-2, along with the results from an annual cross-sectional study. For comparison purposes, the results were converted to percent change in mortality per 10 g/m^3 of $PM_{2.5}$.

Table D-2
Comparison of Long-Term Exposure Mortality Study Results

Study	Time Period	Number of Cities	Particulate Measure	Particulate Mean (g/m^3)	Estimated % Change in Mortality per 10 g/m^3 $PM_{2.5}$
Pope et al. (1995)	1982-1989	50	$PM_{2.5}$	20.2	5.8%
Dockery et al. (1993)	1974-1989	6	$PM_{2.5}$	18.0	7.4%
Ozkaynak and Thurston (1987)	1980	98	$PM_{2.5}$	23.1	2.7%

Time-Series Studies of Acute Exposure

In recent years, numerous studies using time-series methods have indicated acute air pollution exposure impacts daily mortality. In general, time-series studies use observed air quality data and health incidence data aggregated for a population in a defined area to determine whether the incidence is affected by fluctuations in the concentration of the air pollutant(s). The primary strength of time-series studies is that in evaluating a well defined population over a relatively short period of time it can be assumed that the population is not experiencing dramatic socio-economic shifts. As a result, the study population acts as its own control, eliminating the need to define variables to describe and statistically control for changes in a population (e.g., race, income, education).

Many daily time-series studies for cities throughout the world have found statistically significant relationships between daily fluctuations in particulate matter concentrations and daily fluctuations in nonaccidental mortality rates. Dockery and Pope (1994) reviewed the time-series results with regard to PM_{10} and found that the estimated effects ranged from 0.5% to 1.5% change in daily nonaccidental mortality for every 10 g/m^3 change in daily PM_{10}, with an average result of approximately 1.0%. Schwartz et al. (1996) conducted a daily mortality time-series analysis with pooled data from six U.S. cities (the same cities used in the Dockery et al., 1993, prospective cohort study) and $PM_{2.5}$ as the measure of airborne particulate matter. This is the most comprehensive daily time-series analysis conducted to date using $PM_{2.5}$ data. The result

from the pooled analysis shows a 1.5% change in daily mortality for a 10 g/m^3 change in daily $PM_{2.5}$.

D.2.2 Annual Mortality Concentration-Response Parameters

In this analysis, we consider both time-series studies of daily mortality counts and cross-sectional studies of annual mortality rates. Results from a study employing time-series methods are used here to develop the low $PM_{2.5}$-related premature mortality concentration-response parameter while the high parameter is developed from a prospective cross-sectional study. The central parameter is a weighted average of the low and the high estimates. Consistent with the procedure developed by the sulphate panel (Thurston, 1997b), we give a two-thirds to one-third relative weighting of the time series (low parameter) and cross-sectional (high parameter) studies, respectively, in the development of the central parameter. The concentration-response parameters are developed by multiplying the estimates of the percentage change in mortality per g/m^3 of $PM_{2.5}$ associated with the low, central, and high estimates by the prevailing Canadian baseline for all-ages annual nonaccidental mortality of 6.7 per 1,000 (World Health Organization, 1994).

The Schwartz et al. (1996) study is used to develop the low $PM_{2.5}$-related mortality concentration-response parameter because it is a recent study that used $PM_{2.5}$ as its particulate matter measure of air quality and incorporated data from a number of locations. This parameter was derived from the estimated mean mortality effect minus one standard deviation, determined based on the reported 95% confidence interval. From the reported central estimate of a 1.5% increase in daily mortality for a 10 g/m^3 change in $PM_{2.5,}$ and a upper bound effect from the 95% confidence interval of 1.9%, a standard deviation of 0.2% is estimated. With this information the low parameter is estimated as follows: (1.5% - 0.2%) ÷ 10 g/m^3 $PM_{2.5}$ = 0.13% change in mortality per g/m^3 $PM_{2.5}$.

Numerous cross-sectional studies in the literature, as described above, have indicated that, after controlling for potential confounders, places with higher particulate matter concentrations have higher annual mortality rates. The results of the Pope et al. (1995) study support the findings of earlier cross-sectional studies. However, because of its prospective cohort design, it could control for potential confounders (such as smoking) at an individual level rather than at the aggregate city level as in prior ecologic studies. Also, the Pope et al. study included a large study sample across a large number of North American cities. As a result, we employ the results of the Pope et al. (1995) study to develop our high $PM_{2.5}$-related mortality concentration-response parameter. The high parameter is derived from the Pope et al. (1995) study by calculating the mean mortality effect plus one standard deviation as follows: the reported $PM_{2.5}$ risk ratios are for a 24.5 g/m^3 change in annual median $PM_{2.5}$ from the most to the least polluted city, we estimate that this is

equivalent to a 27.2 g/m^3 change in annual mean $PM_{2.5}$.[1] With this change in $PM_{2.5}$, the reported central risk ratio value of 1.17, and the upper 95% confidence interval value of 1.26, the associated premature mortality coefficient was calculated as follows:

$$\text{High mortality coefficient} = 1 - (\exp(\ln(1.17 + 0.045) \div 27.2)) = 0.0072 . \quad \text{(D-3)}$$

Following the procedure described above, the central parameter is thus:
$(0.13\% \times 0.67) + (0.72\% \times 0.33) = 0.32\%$. The estimated percent change in mortality per g/m^3 annual average $PM_{2.5}$ are thus:

low	0.13%
central	0.32%
high	0.72%.

$PM_{2.5}$-related mortality concentration-response parameters are developed using the average annual Canadian nonaccidental mortality rate of 6.7 per 1,000 people and the low, central, and high percentage changes selected above. For example, the central estimate is 0.53% of 6.7 divided by 1,000. The selected mortality risk concentration-response parameters and calculation procedures are thus:

$$\text{low annual } PM_{2.5} \text{ mortality risk} = 0.87 \times 10^{-5} \times POP_j \times PM_{yj} \quad \text{(D-4a)}$$
$$\text{central annual } PM_{2.5} \text{ mortality risk} = 2.14 \times 10^{-5} \times POP_j \times PM_{yj} \quad \text{(D-4b)}$$
$$\text{high annual } PM_{2.5} \text{ mortality risk} = 4.82 \times 10^{-5} \times POP_j \times PM_{yj} \quad \text{(D-4c)}$$

where:

POP_j = total population in area j
PM_{yj} = change in annual average $PM_{2.5}$ (in g/m^3) in area j.

Consistent with the PM_{10} and sulphate results, these concentration-response parameters are assigned the following probability weights: low, 22%; central, 67%; high, 11%.

Evidence on Who Is at Risk

The results of the Philadelphia study (Schwartz and Dockery, 1992a) provide estimates of elevated mortality risks separately for those over and under 65 years old. These results suggest that about 90% of the premature deaths associated with particulate matter occur in the over-65 age group. This finding is consistent with the results of an early cross-sectional mortality study

1. The Pope et al. (1995) study reports a relative risk (RR) of 1.17 for an incremental change of 24.5 g/m^3 in annual median $PM_{2.5}$ from the least to the most polluted cities. Based on our analysis of available monitor data for $PM_{2.5}$ in the United States, the average ratio between annual median and annual mean concentrations is 0.9. This is therefore equivalent to an increment in annual mean $PM_{2.5}$ of about 27.2 g/m^3.

(Lave and Seskin, 1977). Ostro et al. (1996) found that about 80% of the premature deaths associated with particulate matter were in the over-65 age group in their Santiago, Chile, study. In the United States, about 70% of all deaths are individuals 65 or older, so it appears that risks associated with air pollution exposure fall in somewhat greater proportion on the elderly.

The results from Pope et al. (1995) show that the greatest association is with deaths associated with cardiopulmonary illness and lung cancer, and that elevated mortality risks are similar for both smokers and nonsmokers in higher pollution locations. Some of the time-series studies (e.g., Schwartz and Dockery, 1992a) have also found significant cause-specific mortality associations indicating that most pollution-associated deaths are cardiopulmonary-related. Some of those at risk therefore probably suffer from chronic diseases that might be expected to shorten life expectancy even in the absence of air pollution. This does not, however, rule out the possibility that some of these chronic illnesses could themselves be related to air pollution exposure.

As discussed in Chapter 5, the age of the individual at risk of premature mortality may have some bearing on the monetary value of changing that risk. For the purposes of this analysis, it is presumed from evidence in Ostro et al. (1996) and Schwartz and Dockery (1992a) that 85% of the individuals at risk of premature mortality associated with PM are 65 years old or older.

D.3 CHRONIC AND ACUTE MORBIDITY

In this section we describe the development of concentration-response parameter estimates for the $PM_{2.5}$ morbidity effects. Epidemiologic studies have found associations for $PM_{2.5}$ with morbidity effects ranging from chronic bronchitis and elevated hospital admissions rates to small differences in lung function measurements. The studies selected as the basis for quantitative estimates in this report provide evidence for a ranges of illnesses and symptoms likely to have some economic significance; this means symptoms that are noticeable to the subject and can be expected to have some impact on the individual s well-being. For this reason, studies that only evaluate effects on lung function have not been included. Although this may be a medically relevant health endpoint, it cannot at this time be translated into changes in symptoms or illness that can be readily valued.

D.3.1 Chronic Respiratory Disease

For at least the past two decades, there has been some evidence suggesting that higher ambient particulate matter exposures are associated with higher rates of chronic respiratory disease. Much of this evidence, however, has been based on cross-sectional analyses, comparing disease or symptom prevalence rates in different communities with different average pollution levels (e.g., Ferris et al., 1973; 1976; Hodgkin et al., 1984; Portney and Mullahy, 1990). These studies

can suggest a possible association, but are difficult to use for quantitative estimates of specific concentration-response functions because they look at differences in prevalence rather than just new cases of chronic illness.

Recently published articles (Abbey et al., 1991; 1993) have reported results of a 10-year cohort study conducted at Loma Linda University in California with a large sample of nonsmoking adults. The follow-up evaluations in this study allowed for the development of information on changes in chronic respiratory disease incidence over time and exposure measures for the 10-year period. Thus, new cases of disease were analysed in relation to pollution exposure for a matching time period. This study provides, for the first time, a concentration-response function for new cases of chronic respiratory disease. However, uncertainties about the nature of the exposure that leads to chronic illness, and lag times between exposure and illness onset still exist with these findings. This difficulty stems primarily from uncertainty about how to characterize the relevant exposure units, in particular the time aspects of exposure. Chronic symptoms presumably occur as a result of long-term exposures, but cross-sectional analyses are not very enlightening about whether, for example, it is the five-year average, the twenty-year average, or the number of times a given concentration is exceeded that is the relevant exposure measure. Application of the concentration-response function from Abbey et al. in this analysis therefore requires some assumptions on this that are explained below.

The Loma Linda University Study

In the first stage of the Loma Linda University study, a large sample of approximately 7,000 Seventh Day Adventists (selected because they do not smoke) was interviewed in 1977. Health histories, current respiratory symptoms, past smoking and passive smoking exposure, and residence location histories were obtained. Hodgkin et al. (1984) compared the chronic respiratory disease status of respondents who had lived for at least 11 years in either a high or low pollution area in Southern California. After adjusting for sex, race, age, education, occupational exposure, and past smoking history, residents of the higher pollution area had a rate of chronic obstructive pulmonary disease (COPD) (including chronic bronchitis, asthma, and emphysema) that was 15% higher than for residents in the low pollution area. Using the same 1977 Loma Linda sample, Euler et al. (1987) reported results showing a statistically significant association between past TSP exposure, based on residence ZIP code history, and the prevalence of chronic respiratory disease.

Abbey et al. performed a cohort study with the Seventh Day Adventist sample in 1987, which provides better quantitative concentration-response information. Nearly 4,000 subjects who were at least 25 years old when initially interviewed in 1977 were interviewed again in 1987. Estimates of air pollutant exposures were developed based on subjects reported residence locations and pollutant measures from stationary outdoor monitors from corresponding locations over the 10-year period.

Several health outcomes were examined in 1987 including the incidence of new cases of emphysema, chronic bronchitis, or asthma among those who did not report any definite symptoms of these diseases in 1977. Disease definition was based on self-reported symptoms using the standardized respiratory symptoms questionnaire developed by the National Heart and Lung Institute for the United States. Respondents were classified as having *definite* symptoms of emphysema, chronic bronchitis, or asthma if they met specific criteria for the disease diagnosis. Having definite symptoms of any one of these three diseases was defined as definite airway obstructive disease (AOD). Having definite chronic bronchitis was determined based on having symptoms of cough and/or sputum production on most days for at least three months/year for two years or more. Emphysema and asthma required physician s diagnosis as well as associated symptoms. Respondents with some respiratory symptoms, but who did not meet the full criteria, were classified as *possible* for that disease.

Logistic models were estimated for mean concentrations of air pollutants and for hours above selected levels for each pollutant. The regressions included independent variables for past and passive smoking exposure, possible symptoms in 1977, childhood respiratory illness, gender, age and education. Abbey et al. (1993) report a statistically significant association between average long-term TSP exposure levels and AOD, as well as with chronic bronchitis alone. About 85% of AOD cases included a diagnosis of chronic bronchitis.

Abbey et al. (1995b) report statistically significant associations between TSP exposure and new cases of AOD, as well as with new cases of chronic bronchitis and new cases of asthma (which are two types of AOD). The magnitude of the TSP results from this analysis was consistent with the previously reported results (Abbey et al., 1993). The authors also report a statistically significant association between new cases of chronic bronchitis and $PM_{2.5}$, and between new cases of asthma and the sulphate measure. The magnitudes of the reported odds ratios for new cases of AOD were similar for selected changes in TSP, $PM_{2.5}$, and sulphates, but the result was statistically significant only for the TSP measure. The authors note that there is probably more measurement error in the $PM_{2.5}$ exposure estimates because of the approximation from airport visibility.

Abbey et al. (1995b) also report evidence of a statistically significant association between increased severity of AOD and TSP, $PM_{2.5}$, and sulphate exposure for those who reported definite symptoms in 1977. Thus, it appears that particulate matter exposure both causes new cases and aggravates existing cases of AOD. Also very important, is the authors conclusion that exposures to gaseous pollutants did not appear to be a significant confounding factor in the measured association between particulate matter exposure and incidence of chronic respiratory disease.

Two uncertainties in the quantitative estimates based on Abbey et al. (1993) should be noted. First, the authors report that a few subjects who initially describe symptoms that are classified as chronic bronchitis do not continue to report these symptoms in follow-up evaluations. This suggests reversibility in the symptoms for some subjects that is not consistent with how chronic

bronchitis is defined in the economics studies that have estimated monetary values for reducing risks of developing chronic bronchitis. This does not invalidate the relative risk for self reported symptoms in relation to pollution exposure, but it raises some questions regarding monetary valuation of these cases. This is discussed further in Chapter 5. The second uncertainty is how long a change in $PM_{2.5}$ exposure must exist before a change in chronic bronchitis incidence occurs. The estimates are annualized here based on the assumption that the change in risk begins as soon as the change in air pollution exposure begins. This probably overstates the change in new chronic bronchitis cases in the first few years after a reduction in $PM_{2.5}$ concentrations, but there is not enough information from the study determine what the lag between changes in exposure and changes in risk may actually be.

Selected Chronic Respiratory Disease Risk Estimates from Abbey et al. (1995b)

We have selected the $PM_{2.5}$ chronic bronchitis results from Abbey et al. (1995b) to develop a chronic bronchitis concentration-response function for this analysis. The estimates used in this analysis reflect only the development of new cases, not the aggravation of existing cases. Limitations in both the $PM_{2.5}$ and the sulphate data available for this analysis contribute to the ambiguity in the findings. The somewhat weak statistical significance of the $PM_{2.5}$ results from the study is troubling with respect to this quantification approach, but the limitations in the available $PM_{2.5}$ data forced a smaller sample size. The magnitude of the $PM_{2.5}$ effect is supported by the more robust TSP results.

Abbey et al. (1995b) report a central relative risk for developing a new case of chronic bronchitis of 1.81 associated with an increase in average $PM_{2.5}$ exposure of 45 g/m^3 over the study s 10-year follow-up period. This means that the incidence of new cases of chronic bronchitis is 1.32% higher, $\ln(1.81) \div 45$, for every 1 g/m^3 increase in average $PM_{2.5}$ concentrations. The 10-year incidence of new cases of chronic bronchitis was about 6.26% (117 new cases out of 1,868 individuals in the sample for which $PM_{2.5}$ exposures were estimated). Thus, an individual s probability of developing chronic bronchitis in the 10-year period per 1 g/m^3 increase in average $PM_{2.5}$ concentration is $0.0132 \times 0.0626 = 0.000827$. We divide this risk by 10 to obtain an annual central concentration-response parameter for $PM_{2.5}$-related chronic bronchitis. The high and low parameters are based on the study s estimate of the impact of a 1 g/m^3 increase in average $PM_{2.5}$ concentrations plus and minus one standard error, determined from the reported 95% confidence interval for the relative risk in the study, respectively. The selected low, central, and high concentration-response parameters for $PM_{2.5}$-related chronic bronchitis are thus:

$$\text{low annual new cases of CB} = 4.13 \times 10^{-5} \times POP_{25j} \times PM_{yj} \quad \text{(D-5a)}$$

$$\text{central annual new cases of CB} = 8.27 \times 10^{-5} \times POP_{25j} \times PM_{yj} \quad \text{(D-5b)}$$

$$\text{high annual new cases of CB} = 12.4 \times 10^{-5} \times POP_{25j} \times PM_{yj} \quad \text{(D-5c)}$$

where:

CB = adult chronic bronchitis
POP_{25j} = population 25 years and older in area j
PM_{yj} = change in annual average $PM_{2.5}$ in area j.

We give the central parameter a 50% weight and the low and high parameters weights of 25% each and apply the estimates to the adult population age 25 and over because this is the minimum age of the individuals initially included in the Abbey et al. study group.

Threshold Evidence for Chronic Respiratory Disease

The same uncertainty exists regarding the potential existence and level of a threshold for chronic effects of long-term particulate matter exposure as for health effects associated with short-term exposures, but some additional comments are warranted. There is no clear a priori reason to expect that a threshold for short-term exposures would necessarily be the same, higher, or lower than a threshold for long-term exposures.

Two studies conducted to date provide some suggestive evidence that there may be a threshold level for chronic respiratory effects associated with particulate matter exposures. As noted above, Abbey et al. (1991, 1993) report no significant relationship between any chronic respiratory effects and hours above 60 or 75 $\mu g/m^3$ TSP, but do report a significant association for hours above 100 $\mu g/m^3$. They also report a significant association with mean TSP levels, and report that about 25% of the sample was exposed to mean TSP levels of 75 $\mu g/m^3$ or less. These results do not prove whether or not it is mean exposure or peak exposure, or some combination of the two, that causes the elevated risk, nor do they prove the existence of a threshold. In this analysis, concentration-response parameters for chronic bronchitis based on average particulate matter exposures is selected, but this does not exclude the possibility that it is the peak levels associated with a given average level that actually cause the risk rather than chronic exposure to low or moderate levels.

The Abbey et al. results suggest that if hourly levels of TSP do not exceed 100 $\mu g/m^3$, there does not appear to be an elevated risk of developing chronic respiratory disease. However, hourly peaks of TSP above 100 $\mu g/m^3$ are quite common in urban areas even when annual average TSP concentrations are well below 70 $\mu g/m^3$ (the current Canadian acceptable objective of annual average TSP).Given this and that the current Canadian acceptable objective for TSP is 120 $\mu g/m^3$ for a 24-hour average, these findings suggest that if a threshold exists it is well below the current Canadian acceptable objective for particulate matter.

Chestnut et al. (1991) report that lung function is lower in locations with quarterly TSP levels above 60 $\mu g/m^3$ TSP. This translates to about 33 $\mu g/m^3$ PM_{10}. A quarterly average can exceed

33 g/m^3, although annual averages are below this level. In any case, 33 g/m^3 is well below the current U.S. federal annual average PM_{10} standard of 50 g/m^3.

Neither of these studies provide definitive information on whether a chronic effects threshold exists or, if it does, what it would be in terms of annual average particulate matter levels, except that it appears to be well below current federal standards.

D.3.2 Hospital Admissions

Several studies have used Canadian hospital admissions and air pollution monitoring data to examine the relationship between airborne particulate matter levels and hospital admissions with a primary diagnosis of respiratory or cardiac disease (Burnett et al., 1994, 1995, 1997; Thurston et al., 1994). Following the study selection criteria discussed in section 4.1.1, we have selected the results from the Burnett et al. (1995) study to develop concentration-response parameters for $PM_{2.5}$-related respiratory and cardiac hospital admissions. The Burnett et al. results were selected because the study accounted for year round admissions in both admissions categories in a sizable, but well defined, population using models that included controls for ozone. In addition, because the sulphate measure of particulate matter used in the study is a component of $PM_{2.5}$ the results provide a strong basis for extrapolating to $PM_{2.5}$ results.

Burnett et al. (1995) examined the impact of sulphate and ozone on respiratory and cardiac hospital admissions using admissions data from 168 acute care hospitals below the 47th parallel in Ontario, Canada and air pollution data from a large network of monitors existing throughout Ontario for the period 1983-1988. Time-series regression models were used that controlled for the influences of temperature, day-of-week effects, slow moving serial correlations due to seasonal patterns, and differences between hospitals while including measures of sulphate and ozone as explanatory variables. It is important that this analysis controlled for ozone while examining the effect of particulate matter because in this region sulphate and ozone concentrations are highly collinear and a model that includes only a particulate matter measure chances overstating the effect attributable to particulates.

The other hospital admissions studies using Canadian data provide support for the results from Burnett et al. (1995) but have some limitations that make them less suitable for use in this analysis. The Burnett et al. (1994) study used the same data as in the 1995 analysis but considered different regression models where several sulphate and ozone variables were in the models simultaneously. In addition, the Burnett et al. (1994) study only reported the significance of the entire model instead of presenting estimates of the significance of the individual variables. The Burnett et al. (1997) study used data from 16 Canadian cities from 1981 to 1991 but only examined respiratory hospital admissions and used coefficient of haze as the measure of particulate matter. The Thurston et al. (1994) study examined the relationship of air pollutants to

respiratory hospital admissions in metropolitan Toronto, but covered only a six-week period in July and August in 1986-1988.

Respiratory Hospital Admissions

Burnett et al. (1995) report a 3.5% increase in respiratory hospital admissions (RHAs) associated with a 13 g/m^3 increase in sulphate, based on the results from a model that included ozone. The average number of RHAs per day for the study period was 16.0 per million population. As a result, 3.5% of the 16.0 daily RHA are attributed to a 13 g/m^3 increase in sulphate. Therefore, the daily RHA risk factor per g/m^3 sulphate is: $0.035 \times (16.0 \times 10^{-6}) \div 13 = 4.31 \times 10^{-8}$. This sulphate-based result is converted to its $PM_{2.5}$ equivalent using an estimated average sulphate to $PM_{2.5}$ ratio of 0.28 for Ontario based on air pollution monitoring data from all seasons during the period 1984-1997 (Tom Dann, personal communication, 1999).[2] Multiplying the sulphate-based result by this conversion factor provides the central daily RHA concentration-response parameter per g/m^3 of $PM_{2.5}$. Thus, the $PM_{2.5}$ concentration-response parameters for RHAs are as follows, with the low and high parameters representing the central minus and plus one standard error respectively (the standard error associated with the initial estimate can be calculated based on the reported 95% confidence interval of 2.3% to 4.7%):

$$\text{Low daily RHA for } PM_{10} = 1.00 \times 10^{-8} \times \quad PM_{2.5} \times POP_j \qquad \text{(D-6a)}$$

$$\text{Central daily RHA for } PM_{10} = 1.21 \times 10^{-8} \times \quad PM_{2.5} \times POP_j \qquad \text{(D-6b)}$$

$$\text{High daily RHA for } PM_{10} = 1.42 \times 10^{-8} \times \quad PM_{2.5} \times POP_j \qquad \text{(D-6c)}$$

where:

POP_j = total population in area j
$PM_{2.5}$ = change in daily (24-hour) $PM_{2.5}$.

We apply a 50% probability to the central parameter and a 25% probability to both the low and the high estimates.

Cardiac Hospital Admissions

Burnett et al. (1995) also report a 3.3% increase in cardiac hospital admissions (CHAs) associated with a 13 g/m^3 increase in sulphate based on the results from a model that included ozone. The average number of CHAs per day for the study period was 14.4 per million population. Following the procedure outlined above for RHAs, the daily CHA concentration-response parameter per g/m^3 sulphate is: $0.033 \times (14.4 \times 10^{-6}) \div 13 = 3.66 \times 10^{-8}$. This sulphate-

2. This adjustment factor errs on the side of understating the effect of $PM_{2.5}$ on hospital admissions because it assumes that only the sulphate share of $PM_{2.5}$ is responsible for additional hospital admissions. We make this assumption because the original study examined only sulphates.

based result is converted to its $PM_{2.5}$ equivalent using an estimated average sulphate to $PM_{2.5}$ ratio of 0.28 based on air pollution monitoring data from all seasons in Ontario during the period 1984 to 1997 (Tom Dann, personal communication, 1999). Multiplying by this conversion factor provides the central daily CHA concentration-response parameter per g/m^3 of $PM_{2.5}$. Thus, the $PM_{2.5}$ CHA concentration-response parameters are as follows, with the low and high estimates representing the central minus and plus one standard error (the standard error associated with the initial estimate can be calculated based on the reported 95% confidence interval of 1.7% to 4.8%):

$$\text{Low daily CHA for } PM_{10} = 0.79 \times 10^{-8} \times \Delta PM_{2.5} \times POP_j \quad \text{(D-7a)}$$

$$\text{Central daily CHA for } PM_{10} = 1.02 \times 10^{-8} \times \Delta PM_{2.5} \times POP_j \quad \text{(D-7b)}$$

$$\text{High daily CHA for } PM_{10} = 1.26 \times 10^{-8} \times \Delta PM_{2.5} \times POP_j \quad \text{(D-7c)}$$

where:

POP_j = total population in area j
$\Delta PM_{2.5}$ = change in daily (24-hour) $PM_{2.5}$.

We apply a 50% probability to the central parameter and a 25% probability to both the low and the high parameters.

D.3.3 Emergency Room Visits

Studies in the United States have found an association between particulate matter and the incidence of emergency room visits (ERVs) for all causes (Samet et al., 1981) and for asthma-related diagnoses (Schwartz et al., 1993). To estimate the $PM_{2.5}$ ERV concentration-response parameters we follow the approach taken in the sulphate panel report, assuming ERVs are proportional to the pollution-related respiratory and cardiac hospital admissions described in the previous section.

The Saint John Particle Health Effects Study (Stieb et al., 1995) provides data that indicate that for each RHA in Saint John, New Brunswick, there are 5.3 respiratory-related emergency department visits and for each CHA there are 1.4 cardiac emergency department visits. For example, the low ERV concentration-response parameter is thus: $(5.3 \times 1.00 \times 10^{-8}) + (1.4 \times 0.79 \times 10^{-8}) = 6.41 \times 10^{-8}$. Assuming these ratios apply elsewhere in Canada, and using the concentration-response parameters for RHAs and CHAs derived above this yields the following total ERV concentration-response parameter estimates:

$$\text{Low total daily ERV} = 6.41 \times 10^{-8} \times (\ \ PM_j) \times POP_j \quad \text{(D-8a)}$$
$$\text{Central total daily ERV} = 7.84 \times 10^{-8} \times (\ \ PM_j) \times POP_j \quad \text{(D-8b)}$$
$$\text{High total daily ERV} = 9.29 \times 10^{-8} \times (\ \ PM_j) \times POP_j\ , \quad \text{(D-8c)}$$

where:

POP_j = population in location j
PM_j = change in daily average $PM_{2.5}$ in area j.

To estimate the net ERV concentration-response parameters the corresponding low, central, and high concentration-response parameter estimates for the RHA and CHA endpoints are subtracted from the ERV estimates above. This adjustment results in the following net ERV concentration-response parameter estimates:

$$\text{Low net daily ERV} = 4.62 \times 10^{-8} \times (\ \ PM_j) \times POP_j \quad \text{(D-9a)}$$
$$\text{Central net daily ERV} = 5.61 \times 10^{-8} \times (\ \ PM_j) \times POP_j \quad \text{(D-9b)}$$
$$\text{High net daily ERV} = 6.61 \times 10^{-8} \times (\ \ PM_j) \times POP_j \quad \text{(D-9c)}$$

where:

POP_j = population in location j
PM_j = change in daily average $PM_{2.5}$ in area j.

As with the hospital admissions parameters, we apply a 50% probability to the central parameter and a 25% probability to both the low and the high parameters.

D.3.4 Aggravation of Asthma Symptoms

Several studies have related air pollutant concentrations to exacerbation of asthma symptoms in individuals with diagnosed asthma. Two epidemiologic studies with study populations of currently diagnosed asthmatics provide the information needed to develop a concentration-response function relating the frequency of elevated asthma symptoms to fluctuations in ambient particulate matter concentrations (Whittemore and Korn, 1980; Ostro et al., 1991).

These studies had subjects (diagnosed asthmatics) record daily asthma symptoms during the duration of the study. An elevation of asthma symptoms, an asthma symptom day (ASD), was defined for each subject based on each individual s manifestation of symptoms. This typically meant a notable increase in symptoms, such as shortness of breath or wheezing, and/or an increase in use of medication relative to what was normal for that individual. Daily particulate matter and ozone levels were then examined for correlations with day-to-day fluctuations in asthma symptom frequency, controlling for other factors such as weather and previous-day symptoms.

Whittemore and Korn (1980) studied asthmatics (adults and children) living in six different communities in the Los Angeles area. Each subject reported asthma symptoms during one or more 34-week period between 1972 and 1975. A total of 443 subject-periods of data were obtained (some subjects provided data for more than one period). The study used a statistical approach to estimate both individual-level and group effects.

Ostro et al. (1991) examined the association between several different air pollutants, including sulphates, $PM_{2.5}$, and acidic aerosols, and increases in asthma symptom days among adults during winter months in Denver. A significant association was found between the probability of moderate or severe asthma symptom days (measured as shortness of breath) and sulphate particulate levels after controlling for temperature, day of week, previous-day illness, and use of a gas stove. Ozone levels were very low, near background levels, during the study period and do not create a confounding influence.

The logistic model used by Whittemore and Korn (1980) generates an equation with a nonlinear first derivative. Thus, we need a baseline probability rate for ASDs to predict the frequency of ASDs per unit of particulate matter exposure. The average rate of ASDs for the Whittemore and Korn study sample is available, but it appears to be quite high. This corresponds with what appears to be an over representation fairly severe levels of asthma in the study sample. The authors report that additional potential subjects were excluded because of insufficient manifestation of any asthma symptoms during the study period. More representative data on average asthma symptom frequency is, however, not available at this time. It is therefore necessary to make some reasoned assumptions about what an average rate may be.

In the Los Angeles study sample, about 26% of the sample experienced elevated asthma symptoms on any given day. If all of the excluded potential subjects are presumed to have had no elevated asthma symptoms during the study period and this is factored into the calculation, the average daily symptom rate is reduced to 15%. This is similar to the 15% shortness of breath frequency reported by Ostro et al. (1991). Also, Holguin et al. (1985) report an average daily asthma symptom rate of 15% for their study sample, or 13% if those excluded from the study are factored in. As a check on the plausibility of these rates as representative of the active asthmatic population, we consider asthma severity information reported by the National Center for Health Statistics (1980). They report that of all active asthmatics in the United States, 55% have mild symptoms, 32% have moderate symptoms, and 13% have severe symptoms.[3] If we assume that mild means one symptom per month, moderate means one symptom per week, and severe means one symptom every other day, the average daily symptom rate would be 13%. We select this

3. The 1979 Health Interview Survey report (National Center for Health Statistics, 1980) gives the frequency of bother and the severity of asthma for respondents with diagnosed asthma. We calculated a frequency of asthma symptoms by cross-tabulating this descriptive information. We defined mild as those who report being bothered *some* by asthma symptoms *once in a while*. We defined severe as those who report being bothered *a great deal* by asthma symptoms *often* or *all the time*. We defined moderate as everything in between.

lower approximated rate to minimize the chance of overstating the expected effect of $PM_{2.5}$ on the average asthmatic.

Using a logistic model with both particulate matter and ozone included, Whittemore and Korn (1980) obtained a coefficient for daily (24-hour) TSP (g/m^3) of 0.00079, with a standard error of approximately 0.00034. The probability of an ASD as a function of PM levels is given by the following relationship in a logistic specification:

$$Pr/ \quad PM = b \times Pr \times (1 - Pr) \qquad \text{(D-10)}$$

where:

Pr = probability of elevated asthma symptoms on a day
b = the estimated logit coefficient for PM.

Using 13% as the baseline probability that an asthmatic will experience elevated asthma symptoms on a given day, and the estimated TSP coefficient (adjusted to PM_{10} by dividing the b by 0.55 in Equation D-10), provides the following result from Whittemore and Korn (1980):

$$\text{Daily ASD for } PM_{10} = 1.62 \times 10^{-4} \times \quad PM_{10}\,. \qquad \text{(D-11)}$$

The adjustment of the TSP coefficient for the average share of TSP that is PM_{10} assumes that the entire health effect associated with TSP is attributable to the PM_{10} component of TSP. This assumption is consistent with the scientific evidence that it is the particulates that are 10 microns in diameter and smaller that are responsible for the observed health effects. We apply the PM_{10} concentration-response parameter to changes in ambient $PM_{2.5}$ without further adjustment on the assumption that $PM_{2.5}$ aerosols are equally as harmful on a per microgram basis as any other constituent of PM_{10}.

The Ostro et al. (1991) results suggest the following relationship between elevated asthma symptoms and daily sulphate (SO_4) concentrations:

$$\text{Daily ASD for sulphate} = 0.0077\ (\pm\ 0.0038) / SO_4\,. \qquad \text{(D-12)}$$

Using the reported SO_4 mean for the study of 2.11 g/m^3 to linearize the function and converting from sulphate to $PM_{2.5}$ by multiplying by a conversion factor of 0.1 (based on the reported means of the two measures in the Denver study) yields the following daily ASD concentration-response parameter based on the Ostro et al. (1991) results.

$$\text{Daily ASD for } PM_{2.5} = 3.65 \times 10^{-4} \times \quad PM_{2.5}\,. \qquad \text{(D-13)}$$

We take an average of the Ostro et al. (1991) results (converted to $PM_{2.5}$) and the Whittemore and Korn (1980) results (converted to PM_{10}) for the central ASD concentration-response parameter. The low concentration-response parameter is based on Whittemore and Korn, and the high concentration-response parameter is based on Ostro et al. (1991). The resulting ASD concentration-response parameters are applied to the diagnosed asthmatic population (estimated to be 6.0% of the Canadian population, Statistics Canada, 1994) as follows:

$$\text{low daily ASD for } PM_{2.5} = 1.62 \times 10^{-4} \times PM_{dj} \times POP_{aj} \quad \text{(D-14a)}$$

$$\text{central daily ASD for } PM_{2.5} = 2.64 \times 10^{-4} \times PM_{dj} \times POP_{aj} \quad \text{(D-14b)}$$

$$\text{high daily ASD for } PM_{2.5} = 3.65 \times 10^{-4} \times PM_{dj} \times POP_{aj} \quad \text{(D-14c)}$$

where:

POP_{aj} = asthmatic population in location j (6.0% of POP_j)
PM_{dj} = change in daily $PM_{2.5}$ in area j.

These parameter estimates are assigned the following probability weights: low, 33%; central, 34%; high, 33%.

D.3.5 Restricted Activity Days

Restricted activity days (RADs) include days spent in bed, days missed from work, and days when activities are partially restricted due to illness. Ostro (1987) examined the relationship between adult all-cause RADs in a two-week period and $PM_{2.5}$ in the same two-week period for 49 metropolitan areas in the United States. The RADs data were developed from the U.S. Health Interview Survey (HIS) which is conducted annually by the National Center for Health Statistics. The $PM_{2.5}$ data were estimated from visual range data available for airports in each area. Because $PM_{2.5}$ has a more significant impact on visual range than do large suspended particles, a direct relationship can be estimated between visual range and $PM_{2.5}$.

Separate regression estimates for the impact of $PM_{2.5}$ on RADs were obtained for six years, 1976 to 1981. A statistically significant relationship was found in each year and was consistent with earlier findings relating RADs to TSP by Ostro (1983). The mean of the estimated parameters for $PM_{2.5}$ across the six years indicated approximately 91,200 RADs each year per 1 million population for each 1 g/m^3 increase in annual average $PM_{2.5}$. The impact of $PM_{2.5}$ on RADs implied by the individual parameters ranged from a low of 53,200 RADs each year per 1 million population for each 1 g/m^3 increase in annual average $PM_{2.5}$ for the 1981 parameter to a high of 171,000 RADs each year per 1 million population for each 1 g/m^3 increase in annual average $PM_{2.5}$ for the 1976 parameter.

Additional work conducted by Ostro and Rothschild (1989) added ozone measures to the regressions and found the estimated relationship between RADs and $PM_{2.5}$ to be essentially unchanged. This suggests that the RAD/$PM_{2.5}$ relationship was not confounded by the exclusion of ozone levels and is independent of ozone exposures. The newer work estimated the relationship between respiratory RADs (RRADs) and $PM_{2.5}$ for employed individuals only. It was expected that this relationship might be more stable than that between all-cause RADs and $PM_{2.5}$ for all adults for two reasons: (1) it is expected that pollution-induced RADs might be predominantly related to respiratory illness, and (2) workers might define a RAD more consistently than the entire adult population. It was expected, though, that confining the data to RRADs for workers might result in a smaller total number of predicted restricted activity days for a given level of pollution, because all effects might not be classified as respiratory and workers may be on average a healthier, and therefore less sensitive, group than all adults. The findings are consistent with this expectation. The average of the $PM_{2.5}$ parameters for the six years suggested an annual increase of approximately 47,100 RRADs per 1 million workers for each 1 g/m^3 increase in annual average $PM_{2.5}$, and ranged from a low of 30,800 RRADs for the 1978 parameter to a high of 54,700 RRADs for the 1980 parameter.

The mean result over the six years from Ostro (1987) for all-cause RADs for all adults (mean parameter = 0.0048) has been selected for the central concentration-response parameter for this analysis. The mean result from Ostro and Rothschild (1989) for RRADs for workers (mean parameter = 0.0158) was selected for the low estimate. The selected high parameter is the mean of the two highest coefficients in the six-year analysis (mean parameter = 0.0076) by Ostro (1987). The Ostro (1987) and Ostro and Rothschild (1989) parameter s give percentage changes in RADs or RRADs for a 1 g/m^3 change in $PM_{2.5}$. Daily average estimates from the studies based on HIS baseline incidence data of 0.052 RADs and 0.0083 RRADs per person are used to determine the relationship between number of RADs and $PM_{2.5}$. The selected concentration-response function determined by multiplying the percentage changes by the baseline incidence values and are thus:

$$\text{low daily RAD} = 1.31 \times 10^{-4} \times \quad PM_{dj} \times POP_{20j} \qquad \text{(D-15a)}$$

$$\text{central daily RAD} = 2.50 \times 10^{-4} \times \quad PM_{dj} \times POP_{20j} \qquad \text{(D-15b)}$$

$$\text{high daily RAD} = 3.95 \times 10^{-4} \times \quad PM_{dj} \times POP_{20j} \qquad \text{(D-15c)}$$

where:

POP_{20j} = population 20 years and older in location j
PM_{dj} = change in daily $PM_{2.5}$ in area j.

These concentration-response parameters are applied to the adult population 20 years and older. In addition, because daily symptom concentration-response parameter estimates for asthmatics are available based on studies focused specifically on those with diagnosed asthma (see Section D.3.4), we exclude the asthmatic population from the calculations of RADs. Although asthmatics

were not specifically excluded from the RAD studies, nonasthmatics are more representative of the response of the general population because only a small fraction of the general public has diagnosed asthma. We therefore apply the RAD concentration-response function parameter estimates to the nonasthmatic portion (94.0%) of the Canadian population.

The central RAD concentration-response parameter estimate is given a 50% weight while the low and high parameters each receive 25% weights.

D.3.6 Acute Respiratory Symptoms

Krupnick et al. (1990) estimated a relationship between the daily occurrence of acute upper and lower respiratory symptoms (ARS) among a panel of adults and children in Southern California and daily levels of air pollution. Krupnick et al. (1990) used pooled cross-sectional and time-series data based on a health survey conducted in 1978-1979 of families living in Glendora, Covina, and Azusa, California. Health diaries were maintained for 182 days by; 290 participating families. ARS is a binary variable reflecting the presence or absence of any of 19 respiratory-related symptoms, including chest discomfort, coughing, wheezing, sore throat, head cold, chest cold, sinus trouble, hay fever, headache, and doctor-diagnosed flu. The ARS endpoint includes some days with symptoms bothersome enough to result in a restricted activity day, but also includes days when noticeable symptoms are present but no change in activities occurs. To account for this potential overlap, total RADs are subtracted from the total ARSs to develop net ARS concentration-response parameters (see section D.1 for more details).

Krupnick et al. (1990) applied a Markov process model to determine the relationship between air pollution and respiratory symptoms. The model incorporated the probability of illness on the prior day and controlled for autocorrelation. Air pollution variables for coefficient of haze, ozone, and sulfur dioxide were included in the model along with independent variables for socioeconomic measures, presence of a chronic condition, and smoking habits. The initial results with multiple pollutants in separate equations for adults and children showed statistically significant parameters of roughly similar magnitudes for COH, a measure of visibility impairing particles in the air, in the adult and children equations.

The COH parameter in the Krupnick et al. (1990) Equation 3 specification is 0.0088, with a standard error of 0.0046. Data provided to us by the authors show a ratio of COH (units/100 ft.) to TSP for the study period of 0.116. Using the PM_{10}/TSP ratio of 0.55, this gives a COH to PM_{10} ratio of 0.211. The marginal effect of COH was calculated by incorporating the stationary probabilities as described in the paper.[4] Because the study did find symptom effects for children

4. The authors provided mean estimates of the transitional probabilities to us for the all-adults sample, which were not reported in the paper. For all adults, p_1 averages 0.7775 and p_0 averages 0.0468. P_1 is the probability of reporting symptoms given that symptoms were present on the previous day, and p_0 is the probability of reporting symptoms given that no symptoms were present on the previous day.

in some specifications, we apply these calculations to the entire population. The central ARS concentration-response parameter is based on the regression coefficient from Equation 3 in Krupnick et al. The high and low parameters are based on this result plus or minus one standard error of the regression coefficient. We assume for this purpose that $PM_{2.5}$ aerosols are equal to all PM_{10} in terms of their harmfulness per microgram. The resulting $PM_{2.5}$ concentration-response parameters for ARSs are as follows:

$$\text{low total daily ARS} = 2.20 \times 10^{-4} \times PM_{dj} \times POP_j \quad \text{(D-16a)}$$

$$\text{central total daily ARS} = 4.61 \times 10^{-4} \times PM_{dj} \times POP_j \quad \text{(D-16b)}$$

$$\text{high total daily ARS} = 7.02 \times 10^{-4} \times PM_{dj} \times POP_j \quad \text{(D-16c)}$$

where:

POP_j = population in location j
PM_{dj} = change in daily $PM_{2.5}$ in area j.

Because the definition of ARSs includes days that also fall into the category of restricted activity days, we subtract RADs to obtain net ARS parameters. The RADs parameters apply only to the population age 20 and older, so we multiply the RADs parameters by 0.728 (the share of the population in Canada that is age 20 and over in the 1996 census) and then subtract these from the ARSs parameters. The resulting net ARSs concentration-response parameters are as follows:

$$\text{low net daily ARS} = 1.25 \times 10^{-4} \times PM_{dj} \times POP_j \quad \text{(D-17a)}$$

$$\text{central net daily ARS} = 2.79 \times 10^{-4} \times PM_{dj} \times POP_j \quad \text{(D-17b)}$$

$$\text{high net daily ARS} = 4.14 \times 10^{-4} \times PM_{dj} \times POP_j \quad \text{(D-17c)}$$

where:

POP_j = population in location j
PM_{dj} = change in daily $PM_{2.5}$ in area j.

We give the central parameter a 50% probability weight. The low and high parameters each receive a 25% probability weight.

D.3.7 Child Acute Bronchitis

Dockery et al. (1989) studied the relationship between lower respiratory illness in children and particulate matter concentrations in six cities in the United States. The study related annual concentrations of TSP, PM_{15}, $PM_{2.5}$, sulphate, and sulfur dioxide to the presence of chronic cough, acute bronchitis, chest illness, persistent wheeze, and asthma. These illnesses were noted during a health examination and intake questionnaire taken for the sampled children in each city. A condition of asthma or acute bronchitis was based on a physician s diagnosis in the previous

year. Chronic cough was defined as a cough being present for at least three months in the past year. The results of a logistic regression analysis show a statistically significant relationship between annual average particulate matter levels and the probability of the child having bronchitis or chronic cough in the past year.

A recent study (Dockery et al., 1996) replicates the above study using 18 U.S. and 6 Canadian cities. Children ages 8 to 12 were assessed via questionnaire between 1988 and 1991. Among the cities, the annual incidence rates for acute bronchitis in children (B) in the past year ranged from 3% to 10%, with an average of 6.5%. The logistic regression analysis controlled for sex, history of allergies, parental asthma, parental education, and current smoking in the home. Particulate matter measures used in the analysis included sulphates, $PM_{2.1}$, and PM_{10}.

Statistically, the strongest results were for incidence of prevalence of acute bronchitis in children (within the past year) and the particulate matter measures (the impact of gaseous air pollutants such as ozone were also evaluated in the study). Specifically, a 6.8 g/m^3 increase in annual sulphate was associated with an relative risk of 1.65 (95% CI = 1.12, 2.42) for a 14.9 g/m^3 increase in $PM_{2.1}$ was associated with an relative risk of 1.50 (95% CI = 0.91, 2.47). The associated regression parameters are estimated as follows: $\ln(1.65)/6.8 = 0.0736$ with a standard error of 0.029 for sulphate, and $\ln(1.50)/14.9 = 0.0272$ with a standard error of 0.017 for $PM_{2.1}$. Even though the $PM_{2.1}$ results are less statistically robust, we select the $PM_{2.1}$ results as the basis for the B risk factor estimates because they are based on a PM measure very close to the one of interest in this analysis. These estimates are slightly lower than, but very similar in magnitude to, what we would obtain if we adjusted the sulphate results for the average sulphate share of $PM_{2.1}$ based on the reported study data. Based on the $PM_{2.1}$ results, a one g/m^3 change in $PM_{2.1}$ generates a central B concentration-response parameter of $0.0272 \times [0.065 \times (1 - 0.065)] = 1.65 \times 10^{-3}$. We presume for the purposes of this analysis that $PM_{2.1}$ is essentially equivalent to $PM_{2.5}$ and therefore make no adjustment to the parameter for the difference in the PM measure. For the low and high parameters we use the central estimate plus or minus one standard error respectively. Therefore the annual B concentration-response parameters are as follows:

$$\text{low annual B} = 0.62 \times 10^{-3} \times \quad PM_{yj} \times POP_{j<20} \qquad \text{(D-18a)}$$

$$\text{central annual B} = 1.65 \times 10^{-3} \times \quad PM_{yj} \times POP_{j<20} \qquad \text{(D-18b)}$$

$$\text{high annual B} = 2.69 \times 10^{-3} \times \quad PM_{yj} \times POP_{j<20} \qquad \text{(D-18c)}$$

where:

$POP_{j<20}$ = population under 20 years in location j
PM_{yj} = change in annual average $PM_{2.5}$ in area j.

We apply these concentration-response parameters to the population under age 20 and give the central parameter a 50% probability while the low and high parameters each receive 25% probabilities.