Health and Air Quality 2002 – Phase 1

Methods for Estimating and Applying Relationships between Air Pollution and Health Effects

FINAL Report

Prepared for:

British Columbia Lung Association

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[RWDI Project: W02-304]

May 2003

ACKNOWLEDGEMENTS

The project was carried out by the British Columbia Lung Association (BCLA) on behalf of a consortium of funding partners and other interested agencies. BCLA acknowledges the participation of the project steering committee, which developed the terms of reference for the study and recruited the members of an expert panel. The committee members and their affiliations are as follows. Funding agencies are indicated by an asterisk (*).

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BCLA, the expert panel members and the consulting team acknowledge and thank all of these participants for their guidance and helpful comments throughout the project. The findings, conclusions and recommendations remain the responsibility of the Panel and consultant and do not necessarily reflect policy of any of the participating agencies.

SUMMARY

The determination of relationships between exposure to air pollutants and effects on human respiratory and cardio-vascular health – both illness and premature death – is a work in progress. A number of methodologies have been used to estimate the strength and robustness of observed relationships, and the relative merits of various approaches to the analysis are being evaluated. A subject of the ongoing research is the applicability of the available results from epidemiological and toxicological studies to other specific geographical regions or neighbourhoods.

An expert panel was struck under the auspices of the British Columbia Lung Association to address a series of questions about relationships between exposure to air pollution and effects on human health that had been developed by a steering committee. The questions comprised the basis for the outline of this report, as well as lending structure to the Panel's thinking about their mandate.

This report is intended to illuminate the available information, offer some conclusions and provide recommendations on how the literature may be interpreted with respect to application to practical assessment problems in British Columbia (BC) and the Pacific Northwest (PNW). The report provides expert opinion on how to interpret and apply the literature in the context of approaches to air quality management in the region.

The users of this report are expected to be regulatory managers, planners, representatives of public interests, project proponents, researchers and physicians – all of whom wish to apply the available knowledge to assess the impacts of projects and of broader economic and social development. Some specialised language has been used in the report, but an attempt has been made to provide explanatory wording, and a glossary is provided. The report is intended to provide guidance – not prescribe procedures – for application of the knowledge base at the various levels at which air quality impact assessment is carried out.

The review included the literature relevant to the effects of the principal common air contaminants that have long been associated with direct human health effects –

- nitrogen oxides [NOx (as NO_2) and particulate nitrates (NO_3^-)]
- sulphur oxides [SOx (as SO_2) and particulate sulphates ($SO_4^=$)]
- carbon monoxide [CO]
- ozone [O_3]
- fine particles [$PM_{2.5}$] and inhalable particles [PM_{10}].

It did not include consideration of specific air contaminants that may be considered to be cancer-causing agents (carcinogens) – the so-called toxic, or hazardous, air pollutants. The general issue of the association between air pollution and elevated cancer rates in exposed populations (primarily urban), however, was considered.

Studies of air quality effects on human health in the Lower Fraser River Valley, elsewhere in British Columbia and in the Pacific Northwest were considered to have the greatest potential relevance to the users of the knowledge base. There is reason to believe that studies from elsewhere in urban areas with source mixes similar to the larger cities in BC and the PNW are directly relevant. It is likely that Vancouver and Victoria (and

perhaps Kelowna), as well as the cities of the Puget Sound and Portland areas, have more in common with large cities outside of the region than they do with smaller communities in the interior areas of BC or the PNW. The settings and source mixes of the various communities are important in generalising the applicability of the study results to the diverse communities in this region. The broader Canadian and international literature was reviewed because of its great breadth and depth of potentially relevant information.

The Panel considered a number of aspects of the literature on health effects of air pollution, including:

- pollutant mix and exposure patterns
- different types of studies (mainly epidemiology studies)
- sources of uncertainty
- a number of criteria for judging the robustness of the relationships (and their applicability to local situations).

The report summarises the detailed knowledge base of health effects, first by individual pollutants, then by health effects themselves (which may be associated with several pollutants). The Panel then provides an interpretation of the knowledge base, structured according to the questions posed by Steering Committee. Their findings may be summarised in the following conclusions and recommendations.

Conclusions and Recommendations

The Panel reached the following conclusions and associated recommendations.

1. Levels of some air pollutants, particularly $PM_{2.5}$ and its wood smoke component, and ozone, in British Columbia are at levels which, on the basis of comparisons with international data, would be predicted to be causing adverse health effects. Since population-level (as compared with individual or panel-level) thresholds for adverse effects have not been shown to exist in the cases of particulate pollution and ozone, current air quality objectives should not be interpreted as bright lines between 'safe' and 'unsafe' levels.

   The field of air pollution management, with its attendant politics, is driven by the demonstrated adverse health effects of a number of pollutants to which people are currently exposed. When proposed developments will increase exposure to pollutants, prospective public health protection requires that, if possible, adequate safety margins are embodied in proposals; this task has been made more difficult by the absence of demonstrated exposure thresholds and by the fact that exposures to a number of air pollutants are already in the range that has been shown to cause adverse health effects. The literature also indicates that health improvements are associated with air quality improvements (from studies of situations in which air quality changed dramatically as a result of substantial changes in emissions over short periods of time).

   Recommendation: It needs to be recognised that any improvement in air quality for PM or ozone would result in fewer negative health impacts. In the Panel's opinion, also required is a stringent approach to proposals that would entail any increase in public exposure levels to these two pollutants.

2. Based on studies carried out in the Lower Fraser Valley, it appears that the increased risk of dying prematurely due to exposure to air pollution is comparable to some common risks, within broad uncertainty. This may also be the case elsewhere in BC.

   For example, using concentration-response factors (CRFs) from studies elsewhere, the daily risk of dying for people 65 and older is increased by about 4% at an ambient PM_{10} level of 50 $\mu g/m^3$ (i.e., a high pollution day) compared with that at 10 $\mu g/m^3$ (i.e., a relatively low pollution day). The estimated uncertainty range of the increased risk is roughly 0.8% to 4.4 % (i.e., a factor of 5 lower to a factor of 1.1 higher). Over a long-term exposure, the analogous risk for the over-65 population would be about 4% excess risk of premature death for living in a community with a long-term average PM_{10} concentration of 20 $\mu g/m^3$ compared with one at 10 $\mu g/m^3$.

   Recommendation: Communicating exposure-response information in a risk context is essential. It is important that affected communities understand that risk increases with level of exposure – risk of health effects is very low at the lowest ambient concentrations in BC and increases proportionally to ambient concentrations of PM and ozone.

3. Study size, as defined as the number of outcomes multiplied by years of monitoring data, is a determining factor in deciding whether new, local studies of air pollution impacts on health should be considered. Preliminary data supplied in the report indicate that outside of the Lower Mainland of BC and the Puget Sound area of Washington, the population of the smaller communities is a limiting factor. This means that many years of monitoring and health data are necessary to provide statistically reliable results for mortality – fewer years for morbidity studies. For example, a reliable study of relationships between air pollution and mortality in a community of 100,000 people might require 10-15 years of data, and perhaps 3-5 years of data for a morbidity outcome such as emergency room visits.

   Recommendation: Consider the feasibility of pooling health and monitoring data across a number of communities if new health studies are desirable. Pooling requires careful characterisation of potential differences across communities in exposure and other variables.

4. Studies of farm workers in the Fraser Valley, and of asthmatic schoolchildren in Port Alberni, for example, have provided important assurances that generalisations from studies done elsewhere are reliable. The available local study results can be transferred to similar communities in BC. If pollutant mixes and exposure patterns for a community lacking study data are very different, however, there is no choice but to carry out a community-specific study and provide the necessary exposure monitoring.

   Qualitative estimates of the potential incidence of effects can be made based on available data, but the only way of determining whether current levels in some areas of British Columbia are of major concern is to fund the research needed to investigate possible effects. A major deficiency in reliably estimating air pollution-related health effects in Interior BC communities is the predominance of resource sector emissions in the pollutant mix in these communities, especially wood smoke (including the residential space heating contribution to the latter). At present these types of sources impact neither the major BC coastal cities nor most of the US and European cities for

which much of the work on air pollution effects has been conducted. Kelowna is probably the only major BC Interior city that is not significantly affected by wood smoke and thus has a comparable source mix to the Lower Mainland and Victoria. Communities in northeast BC are impacted by emissions from both oil & gas and forest products sectors and are likely to have unique air pollution exposure patterns requiring special study.

Recommendation: Carry out community-specific health studies where comparison with results from similar communities is not feasible. Adequate exposure monitoring would need to be provided. A specific example of such a study that has a good chance of providing reliable information is a pooled study of hospital admissions (or other effects) in Interior BC communities that are currently monitored for PM_{10} (preferably adding $PM_{2.5}$ monitoring where feasible). Comparison of communities with significant wood smoke exposure with similar communities with low wood smoke exposure would be valuable. The Candidate communities are Fort St. John, Quesnel, Houston, Williams Lake and Prince George for the wood smoke-impacted group (pooled population: 117,000). Communities in the Kootenay region (e.g., Cranbrook and Nelson) might also be included. The Fort St. John-Taylor-Chetwynd area with its oil & gas component may also be a candidate for a special effects study, although the population is small and spread out, so that the likelihood of successfully finding statistically significant results is small.

5. Growing evidence of traffic-related impacts in urban areas suggests that the proximity of these emissions to populated areas causes high exposures relative to typical ambient monitoring sites – and associated health effects. Traffic-related PM may be more potent in observed health effects than general PM, and concentrations of PM near roadways may be considerably higher than at locations away from roadways. The combined effect of potency and concentration might increase risk of effects in residents living near a roadway by 1.5-2 times that of the general population (based on European studies).

   Recommendation: Careful study of the results of traffic studies elsewhere should be undertaken to determine likely impacts in BC's major urban centres. Expanded exposure monitoring for PM (and its components) along roadways, such as is currently being conducted in Vancouver in a limited way, is necessary to determine whether to expect similar effects impacts as have been observed elsewhere (in US and European cities). The impacts can be quantified using available concentration-response data.

6. Continuing economic estimates of the costs of current levels of air pollution, both direct to the health care system, and indirect to society as a whole, are required.

   Recommendation: The concentration-response factor databases recommended in this report can be used in conjunction with local monitoring data as the basis for providing a preliminary estimate of air pollution-related disease outcomes in any such economic analysis. The benefits of reducing pollution levels by specific amounts should be estimated, rather than the absolute total impact values.

7. Proposed new facilities that have significant emissions known to cause adverse health effects require critical health risk assessments before approval. The examination of such assessments should be, in the Panel's opinion, in the public domain.

   Recommendation: Include specific health risk analysis in all major project assessments. Such analysis should take into account the specific demographics and health status of exposed populations and should apply the effects estimation methodology outlined in this report where feasible using estimated population-weighted incremental air pollutant exposures.

8. Concentration-response factors for PM_{10} (or $PM_{2.5}$) exposure and mortality and lung cancer are recommended by the Panel for use in BC and the PNW – with respect to urban populations. The Panel's preferences are provided for short-term and long-term exposures. For these populations, the morbidity concentration-response factors from established Canadian databases, such as those in the Air Quality Valuation Model (AQVM) or Illness Costs of Air Pollution (ICAP) model, are acceptable as starting points.

   Recommendation: Since the available BC and PNW mortality and morbidity studies are quite limited, the feasibility of carrying out meta-analysis to compile a region-specific concentration-response factor database for application to this region is questionable. As the most practical approach, the Panel recommends using the most up-to-date version of the AQVM CRF database for morbidity CRFs.

   Recommendation: Carry out screening-level analysis of potential health effects impacts in smaller BC communities using the data produced from the analysis of the available local and other relevant studies (summarised in the report) to estimate the possible incidence of air pollution-related impacts. This will help in assessing priorities for mitigation in impacted airsheds and for identifying areas where health risk studies might be considered.

   Recommendation: Where reliable health study data are not available for a smaller community, ambient air quality can be used as a reasonable measure of relative risk of health effects in that community in comparison with similar communities in which observations have been made – or even in comparison with larger communities with appropriate recognition of the potential uncertainty in such comparisons.

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1. Introduction

1.1. General background of the project

The determination of relationships between exposure to air pollutants and effects on human respiratory and cardio-vascular health – both illness and premature death – is a work in progress. A number of methodologies have been used to estimate the strength and robustness of observed associations, and the relative merits of various approaches to the analysis are being evaluated. A subject of the ongoing research is the applicability of the available results from epidemiological and toxicological studies to other specific geographical regions or neighbourhoods. This report is intended to illuminate the available information, tender some conclusions and provide recommendations on how the literature may be interpreted with respect to application to practical assessment problems in British Columbia (BC) and the Pacific Northwest (PNW). The report provides expert opinion on how to interpret and apply the literature in the context of approaches to air quality management in the region. In this context, both qualitative associations (i.e., strength of association) and quantitative associations (e.g., magnitude of association – exposure-response factors) between exposure to air pollutants and health outcomes are useful in assessing air quality impacts. This paper addresses both of these kinds of data from the literature.

The users of this report are expected to be regulatory managers, planners, representatives of public interests, project proponents, researchers and physicians – all of whom wish to apply the available knowledge to assess the impacts of projects and of broader economic and social development. Interpreting and communicating this information is complicated by scientific jargon. Some specialised language has been used in the report, but an attempt has been made to provide explanatory wording, and a glossary is provided. The report is intended to provide guidance – not prescribe procedures – for application of the knowledge base at the various levels at which air quality impact assessment is carried out.

1.2. Terms of reference/questions to be answered

An expert Panel was struck under the auspices of the British Columbia Lung Association to address a series of questions that had been developed by a steering committee. The Terms of Reference for the project and the questions that guided the expert Panel's work are provided in Appendices 1 and 2. The questions comprised the basis for the outline of this report, as well as lending structure to the Panel's thinking about their mandate.

The project includes peer review of the Panel's report. The process for the expert Panel and peer review included engaging the peer reviewers – all experts in the various aspects of the work – early in the project and keeping them apprised of the development of the report in progress, so that their eventual review of a draft document could be more focused and efficient.

Since the purpose of this document is to assist those who must apply information about air pollution and health effects in their day-to-day jobs, especially in interacting with the

public on such matters, the Panel kept in mind the most relevant aspects of the literature in this context.

1.3. POLLUTANTS TO BE ADDRESSED

The review included the literature relevant to the effects of the principal common air contaminants that have long been associated with direct human health effects –

- nitrogen oxides [NOx (as NO_2) and particulate nitrates (NO_3^-)]
- sulphur oxides [SOx (as SO_2) and particulate sulphates ($SO_4^{=}$)]
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It did not include consideration of specific air contaminants that may be considered to be cancer-causing agents (carcinogens) – the so-called toxic, or hazardous, air pollutants. The general issue of the association between air pollution and elevated cancer rates in exposed populations (primarily urban), however, was considered.

1.4. BACKGROUND/CONTEXT

Studies of air quality effects on human health in the Lower Fraser River Valley, elsewhere in British Columbia and in the Pacific Northwest were considered to have the greatest potential relevance to the users of the knowledge base. There is reason to believe that studies from further afield in urban areas with source mixes similar to the larger cities in BC and the PNW are also relevant. It is likely that Vancouver and Victoria (and perhaps Kelowna), as well as the cities of the Puget Sound and Portland areas, have more in common with large cities outside of the region than they do with smaller communities in the interior areas of BC or the PNW. The settings and source mixes of the various communities are important in the generalisation of applicability of the study results to the diverse communities in this region. The broader Canadian and international literature was reviewed because of its great breadth and depth of potentially relevant information.

A number of factors influence the quality of exposure-response relationships derived from observations. Some of these factors include –

- the relative risks of air quality impacts across the region associated with the mix of pollutants and levels of exposure of the populations (and sub-populations) in the region
- differences in pollutant mixtures or sources between regions, differences in population makeup (e.g., age, susceptibility)
- the different types of effects studies and their relative merits for specific interpretation and application
- the sources of uncertainty and the implications for interpretation and application
- the appropriate criteria for evaluating the quality of the individual studies and the body of air pollution health effects literature as a whole.

Pollutant mix and exposure – The Panel evaluated the literature on effects of mixtures of pollutants and the ability of studies to identify specific pollutants in the mix as agents of specific effects. Paths of exposure of affected populations in the various studies were also considered carefully.

The relative risks of health outcomes associated with exposure to different pollutants and mixes of them and different levels of exposure (concentrations in ambient air) were an important consideration for the Panel. As is stated later in this report, it now appears that the relative risk of experiencing many health outcomes does not show a population-level threshold of response at current levels of air pollution (in part due to the limitations of study designs used to assess health impacts). The implication of this observation is that it may not be possible to detect population-level thresholds. The importance of this concept for regulatory purposes is emphasized in this report. The absence of an observed threshold at current levels of air pollution, however, does not mean that one does not exist at lower pollutant concentrations.

Types of study[1] – The main source of information for this work is ecological[2] epidemiology studies for both morbidity (illness) and mortality (death). The main types of study considered for this review were three kinds of epidemiological study – either based on temporal or spatial variation of exposure.

Studies based on temporal variation of exposure:

- *longitudinal analysis* (observation of pollutant exposures and chronic outcomes in a defined population cohort over a number of years – may also include spatial variation)
- *time-series analysis* (observation of day-to-day patterns in pollutant exposures and acute outcomes in the general population in a geographic region over a limited period of time, which may still cover several years); includes studies of specific episodes
- *panel studies* (smaller groups of individuals followed clinically, and often by environmental monitoring, over time for specific outcomes)
- *indices of morbidity* (studies based on statistics such as emergency room visits or hospital admissions over time).

Studies based on spatial variation of exposure:

[1] More details about the relative merits of the various types of epidemiology study may be found in Section 3.1.1.

[2] In this context, 'ecological' means in the experience of a group of people in their environmental surroundings rather than individuals in controlled environments. Thus, epidemiological studies of this nature aim to extract information about factors in the environment(s) of a group of people that are associated with health outcomes, perhaps to the point of being able to conclude cause and effect relationships with specific causative agents (e.g., pollutants). The information is extracted by analysis of the variability of outcomes in the group with exposure of the group to pollutants over space or time (or both). Without variability of exposure and response, completely different kinds of research would be necessary (e.g., controlled exposure, clinical or toxicological).

- *cross-sectional analysis* (analysis of outcome data in populations that have experienced different environments over the long-term, either within a geographic region or in geographically diverse regions).

Studies with combined temporal and spatial variation of exposure also appear in the literature. Each of these types of study has advantages and disadvantages in extracting reliable exposure-response information from the variability of exposure (air quality as measured by ambient concentrations of pollutants) and response (health outcomes) over time and space, and in terms of comparison with other geographic regions.

Some further information examined by the Panel came from other types of studies, such as controlled exposure clinical studies of individual people, or animal toxicological studies (where dose and response can be carefully controlled and observed), but the bulk of the conclusive information relevant to the purpose of this report came from epidemiology studies as described above.

The Panel looked carefully at the methods used in each study to reach conclusions about the appropriate approaches to interpreting and applying the literature findings for use in the context of pollution regulation and impact assessment.

In the context of this report, study design is important as a factor in retrospective evaluation of the quality and applicability of results and as guidance for prospective design of studies in the region. The pros and cons of study design are addressed from both points of view here.

Sources of uncertainty – The main types of study all rely on sophisticated statistical analysis to extract information about pollutant effects from the observed relationships between exposure and response of exposed individuals or populations. The Panel addressed, at a general level, uncertainties in the statistical analysis and other sources of error, such as population characterisation and confounding and surrogate factors.

Since large groups of exposed individuals are involved in these studies, error arises in characterising exposure of the group to air pollutants associated with their movements within the study area and to attendant changes in the pollutant concentrations. Many factors may influence a given health outcome in a study group or the characteristics of the exposure, and these must be controlled for carefully in the analysis. Cigarette smoking is an example of a factor that influences respiratory and cardio-vascular health in ways similar to air pollution, so that it must be accounted for in certain types of study. These personal life style factors are not important in time series studies as they are not correlated with changes in air pollution. Physical atmospheric factors such as temperature may also play a role. For example, elevated ozone concentrations occur at elevated temperatures (i.e., greater than 25C), and higher temperature may be associated with certain health outcomes; therefore, temperature must be accounted for in studies of ozone epidemiology.

Criteria for evaluation – The quality of dose-response factors determined from epidemiological studies may be assessed against a number of criteria. Some of these criteria that characterise the quality of association between air pollutants and effects are[3]–

- strength (magnitude) of association between exposure and outcomes (response)
- consistency of association between exposure and response (robustness over space and time)
- coherence of supporting results across outcomes and studies – comparable associations for mortality and morbidity
- local or locally-relevant studies describing exposure-response outcomes that are relevant to BC and the PNW (urban areas and smaller communities),
- generalisability – results that were replicated or showed little variability over diverse geographic regions indicating general applicability.

These criteria influenced the Panel's approach to scrutinising the literature and selecting those studies for inclusion in the report. The criteria were not applied in a quantitative or rigorous analytical sense, rather as general guidance. Other factors such as the type of analytical model used (single- or multi-pollutant or statistical approach used, for example) played a role in the Panel's considerations, but again, not in a systematic way. The Panel's conclusions have their basis in consensus professional judgement among the panelists.

The Panel considered the World Health Organization's guidelines for epidemiological review and comparative risk assessment (WHO, 2001a,b). These guidelines were designed to support the Global Burden of Disease Project (GBD). WHO provides guidance for national departments that are preparing formal estimates of mortality and disease disability for submission to the GBD Project. The guidelines describe how to assemble credible epidemiological data for the full range of diseases covered. One of the recommendations from the WHO guidelines is "... choose the highest quality studies from populations similar to the target population." The output from this process is "(a) set of estimates coming from the most valid and representative sources." The process followed by the Panel is consistent with these guidelines, recognizing that the Panel members have done initial screening of the literature and other available data in the course of developing their personal databases – on which their expert opinions as reflected in this report are based.

The Panel is also aware that the US Environmental Protection Agency is in the process of carrying out a study of methodologies for PM risk analysis (PM_{10}, $PM_{2.5}$ and the coarse fraction, $PM_{10-2.5}$). Available draft information about that project covers much of the ground addressed in the Panel's report (*Proposed Methodology for Particulate Matter Risk Analyses for Selected Urban Areas*, Technical Support Document by Abt Associates Inc., January 2002 and updated April 8, 2003 to account for reanalyzed study results). The approach proposed by EPA's consultants is closely similar to the approach discussed

[3] See Zidek and Bates, 2002 for a summary of Bradford Hill's original criteria for assigning causality, on which the criteria are based in part.

in the Panel's report and should be considered as it develops for instructive analysis and recommendations.

It is important for the reader of this report to recognize some general observations that apply to all efforts to summarise existing scientific data and apply the outcome to local air pollution issues.

- Only a very small sample of the existing information can be directly quoted and described. For example, in preparing its air quality criteria documents, the US Environmental Protection Agency has to present summaries of all of the existing literature on a particular pollutant, and these often run to several volumes each of several hundred pages with hundreds of cited references.
- All summaries and databases on health effects and air pollution, even though continuously updated, are based on some evidence that is outdated by more recent studies and do not take account of some more recent information that is relevant to their applications.
- Decisions on the "acceptability" of increased risk are complex; they involve a number of considerations, including
    - whether the impacted people can avoid the risk
    - whether the people taking the risk derive any benefit from their exposure
    - whether the risk is under the control of the affected people.

  It has been shown that cultural and even ethnic factors may influence an opinion about "acceptability." Such issues are important in the interface between science and public policy, and strongly held differences of opinions are to be expected. This report does not enter these fields of controversy; instead it provides an assessment of the current content of the available objective information as a basis for such discussion.

1.5 STRUCTURE OF THE REPORT

This report continues with a summary of the relevant knowledge base for relationships between air pollutants and risk of health effects in Chapter 2. Interpretation of the knowledge base follows in Chapter 3, based on the questions that the Panel had been directed to answer (Appendix 2). Chapter 4 describes how the information might be applied in practice in the form of several situational scenarios that arise frequently in air pollution health effects assessment. Chapter 5 provides conclusions and recommendations for the application of information presented in this report and for the path forward.

Readers will find some of the material redundant, since it may be repeated along the several dimensions of the analysis in different parts of the report: (1) basic knowledge about individual pollutants and their associated effects, (2) health outcomes and associated pollutants and other factors, (3) interpretation of the knowledge base and (4) application of the cross-tabulated information to practical problems. Cross referencing is provided to enable readers to find references to the material in other contexts in the report.

2. The Knowledge Base

This chapter summarises the information that the Panel has gathered about what is known, how it is known and what to keep in mind for future application. The information is categorised first by pollutant and then by the suite of effects that are associated with individual pollutants and mixtures.

2.1. SUMMARY OF KNOWN EFFECTS BY POLLUTANT

In summarising the reports of adverse health effects of air pollutants, there are four major difficulties in the interpretation of such studies which have to be defined:

1. Almost all air pollutants occur in the presence of other pollutants. Often the correlations between them are strong (e.g., ozone and sulphates in the Northeast US and pollutants derived from vehicle emissions such as CO, NO_2, and formaldehyde).
2. Separation of the effects by regression analysis and special statistical methods is generally unreliable.
3. It is often necessary to compare studies conducted in different environments to throw light on the role of individual pollutants. Thus, data from Brisbane, Australia is valuable in the BC context because in both regions, levels of sulphates are low and concomitant $PM_{2.5}$ levels are similar and lower than in many regions.
4. There may be significant differences in personal exposure to air pollutants (by comparison with ambient monitor values) depending on climate, time-activity patterns and open *vs* closed houses with air conditioning, for example.

2.1.1 Particulate matter

As soon as routine measurements began of particles less than 10 microns in size (PM_{10}), and in some regions also of particles less than 2.5 microns in size ($PM_{2.5}$), it became clear that many adverse outcomes were associated with both of these:

- Cardio-respiratory effects
    - risk of heart attacks
    - risk of pneumonia
    - aggravation of chronic lung disease
    - increased risk of lung cancer
    - reduced survival in longitudinal studies
- Effects on asthmatics
    - increased hospital emergency visits
    - increased hospital admissions
    - increased family physician visits

    - increased symptoms and reduced peak expiratory flow rate (PEFR)
    - increased airway inflammation
    - increased prevalence[4] (possible)
- Effects on sub-populations
    - tachycardia (rapid heart rate) in the elderly
    - increased heart rate variability
    - increased angina
    - increased blood viscosity
    - increased blood *fibrinogen*[5]
    - mobilization of cells from bone marrow (banded *neutrophils*)
- Effects on normal subjects of concentrated urban particles
    - increased blood *fibrinogen*
    - increased *cytokine*[6] mobilization
    - white blood cell mobilization
- Effects seen in animal studies
    - increased mortality in frail animals
    - increased neutrophil cell mobilization from bone marrow
    - increased pro-inflammatory *cytokines*
    - cardiac arrythmia (irregularities)
    - macrophage anti-bacterial function

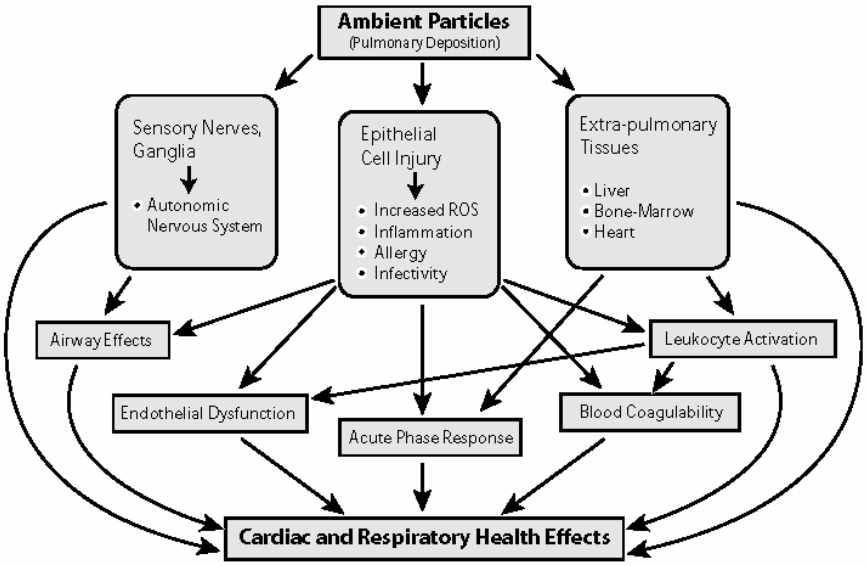
Taken together, the body of information from which this brief summary is derived shows reasonable coherence; and since the studies came from many parts of the world, relative insensitivity to climatic or demographic factors. Several important aspects remain to be elucidated, as follows:

- The relative toxicity of $PM_{2.5}$ particles of different composition. At present there is evidence that the metal content increases the toxicity (animal and human data); that crustal dust particles are relatively inert; and that all combustion-derived particles should be treated as capable of producing the above-indicated effects. Ultrafine particles may be more potent than fine particles (smaller than $PM_{2.5}$, nominally $PM_{0.1}$; 0.3 micrometer and smaller may be most effective physiologically).
- The precise mechanisms linking the particles to a specific health outcome, especially the adverse effects on cardiovascular disease.
- The possibility of interactive effects with gaseous pollutants. It is not thought that SO_2 contributes to the mortality ascribable to PM_{10}, but it may be associated with other outcomes, possibly in asthmatics. Possible interactions with ozone are difficult to determine because of the seasonal nature of ozone exposure. NO_2 may impair the ability of the host to deal with infective organisms or inhaled particles.

[4] Prevalence is the rate of occurrence of a disease in the population, i.e., the fraction of a group or age sub-group of people with a disease.
[5] A blood protein important in the clotting process.
[6] Hormone-like substances produced by cells of the immune system

The following figure (Figure 1) summarises the current hypotheses for the mechanism of health effects of PM. Many of the pathways and effects shown in Figure 1 have been established through both clinical and cohort studies in recent years. The previous criticism that there was no plausible mechanism for the health effects that have been associated with PM is no longer valid.

Figure 1. Mechanisms for PM health effects



Hypotheses for health effects of PM. Source: Utell & Frampton (2000)

Note: ROS = reactive oxygen species.

2.1.2 Ozone

Studies using controlled exposures of normal subjects to ozone have permitted relatively precise information on the acute effects of this gas to be obtained. With moderate exercise, an hour of breathing air with 60 ppb of ozone reduces lung function, probably indicating that inflammation has been induced in the lung. The initial effect of ozone is to activate the C-fibre system, leading to changes in lung function (as evidenced by decreases in FEV_1 and FVC). Ozone may also cause pain on deep inspiration. There is a wide range of susceptibility in normal subjects, but the response is repeatable. If breathed sequentially on successive days, the response is reduced after the first day. This is thought to be due to the protective effect of the inflammatory secretions produced by the first day's exposure. The following adverse health effects have been shown to be associated with increases in tropospheric ozone (the list is not necessarily exhaustive):

- Mortality
    - There is conflicting evidence on this effect; there has been less consistency in findings than there have been for PM, for example, although some studies have found statistically significant responses.
- Effects on susceptible people
    - increases in emergency visits and hospital admissions in asthmatics
    - increased pneumonia in the elderly
    - exacerbation of asthma
- Effects on the general population
    - increased hospital admissions for bronchiolitis (inflammation of the intermediate airways) in infants under the age of two
    - declines in pulmonary function with outdoor exercise found in children at summer camp; hikers on Mount Washington; field workers at Abbotsford; and lunchtime joggers
    - increased school absences due to respiratory illness in school children
    - exposure to ozone increases the subsequent response to SO_2.
- Possible long-term effects
    - demonstration of induced airflow reduction in lungs of non-smoking entering university students
    - reduced lung function growth
- Effects on animals
    - The most relevant data are the ongoing young rhesus monkey exposures which indicate an important ozone/allergen interaction leading to irreversible structural changes in the developing lung. These studies are being conducted at the Primate Center at the University of California, Davis.
    - Ozone affects the function of alveolar macrophages, leading to higher death rates in mice and rats when pre-exposed to ozone and then exposed to bacterial aerosols.

2.1.3 Sulphur dioxide

Sulphur dioxide was historically one of the first to be recognized as an air pollutant. It is now so closely associated with particulate pollution that it is difficult to be sure of independent effects. In present day cities, it is not thought to be a contributor to mortality, although it is correlated with particulate pollution.[7] Its effects can be summarized as follows:

[7] SO_2 has occasionally been associated with mortality, but on balance, most relevant recent studies have not found a convincing independent effect from PM, in the panelists' judgement. A recent study in Hong Kong, for example, that purported to find an independent effect for SO_2 has been challenged on the basis that many factors other than SO_2 concentration had changed over the study period.

- It is probably responsible for chronic mucus gland swelling leading to chronic productive cough. The prevalence of this in schoolchildren has been shown to be reduced when high levels of SO_2 and particulate pollution (as in East Germany) have been ameliorated. See Heinrich et al. (2002). Additional recent evidence from Dublin confirms this association (Clancy et al., 2002).
- Asthmatic subjects are much more sensitive to inhaled SO_2 than are non-asthmatics. If inhaled while exercising, about 20% of asthmatics have a doubling of airway resistance at levels of 0.5 parts per million (ppm) of SO_2 (and about 50% have decrements in $FEV_1 > 10\%$).
- The increased lung cancer occurrence in urban regions might be attributable to sulphate aerosol inhalation, but gaseous SO_2 might be important as well. The recent finding of an increased risk of lung cancer in pulp and paper workers exposed to SO_2 lends some support for this association (Lee et al., 2002)

2.1.4 Oxides of nitrogen

The following summarises our understanding of the effects of nitrogen dioxide (NO_2). The other component of the mixture NOx is NO, and there is no indication in the literature that NO is of concern on its own (although there are few studies upon which this conclusion is based). Other oxides of nitrogen, such as nitric acid (HNO_3) are not addressed here, other than for their presence in the form of particulate nitrates (see below). The studies on which the conclusions are based have involved both outdoor exposures and indoor levels of NO_2 gas, levels of which are higher in homes that use natural gas cooking. The effects may be summarised as follows:

- Studies not confounded by particulate pollution exposure have indicated that in asthmatic children, higher exposure levels are associated with an increased incidence of lower respiratory illness. A precise dose-response relationship has not yet been defined.
- Prior exposure to NO_2 increases the subsequent response to ozone in normal subjects.
- Higher personal exposure to NO_2 is associated with a lower forced vital capacity (*FVC*) and forced expiratory volume in one second (FEV_1) in population studies. Reductions in these lung function parameters indicate compromised lung function.
- Aggravation of asthma has been shown to occur in panel studies, but the dose-response function is not precisely known.
- NO_2 exposure is an indicator of exposure to vehicle traffic exhaust; which is associated with a reduced rate of lung growth in children, but it is not known whether NO_2 alone is responsible for this.
- A recent paper from Austria (Neuberger et al., 2002) of a longitudinal study of school children indicates that a reduction in NO_2 exposure from an annual mean of about 80 $\mu g/m^3$ to about 30 $\mu g/m^3$ resulted in an improvement in measured terminal airflow velocities. This reversibility confirms a conclusion from the Southern California Children's Health Study.

- It is doubtful whether NO_2 is an 'effective pollutant' in acute mortality.

There is a large number of studies, from Europe, Australia, and the US, involving schoolchildren in which NO_2 exposures have been measured on each child with Palmes tube passive samplers attached to their clothing; various respiratory outcomes have been logged. These observations have involved large numbers of children studied over periods of months. Unfortunately, it is not possible to reduce these results to any definite dose-response function since it is not known whether children in the highest quartile of exposure might have encountered several short-term peaks in exposure (from traffic exhaust, for example) which determined the outcome. This uncertainty is reflected in current guidelines for exposure, which vary from a one-hour level of 70 micrograms/m^3 in Finland, to 400 micrograms/m^3 in Canada, to 475 micrograms/m^3 in California, with WHO and Australia being at 200 micrograms/m^3.

An recent issue of the *Health & Clean Air Newsletter* (issue #4) sponsored by the California Air Resources Board addresses evidence for the effects of air pollution on children, and an upcoming issue (#5) includes those of NO_2. Link: http://healthandcleanair.org/.

2.1.5 Carbon monoxide

Carbon monoxide (CO) ambient levels have decreased markedly almost everywhere in the past 20 years. Some effects have been demonstrated at levels much higher than current typical ambient concentrations.

The adverse health effects of CO are well known and include:

- Increased myocardial *ischemia*[8] consequent upon the binding of CO with *hemoglobin*. A recent study in Boston found an association between ambient CO and myocardial infarcts.
- CO levels (now very low relative to levels of concern almost everywhere) have been associated with cardiac hospital admissions.
- CO does not affect the respiratory system – hence reported associations with respiratory disease indicate that CO is serving as a marker for exposure to traffic exhaust.

2.1.6 Volatile organic compounds

Volatile organic compounds (VOCs) are a broad class of pollutants consisting mainly of hydrocarbons and other organic gases. This class of pollutants contains many photochemically reactive compounds which are of concern as precursors of ozone formation more so than as toxins. VOCs also include many chemicals that are potentially toxic at ambient levels. Most evidence of health effects comes from occupational exposures at much higher levels than ambient. The only associated direct health effects, based on studies of a valley in the US with a high concentration of chemical industries,

[8] Restricted blood flow to heart muscle tissue.

are general symptoms which include headache, loss of appetite, and nausea. Some VOCs are carcinogens, but this represents a small fraction of the total class of these compounds. Studies using controlled exposures to mixtures of VOCs have indicated upper airway irritation and eye irritation (see Bates & Vedal, 2002; Hempel-Jorgensen et al., 1999; Kjaergaard, 1992 and Harving et al., 1991). Carcinogenic VOCs in vehicle exhaust have been implicated (along with diesel particulate matter) as significant contributors to urban excess lung cancer risk, namely, benzene, 1,3-butadiene, acetaldehyde and formaldehyde. An initial assessment of measurements of these and other hazardous air pollutants in the VOC class in Vancouver in 1998 indicated that about 20 merited further consideration on the basis of potential exposure and cancer risk. Their public health significance has not been fully assessed.

2.1.7 Aerosol sulphates

In many regions, aerosol sulphates (e.g., ammonium sulphate, $(NH_4)_2SO_4$)) are closely correlated with ozone in summer and are an important constituent of $PM_{2.5}$. It is not known whether the effects of $PM_{2.5}$ can be attributed in whole or in part to their sulphate content, but some animal experiments indicate that this is unlikely at the concentrations encountered.

Brauer's group at UBC have results from a few panel studies in which they looked at sulphate in relation to lung function, cardiac outcomes. In these local examples they never saw any relationship between sulphate and any of the health effects evaluated (more detail is provided below in the local studies section, 2.3.3). The Ontario studies have also generally failed to show a significant independent sulphate effect as long as there were other fine PM markers measured and included in the analysis. There are, of course, examples of associations with sulphate, but this is mostly in cases where either sulphate is a major contributor to PM and/or where sulphate was the only measure of fine PM. Accordingly, there is little evidence to support a specific sulphate effect in the Northwest.

2.1.8 Aerosol nitrates

Although aerosol nitrate compounds are important in impairing atmospheric clarity as a component of haze, no known adverse health effects are associated with them, other than their generic contribution to PM. Studies of the effects of particulate nitrates separate from other components of PM are few.

2.2 SUMMARY OF KNOWLEDGE ABOUT SPECIFIC OUTCOMES AND FACTORS

This section summarises information about some specific diseases and other physiological effects of air pollutants. It does not address indirect effects such as restricted activity days resulting from air pollution-related illness, lost school days or other quantifiable but nonetheless indirect effects.

2.2.1 Respiratory effects

ASTHMA

Since asthma prevalence is significant in Canada (and world-wide), and air pollution is indicated as a factor in its incidence elsewhere (not shown definitively in Canada yet), we treat it in some detail here. Asthma is a chronic inflammation of the airways characterised by:

- Infiltration into airways of cells from connective tissue, blood and lymph systems
- Recurrent episodes of wheezing, coughing, and shortness of breath
- Commonly, nocturnal awakening
- Variable reversible airflow restriction
- Bronchial hyperresponsiveness.

Magnitude of the problem

Asthma affects about 150 million individuals world wide, and the prevalence is increasing. In the US, childhood asthma prevalence increased on average at a rate of 4.3% per year between 1980 and 1996, but appears to have levelled off since then (although this change in trend may be due to a change in data collection methodology).

The total prevalence of asthma in Canada in 1998-99 was 8.4% of the population (2.5 million cases). In Canada, 7-14% of the pediatric population (0-19 years) experience asthma attacks (dependent on age group). In 1998-99, Canadian boys 10-14 years old had a prevalence rate of 13.5% and girls 15-19, 14.3%. About 20 children and 500 adults in Canada die from asthma each year (1998 data). The asthma mortality rate for BC was about 1.7 per 100,000 in 1996-98 (about 65 deaths per year), compared with the Canadian average of 1.2 per 100,000.

The economic burden of asthma in the US is estimated at $12.7 billion in 1998, or about $42 per capita (Weiss and Sullivan, 2001). Canadian asthma costs were estimated in 1996 for 1990 as about $7.50 per capita, or somewhat more than $200 million CAD (Krahn et al., 1996). US data cited in the Canadian report indicated costs of about $3 billion US ($11.50 per capita) for the same time period. If the comparative data are correct, they imply that US asthma costs quadrupled in the 1990s. Since the prevalence rates are similar in Canada and the US, it is reasonable to assume that Canadian costs have increased similarly since 1990.

Risk factors for asthma prevalence are:

- Family history and atopy (hereditary disposition)
- Allergen exposure and sensitization
- Gender
- Environmental tobacco smoke (ETS)
- Premature birth – low birth weight
- Air pollution (possible, but not definite).

Table 1 summarises air pollution effects in subjects with asthma (Koenig 1999).

Table 1. Relationships of asthma endpoints with pollutant exposures

Asthma Endpoint	SO_2	NO_2	PM	Ozone
PFTs[c]	++	0	+	++
BHR[d]	+/-	+/-	*	++
Emergency admissions	*	+	++	++
Symptoms/Med use	*	+	+	+
Inflammation	++/-	+/-	*	++
Allergen response[a]	*	+	*	+/-
Immune effects[b]	*	+	*	+

* Indicates no or limited evidence
+ Indicates a statistically significant association (++ a strong one); - indicates no significant association; +/- indicates mixed reports
a. Potentiation of allergen response by prior exposure to air pollutants
b. Immune system changes associated with air pollution exposures
c. Pulmonary function tests
d. Bronchial hyperresponsiveness

Aggravation of asthma & possible influence on prevalence

There are many studies showing increased asthma emergency visits and hospital admissions in association with PM_{10} and independently with ozone. Some of the ozone studies (in Toronto, Atlanta and New Jersey, for example) are questioned because of the close association (high correlation) between ozone and aerosol sulphates in the summer. That ozone alone can cause aggravation of asthma has been shown in Brisbane, Australia (Petroeschevsky et al., 2001), where an analysis of more than 13,000 asthma admissions to hospital found a strong association with ozone; but sulphates are virtually non-existent. The relative risk for asthma hospital admissions was found to be 1.090 (CI: 1.042-1.141) per 10 ppb increase in 8-hour average ambient ozone concentration (lagged 5 days). The Brisbane data are important because of the similarity of the geographic setting and pollutant mix to Vancouver's (although climatological differences may be significant, and ozone levels are somewhat higher).

A recent panel study of moderately severe asthmatic subjects, who were clinically closely followed over an eight month period, conducted in Paris, France (Just et al., 2002 and Desqueyroux et al., 2002) showed a close association between asthma worsening and particulate pollution levels; and another recent study in Barcelona, Spain (Sunyer et al., 2002) found an association between NO_2 levels and the risk of mortality in severe asthmatics.

It is not easy to imagine an exposure that can cause worsening of asthma that would not also have an influence on prevalence.[9] Studies of asthma prevalence between different

[9] A reviewer considered this statement to be unsupported, but it stands in the judgement of the Panel. The paragraph that follows contains sufficient cautionary qualification.

communities with different levels of air pollution have generally been negative. However, there are other studies suggesting that asthma prevalence is affected by increased exposure to traffic emissions, and the Southern California Children's Health Study found an increased risk of asthma in a prospective analysis in physically active children in high ozone communities (generally much higher than experienced in BC and the PNW). None of these observations can be taken as definitive, as it is difficult to control for other factors, and in the Southern California study, as it was prospective, the sample of children developing asthma while in the study was relatively small in number. An international study of asthma prevalence concluded that differences in asthma prevalence were probably to be ascribed to environmental factors, but these have not yet been elucidated. Zwick et al. (1991) found higher prevalence in high ozone areas of Austria. Dutch studies have also found association between traffic-related pollution and asthma prevalence (Brauer et al, 2002), although in an age group of children (0-2 years) in which a diagnosis of asthma is not normally ascribed.[10]

In any health risk assessment process, evaluation of possible future exposures of vulnerable populations to air pollutants has to be an essential component.

PNEUMONIA HOSPITAL ADMISSIONS OR BRONCHIOLITIS

An increased risk of hospital admissions for pneumonia in the elderly has been found to be associated with PM_{10}. Ozone (together with sulphates) has been shown to be associated with hospital admissions for bronchiolitis in infants under the age of 2 years; a similar study in Mexico City showed an increase in respiratory infections requiring medical care in infants aged from 3 months to two years, associated with levels of particulate pollution.

RESPIRATORY MORTALITY

Time-series studies have consistently shown an increase in respiratory mortality in association with PM_{10} levels. Although an association exists between daily mortality and SO_2 and sulphate levels, it is not generally considered that these pollutants should be considered as causative. In regions where uncontrolled coal burning, leading to high levels of SO_2 and particulate pollution, still exists (as in North Bohemia), it has been shown that respiratory mortality in the post neonatal period is associated with higher levels of pollution, probably on account of an increased risk of pneumonia.

RISK OF RESPIRATORY INFECTIONS

In children, respiratory infections are so common in the first few years of life that it has been difficult to determine the influence of air pollutants. However, there is now definitive evidence that school absences in Los Angeles for a respiratory illness, are associated with the community ozone level for the previous 48 hours. There is also

[10] Unpublished extended analysis of these data confirm the association with asthma prevalence in an older age group of children in which asthma may be properly diagnosed (M. Brauer, personal communication, 2003).

evidence that respiratory infections are commoner in children living within 100 metres of a heavily traveled vehicle throughway, particularly if this carries a high level of heavy diesel traffic. In Provo, Utah, it was shown that when the local steel mill (responsible for 60% of the ambient PM_{10}) was shut down, there was a dramatic reduction in the admissions of children with acute *bronchiolitis* to the local hospital.

RISK OF LUNG CANCER

The American Cancer Society (ACS) cohort of more than 500,000 adults followed for 16 years showed that lung cancer rates were higher in more polluted counties, after all other factors had been allowed for. Previous analyses had indicated an approximately 30% increase in risk of lung cancer between the most polluted and the least polluted counties, and the ACS study confirmed this conclusion. Specific association with fine particles in the pollution mix has been demonstrated recently for the ACS cohort, with a response factor of 8% increase in lung cancer mortality for each 10 $\mu g/m^3$ increase in long-term $PM_{2.5}$ exposure (1983-1998). To quote from a recent paper on the ACS study: "Long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality." (Pope et al., 2002).

2.2.2 Cardio-vascular effects

DAILY MORTALITY AND PM_{10}

The time-series data showing a significant association between daily mortality and exposure to ambient PM_{10} have been confirmed in over 100 different studies. Recently, the magnitude of the increased risk has been revised downward in some series, but this has not affected the general conclusion of robustness. Respiratory mortality is more strongly associated with daily PM_{10} than is cardiovascular mortality, but since cardiac deaths in North America outnumber respiratory deaths by a large factor, cardiovascular mortality plays an important part in determining the overall significance of the relationship. This finding launched a major research initiative to discover the mechanism behind the association. So far, the following findings have been reported on the mechanism of the effects.

- Fine particle inhalation (concentrated $PM_{2.5}$) induces cytokine release from the lung, indicating a pro-inflammatory response.
- Increased PM_{10} levels are associated with tachycardia in the elderly, earlier onset of angina in those with heart disease, and significant changes in the blood including increased viscosity, increased fibrinogen, and an increase in the neutrophil cell count, indicating that neutrophils have been released from the bone marrow. The released cytokines might have the effect of rendering plaques in the coronary circulation unstable or more easily detached, so that they may accumulate elsewhere causing blockage.
- Decreased heart rate variability (a premonitory sign of heart attack) has also been reported.

- Increased plasma endothelin (a vasoconstrictor) and reduced arterial diameter

Although the exact phenomena underlying the association between PM_{10} and the risk of cardiovascular mortality have yet to be defined, these results, taken together, provide a convincing body of evidence supporting the time-series observations summarised above.

HOSPITAL ADMISSIONS

General hospital admissions for cardiovascular diagnoses have been shown to be associated with particulate levels. An important study conducted in Boston of 770 hospital admissions for acute myocardial infarction over an eight month period showed a strong association with $PM_{2.5}$ levels during the preceding 48 hours. This provided important confirmation of the evidence for the association between PM exposure and effects from the time-series studies. However the data are not unanimous, since a recent analysis of over 5,000 admissions to hospital for myocardial infarction in Seattle did not find an association with PM_{10} (J. Koenig – personal communication, 2002).

2.2.3 Air pollution exposure issues of current concern

WOOD SMOKE[11]

Wood smoke is an especially important component of PM in BC and the PNW and is treated here in some detail. A great deal of relevant work has been published on the impacts for wood smoke – that is, fine PM produced from the combustion of woody biomass. Appendix 4 provides more detail from the literature on the subject for residential wood combustion, forest fires and agricultural burning.

The main findings of that work are as follows:

- Exposure to emissions from wood stoves in the home is associated with increased risk of respiratory illness and increased respiratory symptom reporting in children.
- From a case-control study of infants, there is an increased risk of acute lower respiratory illness in 1-24 month-olds for children living in a home that cooked with any wood or had indoor particle concentrations above 65 $\mu g/m^3$.
- Adults are also affected by indoor combustion sources, including wood stoves, which have been associated with increased asthma exacerbation.
- In areas of Oregon and Montana, where wood smoke may comprise very high percentages of winter PM, associations were found between decreased lung function and exposure to higher levels of wood smoke.
- In Seattle, 1-5 year-olds showed significantly higher levels of congestion and wheezing in the higher pollution areas, and in the high exposure area, significant

[11] From residential space heating, agricultural biomass burning, forest fires and waste burning.

decreases in lung function were found in another study that found significant lung function decrement in asthmatic subjects with increasing exposure to wood smoke. Significant association between exposure to PM_{10} in Seattle and asthma emergency room visits. At the annual average PM_{10} during the study period of about 30 $\mu g/m^3$, PM_{10} seemed to be responsible for 12% of the asthma emergency room visits. As much as 60% of fine particle mass annual mean level in some Seattle residential neighbourhoods can be from wood burning.

- A detailed comparison of recent data for PM_{10} impacts from Christchurch, NZ with hospitalization data for the central interior regions of BC is shown in Appendix 5. During winter, a major part of Christchurch's PM loading is from wood smoke. The NZ data show, for example, a significant increase in acute respiratory infections between daily PM_{10} ambient concentration between highest and lowest concentration quartile days. The NZ data also show a significant increase in cardiac conditions for the same PM_{10} concentration difference. The central BC hospitalization data are very similar to the NZ data – with potentially similar exposure to wood smoke, which suggests that information from studies such as the Christchurch study may be used to carry out health risk evaluations for analogous areas of BC and the PNW. More detailed research would need to be carried out with proper study design to expand on the suggestive similarities in Appendix 5.
- The studies of the impacts of forest fires and agricultural burning summarized in Appendix 4 generally show significant respiratory effects from exposure in both occupational and general environmental settings to the sometimes very high concentrations of fine particles produced from forest fires and agricultural burning.

DIESEL EXHAUST PARTICULATE

The suspicion that diesel exhaust particles might be an important constituent of urban $PM_{2.5}$ in terms of inducing adverse effects rests on the following observations.

- The diesel on-road and off-road fleets emit a disproportionately high proportion of particles compared with the far more numerous gasoline-powered vehicles – the two classes may contribute about equally to emission totals.
- Controlled human exposures to diesel exhaust indicate that it is capable of inducing lung inflammation and increasing the response to an inhaled allergen.
- Acute respiratory illnesses in children are increased if they live close to a freeway with heavy diesel traffic (responses increasing with proportion of diesel traffic).
- Experimental studies show that diesel exhaust exposure increases the response to an allergen.
- Diesel exhaust is thought to be a carcinogen based on occupational studies of cohorts of train drivers, truck drivers, men underground in mines using diesel engines, forklift operators, and others. A special scientific panel advised the Governor of California in 2000 that diesel emissions should be classified as a human carcinogen.

- A recently completed US EPA assessment concluded that diesel emissions are a "probable" human carcinogen (confirming an earlier International Agency for Research on Cancer (IARC) finding).

It may be concluded that exposure to diesel particles is an important component of urban particulate exposure as a whole, and there is a considerable body of evidence suggesting that diesel particles may constitute a particularly toxic fraction of $PM_{2.5}$. It is clear from the statistics of diesel traffic volume that some segments of the population may have experienced a substantial increase in exposure to diesel particles over the past fifteen years, although emission inventories generally show a decrease in diesel PM emissions as cleaner engine technology penetrates the market. Inventories do not yet address ultrafine particles, and estimates of $PM_{2.5}$ emissions are still only approximate.

A number of studies of the impacts of traffic generally on health have been published. Appendix 3 summarizes the findings from the individual studies. Many of these studies used elemental carbon[12] as an indicator of exposure, which implicates traffic-related PM with asthmatic symptoms, asthma incidence, decreased lung function and now mortality (Hoek et al., 2002). It is not clear if these studies point directly to diesel exhaust (although in at least one set of studies it was the relationship to truck counts and not total vehicle counts that was the important predictor) or to traffic emissions in general or even if that is an important distinction given that trucks and cars travel on the same roads. The study by Hoek et al. (2002) – the Netherlands Cohort Study on Diet and Cancer (120,000 + participants) – found that cardio-pulmonary mortality was significantly associated with living near a major road and that the association was much greater than for total deaths. A recent study from New York City has provided important new information on public exposure to diesel vehicle emissions (see Lena et al., 2002 and discussion in Chapter 4). Lena et al. found that sidewalk concentrations of elemental carbon in a South Bronx neighbourhood in New York City were nearly three times those at a control monitoring site. The neighbourhood was heavily impacted by heavy-duty truck traffic.

A European study by Kunzli et al. (2000) attributed about 50% of air pollution-related mortality and morbidity in France, Austria and Switzerland to motorised vehicle traffic. This study is referred to in Section 3.2.1 as an example of the application of the estimation methodology recommended by the Panel.

In addition to the linear relationship between $PM_{2.5}$ and mortality shown in Figure 2, (see section 2.4.5 below) Schwartz et al. (2002) found a similar linear relationship between the traffic-related $PM_{2.5}$ (determined by source apportionment techniques) and total mortality. This does not implicate diesel emissions explicitly.

[12] Much of the particulate matter emitted from internal combustion engines is soot composed mainly of elemental carbon. Until recently, elemental carbon was thought to be a good tracer for diesel emissions, but detailed sampling and analysis programs have shown that gasoline engines also emit elemental carbon. The much larger number of gasoline-powered vehicles on the road makes the total particulate emission about equal for the two classes of vehicles (A. Gertler, Desert Research Institute, private communication, 2002).

COAL-FIRED POWER PLANT EMISSIONS

Some studies have identified specific impacts for particulate emissions from coal-fired power plants, but it is difficult to separate the effect from other components of PM. This may be an important factor in some regions of North America – including some areas in the PNW – at present there are no coal-fired power plants in BC. See Section 2.3.1 and research described in Laden et al. (2000), Levy et al. (1999) and Levy et al. (2002a).

Laden et al. found that for the Harvard Six-Cities Study pooled database, source apportionment resolved $PM_{2.5}$ data indicated a 1.1% increase in daily mortality for a 10 $\mu g/m_3$ increase in coal-fired power plant $PM_{2.5}$ (95% CI: 0.3-2.0%). This compared with a 3.5% increase in daily mortality for a 10 $\mu g/m^3$ increase in traffic-related $PM_{2.5}$ (95% CI: 1.7-5.2%) and an overall increase of 1.5% in daily mortality for 10 $\mu g/m^3$ of total $PM_{2.5}$ (Schwartz et al., 2002). That is $PM_{2.5}$ from coal-fired power plants had about the same effect as $PM_{2.5}$ generally, but traffic-related $PM_{2.5}$ had more than twice the effect.

Levy et al. estimated the impacts of power plants in the Boston and Chicago areas. The impacts of the known emissions of fine particles and secondary-pollutant-forming gases from the plants were estimated by dispersion modelling and application of CRFs for PM_{10} (Boston) or $PM_{2.5}$ (Chicago) that were not specific to coal-fired emissions (i.e., general PM CRFs). For the Chicago area, the reseachers found a population-weighted (i.e., exposure-weighted) annual average impact from nine plants to be 0.3 $\mu g/m^3$ (total of primary and secondary PM). The size of the population exposed at this level was 33 million, so the incremental exposure due to the plants was estimated to account for 320 premature deaths per year. Their approach to this estimate was similar to the one which we recommend in Section 3.2. This analysis has been challenged by Ames et al. (2002), but the rebuttal does not affect the main conclusion of the work.

Levy et al. (2002b) applied a similar analysis to exposure of the demographically-characterised sub-populations of Washington, DC in relation to coal-fired power plants. They concluded that application of 'Best Available Control Technology' to the five plants analysed would result in 240 fewer premature deaths per year in the greater Washington, DC area.

SO_2/SULPHATE EXPOSURE ISSUES

Most particulate sulphate is formed from oxidation of SO_2 in the atmosphere rather than being emitted directly (as sulphuric acid mist or other forms). The atmospheric chemistry that contributes to that transformation is complex and involves, essentially, NOx, ammonia and water vapour, in addition to SO_2. From the human health perspective, reducing SO_2 emissions, thereby, in principle, reducing sulphate concentrations, would appear to be unquestionably beneficial. Acid sulphates (e.g., sulphuric acid), however, compete with acid nitrates (primarily nitric acid) for available ammonia to form stable particulate sulphates and nitrates, respectively. At issue, then, is whether reducing exposure to SO_2 will in fact lead to concomitant reduction in exposure to total particulate matter, for although the sulphate component of PM is reduced, the mass contribution to PM can be made up, in principle, by additional nitrate that is formed by reaction with the available ammonia to form ammonium nitrate. The chemical reactions that control the

sulphur-nitrogen-ammonia-water vapour system are complicated and sensitive to temperature and humidity and need not be detailed here. In principle, a more or less equivalent mass of ammonium nitrate could be added to PM to compensate for the nominal reduction due to reduced ammonium sulphate. Simultaneous reductions in SO_2, NOx and VOCs, moreover, complicate matters even further, since the balance of oxidants in the atmosphere could end up producing either more or less particulate sulphate, depending on the relative changes in the various contaminants.

Pandis has estimated this effect for Vancouver in a national study of air quality impacts of reducing sulphur content of gasoline and diesel fuels (Atmospheric Science expert Panel, 1997) and more recently, in detail for Ontario (Pandis, 2002). The sulphate-nitrate transfer is probably significant only in areas where high rates of conversion of SO_2 to sulphate occur, namely, under oxidizing, smog-forming conditions. Outside of the Lower Mainland, the Okanagan Valley and Victoria, most of BC does not appear to be in the latter category. The Sulphur-in-Fuels Study indicated that the effect of nitrate compensating for reduced sulphate would be small in the Vancouver region.

Reduction in SO_2 / sulphate does contribute to overall reduction in PM and reduction in gasoline S levels also leads to reduced nitrate aerosols due to improved catalytic converter performance and in diesel fuel to reduced diesel PM emissions.

On balance, it is reasonable to assume that reduction in SO_2 emissions will lead to reduced exposure to both SO_2 and particulate sulphates – as well as fine particles – in ambient air.

2.3 Effects of demographic factors, pollutant source mix, pollutant characteristics

Many factors influence epidemiology studies and can determine the transferability of the results to other locations and populations. In this section, we summarise some of these.

2.3.1 Evidence for specific source impacts

There are two major approaches to evaluating specific source impacts. The first of these is to conduct panel studies in areas where specific source(s) predominate – and couple epidemiological analysis with careful characterization of source impacts. This is an indirect approach, but if replicated in different locations can provide meaningful input. Examples are studies of wood smoke-impacted communities – these studies, as well as those conducted in urban areas where wood smoke is a major source, do suggest that wood smoke is a source category that is associated with adverse health impacts (see above).

Of course, it is very difficult to conduct these source-specific studies in most urban areas, given the fact that the major sources of air pollutants in urban areas are common. One exception are locations where windblown dust is a seasonal contributor to particulate matter concentrations - studies in Spokane, Palm Springs and in the Central Valley of California have all been conducted.

The Spokane PM Health Effects Study was initiated in 1994. The objective was to determine the differential effects of wind blown dust and combustion PM. The investigators used an atmospheric stagnation persistence index as a surrogate for combustion PM concentration. That analysis found an association between asthma visits to emergency departments at the four Spokane hospitals and stagnant air. The relative risk of a hospital visit between stagnant days and windy days was 1.12 (95% CI 1.05-1.19) for an increase of 11 hours of low wind speed (Norris et al., 2000). Schwartz (and University of Washington co-investigators) later published an analysis showing no association between mortality and 19 episodes of wind-blown dust in Spokane during the time period. Further analyses of the Spokane data are forthcoming.

Studies that evaluate within-city variability in air pollution concentrations are a modification of this approach. Studies of this type have mainly been conducted in Europe, although there are a few examples of similar studies in North American locations. In most cases these studies have focused on traffic-related emissions (see Appendix 3), although there are examples of studies in which specific point sources have been evaluated (Hruba et al., 2001).

The second approach is to study multiple cities and/or multiple time periods simultaneously and to use a source apportionment approach to assign measured health impacts to specific source categories, based on factor analytic approaches. The study by Laden et al. (2000) is an example in which impacts appeared to be strongest for motor vehicles and power plant source factors. They found that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ from mobile sources accounted for a 3.4% increase in daily mortality (95% confidence interval (CI): 1.7-5.2%), and the equivalent increase in fine particles from coal combustion sources accounted for a 1.1% increase (CI, 0.3-2.0%). $PM_{2.5}$ crustal particles were not associated with daily mortality.

A similar analysis was conducted in Toronto (Ozkaynak et al., 1996) and highlighted the importance of local motor vehicle-related emissions relative to long-range transported pollutants.

Recently, Schwartz et al. (2002) found for the cities in the Six-Cities Study that $PM_{2.5}$ from traffic-related particles (determined by source apportionment techniques) is linearly associated with mortality. The relationship was a 3% increase in mortality per 10 $\mu g/m^3$ increase in traffic-related $PM_{2.5}$ (compared with about one-half that effect for $PM_{2.5}$ generally).

2.3.2 Attribution of effects to individual pollutants or groups of pollutants

The evidence for specific pollutant effects has been presented above under the individual pollutant headings. In summary, many studies seem to implicate motor vehicle emissions as a major source contributing to health impacts, as well as biomass burning (wood, agricultural burning, forest fires) and to a lesser degree coal-fired power plants (see Levy et al., 1999 & 2002 for a summary). Motor vehicle emissions contribute significantly to

urban emission inventories (for NOx and VOCs especially), and studies have indicated that traffic-related PM may be more potent than general ambient PM.[13]

There is very little direct and consistent information on specific PM components that are associated with health impacts. Particle size has been evaluated, with the predominance of evidence suggesting effects most closely linked to the fine fraction – as measured by $PM_{2.5}$ or PM_1, although several studies have implicated coarse particle sulphur as well. Data on ultrafine particles (< 0.1 micrometer) is still quite mixed – both positive and negative studies.

Gaseous pollutants have also been associated with health impacts, in particular CO and O_3, and to a lesser extent SO_2. While the case for specific impacts of ozone is strongest (both acute and chronic health effects), the effects associated with CO and SO_2 may in fact be due to the role of these compounds as surrogates for PM from source categories from which they are co-emitted (vehicle emissions in the case of CO and coal-fired power plants in the case of SO_2). NO_2 may also be a surrogate for particles with which NOx is associated with vehicle emissions, although there is evidence of an independent effect on morbidity.[14]

2.3.3 Importance of PM physical and chemical characteristics and source origins

The literature on PM effects has been reviewed in earlier sections of this chapter. The effect of PM size has been studied extensively – see Schwartz et al. papers for examples where $PM_{2.5}$ (fine fraction), PM_{10} (inhalable fraction) and coarse fraction (PM_{10} minus $PM_{2.5}$) have been compared. In those analyses, $PM_{2.5}$ has a much stronger relationship to health effects than other metrics. However, this is not a universal conclusion – studies from Mexico City, Santiago and Phoenix also implicate the coarse fraction. This may have to do with particle composition and/or correlations amongst different pollutants. Most consistent results are seen for $PM_{2.5}$, but this is not universal. The coarse fraction has generally, but not always, been found to be less potent than the fine fraction.

The finding of Laden et al. (2000) and Schwartz et al. (2002) that traffic-related $PM_{2.5}$ may be more potent in its association with mortality than general $PM_{2.5}$ is the most striking example of differentiation among source origins. It is not yet clear to what extent the traffic-related PM effect is influenced by diesel PM, since diesel and gasoline-powered vehicles appear top emit approximately equal fractions of traffic-related PM emissions. Studies from Europe have implicated roadways with predominantly diesel traffic more strongly in effects than those with mixed or predominantly light-duty vehicle traffic, but definitive results are not yet available.

Several studies have found that soil-related PM is less potent than combustion-related PM in associations with health effects, but others have found no such effect. There is insufficient evidence to conclude that there is a definite difference in potency. See

[13] A reviewer pointed out that studies have not clarified whether traffic particles are more potent than other combustion particles or elevated exposure near roadways on its own is implicated in the elevated mortality and morbidity results.

[14] A recent report from Hong Kong appeared to identify an independent effect of SO_2 on mortality, but this finding has been successfully challenged by other researchers in Hong Kong, in the Panel's opinion.

Section 2.3.1 for references to the Spokane PM Health Effects Study as a local example of the finding that soil-derived particles have low potency.

2.4 FACTORS THAT INFLUENCE HEALTH EFFECTS STUDIES RESULTS

2.4.1 Effects of socio-economic status

There is ample evidence that socio-economic status impacts the susceptibility to the adverse health impacts of air pollution. Samet et al. (2001) summarize the deliberation of an American Lung Association (ALA) workshop where this issue was discussed in detail.

Among the key observations from the ALA workshop were:

- Socioeconomic factors are important because certain conditions such as asthma are more prevalent in low-income communities,[15] but also because other factors common to those living in poverty may also increase exposure to air pollutants or other toxins, increase susceptibility, or both. Other community-level factors may also be important in determining the impact of air pollution, including information flow, existing networks, strengths of networks, extent of resource sharing, and the degree of the community's political empowerment or lack thereof, as well as cultural factors. The reports indicate that there are few data available to explain the mechanisms by which the social context of a community may contribute to increased susceptibility to ambient air pollutants.
- Socioeconomic status may impact air pollution exposure due to disparities in proximity to major air pollution sources, especially point sources (Jerret et al., 2001). In addition, recent studies from California, New York and Europe describe within-urban area differences in NO_2 and PM concentrations in relation to major roads, indicating that residents of different sections of urban areas may be differentially exposed to some air pollutants. If location of residence corresponds to socio-economic status differences, then the result would be an impact of socio-economic status on the health effects of air pollution.

The recent reanalyses of the Harvard Six Cities and the ACS cohort studies found that the air pollution mortality risk was only significant for those with the least amount of education. Data limitations did not allow the underlying cause of this relationship to be explored further – exposure differences or differences in personal characteristics. This socio-economic status impact was also confirmed in the follow-up of the ACS cohort (Pope et al, 2002).

A recent approach to assessing socio-economic status as a factor in effects studies has been to examine variability in effect estimates across different locations within a study area – when these are analyzed in a common study. Jannsen et al. (2002) indicate that for hospital admissions (16 largest US cities), regression coefficients were associated with the percentage of PM emissions from highway diesel vehicles and with the percentage of homes using air conditioning – both of which are correlated with socio-economic status.

[15] It is not known whether this is true in Canada.

2.4.2 Harvesting and lag effects; survival rates

The large number of epidemiologic studies consistently showing association between PM and premature mortality have engendered a number of criticisms. Primarily, the concern is that the association is simply a case of mortality displacement ("harvesting"), and that individuals dying at increased numbers when air quality deteriorates are frail and would die within days without influence from air pollution. If that were true, one should see a decrease in death counts following the increase seen during air pollution episodes – which is not observed. Both Schwartz and Samet have addressed this problem, and in general have shown that such "harvesting" is not a significant factor in premature death. Using a series of moving averages of mortality, Schwartz (2000) examined whether the strength of associations varied over several averaging periods, and found that the associations between death and PM remained significant and in fact grew stronger over longer averaging periods. This study and a number of others indicate that a harvesting of frail individuals at a threshold of death is not occurring.

The NMMAPS studies (and other time-series studies) have found that the maximum concentration-response factor (CRF) is for deaths lagged one day from the exposure change. A two-day lag time produced a smaller coefficient. Recalling that time-series studies evaluate day-to-day acute response to changing exposure, the fact that a one day lag produced the maximum response does not necessarily support a harvesting hypothesis. The results from long-term cohort studies show significantly reduced life expectancy in communities in North America with high pollution compared with low pollution areas – by several years in some cases. Studies of life-years lost in response to air pollution exposure are the subject of current research.

2.4.3 Evidence for fetal or neonatal effects of air pollution

Studies that suggest air pollution may influence birth outcomes are beginning to appear. Low birth weight of infants is a considerable public health concern. In the US, 65% of infant deaths occur among low birth weight infants[16]. According to the latest data, 7.6% of infants in the US are born in the category of low birth weight. There is no doubt that environmental factors affect birth weight. The most carefully studied example is environmental tobacco smoke (*ETS*). Exposure to *ETS* during pregnancy is known to be statistically related to birth weight of the infant. Maternal smoking, of course, is an important factor in a number of fetal and neonatal effects. The cause is likely reduced oxygen supply to the fetus mediated by CO, but this is not demonstrated.

Recent studies suggest that exposure to ambient 'criteria' air pollutants such as particulate matter (PM), ozone, and sulphur dioxide is also associated with low birth weight. In US studies, researchers have found associations with PM and ozone/sulphur dioxide, respectively, and birth weight. Another study using data from six eastern US cities also reported a significant association between birth weight and both ozone and SO_2. The relationship between non-criteria pollutants, the so called 'air toxics' and birth weight in the US is uncertain. The mechanisms underlying these effects are largely unknown.

[16] Defined as birth weight below 2.5 kilograms

There are several studies linking proximity to traffic and traffic-related pollutants with low birth weight and premature births (all conducted in Los Angeles). See table in Appendix 3 and references there to work by Ritz et al. and Wilhem and Ritz.

2.4.4 Vulnerable populations at risk

Children, the elderly, and people with pre-existing diseases such as diabetes, respiratory disease, and cardiovascular disease appear to constitute susceptible populations (EPA, 2002).

SUSCEPTIBILITY FOR MORTALITY

Identifying the populations most sensitive to adverse health effects of air pollution has been a goal since the early 1990s when premature deaths attributable to PM exposure became prominent. Schwartz published a recent paper related to identification of susceptibility entitled *Are there sensitive subgroups for the effects of airborne particles?* (Zanobetti et al., 2000). Data described in this paper were for hospital admissions in Cook County, Illinois (Chicago). The following risk factors related to PM exposure in the admitted group were identified.

Specific high relative risks associated with PM_{10} exposure:

- for all patients, admissions for pneumonia
- for patients with previous asthma diagnosis
- for patients with cardiovascular disease (CVD) conduction disorders
- for patients with cardiac dysrhythmias (irregular heartbeats)
- for patients under treatment for congestive heart failure

For all patients, the following groups had significant relative risks associated with PM_{10} exposure:

- for all patients, admission for CVD
- patients with concurrent respiratory disease
- patients with acute bronchitis
- patients with acute respiratory infections
- patients with pneumonia
- patients with COPD

These findings are consistent with other studies that have identified similar relationships.

A series of studies in Montreal by Goldberg et al. (e.g., 2000, 2001, 2003) identified that some groups of persons with cardio-vascular disease are more susceptible to exposure to ambient particles than others. Significant associations were found between exposure to PM (as measured by coefficient of haze) and mortality due to acute lower respiratory diseases, chronic artery diseases and congestive heart failure.

SUSCEPTIBILITY FOR MORBIDITY

Age may be a susceptibility factor, although it has not been studied systematically. McDonnell (1993) analyzed over 200 subjects who had participated in controlled ozone

exposures at the US EPA laboratory. The study found a negative relationship between FEV1 decrement and age, over an age range of 15 to 35 years. Other studies indicate that subjects over 55 years of age show less ozone-induced decrements in lung function than younger adult subjects. Several researchers have reported that children do not report respiratory symptoms upon exposure to ozone to nearly the same degree as adults. The concern is that lack of symptoms may remove an important feedback mechanism for prevention of adverse respiratory effects.

Cardio-vascular effects have been shown by panel studies that have seen changes in heart rate variability (*HRV*) and increases in indicators of inflammation and thrombosis. It is anticipated that US EPA is soon to publish effects from common air contaminant exposures in healthy elderly showing *HRV*, *fibrinogen* and other adverse cardiac events. This body of information indicates that the elderly are a susceptible group.

Mann et al. (2002) found (in Southern California) significant association between exposure to air pollutants (especially CO and NO_2) and hospital admissions for ischemic heart disease in those patients with accompanying congestive heart failure and/or arrhythmia.

CHILDREN

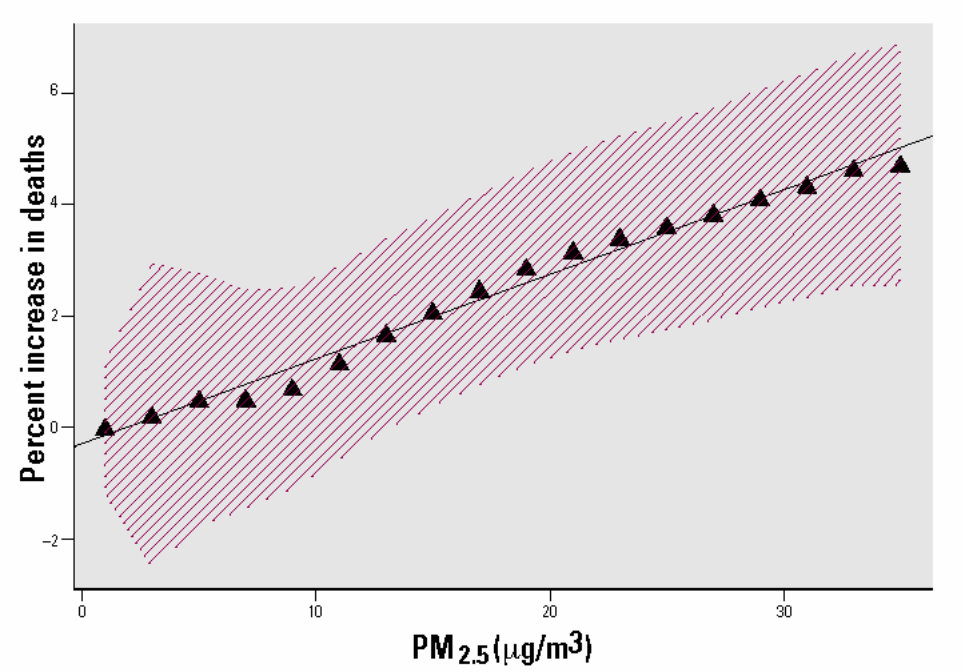
A large body of literature is developing on the effects of air pollutants on the respiratory and cardiovascular health of children. As noted in the discussion of effects of NO_2 on children above (Section 2.1.4), a forthcoming issue of the *Health & Clean Air Newsletter* (Issue #4) will be devoted to effects on children (http://healthandcleanair.org/). Since the body of evidence is still relatively sparse, the Panel cannot classify the certainty of the associations with the same confidence that is possible for some of the other relationships cited above. Studies have demonstrated effects associated with exposure to NO_2, $PM_{2.5}$ and ozone for –

- prevalence and aggravation of asthma
- small airway disease (chronic bronchiolitis)
- respiratory infections
- congenital heart defects
- premature mortality.

2.4.5 Thresholds

A recent study by Schwartz showed convincingly the absence of a threshold in the association between $PM_{2.5}$ and daily mortality (Schwartz et al., 2002). The following figure, Figure 2, reproduced from Schwartz et al. (2002) shows the robustness of the relationship for $PM_{2.5}$ down to levels that are essentially urban background (about 2 $\mu g/m^3$). The results are from the Harvard Six-Cities Study. This observation (and other similar findings in searching for a threshold in exposure-response relationships) supports the argument that at current levels of exposure in North America, Europe and elsewhere there is no discernible threshold of response to PM exposure with respect to mortality. The consistency argument would extend this conclusion to PM-related morbidity as well.

Figure 2. Overall estimated dose-response relation between $PM_{2.5}$ and daily deaths (from Schwartz et al., 2002)



(Data from the Harvard Six Cities Study showing daily deaths plotted against $PM_{2.5}$ values)

Other searches for thresholds of response to PM and other air pollutants have not found evidence of other than a continuing linear concentration-response function with decreasing concentration. No sign of a threshold was found in the NMMAPS analysis, for example.

Brauer et al. (2002a) have shown analytically that an individual's threshold (or uniform population threshold) could be masked by lack of specificity or uncertainty in exposure estimation in time series studies. In the likely case, however, that there is a distribution of individual thresholds in a population – some people having relatively low thresholds of response (chronically or temporarily 'frail' people) – it may be that individuals with lower thresholds of response contribute to the mortality and morbidity results.

One of the problems in determining whether a threshold exists for air pollution-related mortality or morbidity is inherent in the different statistics of clinical studies of individuals and population or cohort studies. Individual thresholds, if they exist, can be determined fairly accurately in a controlled exposure environment for a group of individuals under observation. Determining the group mean threshold, whether in a clinical study group or a large population, at a level of statistical significance introduces uncertainty in such an estimate. It is the level of statistical significance of that determines whether a threshold can be defined in epidemiological studies. See Hazucha (1987) for an analysis of the issue of individual *versus* group threshold determination. Exposure to a mixture of pollutants, as in ambient air, also makes definition of the precise pollutants(s) responsible for observed effects difficult.

In any case, the issue of whether effective thresholds of response to air pollutants exist is essentially moot, since statistically significant associations between exposure to air pollutants and health outcomes (morbidity and mortality) have been observed at concentrations of PM (and some other pollutants) in the range of current concentrations in this region. See Vedal et al. (2003) and the following section.

2.5 LOCAL STUDIES AND APPLICABILITY OF STUDIES IN OTHER REGIONS TO LOCAL CONDITIONS

Given that the major emission sources present in large urban areas of BC and the PNW (especially the Lower Mainland and southern Vancouver Island in BC and the Puget Sound area in Washington) are similar to those present in other urban areas and that the larger studies (NMMAPS[17], ACS[18], APHEA[19]) have included a diverse range of locations with different air pollution mixtures, it is reasonable to conclude that the results of these studies can be applied to this region. The similarity of the ranges of pollutant concentrations (exposures) covered in the studies considered to those experienced in BC and the PNW, and the fact that concentration-response factors for health effects are uniformly linear with ambient concentration where they have been measured, together suggest that study results from elsewhere ought to be generally applicable to this region. Exceptions for dissimilarities in pollutant mix and other factors are taken into account where appropriate.

The impacts of BC Lower Mainland air pollution on health have also been demonstrated in direct studies. The fact that adverse health impacts have been observed even though the Lower Mainland air pollutant levels are on the low end of those observed even in western cities of comparable population supports the applicability of other studies to this region (see the NMMAPS PM air quality distribution and Brauer et al., 2000, which compares the Lower Mainland to western US cities of comparable size for all criteria pollutants). With respect to smaller locations, the specific air pollution mixture needs to be examined in more detail. Vedal et al. (2003) have found associations between 'air pollution' and total, respiratory and cardio-vascular mortality in Vancouver at statistically significant levels, but without resolution of the specific pollutant or pollutants in the mix that may be responsible. That the associations were observable at the low levels of PM_{10} and ozone present in Vancouver during their study period further underscores the apparent absence of a response threshold, but also points out the difficulty of finding definitive results for the relatively clean air of the Lower Mainland.

While the Lower Mainland reports pollutant concentrations that are generally lower than those measured in other locations where health impacts have been measured, the same cannot be said for many interior communities (see table from BC *State of Knowledge Report* excerpt in Appendix 6). Vancouver, Victoria and Seattle have low pollution levels relative to other cities of comparable size, but elsewhere in BC and the PNW, resource-

[17] *National Morbidity, Mortality and Air Pollution Study* – carried out for the Health Effects Institute by Johns Hopkins University for 90 US urban areas. See reference listing for NMMAPS, 2000.
[18] American Cancer Society
[19] *Air Pollution and Health: European Approach*, sponsored by the European Commission.

based communities can experience much poorer air quality, especially with respect to PM and sulphur compounds.

Although high PM concentrations may largely be seasonal and associated with wood or agricultural burning in BC and the PNW, concentrations are at the same levels as those observed in other wood burning communities where health impacts have been observed, so again studies conducted elsewhere should be applicable. The Port Alberni study is also important for communities where pulp/paper mills are major PM sources – this study is rather unique in evaluating the impacts of PM in a pulp mill community (see below).

Bates' early study (Bates, et al., 1990) of the relationship between air quality and hospital emergency room visits in Vancouver was of visits to all nine hospital emergency departments in the Lower Mainland over a period of two and a half years (see below) at a time when pollution levels were considerably higher than they are now.

In addition, the traffic studies mentioned above may also be applicable to high traffic areas in any community, although this would depend on measurements of vehicle counts and specific exposure monitoring.

In general, studies elsewhere have been confirmed by the results of studies carried out in BC and the PNW. Examples of these 'local' studies follow.

SPECIFIC FINDINGS FROM STUDIES IN BC AND THE PACIFIC NW

The following research summaries present detailed findings for the BC and PNW regions. The findings are important indicators of local relationships between air pollution and health effects, but they are also valuable as evidence of the applicability to this region of findings from the larger literature. Most of the studies summarised have been discussed earlier in various sections of Chapter 2 under discussions of specific pollutants or outcomes.

British Columbia

There are several studies in Greater Vancouver to draw upon to evaluate exposure *vs* ambient monitoring data. For ozone, it has been shown that exposure is proportional to the amount of time spent outdoors (Brauer and Brook 1995). For ozone it is also important to consider that BC and the PNW have very little air conditioning, so exposures may be higher (relative to ambient monitors) than those in locations with more air conditioning. For PM, exposure is dominated by indoor sources, although locally Brauer and the University of BC group have assessed exposure to PM of ambient origin (using sulphate as an indicator) and shown that personal exposures to PM of ambient origin are highly correlated to ambient levels. These studies have been done in Vancouver as well as in Prince George (Ebelt, Petkau et al. 2000; Noullett, Jackson et al. 2002; Rich, Brauer et al. 2002), and they have shown that exposures to PM of ambient origin are roughly 80% of the measured ambient levels – similar to results for other cities for residents without air conditioning.

Vedal et al. (1998) studied PM and asthma associations in Port Alberni, BC and found acute PM_{10} effects in this community, which has a pulp mill. Increases in PM_{10} associated

with reductions in peak expiratory flow and increased reporting of cough, phlegm production, and sore throat were found in the study population of asthmatic and nonasthmatic children.

- For the subgroup of children with diagnosed asthma, peak expiratory flow in the time period with the highest PM_{10} concentrations fell by an estimated 0.55 L/min (95% CI, 0.06 to 1.05) for a 10 $\mu g/m^3$ PM_{10} increase above the mean daily PM_{10} concentration of 27.3 $\mu g/m^3$, and the odds of reported cough increased by 8% (95% CI, 0 to 16%).
- Children experience reductions in peak lung flow and increased symptoms after increases in relatively low ambient PM_{10} concentrations, and children with diagnosed asthma are more susceptible to these effects than are other children.

Bates et al. (1990) analysed all visits to the emergency departments of nine hospitals serving just less than one million people in Greater Vancouver, logged from 1984 to 1986. No PM_{10} or $PM_{2.5}$ data were available. Their principal findings were:

- There was a peak in asthma visits affecting children and the 15-60 age group but not those over 60, which occurred in the third week of September each year[20], causing at least a doubling of weekly visits for a three-week period. This was not related to temperature changes or to peaks in air pollutants. No cause could be identified.
- There were significant associations between SO_2 levels (probably indicative of inversion conditions) and visits for asthma; and a significant association between NO_2 levels and visits by the elderly.
- Respiratory visits were unrelated to temperature changes, but varied seasonally.

Associations have been found between mortality and gaseous pollutants in studies in the Lower Fraser Valley (Vedal et al., 2003). Analysis of three years of data (1994-1996) showed that the dominant associations were between ozone and total, respiratory and cardiovascular mortality in summer and between NO_2 and total mortality in winter. Some evidence was shown for a PM_{10} effect on respiratory mortality, in summer. The paper raises the possibility that the general absence of associations between PM_{10} and mortality may be due to the very low concentrations of PM_{10} in the Vancouver study region (mean of 14.4 $\mu g/m^3$) – which was lower than mean PM_{10} concentrations in any of the 88 NMMAPS locations – and/or that the observed effects of gaseous pollutants may be due to their acting as surrogates for general features of the air pollution mixture. For example, the association between ambient ozone and cardiac mortality lacks a plausible biological mechanism. Insufficient $PM_{2.5}$ monitoring data were available for the study period. The study was updated to reflect general additive model statistical issues. See also Vedal, Brauer et al. (1999).

A Vancouver panel study of COPD patients evaluated lung function, heart rate, heart rate variability and cardiac arrhythmia (Brauer, Ebelt et al., 2001). 16 subjects were followed

[20] This is an annual nation-wide phenomenon in Canada that is not understood. It does not appear to be related to allergens, since it occurs in the same week across the country, nor does it appear to be related to return to school, since it affects children and adults at the same time.

for 7 24-hour periods in summer 1998. Subjects underwent lung function measurements and 24-hour monitoring for each measurement session. An estimated effect of 1% decrease in daily FEV1 per 10 $\mu g/m^3$ change in $PM_{2.5}$ was observed (not statistically significant). Weak associations were observed between measured $PM_{2.5}$ and cardiac arrhythmia. No consistent associations were observed with heart rate or heart rate variability, symptom severity or bronchodilator use, although of the pollutants measured (PM_{10}, $PM_{2.5}$, sulphate, personal exposures), ambient PM_{10} was most consistently and strongly associated with health outcomes. Additional (as yet unpublished) research by Brauer's group indicates these effects may be due to coarse particles (estimates by PM_{10}-$PM_{2.5}$); removal of data points associated with a transported Asian dust episode further strengthened associations between PM_{10}/coarse PM and health outcomes, especially lung function, heart rate and heart rate variability. No associations were observed with ambient sulphate.

In Vancouver, a panel study of cardiac patients (with implanted defibrillators) was conducted to investigate cardiac arrhythmia. Two analyses of this group of patients were carried out. The first analysis included only routine monitoring network data and showed essentially no associations between arrhythmias and any air pollutants (Vedal et al., 2002). The second analysis (using case-crossover methodology) on a subset of the study period in which additional monitoring data were available showed some evidence for associations – especially in summer and no lag with PM_{10} (but not $PM_{2.5}$ or sulphate), elemental carbon, organic carbon, CO, NO_2, O_3, SO_2, as well as O_3 in winter at longer lag times after exposure (Rich, 2002).

In the Fraser Valley two studies of ozone and lung function (acute and chronic impacts) were done in two consecutive summer seasons. Agricultural workers with high ozone exposures (due to 11—14 hours per day of outdoor work) had lung function measured daily for approximately a 2-month summer period. In both years, acute changes in lung function were observed – the impact on lung function was persistent to the following day and also remained even for days in which the maximum hourly ozone did not exceed 40 ppb. On both years, seasonal declines in lung function were also observed over the course of the study period. Lung function levels returned to initial values between the two study periods. These findings suggest a reversible seasonal effect of ozone on lung function (Brauer, Blair et al., 1996; Brauer and Vedal, 1999).

Brauer et al. (2000) analysed mortality data for the Lower Fraser Valley in relation to air pollution. Table 2 summarises their estimates of air pollution-related mortality in Vancouver. There is a broad range of uncertainty indicated.

Table 2. Estimated air pollution-attributable deaths in the Lower Mainland (BC) (1994-1998) (Source: Brauer et al., 2000)

Cause	Number (attributable deaths/year)
Air pollution*	***0 (low)/600 (mean worst case)***

* The range of air pollution-attributable premature deaths arises from assumptions about threshold of response. The Lower Mainland Medical Health Officers, for whom the report was prepared, interpreted the range of annual air quality-related mortality to lie most likely in the range 15-150. See their interpretation at http://www.southfraserhealth.com/Health_Info-Air_Quality.asp.

The "mean worst case" value in Table 2 is for the assumption that there is essentially no threshold of response to air pollution, that is, at a "low pollution" level cutoff at the 10th percentile of the actual air quality data. The number of annual deaths attributable to air pollution at the "mean worst case" level is 4.6% of total non-trauma deaths in the Lower Mainland region. Care must be exercised in comparing premature deaths attributable to air pollution with other causes of death, since they are specifically assignable causes with unambiguous outcomes (e.g., motor vehicle accidents) and are not necessarily statistical risks faced by the normal population (e.g., drug overdoses or suicides). The low end of the range of estimates – zero – in Table 2 is based on the assumption that no effects occur below the concentration of the reference air quality guideline chosen for each of the pollutants studied (SO_2, NO_2, CO, O_3, PM_{10} and $PM_{2.5}$). Details of the analytical model used for the time-series analysis may be found in the study report (Brauer et al., 2000). It is based on the work of Vedal, Brauer et al. (1999).

Pacific Northwest US

This section gathers together for convenience summaries of work presented in earlier sections of the report.

Studies in Seattle have focused largely on PM exposure outcomes,[21] with particular reference to wood smoke effects due to its prevalence in the region – one study indicated that on an annual basis 60% of the fine particle mass in Seattle residential neighbourhoods is from wood burning (Schwartz and Slater, et al., 1993). A questionnaire study of respiratory symptoms compared residents of high (mean $PM_{2.5}$ of 55 $\mu g/m^3$) and low (33 $\mu g/m^3$) wood smoke pollution areas of Seattle. Although no significant differences were observed between the high and low exposure areas when all age groups were considered, there were statistically higher levels of congestion and wheezing in 1-5 year olds from the high pollution area. This finding supports those of other studies which suggest that young children are particularly susceptible to adverse effects of wood smoke (Browning, Koenig et al., 1990).

A more comprehensive study of 326 children in the same high exposure Seattle area (where 80% of the particulates were from wood smoke) (Koenig et al., 1993) measured significant lung function decrements in the 26 asthmatic subjects, in association with increased wood smoke exposure. The highest $PM_{2.5}$ level measured during the study period (night time 12-hour average) was approximately 195 $\mu g/m^3$ (Koenig, Larson, et al., 1993).

A companion study found a significant association between PM_{10} levels and asthma emergency room visits throughout Seattle (Schwartz, Slater, et al., 1993). The mean PM_{10} level during the 1-year study was 30 $\mu g/m^3$. At this concentration, PM_{10} appeared to be responsible for 12% of the asthma emergency room visits.

The effects of PM_{10} for other health outcomes have also been examined in this region to some extent. However, a recent analysis of over 5,000 admissions to hospital for

[21] Washington has no non-attainment areas for the US National Ambient Air Quality Standard for ozone and the PM non-attainment areas are in eastern Washington. Oregon has an ozone non-attainment area in the Salem area and a number of PM non-attainment areas.

myocardial infarction in Seattle did not find an association with PM_{10} (J. Koenig, personal communication, 2002).

For the broader region, the NMMAPS time-series results for the State of Washington cities in the analysis (Seattle, Tacoma, Spokane, and Olympia), as adjusted to address the general additive model statistical problem, indicate a CRF of 0.2 % change in total mortality per 10 $\mu g/m^3$ change in PM_{10} (with a standard error of 0.1). This range of values is taken by the Panel to represent the lower bound of acute (short-term) responses to exposure to inhalable PM.

Wood smoke has also been studied for lung function of 410 children in high and low exposure areas of Oregon where wood smoke accounts for as much as 80% of winter period particulate (Heumann, Foster et al., 1991). PM_{10} ranged from approximately 50-250 $\mu g/m^3$ in the high exposure area and 20-75 $\mu g/m^3$ in the low exposure area. Lung function decreased during the wood burning season for the children in the high exposure area, but not in the low exposure area.

The differential effects of windblown and combustion PM were investigated in Spokane. An atmospheric stagnation index was used as a surrogate for combustion PM concentration. The analysis found an association between asthma emergency room visits and stagnant air, with a relative risk of 1.12 (95% CI 1.05-1.19) for an increase of 11 hours of low wind speed (Norris et al., 2000). Schwartz and co-investigators later found no association between mortality and 19 episodes of wind-blown dust during the time period.

In summary, the effects on both mortality and morbidity that have been seen elsewhere have been found in a number of local studies in BC (focused on the Lower Fraser Valley) and the PNW – at similar intensities. These findings support the consistency and coherence tests for concluding that the important findings from other geographic areas are applicable to BC and the PNW. Furthermore, premature deaths attributable to air pollution in the Lower Mainland may be comparable in number to some common causes of death, with broad uncertainty. There is reason to believe that this situation may also be true for communities in BC whose pollution exposure is dominated by combustion sources (as opposed to windblown soil, for example), although detailed analysis has not been done. Comparison with other causes of death may be different in other communities because of different demographics and cause of death statistics – or significantly different pollutant mixes.

2.6 SUMMARY OF THE KNOWLEDGE BASE

Table 3 provides a summary of the conclusive information presented in the chapter about relationships between air pollutants and health outcomes. The division of effects relationships into 'definite,' 'probable' and 'possible' is based on professional judgement (Bates and Vedal, 2002) that was confirmed and extended by the Panel for this work. The table does not attempt to summarize the quantitative parameters of the relationships, just the qualitative assignment to the three categories of relative certainty of association.

Effects in the 'definite' column have been so categorized when several reliable studies substantiate one another. They will have shown statistically significant associations but may not have produced numerical data that can be translated into an ambient concentration-response function.

'Probable' effects are those that have been demonstrated in one or more reliable studies but whose range of application or numerical results are not yet considered definitive.

'Possible' effects are those that have been found in one or more studies, but whose quantitative association with a specific air pollutant is not yet convincing, has not been replicated in other studies or whose significance for public health has not yet been determined.

It will be noted that exposure to diesel particles is stated to be associated with an increased risk of lung cancer in the "probable" column. Such a designation requires interpretation. There is convincing evidence that residence in major urban areas over the past 15 years is associated with an increased risk of lung cancer. Exposure to diesel particulate emissions is only one of many exposures that might be responsible. However, the probability that diesel particles may be responsible is supported by animal evidence that these particles may induce cancer; by the fact that public exposure to diesel particles may have increased in the past few years; and by the fact that such particles constitute a major fraction of directly emitted particles from vehicle exhaust. None of this evidence constitutes "proof", and all such decisions are judgements based on the total weight of evidence available from many sources. In this case, an increased risk of lung cancer in men exposed to high levels of diesel exhaust (including underground exposures) should also be taken into account.

Each entry in this table represents a summary judgement from a lot of different evidence, and therefore constitutes a best contemporary educated guess as to where any particular pollutant should be placed.

Table 3. Summary of effects of individual air pollutants and mixtures at current ambient levels of exposure*

POLLUTANT	DEFINITE EFFECTS	PROBABLE EFFECTS	POSSIBLE EFFECTS
Fine Particles (PM_{10}, $PM_{2.5}$)	Time-series and cohort association with daily respiratory and cardiac mortality Aggravation of asthma Increased hospital admissions for respiratory and cardiac conditions Depressed lung function in schoolchildren (acute & chronic) Increased prevalence of bronchitis Increased risk of lung cancer Increased school absences Increase in banded neutrophils	Aggravation of acute respiratory infections Increased risk of wheezy bronchitis in infants 4-12 months Decreased rate of lung growth in children Increased exhaled NO Tachycardia in the elderly Reduced heart rate variability Increased c-reactive protein Increased blood vessel constriction	Decreased birth weight Increased blood fibrinogen Increased asthma prevalence
Diesel Emissions (in addition to particle effects)	Increased response to allergens Increased airway inflammation	Increased risk of lung cancer	
Wood smoke (in addition to particle effects)	Aggravation of asthma Increased hospital respiratory admissions Increased respiratory infections		Increased mortality
Ozone	Increased hospital admissions for acute respiratory diseases Aggravation of asthma Increased bronchial responsiveness Increased response to SO_2 Increased reduced activity days Increased school absences for respiratory illness Reduced lung function	Effect on mortality Increased sensitivity to allergens	Aggravation of acute respiratory infections Chronic bronchiolitis with repetitive exposure Increased prevalence of asthma
Aerosol sulphates & nitrates	Reduced visibility Decreased mucociliary clearance (in rabbits) (H_2SO_4)	May be partly responsible for effects of $PM_{2.5}$ Decreased lung function in adolescents with asthma	May increase all effects of concomitant ozone

POLLUTANT	DEFINITE EFFECTS	PROBABLE EFFECTS	POSSIBLE EFFECTS
Acid aerosols (combined gases & particles)	Aggravation of asthma	Increased prevalence of bronchitis	May increase all effects of concomitant ozone
Sulphur dioxide	Acute bronchoconstriction in asthmatics Increased chronic bronchitis	Increased prevalence of lung cancer Increased nasal congestion (work of breathing)	Interaction with particles in relation to mortality and morbidity effects Increased prevalence of chronic bronchitis
Nitrogen dioxide	Increased respiratory morbidity & infections Aggravation of asthma in children Lowered FVC and FEV1 Increased response to ozone	Increased bronchial hyperresponsiveness to inhaled methacholine Chronic respiratory bronchiolitis	Interaction with particles in relation to mortality and morbidity effects
Carbon monoxide	Increased cardiac ischemia	Increased hospital cardiac admissions Decreased birth weight	Increased cardiac mortality Increased birth defects Interaction with particles in relation to mortality and morbidity effects
Hydrogen sulphide	Central nervous system and respiratory symptoms Eye irritation Mortality at very high concentrations	Chronic sinusitis	

* Adapted from Table 4.3, *A Citizen's Guide to Air Pollution*, second edition, The David Suzuki Foundation, 2002. D.V. Bates & R.B. Caton, eds.)

3. Interpretation of the Knowledge Base

3.1 Strengths and weaknesses: comparison of different methods for estimating air pollution effects

3.1.1 Are there preferred estimation methods?

Methods of study have to be tailored to the question being addressed – but always with the proviso that much important air pollution research has been essentially opportunistic in nature. Researchers have had to deal with available data and situations, rather than being able to plan well-controlled experiments. Clinical studies have the advantage of the latter type of control. Three examples of opportunistic work are the Provo, Utah study of hospital admissions of children for respiratory disease when the local steel mill (which was responsible for about 60% of the PM_{10}) was closed for a year; the analysis of blood viscosity during an air pollution episode in Augsburg (Peters); and the study by Tan and her colleagues of army cadets in Singapore during and after the Indonesia smoke episode in 1998. Another example is the work of Clancy et al. (2002) in Dublin where marked changes in air quality with respect to certain pollutants over a relatively short period of time due to a fuel quality intervention enabled associations to be found that would otherwise have been masked. During the Atlanta Olympic Games, there was a marked reduction in peak ozone levels due to the imposed restrictions on traffic which reduced ozone precursor emissions. The reductions in peak ozone compared with the same time period in other years was paralleled by a proportionate reduction in hospital emergency visits for asthma.

In acute studies, data from hospital emergency visits, hospital admissions, or family practice visits, can all provide useful information. Panel studies of carefully recruited children or adults also have provided very important information – examples, are from Port Alberni, BC, Paris, France, Alpine, California, and several more of these are in process such as the ambitious project in Fresno, California.

Long-term or chronic effects are more difficult to evaluate. The two largest are the American Cancer Society study of more than 500,000 adults; and the Harvard Six-Cities study in which the number of subjects was much smaller, but the quality of the air pollution exposure data was very good, as was the individual knowledge of such variables as socio-economic status and employment history. The detailed characterisation of exposure and other potentially confounding variables in the Six-Cities Study increases the level of confidence in the results, in the Panel's opinion.

Recent experience has shown that comparisons of communities in cross-sectional studies are relatively insensitive indicators of the impact of air pollutants (e.g., the Southern California Children's Health Study). Uncertainty in such studies arises from variability of exposure within a community, socio-economic differences, etc. which interfere with the analysis in (often) uncontrollable ways. Time-series data are much more sensitive, and not affected by some of the important confounders that affect cross-sectional comparisons. Time-series data are influenced by seasonal and climatic factors, but these can be corrected for by various statistical techniques. Such corrections are not straight forward, and there is ongoing debate about the adequacy of controlling the time-varying factors in time series studies. Long-term cohort studies are thought to address potentially cumulative factors in air pollution disease and mortality that would not show up in the

acute effects analysed in time series studies. Both cohort and time-series studies have their strengths and weaknesses.

Panel studies are a preferred method of studying one disease entity, such as asthma, as they offer the advantages of detailed exposure data (often using personal monitors) and daily physiological measurements. Panel studies are generally useful for looking at relatively frequent outcomes in a defined subgroup. An outcome such as an influence of air pollution exposure on lung function development can only be studied by following children through their growth period.

The EPA Research Center for Particulate Air Pollution and Health in the Department of Environmental Health at the University of Washington, Seattle has analyzed models for extracting information about the rate of disease outcomes in a population using a relative risk disease model and personal versus ambient exposure data sets. Lianne Sheppard of that group presented several papers on the subject at the joint conference of the International Society of Exposure Analysis (ISEA) and International Society for Environmental Epidemiology (ISEE) held at the University of British Columbia in August 2002.[22] The remainder of this section is based in large part on her remarks on time-series and panel study designs from the conference presentations, which she kindly provided to the Panel, and subsequent discussions with her.

Practical limitations are important – Panel studies are limited by feasibility and cost. This influences the power of epidemiological studies (ability to extract statistically significant results when the null hypothesis of no effect of pollution is false – i.e., there is a demonstrable effect of air pollution). Time series studies are limited by using ambient concentrations as surrogates for personal exposure and routinely collected health data for outcomes.

Based on estimates of relative variability from measured personal and ambient exposures in Seattle, it was concluded that only about 5% (a range of about 3 to 16%) of the variance in personal exposure comes from ambient exposure. Because there is less measured exposure variation, an ecologic study is considerably less efficient than a personal exposure panel study – the relative efficiency being about 0.05, reflecting the range of variance noted above (for an ecologic study with ambient monitoring compared with a panel study with personal exposure monitoring – assuming that the studies have the same number of people and observation days, which is not feasible for a panel study). The relative power of the two types of study is approximately proportional to the number of subjects, so a reasonable measure of the relative power of an ecologic study is approximately 0.05N, where N is the number of subjects. The larger number of subjects in a time series study can overcome the advantage of specificity of single person-days of observations and monitoring in a panel study.

Some characteristics of panel studies are as follows.

- They have limited statistical power because of limited sample size (due to the cost of maintaining a large group of people as subjects), which is exaggerated by rare outcomes in the group.

[22] *Exposures in time-series studies: Impact on health effect estimates* (Paper 12.02) and *Air Pollution Study Designs: Linking Exposures with Outcomes* (Paper 21.03).

- They are very good for studying personal exposure effects – specificity of exposure and outcome diagnosis. The panel can be selected for prior diagnosis of an existing or potential disease state, and exposure and outcome are linked at the personal level.

Time-series studies, relatively speaking, have the following characteristics.

- They are simple and inexpensive. They use routinely collected monitoring and outcome data and can cover large geographic areas, thus capturing a very large number of subjects.
- Power is a major advantage, since the size of datasets (number of people at risk for a rare event and number of days included) can be very large.
- Because of their large population under study, they lend themselves to estimating acute health effects of ambient pollutants for rare events (that might be lost in a small panel study, for example).
- Varying ambient exposures are able to provide significant signal in the data, which suggests that lack of total personal exposure information need not be a concern (assuming that day-to-day variations in ambient and non-ambient sources are independent).
- Bias in the results due to spatial variation in exposure is probably small for PM (especially $PM_{2.5}$ or smaller) due to the relatively small amount of spatial variation and lack of evidence of spatial-temporal interaction.

Long-term, large-scale cohort studies generally use annual average pollutant concentrations in varying geographic settings as the exposure metric contrasted to the day-to-day concentration metric of time-series studies. There is an issue, then as to whether the longer-term average concentrations and daily (or hourly) ambient concentrations represent effective exposure adequately. Analysis of cohort study design and limitations is incomplete, but factors being evaluated by the University of Washington group include: determining what is lost by replacing cumulative personal exposure with community exposure and understanding cumulative exposure over the study time period (representativeness of fixed sites over time and inter-site variation relative to local contributions and personal contributions).[23]

In summary, time series design is inherently powerful for analyzing population disease acute outcome variation in response to ambient concentrations. The loss of efficiency by using just ambient concentration measurements is more than offset by gains in power from observing events from entire populations (this can be demonstrated analytically). Our best understanding is that long-term cohort studies estimate acute effects plus chronic effects combined and that time series studies estimate only acute effects.

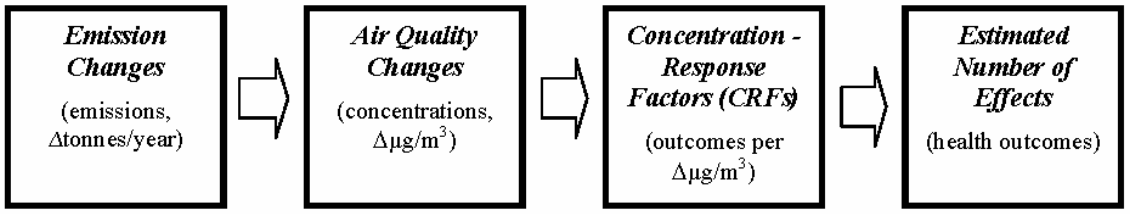
[23] A reviewer commented that it is unlikely that exposure error accounts for differences in effects estimates between cohort and time series studies.

3.2 QUANTITATIVE CONCENTRATION (EXPOSURE) – EFFECTS (RESPONSE) RELATIONSHIPS APPLICABLE TO BC AND PNW COMMUNITIES.

3.2.1 The effects estimation model

In applying the information provided in this report, one of the principal interests of the users will be estimating health outcomes in response to air quality changes in their communities. The following graphic illustrates this stepwise estimation process:

Air Quality Effects Estimation Schematic



The following sections address the Panel's recommendations for preferred CRFs to be used in this context and a brief discussion of the associated uncertainty.

The form in which the results of air pollution effects studies are expressed as CRFs is related to the form of the commonly-used disease model that is generally expressed as a Poisson statistical distribution. The relative risk of a disease outcome can be expressed in simplest form as follows (with ambient concentration, C, as the measure of exposure):

$$RR = \exp[CRF*(C-X)_+]$$

where the relative risk, RR,[24] for a disease outcome is an exponential function of the exposure characterised by ambient concentration, C. X represents a threshold concentration (see WHO, 2002). The subscript "+" indicates that the argument in parentheses is either positive (C>X) or zero (C≤X). If no threshold is discernible, X in the equation is effectively zero.[25] The logarithm of this function, then, is:

$$\ln RR = CRF*C$$

For small changes in RR, this equation reduces to

$$\Delta RR/RR = CRF*\Delta C.$$

The latter equation expresses a linear relationship between the fractional change in the disease outcome (for example, expressed as a percentage change in mortality or a

[24] Some of the data used here are more accurately characterised in terms of 'relative rate' rather than 'relative risk.' Time series studies, for example, determine relative rates of acute, daily responses to daily changes in pollutant levels and do not represent relative risk in a population in the same way that CRFs from cohort studies do. This means that, strictly speaking, CRFs determined from acute response studies (e.g., time series analysis) should not be used to estimate other than day-to-day changes in outcomes (i.e., not used to estimate annual numbers of outcomes). The applications based on the 'RR' equation given here that are suggested later in this report need to be accompanied by appropriate caveats in this regard.

[25] If X is non-zero, i.e., a threshold exists, but at concentrations less than current ambient levels, the issue of whether a threshold actually exists is moot – hence the statement that it is 'effectively' zero. If X is left in the equation, the approach to the analysis suggested here is not affected materially.

morbidity outcome) relative to its base rate of occurrence and a change in ambient concentration. This assumes that ambient concentration is an adequate representation of exposure, and that no effective threshold has been shown to exist, as indicated elsewhere in this report. The CRF is the slope of a graph such as Figure 2 for the relationship between $PM_{2.5}$ concentration and total mortality in the Six-Cities Study.

The above equation, then, can be expressed in terms of changes in disease effects (E) relative to the base rate (BR), as follows:

$$\Delta RR/RR \sim \Delta E/BR = CRF*\Delta C$$

BR must be adjusted as necessary to represent non-traumatic deaths (non-accidental deaths) and to account for any other factors that might not be attributable to air pollution or to avoid double-counting of overlapping morbidity outcomes.[26] A BR is specific to each outcome – that is, the BR for total mortality is different from that for cardio-vascular mortality, lung cancer mortality, lung cancer prevalence or hospital admissions, for example. For any given change in ambient concentration of a pollutant (e.g., $PM_{2.5}$), the estimated number of health effects (outcomes), then, is the product of the appropriate concentration-response factor (CRF), the change in concentration (ΔC), the per capita base disease occurrence rate (BR/POP) and the exposed population (POP). That is, the estimated change in the number of disease outcomes for a given change in concentration (exposure) is given by Equation (1):

Equation (1) $$\Delta E = CRF \times \Delta C \times (BR/POP) \times POP$$

The parameters must all be expressed in compatible units. CRF is usually expressed as a fractional change in disease occurrence rate for a unit change in concentration. If BR is given as an annual rate per person (for example, the number of deaths per year per thousand of population), Equation (1) gives the annual number of health outcomes. If the BR is expressed as a daily rate, the result must be multiplied by 365 to give annual outcomes.

The AQVM CRFs in Appendix 7 are per capita risk factors expressed either in daily or annual terms depending on the health outcome, as noted in the tables, and already incorporate the applicable BR values. Other tables of CRFs may express the values in terms of risk per million people. POP may be the whole population or a subset, such as, people over 65, asthmatics, or children under a certain age. Some CRFs refer only to specific subsets of the population, as determined by the nature of the statistical analysis for each study.

Each health outcome has its own CRF to express the relative risk of that effect (premature mortality, chronic bronchitis, emergency room visits, etc.). The base mortality rate (BR) for the Lower Mainland of BC is currently about 6.2 per thousand people per year (6.2×10^{-3}/y). The analogous base rates for other outcomes are available from hospital data and other health statistics. Baseline vital statistics data are available for all health regions in Canada from Statistics Canada. They show changes from year to year as regional demographics change and controlling factors vary (e.g., disease epidemics, heat

[26] For example, in AQVM, the rate for emergency room visits is adjusted by subtracting the sum of respiratory hospital admissions and cardiac hospital admissions from total emergency room visits, since the hospital admissions would have been preceded by an emergency room visit.

waves, etc.). Such data are sometimes normalised to outcomes in a standard population age distribution for a reference year in order to adjust data for consistent trend analysis (standardised, age-adjusted rates).

The above expression of $\Delta RR/RR$ or $\Delta E/BR$ for a change in relative risk or disease incidence is similar to the WHO use of the terminology "potential impact fraction," which WHO defines as "the proportional reduction in the total number of new cases of a certain disease, resulting from a change in the distribution of the risk factor in the population at risk" (WHO, 2001). In the case of air pollutants, the proportion of the population in an exposure category before and after a public health 'intervention' (which in other common public health contexts might be an immunisation program or control of a disease-bearing pest) corresponds to a change in ambient (or total) concentration as a result of changes in emissions or meteorological conditions or changes in personal behaviour. Changes in the demographics of the exposed population may also contribute to the "potential impact fraction." WHO uses the terminology 'estimates of attributable and avoidable burden' of disease to describe the result of applying Equation (1). See Stieb et al. (2002b), Cifuentes et al. (2001), Schwartz et al. (2002), WHO (2001) and WHO (2002) for more exact equations relating these parameters and additional background discussion. General discussions of the issues related to estimating outcomes from concentration-response or dose-response factors may be found in Chapters 4 and 5 in Bates and Caton (2002) and Lipfert (1994).

A caveat with respect to Equation (1) is that it will be more reliable in estimating relative risk differences between two exposure regimes than in estimating absolute risk of a given exposure level. Equation (1) may be used with caution to estimate absolute risk of current exposure levels with the assumption of the absence of a threshold, so that risk is directly proportional to exposure over the full range of ambient concentrations. Also as pointed out earlier, the use of Equation (1) to estimate absolute numbers of outcomes – body counts, if you will – should be done with caution and with appropriate accounting for uncertainty. That said, most of the applications of Equation (1) outlined later in this report – and commonly elsewhere – use Equation (1) to estimate numbers of outcomes for a given state of air quality or for changes in short-term or long-term air quality. The applications in Chapter 4 are qualified with cautionary language, but the utility of Equation (1) is in providing the most reasonable estimates of air pollution-related health outcomes based on current research. Interpretation of estimates of absolute numbers of outcomes needs to be careful, with appropriate accounting for the generally broad uncertainties in the CRF database. If the latter are always taken into account, serious misinterpretations should not occur.

One could use Equation (1) to calculate estimated relative risks for different levels of exposure (C). For example, the relative risk per person of premature PM-related mortality (or a morbidity effect) between two communities with annual average concentrations C_1 and C_2 would be approximately the ratio of C_1/C_2, again assuming no threshold. That is, a person living in a community with annual average PM_{10} concentration of 30 micrograms/m^3 has a risk of premature death (or any other PM-related outcome) in a given year that is twice that of a resident in a community with annual average PM_{10} concentration of 15 micrograms/m^3 (in the absence of a threshold). This assumes that the population demographics and pollution mix are similar between the two communities, but

it allows a rough comparison and is a useful illustration for public information. If there were a threshold of PM mortality response at, say, 5 μg/m^3, the relative risk would be approximately in the ratio (30 – 5)/(15 – 5) = 2.5 (i.e., not materially different within the known uncertainties and for the purposes of using this type of estimate).

This simple analysis could be extended by using the relative sizes of the affected population (POP) in Equation (1), for example, the relative number of people over 65 in each community. The base disease occurrence rates (BR) may also differ among communities. The CRF factors in Equation (1) may also differ from community to community, if local data are available. The concentrations might also in future be adjusted for specific composition (e.g., combustion source contributions resolved from windblown soil), if more composition-specific health effects information and CRFs become available. Thus, Equation (1) provides a simple algorithm for estimating air pollution-related health outcomes, or it can be applied in more complex form to account for differences in composition or distribution of any of the factors.

Cifuentes et al. (2001) provide a CRF database developed to estimate health co-benefits of climate change mitigation measures for a number of health effect outcomes (mortality and morbidity) for PM_{10} and ozone. The only North American region covered in their analysis is New York City, but the database clearly illustrates the relative hierarchy of the magnitudes of CRFs for mortality and morbidity outcomes. The paper also illustrates the algorithms used in the estimation method. To place the CRF database in context, the following table shows the CRF database from Cifuentes et al. (2001) to illustrate the relative values of the mortality and morbidity CRFs. The 'health impact factor' in the table is equivalent to the CRF terminology used in this report.

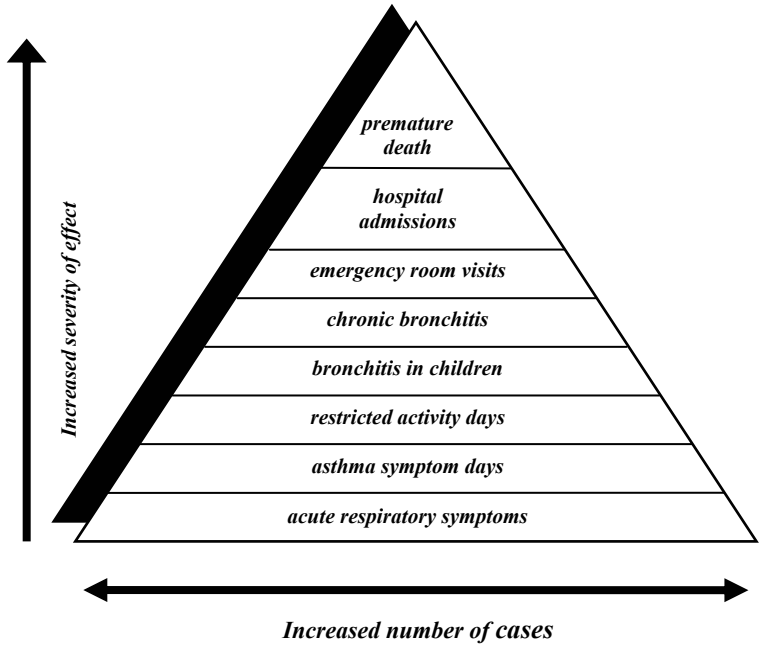
Table 4: Ozone and PM_{10} health impact factors for New York City (Cifuentes et al., 2001)

Health effect outcome	Health impact factor per million inhabitants	
	Central value	95% CI
Ozone impacts (effects per part per billion of annual average daily 1-hour maximum ozone)		
Acute mortality	1.2	(0.0-2.4)
Acute respiratory hospital admissions	5	(3-7)
Acute emergency department visits	40	(25-55)
Acute asthma attacks	1,005	(570-2,747)
Acute restricted activity days	17,000	(7,000-27,000)
Acute respiratory symptom days	50,000	(26,000-78,000)
PM_{10} impacts (effects per microgram per cubic meter of PM_{10})		
Acute & chronic infant mortality	0.21	(0.1-0.3)
Acute & chronic adult mortality	33	(8-52)
Acute respiratory hospital admissions	12	(7-17)
Chronic adult bronchitis	39	(19-59)
Acute bronchitis in children	53	(26-78)
Acute emergency department visits	94	(55-130)

Health effect outcome	Health impact factor per million inhabitants	
	Central value	95% CI
Acute asthma attacks	774	(446-2,589)
Acute work loss days	5,300	(2,700-8,300)
Acute restricted activity days	14,900	(7,616-23,509)
Acute respiratory symptom days	170,000	(81,000-259,000)

The following figure (Figure 3) illustrates graphically the hierarchy of mortality and morbidity outcomes in terms of the outcome severity (vertical dimension) and expected number of cases of each type of outcome (horizontal dimension). This hierarchy reflects the relative values of the impact factors in the Table 4.

Figure 3. Hierarchy of air pollution health effects



A number of the effects designated in summary Table 3 as "definite" or "probable" are not addressed in any of the CRF databases cited here. The underlying studies would need to be reviewed and analysed to extract reliable CRFs to use with Equation (1). It was beyond the scope of the Panel's mandate to carry out such detailed new analysis. The significance of some of the omitted effects for public health protection is not clear. Filling in some of these information gaps is one of the recommended actions following from the Panel's considerations.

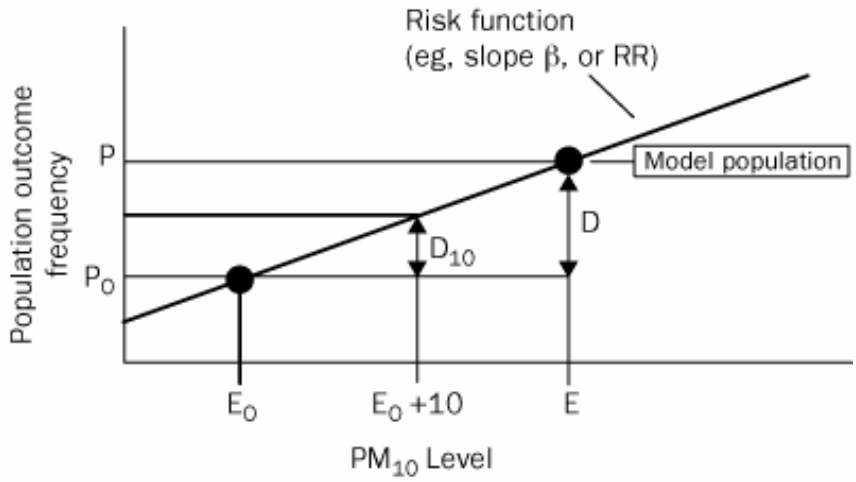
An example of the application of the model

A European study (France, Austria and Switzerland) provides an example of how the quantitative information in this section can be applied to estimate the impact on mortality of reducing air pollution (Kunzli et al, 2000). The study found that 6% of total mortality

in the three countries was attributable to air pollution – 40,000 attributable cases per year. In addition, they attributed about 50% of the air pollution-related mortality to motorised traffic. The traffic-related portion also accounted for 25,000 new cases of chronic adult bronchitis/year, 290,000 episodes of bronchitis in children, more than 500,000 asthma attacks and more than 16 million person-days of restricted activities. The CRF for PM_{10} and mortality was based on the Harvard Six-Cities and ACS cohort studies from 1993 and 1995. Morbidity CRFs are documented in their paper and were similar to the databases recommended here (see the next section).

The Kunzli et al. study characterised exposure for the population of the three countries by estimating the PM_{10} concentration from monitoring data for each km^2. Thus, a population-weighted exposure distribution was created. The effect of an intervention that would reduce PM_{10} levels by various increments was modelled and the resulting reduction in mortality and morbidity was estimated for the populations in each country at each level of exposure (small range of PM_{10} concentration). Thus, the benefit of each intervention step could be seen.

The following graphic from Kunzli et al. shows the basic model for the application of the concentration-response data to estimate impacts of changes in ambient pollutant concentration. The slope of the graph is equivalent to the CRF parameter used here.



P and P_0 in the figure are probabilities of outcomes in the model and reference populations, respectively. E and E_0 are the levels of exposure of the two populations, here defined in terms of PM_{10} concentration. D is the difference in outcome frequency between the model and reference populations at exposure level E. $E_0 + 10$ defines the exposure for an increase of 10 $\mu g/m^3$ PM_{10}, and D_{10} is the difference in outcome frequency (probability or risk) for that change in PM_{10} exposure.

3.2.2 Preferred exposure-response factors

PARTICULATE MATTER

The evidence presented thus far supports most strongly a quantitative relationship

between exposure to PM (PM_{10} or $PM_{2.5}$) and premature total population mortality – both from the time series analysis and the large-scale longitudinal studies.[27] The Panel prefers to base its selection of the most reliable concentration-response factors on the (updated) NMMAPS data for the Northwest US cities, the most recent results from the Harvard Six-Cities Study, the most recent results from the American Cancer Society Study and the Health Canada studies of Canadian cities. Some of this information has been synthesized into the current database used in Health Canada's Air Quality Valuation Model (AQVM) and the Ontario Medical Association's Illness Costs of Air Pollution (ICAP) model.

A similar database of concentration-response factors was created for the cost-benefit analyses of the US Clean Air Act carried out by the US Environmental Protection Agency (US EPA, 1999). One such study was done retrospectively for the period to 1990 (when major amendments were promulgated), and another for the prospective period 1990-2010. The databases prepared for the Canadian versions of the valuation model cited above are preferred. For the purposes of this report, only the CRF databases from the models are needed – the economic valuation of the health outcomes is not within the purview of the Panel.

The mechanics of applying the estimation method are described in more detail in Chapter 4, where the practical applications of the information are discussed. The relative merits of the AQVM and ICAP CRF databases relative to other possible choices of CRFs are discussed below where relevant with respect to the Panel's alternate recommendations. See Appendix 7 for a summary of the AQVM and ICAP CRF databases.

All three major CRF databases – AQVM, ICAP and the US EPA database – have been thoroughly peer reviewed. AQVM was reviewed by an expert panel of the Royal Society of Canada (see below), and ICAP was subjected to a peer review by Health Canada in which reviewer comments on ICAP were broadly similar to those of the Royal Society reviewers on AQVM (D. Stieb, Health Canada, personal communication, February 2003).

The various Health Canada-sponsored studies of air pollution and health effects in Canadian cities (Burnett et al, 1997a, 1997b, 1998, 2000) include Vancouver, but the earlier studies do not include ambient monitoring data for PM and the most recent (Burnett et al., 2000) reports only pooled data for the eight cities together. Thus, the Panel prefers to cite the Health Canada meta-analysis (Stieb et al., 2002b, 2003) of a broader range of studies that included the Canadian city studies.

The World Health Organization's Comparative Risk Assessment Working Group, specifically, the Outdoor Air Pollution Working Group of the WHO Global Burden of Disease Comparative Risk Assessment Project, have proposed a relative risk model for PM that assumes a threshold (WHO, 2002), but the Panel prefers the recent convincing

[27] As noted in the Introduction, the Panel is aware of potential limitations of focusing on a single pollutant, since statistical analyses using single- and multi-pollutant models have produced quite different CRFs on occasion. The Panel has not systematically evaluated these differences, which do not appear to be material for the purposes of this report. One reason for the Panel's preference for selecting PM as the principal air pollutant is to avoid potential double-counting of effects that are in fact redundant among several co-pollutants. Another reason is that the PM literature as a whole (whether for single- or multi-pollutant analyses) is more convincing in the Panel's opinion than that relating the gaseous pollutants with mortality and morbidity outcomes.

evidence from North American studies that have found no threshold of response. The WHO group appears to have adopted the threshold formulation of relative risk in the practical context of the lowest observable effects levels in the underlying studies and does not mean to imply that a threshold has been detected (WHO, 2002).

The WHO model does not differ materially, in effect, from the non-threshold approach recommended by the Panel, since it is linear over the range of exposure above the hypothesized threshold. In any case, for the purposes of this section, the approaches are equivalent, since the typical current ambient levels of PM_{10} or $PM_{2.5}$ in BC and the PNW are close to or greater than the hypothesized thresholds in the WHO work (20 and 10 $\mu g/m^3$, respectively)[28], and essentially the same CRFs for mortality and inhalable and fine PM are used by the WHO group as are recommended by the Panel. The application of the recommended CRFs below the WHO thresholds is supported by the data in Figure 2 (Schwartz et al., 2002) and other studies.

The NMMAPS time-series results for the State of Washington cities in the analysis (Seattle, Tacoma, Spokane and Olympia), as adjusted to address the general additive model statistical problem, indicate a CRF of 0.2% change in total mortality per 10 $\mu g/m^3$ change in 24-hour average PM_{10} (with a standard error of 0.1).[29] This range of values is taken by the Panel to represent the lower bound of acute (short-term) response to exposure to inhalable PM.[30]

The most recent analysis of the Six-Cities data (Schwartz et al., 2002) is expressed in terms of $PM_{2.5}$ and shows a linear relationship with total daily deaths down to about 2 $\mu g/m^3$. See Figure 2. The CRF (slope) of this function is about 1.5% per 10 $\mu g/m^3$ (24-hour average). This is consistent with the Harvard group's earlier analysis based on PM_{10}, which indicated a CRF of about 1% per 10 $\mu g/m^3$ change in 24-hour average PM_{10} (Schwartz and Dockery, 1992) and 1.5% per 10 $\mu g/m^3$ change in 24-hour average $PM_{2.5}$ (Schwartz et al., 1996). Since these analyses are of daily death time series, this CRF also represents short-term (i.e., day-to-day), acute response.

Stieb et al. (2002b, 2003) in their synthesis of world-wide daily time-series analyses, which included their Canadian studies, found a CRF for PM_{10} equivalent to 0.6% change

[28] The WHO Working Group is currently using a practical, lower-end, working truncation of the concentration response function (working 'threshold') for cardio-pulmonary mortality of 7.5 $\mu g/m^3$ for $PM_{2.5}$, with sensitivity analysis that includes no truncation or truncation at 3 or 10 $\mu g/m^3$. The WHO Working Group emphasizes that the preference to truncate the concentration-response function is not intended to represent a 'threshold' but a reference point to add conservatism to the world-wide analysis of air pollution burden of disease. The 7.5 $\mu g/m^3$ value is, in fact, the 1998-2000 mean concentration at the Abbotsford, BC monitoring station. The WHO Working Group is also considering truncating the upper end of the concentration-response function at 30 or 50 $\mu g/m^3$ to indicate a possible flattening off of response at higher exposures, but this approach is still under deliberation. The full linear treatment with no lower or upper truncation increases the estimate of global air pollution-related cardio-pulmonary mortality by about 10% (Personal communication, H. Ross Anderson, speaking on behalf of the WHO Working Group, at the California Air Resources Board, Third Haagen-Smit Symposium, May 2003).

[29] This is based on Dominici's recommendation of using the results of regionally pooled analysis. See the NMMAPS (2000) reference entry for details and Dominici et al. (2003).

[30] It should also be noted that the recent reporting of the corrected NMMAPS results by Dominici et al. (2003) shows that the regionally-pooled CRFs vary by only about a factor of 2 across the 88 US cities for both total and cardiovascular-respiratory mortality.

in total mortality per 10 μg/m^3 change in PM_{10} (95% confidence interval: 0.5-0.8%).

The long-term American Cancer Society study (Pope et al., 2002) found somewhat stronger response, which is interpreted by the Panel (see above) to indicate chronic, cumulative effects that are not captured by the time-series studies. Pope et al. found CRFs of 4%, 6% and 8% increased risk of all-cause mortality, cardiopulmonary mortality and lung cancer mortality, respectively, per 10 μg/m^3 increase in $PM_{2.5}$. Chronic effects with long lag time between exposure and response, such as lung cancer, would not be picked up in the daily time-series studies, and similarly for other cumulative effects that might influence susceptibility. Their earlier study of the same cohort (Pope et al., 1995) had found a similar CRF of 3.6% change in total mortality per 10 μg/m^3 increase in PM_{10}.

The Royal Society of Canada convened an expert panel to critique the Canadian Government's approach to estimating the benefits of implementing the Canada-Wide Standards for PM and ozone (Royal Society of Canada, 2001 – see also Appendix 7). The use of AQVM as the health benefit evaluation tool was thoroughly reviewed. The Royal Society Panel recommended that the CRFs used in AQVM for estimating mortality associated with PM be replaced by the values from the long-term studies, such as the current Panel recommends.

The Royal Society recommended the use of results from long-term cohort studies, namely, the Harvard Six-Cities Study (the original 1993 study results – for the high end of the range of reliable values) and the American Cancer Society study (the original 1995 results – for the central value of the range). They also recommended that the results of Abbey et al. (1999) be used as the 'low' value of the range of the $PM_{10}/PM_{2.5}$ mortality CRF. The current Panel does not recommend the Abbey et al. values, since the study population was relatively small, homogeneous, not representative of the general population (being members of a specific religious denomination) and localised in Southern California. The CRF found by Abbey et al. (1999) is very similar to that found by Schwartz et al. (2002) from their time-series study, so the agreement of the low-end chronic effect study value with the most recent Six-Cities time series value lends credence to the Panel's recommendation that the time series results be taken as the low end of CRF values to be used for effects estimation.

The US Environmental Protection Agency's *Air Quality Criteria for Particulate Matter* (US EPA 2002) provides a compact summary of the large body of literature and the relative risk values for many health outcomes (see Chapter 9, Tables 9-14, 9-15 and 9-17).

Which of the acute or chronic CRFs might be chosen for application in a given situation depends on the context of the issue being addressed. This is discussed further in the applications scenarios presented in Chapter 4. The following table, Table 5, summarises the range of CRFs for PM that are considered by the Panel as the most reliable. The basis for this choice is severalfold, including scale of the supporting studies, statistical robustness and coverage of a diverse range of community settings – all suggesting generalisability to this region. The range of values in Table 5 is similar to that being considered by the WHO Outdoor Air Pollution Working Group (WHO, 2002).

The AQVM database of CRFs assigns weightings to each of the 'low,' 'central' and 'high' values to enable uncertainty distributions to be generated within the AQVM

software. The Panel has not assigned weightings to the distribution points in Table 5 and recommends that the 'high' and 'low' values be used for sensitivity testing estimates. This will not encumber most applications of the recommended values in Table 5 and avoids creating an inappropriate impression that the uncertainties are explicitly defined. More detailed analysis of the data from the various studies that contributed results to Table 5 would be necessary in order to ascribe quantitative weightings (or a complete uncertainty distribution) to the values in Table 5. It is suggested that the range of values be used in sensitivity testing of calculations using Equation (1).

Table 5. Best estimates of PM_{10} or $PM_{2.5}$ mortality concentration-response factors for use in BC/PNW

Context	CRF (%/10 μg/m^3)*	Sources
Acute, daily mortality	Low: 0.2 Central: 1 High: 1.3	NMMAPS (corrected); Dominici et al. (2003) Stieb et al. (2002b, 2003) Six-Cities (updated)
Chronic, total mortality	Low: 1 Central: 4 High: 11	Time series: Stieb et al. (2002b, 2003) PM_{10} & $PM_{2.5}$ similar (Pope et al. 1995 & 2002) Six-Cities Study (re-analysis)
Chronic, lung cancer mortality	Central: 4 (PM_{10}) 8 ($PM_{2.5}$)	Pope et al. (1995) Pope et al. (2002)

* Percent change in total non-accidental mortality in adults per indicated increment of PM_{10} or $PM_{2.5}$ concentration. May be converted to AQVM-compatible units by multiplying the CRF by the per capita base rate for daily, total or lung cancer mortality and dividing by 10 to put the CRFs into units of per capita unit risk per 1 microgram per cubic meter change in concentration.

Morbidity CRFs

As noted earlier, morbidity outcomes are far more numerous than mortality and are very important in assessing impacts of air pollution on public health. The Panel is reluctant at this time to select specific CRFs for morbidity outcomes for PM or other pollutants to be applied generally to this region, other than those in the existing databases that have been cited. A range of possible values has been cited in the previous sections in connection with the studies that have been reviewed. Uncertainty in the morbidity responses may be greater than for mortality, since mortality is a unique, singular, well-defined endpoint, whereas, the various types of morbidity suffer from variability in reporting and diagnosis and availability of reliable statistics. Both morbidity and specific-cause mortality data are subject to uncertainties, for example, because of diagnosis and attribution variability. There are difficulties in mortality assignment, particular as between cardio-vascular and respiratory causes of death. The characterisation of morbidity may also vary considerably from location to location. The Panel did not have the resources to undertake the detailed statistical evaluation that would be necessary to improve on the available databases of morbidity CRFs.

The softness of the morbidity data does not preclude using the AQVM, ICAP or EPA compilations of CRFs, but applying them to quantitative estimates of morbidity effects in communities remote from the original study area must be done cautiously. The estimates are not as reliable as the mortality estimates. The best starting point for application of the CRFs is the most current AQVM database, with the exceptions noted here. See Appendix 7.

Detailed assessment of other individual morbidity CRFs is beyond the scope of the Panel's assignment. That said, some suggestions for using these data qualitatively are provided in the examples in Chapter 4.

Applicability

The applicability of the CRF database for PM to communities in BC and the PNW is qualified by both community size (population) and mix of sources. The Panel concludes that the results of the studies discussed in Chapter 2 should be applied quantitatively only to communities in BC that have general urban source mixes comparable with communities that were the basis of studies elsewhere. The Lower Mainland, Victoria and Kelowna, which have typical urban source mixes not dominated by specific pollutant sources, are in this category. Conclusive evidence for applicability of the available data to smaller BC communities with very different source mixes (e.g., significant wood smoke sources, such as Prince George) is lacking. The general agreement of the CRFs for PM, however, across diverse study regions and populations around the world indicates that for this pollutant, general applicability can be recommended. The relationships can be applied qualitatively with some caution to smaller communities as preliminary indicators of the relative magnitude of effects associated with air pollutants. Some examples are given in Chapter 4.

OZONE

The variability of the ozone mortality data and the scarcity of local studies make it difficult to specify a CRF. Vedal et al. (2003) did find a significant association between ozone and total mortality in Vancouver in summer, with a CRF of about 4% per 10.2 ppb change in daily peak hour O_3 concentration, but with large uncertainty (95% confidence interval: 1-7%). The authors conclude that the effect may be due to unresolved effects of 'air pollution' generally rather than of ozone itself.

Stieb et al. (2002b, 2003) found excess mortality associated with ozone exposure and a CRF for total adult mortality of 0.5% (95% CI: 0.4-0.6%) per 10 ppb change in daily peak hour ozone.

The Panel does not recommend relying on the small database for estimating ozone-related mortality in this region. The CRF for ozone-related mortality in the AQVM database, for example, is about three orders of magnitude lower than that for PM for comparable concentration changes that occur in typical episodes in this region. The team that selected the CRF database for ICAP, which included a member of the current Panel and several other experienced epidemiologists, concluded that the data were insufficient to assign a reliable CRF for ozone and mortality. ICAP does not estimate ozone-related mortality in its current version. Note also that the 95% CI for ozone mortality used by

Cifuentes et al. (2001) includes zero (see Table 4). The 'low' CRF used in AQVM for ozone mortality is 40 times smaller than the 'central' value.

Other typical CRF databases that include both PM and ozone show the PM_{10} mortality CRF as about 30 times as great for unit change in concentration as that for ozone (for PM_{10} incremental unit = 1 $\mu g/m^3$ and for ozone, 1 ppb). During an episode in this region, PM_{10} and ozone would both increase by about the same number of incremental concentration units, i.e., 40 $\mu g/m^3$ for PM_{10} and 40 ppb for ozone, so that the relative CRFs represent roughly the relative impacts during an episode. See Cifuentes et al. (2001), for example.

As noted above, clinical and epidemiological studies have demonstrated significant morbidity effects of ozone, but again, the results are highly variable from study to study. Their application to this region (e.g., using the AQVM database) should be done cautiously. The Brisbane ozone and asthma data should be considered for application in this region for the reasons stated earlier (Section 2.2.1). The relative risk in Brisbane of total asthma hospital admissions associated with ozone exposure was 1.09 per 10 ppb increase in the 8-hour average concentration of ozone (lagged 5 days).[31] AQVM does not use a specific relationship for asthma-related hospital admissions (total respiratory hospital admissions only).

OTHER POLLUTANTS

The analysis in Appendix 5 suggests that concentration-response data from elsewhere may be applicable to assessing the quantitative impact of wood smoke in the Interior of BC. The Panel believes that more detailed analysis of data for these communities is necessary before reaching a conclusion about a reliable CRF. A pooled analysis of monitored PM_{10} or $PM_{2.5}$ with hospital admissions or other morbidity data from the BC Interior communities listed in Appendix 5 (pooled population in excess of 100,000) may provide statistically significant results.

3.2.3 Uncertainty

The problem of uncertainty in the exposure metric is common to all air pollution studies. Considerable refinement of exposure estimates is possible by using time-activity diaries, ambient monitoring data, personal monitors (particularly for NO_2), and measurements (for particulate matter and ozone for example) inside and outside the home, or within schools or offices. An important principle is that any refinement in exposure data increases the power of the study. One of the most detailed efforts to define the exposure metric in an urban population was conducted recently in Helsinki. This study yielded the finding that ambient monitors reflected the NO_2 exposure of those who did not move much away from their home environment (tending to be older people), but that younger residents had a much more variable relationship to static monitored data. Innovative methods have been used to define exposures to vehicle exhaust in relation to homes close

[31] Petroeschevsky et al. (2001) should be consulted for details of the analysis. Specific RR factors for various age groups and exposure metrics are given.

to, or away from, heavily traveled freeways. One might note that if wood smoke exposure is an important risk factor in rural or semi-rural communities in BC, then a biomarker (from urine analysis, for example) of wood smoke exposure might prove to be an important research tool.

It should be noted that the quest for "certainty" is illusory. This word, and also "proof" cannot be applied, in any rigorous sense, to issues that require judgement as to causality.

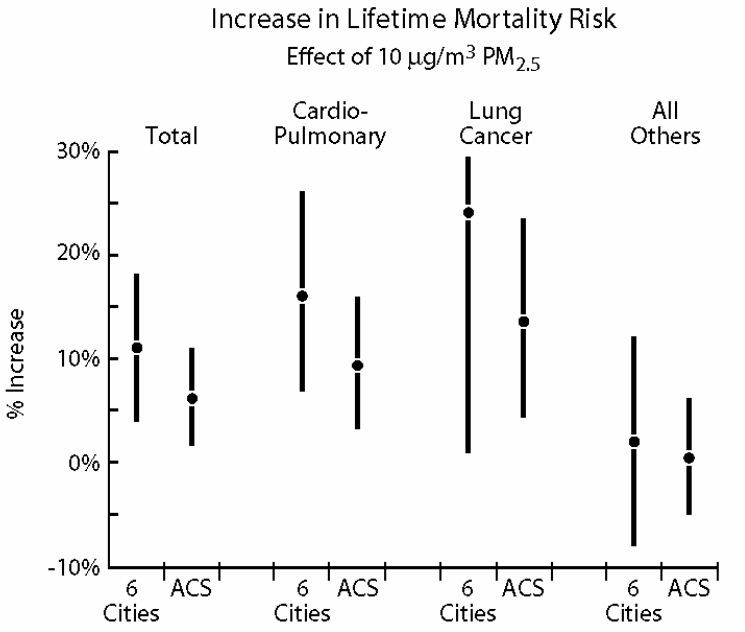
Uncertainty in environmental epidemiological analysis has received a great deal of attention recently in both the scientific community and the general public because of reported errors in the analytical methods for the NMMAPS analysis (NMMAPS, 2000). NMMAPS is managed by the Health Effects Institute, which is a research organization jointly funded by the US EPA and the automobile industry. It has a very reputable track record and is highly regarded by the scientific, regulatory and business communities. The original NMMAPS study was a time-series analysis of total non-accidental mortality and PM_{10} concentrations for the 88 largest US urban areas. The results indicated that on average across the US, the total mortality concentration-response factor for PM_{10} was about 0.4% change for a 10 $\mu g/m^3$ day-to-day change in PM_{10}. After discovering an error in the way that the statistical software used (S-Plus) was applied in calculating analytical iteration convergence in general additive models (GAM), reanalysis indicated that the national average estimate was about 0.2% change per 10 $\mu g/m^3$ PM_{10} change. This discrepancy caused an immediate response in the press and among sceptics of the original results, attempting to cast doubt on all such analyses. Realistically, the factor-of-two error is not a material one in the overall scheme of uncertainty in epidemiological analysis. The corrected data are summarised in Dominici et al. (2003). It should also be recognised that time series results do not reflect the total impact of air pollutants on health, as evidenced in the longer-term cohort studies cited in this report.

The following graphic (Figure 4) summarises the results and uncertainties of the major studies on which the Panel's recommended CRFs for PM are based.

Katsouyanni et al. (2002) recalculated the European (APHEA) data on PM_{10} and black smoke and mortality associations. Their results show little impact of the GAM analysis effect, with a reduction of only 4% and no change when reported with one significant figure. PM_{10} data show an increase of total mortality of 0.6% for a change of 10 $\mu g/m^3$.[32]

[32] A reviewer pointed out that other re-analysis of the European data showed a larger reduction from the APHEA result of about 30%, similar to the NMMAPS re-analysis, such . Such an adjustment does not affect the Panel's selection of data for Table 5. LeTetre et al. (2002) obtained results for nine French cities that confirmed the corrected NMMAPS results.

Figure 4. Summary of Six-Cities and American Cancer Society mortality risk (CRFs). Source: Lippmann et al. (2003)



Mean increase and 95% confidence intervals for annual mortality rate increases per 10 µg/m^3 increment of $PM_{2.5}$ for the 6 cities and ACS cohorts based on 16 years of mortality data.

It has been said that all models are wrong (... but some are useful) – whether of air pollution effects on health or stratospheric ozone depletion, climate change, the Canadian economy or the stock exchange. Each database from which we wish to extract useful information has more complicated underlying structure than we can hope to represent by analytical models, for which we must choose our best estimate of the important parameters. Governments and investors make important decisions every day on information from models that is far more uncertain than a factor of two. In order to analyze any data, it is necessary to choose a model and try to ensure that it reflects as accurately as possible our knowledge of the underlying complexities. We are of course limited by the resources (i.e., budget) available to undertake the work.

Another limitation of the existing database is the practical need to relate outcomes to daily events and monitoring regimes. No shorter term results are possible for mortality, which can only be studied observationally, not experimentally. Thus, there is no information that would relate mortality to very short-term concentrations of PM, such as one-hour averages. Morbidity outcomes for short-term exposures have been discussed in Chapter 2 for several pollutants in connection with clinical experimental studies, but, at present, estimates of mortality outcomes and more severe morbidity outcomes (e.g., hospital admissions) must rely on daily rather hourly data. It is not known what uncertainty this limitation may introduce.

The work that the Panel has selected to rely on for conclusions and recommendations (see above and Chapter 5) has demonstrated robust statistical associations between exposure to pollutants and effects (see the 'Definite' column in Table 3). The certainty of the effect is not at issue. The magnitude of the effect depends on the model chosen for the analysis. It is important to distinguish between certainty of the demonstrated association and certainty of the magnitude of the association. The former is firmly based; the latter is improving.

In practical application of the CRF database, the approach used in AQVM is reasonable. The AQVM CRF database provides 'low,' 'central' and 'high' values with suggested weighting factors to be used in simulations of the uncertainty distribution in the estimated outcome values (see Appendix 7). If the 'central' values alone are used in practice, it must be recognised that the uncertainty distribution implied by the ranges given is implicit in the estimate.

3.3 TRANSFERABILITY OF CONCENTRATION-EFFECT RELATIONSHIPS FROM COMMUNITY TO COMMUNITY FOR VARIOUS OUTCOMES.

3.3.1 When should local data be used instead of values from the literature?

Assumptions about the applicability of epidemiological information between different regions are dependent on a number of issues. As guidelines, the following general statements are relevant:

- Similarity between defined populations in some respects can be assumed. There is no reason to suppose that asthmatic children in Port Alberni are different from asthmatic children in Provo, Utah; and seventy year-old people who have had a myocardial infarct can be assumed to be similar in Victoria BC and metropolitan Boston.
- Some outcomes may be affected by dietetic differences – the effects of ozone may be mitigated by diets higher in antioxidants for example.
- Differences in time-activity patterns may affect aggregate exposure. In Los Angeles for example, children are in air-conditioned schools during the day, in which the ozone level is only 15% of what it is outdoors; however, they come out of school at 3 pm into what is generally the highest ozone of the day; their aggregate ozone exposure is greater than it is for office workers who stay inside buildings until 6 pm.
- Housing and building differences may be important – air conditioning, proximity to freeways, local topography, etc. may all affect exposure patterns to specific emissions. The impact of these factors on effect estimates has been demonstrated (Janssen et al, 2002).

For these reasons, local data should always be used when they are available. In the case of BC, these consist of the Port Alberni study and the Abbotsford fruit pickers study and others cited in Section 2.3.3. The annual peak in asthma emergency visits in the third week of September in Vancouver has to be considered if a panel study of asthmatics is planned.

Other local studies are described in Section 2.3.3 – PM and lung function, PM and cardiac arrhythmia, PM and mortality are all indicated relationships. Seattle studies should also be generally applicable to BC for certain endpoints (mortality, lung function) – it is a bit more difficult to extend Seattle panel studies that focused on inner city asthmatics, ER visits, hospitalizations or studies with symptom outcomes, given the differences in the health care systems and access to health care.

3.3.2 Role of the size of the exposed population (limitation)

The following guidelines may be useful:

1. Increased exposure to air pollution will enhance the risk of an outcome even if the population is too small for an epidemiological study to be able to demonstrate the increased risk.
2. In general, single epidemiological studies do not yield precise enough dose-response metrics for formal risk-analysis methods to be applicable. Reliable risk factors are developed through many reproducible studies on populations of sufficient size to produce statistically significant data.
3. The statistical power of panel studies is often between the two extremes of controlled exposure, clinical studies and epidemiologic studies. Panel studies are uniquely valuable because usually the exposure metric is more precise, and the specific outcome is better specified than, for example, in analyses of hospital admissions.
4. The 'precautionary principle' should be considered applicable whenever a risk has been well defined, irrespective of the size of the impacted population.

As noted above, Sheppard's analysis concludes that for distributions of pollutants and outcomes similar to Seattle's, fewer than about 100 outcomes per day materially reduce the reliability of epidemiological analysis.

Epidemiological studies themselves are not inherently less precise than controlled human exposure studies or animal studies that are used in risk assessment – in epidemiological studies one often trades specificity with respect to the outcome and the exposure for a larger and more representative population (including a much better representation of susceptible individuals) that enhances overall statistical power.

The issues of "thresholds" and the presence or absence of "harvesting" (see Section 2.4.2) can only be resolved when different epidemiological studies are analysed. Often important information can be derived from comparisons of studies in different locations (examples are the Brisbane asthma study where pollutants other than ozone are absent and the Christchurch study where the PM_{10} is derived primarily from wood smoke and other emissions are virtually absent).

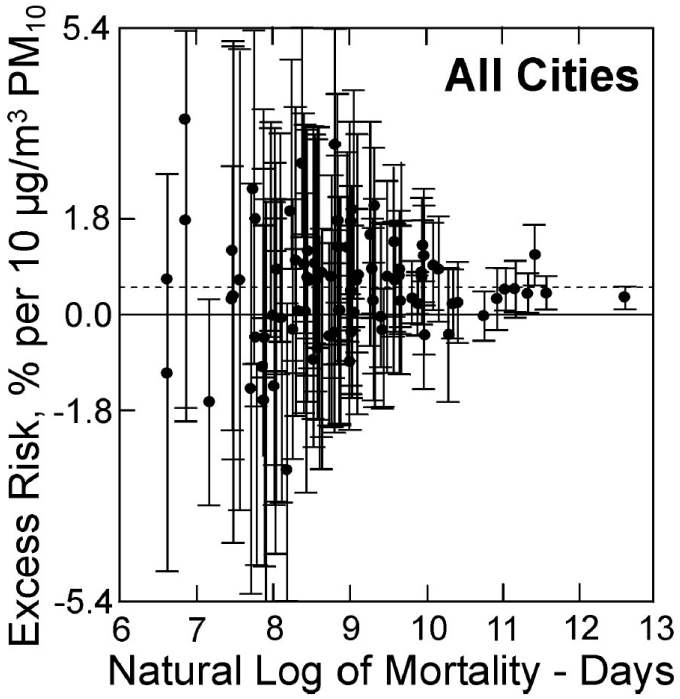
The US EPA carried out analysis of the original (uncorrected) NMMAPS results in terms of the relative magnitude of error with respect to the 'study size.' In this context, 'study size' is the natural logarithm of number of person-years of effects and monitoring data available to be analysed. The following figure (Figure 5) is taken from the Second External Review Draft of the EPA *Air Quality Criteria for Particulate Matter* document

(March 2001), so should be treated with caution, since it does not reflect official analysis of the corrected NMMAPS data. It illustrates the effect of population size on uncertainty in a time series analysis of this sort. The figure appears as Figure 6-12 in EPA (2001: Vol. 2, p.6-261).

The figure shows that the greater the number of mortality-days of observation (effect data and monitoring data), the more precise the effect estimate. The metric used by EPA is the natural logarithm of the number of mortality-days (the 'power' of the analysis) for each city analysed by the NMMAPS study (related to the exponential form of the basic disease model). The EPA analysis shows that the 95% CI uncertainty limits converge toward a stable value for study sizes having the natural log metric greater than about 9. This means that only for a study size of the order of 10,000 death*days do the estimates converge to a stable value – in other words for the 20 or so largest cities in the US for the NMMAPS study.

The analogous figure for the cities in the 'Northwest' region of the NMMAPS study shows a similar convergence toward the national mean risk factor as a function of study size (range of study 'power' ~ ln 7-11).

Figure 5: Effect of study size on precision of excess risk estimates in NMMAPS (uncorrected for GAM effects)



Thus, precise estimates of the relationship between PM_{10} exposure and mortality can be expected only if the study population experiences at least approximately 27 deaths per day over a one-year period. The average death rate in Greater Vancouver, for example, is about 34 per day (6.2 per thousand per year x 2 million people), so the population in this region is just sufficient to produce a reasonably precise estimate of the mortality effect of PM for one year of monitoring data.

In a community in this region with a population of 100,000, for example, we would expect about 650 deaths per year, or just less than 2 per day. Based on the EPA's preliminary analysis, the implication for this smaller population is that about 15 years of mortality and monitoring data would be necessary to extract a reliable estimate of the PM-mortality relationship. This assumes that all important factors in the analysis (including demographics and pollutant mix, for example) could be controlled adequately over the extended period of time to limit the level of uncertainty. This level of control has been possible only in very unusual circumstances, such as the long-term monitoring of all of the important parameters in the communities in the Harvard Six-Cities Study. The period of data coverage could be retrospective or prospective, depending on whether the historical databases for both death records (or other effects data) and monitoring data were sufficiently complete. Relative uncertainty in the exposure metric (PM monitoring) is not reflected in the EPA's preliminary analysis and would also play a role in differences among communities.

There are similar implications for morbidity studies. Morbidity outcomes occur more frequently than mortality, so, generally speaking, the time period of a study can be shortened relative to a mortality study (see Figure 3). This assumes that adequate exposure data are available and that the morbidity outcome(s) to be studied have adequate data, including accurate and precise diagnostic characterisation (well-defined endpoints). For example, if, say, emergency room visits related to PM are estimated to occur at about three times the rate of mortality for a given level of PM, the study might be expected to produce reliable results with 3-5 years of data rather than 10-15 years.

The above model does not apply to all types of epidemiological analysis and is a generalisation that should not be over-extended, but it is strongly suggestive of the broad parameters that would need to be addressed in carrying out new studies in BC and the PNW. Some examples are provided in the scenarios evaluated in Chapter 4.

All studies of adverse health effects are constrained by certain inevitable limitations. For example, it is not possible to show statistically significant outcomes when the population to be studied is too small (as shown above). It is acceptable to aggregate data (as we have suggested might be done with respect to wood smoke effects in the interior cities and communities in BC) but only if the statistical consequences of this are understood (see Zidek and Bates (2002) for examples of this problem). Where subjective issues are involved, such as headaches, or nausea, or attacks of dizziness, it must always be assumed that inevitable personal recall bias has had some influence. This difficulty is avoided if some definitive outcome metric, such as admissions to hospital, can be used.

Although many people commonly believe that comparisons of specific outcomes between small populations, or between a small population and the province or state as a whole, can provide "definitive" information about illness prevalence, such comparisons are very rarely useful and careful control of them has to be exercised if any conclusions are to be drawn.

3.4 APPLICABILITY OF THE VARIOUS METHODS FOR EFFECTS ESTIMATION TO EVALUATING SPECIFIC SOURCE IMPACTS

3.4.1 Is there a preferred methodology for different specific outcomes (health endpoints)

The methodology employed should be carefully tailored to the question being addressed. Thus, for aggravation of asthma, a panel study (as in Port Alberni) provides a sensitive indicator of effect. Hospital emergency visits or family practice consultations can be used if the data are carefully screened, for the same purpose. As only a small proportion of cases seen in the emergency department need hospital admission, such admissions, unless studied over a long period of time, are likely to be less sensitive.

In the case of the effect of $PM_{2.5}$ on cardiovascular mortality, a correlative study between deaths so certified and pollutant levels can be undertaken; or, as was done in Boston and Seattle, every case admitted to hospital with a myocardial infarct can be analysed in terms of probable pollution exposure during the 48 hours prior to admission.

If in a community, there is a specific source of a pollutant known to be associated with adverse effects (as the PM_{10} from wood smoke in the Bulkley Valley), the strongest indicator of effects would be a study which used all available health outcome data over the same period of time as monitoring data – i.e., family practice visits, measurements of flow rate and medication use in an asthmatic panel, hospital emergency visits, and hospital admissions for respiratory disease.

In short, no specific methodology is preferred for any of the outcomes. The study approach must take account of the adequacy of available (or likely to be available) data, the characteristics of the impacted population and an hypothesised relationship between the components of the local pollutant mixture and the specific outcomes of interest. Each study will be defined in its own terms.

3.5 DATA GAPS AND SIGNIFICANT SOURCES OF UNCERTAINTY

3.5.1 Approaches to reducing uncertainties for application to the BC & PNW context

The overall level of uncertainty in applying the model of excess risk *vs* pollutant concentration (Equation (1) or equivalent) arises from the uncertainty in the CRFs and the measurement of concentration (exposure). The range of uncertainty in the CRFs can be seen in the ranges provided for the AQVM and ICAP databases in Appendix 7 or in Tables 4 and 5 above. It will be difficult to improve on the uncertainties in the CRF database by carrying out local studies, for reasons stated above. The uncertainty in the overall estimates due to error in exposure classification from inadequate or highly variable concentration monitoring is also difficult to address. The latter factor is under the control of monitoring agencies but is not easy to characterise for the purpose of health effects studies.

Several approaches would reduce uncertainty in application of the knowledge base to BC and the PNW context:

- Better characterisation of exposure in local communities would permit closer comparison with the exposure regimes of other studies.

- Better source apportionment data to define the nature of the pollution in different regions would assist in comparing local exposures with the literature and, hence, make more definitive conclusions possible.
- Studies of representative exposures (schoolchildren to NO_2, elderly subjects in rest homes, exposures to specific emissions, etc) would also assist by permitting closer comparison of exposures with the literature.
- Much more emphasis on local epidemiological studies to define relevance of other data to the local region is the most certain method for connecting the local information to the wider literature – bearing in mind the inherent limitations of statistical power in small data sets, as noted earlier.

More detailed statistical (meta-) analysis of data from local studies and from the broader region (e.g., Seattle) is not expected to provide more conclusive concentration-response factors for morbidity from the available data than the database recommended here (i.e., AQVM) can provide.

In the context of the scope of the Panel's review, major data gaps exist in relating the multitude of morbidity studies to risk factors that may be used for public health assessment purposes. This is especially true for the gaseous pollutants (perhaps most importantly, NO_2). Estimating ozone impacts at the relatively low exposures that occur in BC and the PNW suffers from significant uncertainty, as reflected in the Panel's reluctance to specify region-specific CRFs for ozone mortality or morbidity.

The specific characteristics of PM that are implicated in the epidemiological results to-date are not explicit enough to allow specific analysis of the health impacts of wood smoke or diesel particulate matter – as distinguished from the general effect of PM – with confidence. Exposure data specific to wood smoke or diesel particulate matter are also lacking.

The power of studies in the local region is limited by the relatively low population density in much of the region – the entire population of BC, for example, is smaller than that of some of the US cities included in NMMAPS. The approach suggested above, namely of concentrating on a defined region of BC and designing a study that would include many factors such as physicians' practice data, hospital admissions data and enhanced monitoring might be able to produce reliable results by pooling the data for a number of communities with similar exposures. Focusing on wood smoke would make sense in this context.

3.5.2 How much uncertainty is introduced by using different sources of exposure-response relationships?

In some instances – as for lung cancer incidence and cigarette smoking, for example – it can be assumed that the defined risk of the outcome in terms of the exposure is generally applicable. Observed differences between populations are usually attributed to differences in the nature of the tobacco smoked or in the prevalence of deep inhalation. There has also been some evidence that dietetic differences may influence the risk. In air pollution terms, some factors have been summarised in 3.3.1 above. From the point

of view of public health policy, the general applicability of well established dose-response data should be assumed unless it has been specifically disproved.

The Panel prefers the use of local data for the most part, although generally speaking, local studies, as summarized above, confirm exposure-response data from elsewhere. There is a degree of uncertainty introduced by using exposure-response data from studies in areas with different geographic and demographic characteristics, but the larger-scale studies that the Panel relied on (both time-series and longitudinal) are robust enough to travel and be applied elsewhere.

The question assumes that some specific measure of 'uncertainty' might be derived here, but because of the difficulty of synthesizing results from studies of different designs and database sizes, it is not possible to define a statistical 'confidence limit' for uncertainty that might be introduced by transferring coefficients from studies elsewhere to this region.

Meta-analysis, which attempts to synthesize joint coefficient values and confidence intervals from comparable studies, can provide added support to selection of particular concentration-response factors. See Stieb et al. (2002b, 2003) for an example of the potential power of meta-analysis (in this case applied to word-wide time-series studies for all of the major pollutants). In the 2002 paper, the authors conclude that the current suite of results is robust enough that "... there is little need for simple replication of these results in additional locations." Results from those studies have been used in section 3.2.1.

3.6 Appropriateness of applying various methods to estimate mortality and morbidity from air pollution related diseases to different population sizes and geographic scales

3.6.1 Are the same methods applicable to provincial/state, regional, airshed, community and neighbourhood levels of exposure?

The general answer to this question is 'yes.' Other factors may skew the results however, and these include those listed in 3.3.1 above; there may also be important differences in the validity of assumptions made about exposures. In general, all methods are applicable to any region. Comparisons between regions have to take account of differences in associated pollutants – it is for this reason that Brisbane, Australia is important from the point of view of comparisons with Vancouver. Although Brisbane is warmer, the level of ozone is not greatly different (although somewhat higher), and in both locations the ozone is not accompanied by aerosol sulphates (as is the case in the Northeast US and Eastern Canada, for example).

The main determining factor for studies of progressively smaller populations is the statistical power of the analysis as limited by the size of the available database. See the discussion in section 3.3.2 and Figure 5.

3.6.2 Can the methods be used to estimate impacts from specific sources or classes of sources, or only to overall ambient exposures?

Where a single source such as the Geneva Steel mill in Provo in Utah is responsible for 60% of the PM_{10}, valid comparisons could be made between the level of hospital admissions of children when the mill was, or was not operating. Subsequent studies involving analyses of the chemical composition of the PM_{10} from collected filters, have demonstrated that the material was much more toxic to animals when the mill was operating than when it was closed down. In towns with local industries from which the emissions have been characterized, reductions in pollution levels have been shown (in Finland for example) to be associated with a drop in respiratory symptoms in children. Prince George would constitute a comparable environment.

A recent study of the emissions from nine coal-fired power plants in Illinois involved calculation of their specific emissions and the resulting $PM_{2.5}$, modelling dispersion from the plants and then using this information to calculate the adverse health impact on the 13 million people who live within 50 kilometers of the plants (see Levy et al., 2002). This hypothetical scenario is similar to analysis carried out by Ontario Hydro (now, Ontario Power Generation) in the 1980s for its thermal power plants. The only epidemiological study that specifically resolved coal-fired power plant emissions from the receptor perspective is Laden et al. (2000), as summarised above.

If a detailed analysis of wood smoke exposure and morbidity and mortality data could be carried out, the impact of wood smoke sources could be quantified on the basis of the available data. Some communities in the Interior of BC are impacted predominantly by PM from wood burning, which would be reflected in local monitoring data.

Most community exposures are to pollutants from a mix of sources, so that specific source impact is difficult to identify. For the examples given above, dominant source(s) in a community allowed detailed source apportionment and impact characterisation. Detailed information of this sort is not available for many communities.

3.7 APPROPRIATE METHODS FOR ESTIMATING EFFECTS OF SHORT-TERM EXPOSURE TO HIGH LEVELS OF POLLUTANTS

There is a considerable literature on the measurement of exposures to industrial toxicants such as occurs when there are accidental spills (of such chemicals as chlorine, ammonia and sulphuric acid). This is not particularly relevant to ambient exposures under normal circumstances. For SO_2 and NO_2, short-term effects were described in Chapter 2, and guidelines have been provided by WHO for exposure to those pollutants (10-minute and one-hour values, respectively). For other pollutants, specifically PM, data are not available to provide an answer to this question. The question is moot with respect to ozone, since concentrations of this pollutant develop slowly through a day and are not produced in short-term bursts.

Assuming that the 'high levels' are daily values within the range of the epidemiology studies that have been cited here (which is some cases include peak PM concentrations of 150-200 $\mu g/m^3$), the fact that the concentration response functions do not seem to show significant fall off at the higher exposures means that the CRFs cited or recommended

here can be used to assess responses to short-term exposure peaks within the range of typical variation in ambient levels.

3.7.1 What time periods are most appropriate for estimating the various health impacts (hourly, daily, weekly, monthly, annually)?

There is no preferred time period for estimating effects. The reported results of the epidemiological studies that the Panel has relied on have been constrained by the logistics and available resources of estimating exposure and quantifying responses, rather than by any inherent preference for time-scales based on medical criteria. As discussed above, daily time-series and long-term cohort evaluations both have their place in estimating the relationships between air pollution and health effects. In various studies, seasonality of exposure and effects has been a factor, and for various pollutants, shorter or longer time periods have been selected because of the format of monitoring data (e.g., PM data have only recently become available in other than a daily average format). The exposure metric for ozone effects evaluation has been the peak hour daily value.

Acute effects on sensitive populations in panel studies can use hourly data. 24-hour exposures are often used in time-series studies both of mortality and morbidity. Weekly exposures are used when personal passive sampling tubes are worn by a panel subject (often children) and left in different regions of a house to estimate the aggregate exposure to NO_2 over the course of a week. Ozone badges and other passive monitors have been similarly used. Annual exposures (which give a general indication of the level of pollutants experienced in a specific county or region) have been used in longitudinal survival studies, since they provide broad comparisons of pollution levels in different regions and can be compared to specific causes of mortality. They have also been used in comparisons of such outcomes as birth weight and neonatal and postnatal mortality.

The study size discussion in Section 3.3.2 suggests general guidelines for how long a community might need to be monitored to produce significant health effects analytical results from a general community health study.

4. Applications of the Effects Estimation Methodologies

4.1 Overview of practical application method

To estimate potential health outcomes due to exposure to air pollutants based on applying the database of CRFs, the Panel recommends using the 'chronic' values for PM and mortality given in Table 5. Given the uncertainties expressed in Chapters 2 and 3, a reasonable approach to morbidity estimation is to use the existing AQVM CRF database (see Appendix 7), recognising that these values will change with time as new critically evaluated data become available. Health Canada reviews the AQVM database continually and revises it accordingly. Substitution of the 'chronic' CRFs for the time-series values used in the AQVM database that we have recommended addresses the only major criticism of AQVM's effects estimation methodology raised by the Royal Society Expert Panel.

Our recommendation (Table 5) also adds a lung cancer mortality factor to the database. Lung cancer deaths are included in the total deaths data, so if this factor is to be used, the implication for a more relevant parameter, for example, 'annual new cases of cancer,' would need to be determined based on cancer survival rate data and other information.

To estimate health effects in specific communities or neighbourhoods by means other than the CRF estimation method (Equation 1 or similar), clinical or epidemiological studies must be carried out. The circumstances in which such studies would provide useful information vary from case to case. A critical factor in being able to perform any meaningful new studies will be the adequacy of exposure monitoring. If the day-to-day variation or long-term trend in ambient air quality in a community in which a study is contemplated is not substantial, extracting meaningful information will be difficult in any case. As noted in Section 3.3.2, the statistical importance of 'study size' ('power') in considering any new studies in BC or the PNW needs to be taken into account. This point is emphasised below in the scenario examples.

It is important to recognise that the proponents of all of the principal CRF databases that we have cited in this report do not recommend using the factors to estimate absolute rates of mortality or morbidity. Rather, the CRFs should be used as intended to estimate effects for marginal changes in air quality from current levels. The model from which they are derived assumes that the relative changes in effects are small fractions of the base rates. The estimation methods can be used to estimate absolute numbers of outcomes rather than incremental or relative numbers, but it must be recognised that there is an unknown level of uncertainty in such estimates.

4.2 Scenario examples

The following hypothetical scenarios represent typical situations that regulatory authorities and the public experience in project impact assessment, regional air quality management or jurisdiction-wide planning.

There are two approaches to these scenarios depending upon whether the issue is how to apply the available literature information to addressing the problems presented by these cases, or whether the issue is how to design a new study to provide information specific

to the scenario. Which of these pertains depends on the timeframe. For example, in some of the cases below, the scenario allows a reasonably long time for carrying out an assessment; whereas, in others, the time available for doing the assessment may be only a few weeks or months – precluding new local studies. These offer examples of the differences between retrospective and prospective applications of the literature information. At issue as well is how detailed an evaluation is warranted by the potential risk presented by the situation. The latter can be estimated by application of the literature as a factor in deciding whether a new study is warranted.

The principal question being addressed is how are the findings of this report best used to inform the approach to each scenario – which types of study results should be applied to each situation and which scenarios are amenable to quantitative or semi-quantitative analysis using concentration-response factors or other data presented here.

SCENARIO 1: ASSESSING THE HEALTH BENEFITS OF MEETING AIR QUALITY STANDARDS IN AN URBAN REGION

If the issue is trying to assess the health benefits of a suite of emission reduction measures that are proposed, or whose relative benefits need to be assessed prior to implementation, there are several ways in which the health benefits can be assessed. The most reliable would be to use the long-term concentration-response factors from, say, the ACS study to estimate avoided health impacts of improving air quality to levels that would meet air quality standards such as the Canada-Wide Standards for PM and ozone or the US National Ambient Air Quality Standard for $PM_{2.5}$. In the simplest approach, the change in average air quality required to meet the standards would be equated to a corresponding change in total (or respiratory or cardio-vascular) mortality, hospital admissions, asthma attacks, and so on.

If the scenario involves closure of industrial facilities, the simplest approach is a carefully planned prevalence survey of respiratory disease in children in the vicinity of the facilities before, and after, the closures. It might be possible to use hospital emergency visits or even admissions as an indicator, but it would be difficult to exclude other factors which might affect these outcomes. A large population would be needed for any comparative data on mortality to be useful, but some studies of that kind have been done. This approach cannot be used in a predictive mode.

Economic estimates of costs can be made of specific diseases such as asthma – these were noted earlier. The data from Christchurch (see Appendix 5) provide a framework around which estimates could be made of the probable current costs of wood smoke emissions in terms of hospital costs for respiratory disease.

The current suites of CRFs in Air Quality Valuation Model (AQVM) and OMA's Illness Costs of Air Pollution model (ICAP) are distillations of values from the literature that cover mortality and the principal morbidity outcomes. The cost-benefit model for the Lower Fraser Valley that has been used and updated since 1994 has a similar set of CRFs to the 1999 AQVM set. The ICAP model has CRFs very similar to AQVM's. A target change in air quality, or an estimated change in air quality resulting from specific emission reductions, when provided as input to these models produces an estimate of the

social welfare benefit of the action (either in terms of avoided health outcomes or monetisation). The CRFs used in these models can be changed to accommodate research of the sort reviewed here – and recommended in Chapter 3.

The tendency has been to use the time-series or similar values for mortality response, but as noted above, it is more advisable to use the long-term study CRFs to account for cumulative, chronic effects that are not captured by the time-series studies. The models all use ranges of CRFs, with appropriate weightings or defined distribution functions, to provide output of the range of possible estimates of benefit. The CRF data in Table 5 are relevant here, and the AQVM dataset for morbidity CRFs is a reasonable starting point for preliminary analysis.

Such models can be built for any airshed, and local CRFs can be inserted whenever appropriate. There is no inherent limitation imposed by the structure of the models. It is important to address the relative uncertainty weighting given to locally-determined CRF values.

The following is a practical example of applying these tools to a large urban airshed. The methodology has been documented for this type of analysis for the Lower Fraser Valley in the reports on the several applications of air quality cost-benefit analysis that have been done since 1993.

Assumptions –

Community size: 100,000 population

Current average PM_{10} concentration: 25 $\mu g/m^3$, with numerous daily peaks above BC's 50 $\mu g/m^3$ objective.

Emission reduction measures: estimated to reduce the annual average to 20 $\mu g/m^3$ and eliminate most of the excursion above the 50 $\mu g/m^3$ objective.

Annual total non-accidental mortality rate: 6.2 per thousand people (6.2×10^{-3} - typical for BC). For this community, the rate is then 620 people per year. Actual, current mortality rate data for a community, with characterisation of the local population demographics, should be used whenever possible.

The question is "Approximately how many prematurely shortened lives will be saved each year by implementing the emission reduction measures?"

Analysis –

The estimated change in ambient concentration of PM_{10} is 5 $\mu g/m^3$. The preferred 'central' value CRF for PM_{10} and mortality is 4% change in total mortality per 10 $\mu g/m^3$ change in daily PM_{10}, assumed to persist for many years. The estimated number of avoided premature deaths annually, then, is a 2% reduction in total mortality or 12 lengthened lives per year. The range of the estimate based on the recommended 'low' and 'high' CRF values (1% and 11%, respectively) is between 3 and 34 lengthened lives per year.

If the time series CRFs were used instead of the 'chronic' study values from Table 5 to represent short-term response to the reduced PM concentration, these estimates would be reduced to a range of <1 person per year to 4 per year.

Comment –

The various morbidity CRFs from the AQVM database can be applied similarly to the foregoing analysis. The data are presented as per capita change in risk for a 1 $\mu g/m^3$ change in PM (or 1 ppb change in ozone), so the underlying total mortality rate (assumed uniform across Canada) and the pollutant increments from the literature have already been accounted for. Consult the AQVM 'Methodology' manual (AQVM version 3.0), which is available from Health Canada, for details of the CRF database.

SCENARIO 2: ASSESSING THE HEALTH IMPACTS OF EMISSIONS FROM A PROPOSED INDUSTRIAL PLANT NEAR AN URBAN REGION IN A FORMAL ENVIRONMENTAL IMPACT PROCEEDING

Bates (2002) has recently published some notes for carrying out health risk assessment of industrial facilities. He proposes that each of the four steps that follow is necessary to assessing the impacts of an industrial plant (or other new facility):

1. Quantitative assessment of emissions under the full range of operating conditions.
2. Description of potentially impacted population, including sensitive or vulnerable sub-populations.
3. Modelling pollution impacts using worst case emissions to estimate maximal likely impact.
4. Estimates of increased risk attributable to the estimated ambient concentrations using reliable concentration-response factors (such as those evaluated here) – reference to compliance with standards or objectives is not sufficient.

This risk estimated in this assessment procedure may be translated into estimated number of excess cases of illness or premature death in the impacted population. Since acute responses to changes in air pollutant levels are being assessed, the concentration-response factors to be used in this situation would be the results of the time-series studies (as a low estimate), and of some panel studies. Numerical values of the concentration-response factors to be used in this situation are suggested in Table 5. Assessing longer-term impacts would require estimating changes in total exposure of the impacted community including the new facility over an extended period of time. If such information were provided, use of the long-term CRFs in Table 5 would be appropriate. If the impacted community is small, the CRFs determined from the large-scale studies need to be applied with caution, since the number of exposed individuals can no longer be treated as a normally distributed population – individual susceptibilities need to be taken into account. The estimated changes in short-term and long-term ambient concentration incremental impacts of the facility, combined with local monitoring data, could be used

with the CRF database to provide a rough estimate of the increased risks of mortality or morbidity.

For more facility-specific impacts, for example, measurements of blood lead levels in a population impacted by a lead smelter have been reported, but exposures may continue after the emissions have ceased. Detailed studies of possible outcomes might be possible in relation to very large installations (such as in Fort McMurray, for example). Body burdens of fluoride might be indicators of exposures (in Kitimat, for example). Plant emissions have not been implicated in relation to asthma prevalence as far as the Panel is aware. See 3.6.2 for a recent attempt to link emissions from pre-1930 power plants in Illinois to health impacts calculated on the basis of $PM_{2.5}$ exposure. Time-series study results from opportunistic abrupt changes in exposure due to closure of a specific type of facility, such as the Utah Valley study of Pope et al., could be used for specific industry types.

Population survey results are often difficult to evaluate because of personal perception bias. This would be the case with attempts to evaluate the impact of episodic formaldehyde emissions (or other odorous substances), for example. Recent studies have permitted a more precise and objective evaluation of the impact of hydrogen sulphide emissions (see Bates and Vedal, 2002).

Practical example, impact of facility emissions of $PM_{2.5}$:

Assumptions –

Community size: 10,000 population

Facility-related estimated annual average $PM_{2.5}$ incremental impact: 0.2 $\mu g/m^3$.

Question: What is the estimated annual mortality impact of the facility's incremental PM emissions?

Analysis –

The community's annual mortality rate would be about 62 per year (assuming a base mortality rate of 6.2 per thousand population per year). An increment of 0.2 $\mu g/m^3$ in the annual average ambient concentration would increase the rate by about 0.08%, or 0.05 deaths per year, or one excess premature death per 20 years.

The range of values based on the range of CRFs for PM mortality would be 0.01 to 0.2 per year.

Comment –

The above result demonstrates a weakness of applying the simple CRF analysis to such a small community. The individual risk has been increased in this small community by the same increment as in a large community, but the societal risk (total number of people affected) increases far less in the small community because of the small population. The above result might not be meaningful in communicating the impact of that facility. If its emissions were impacting on a large city, the mortality increment would be 20 times larger for 2 million exposed people, or 1 premature death per year. In this situation,

explaining the change in relative risk without extending the calculation to an estimate of outcomes may be more meaningful.

This case would also need to be evaluated on the basis of other factors, such as excursions above short-term air quality objectives, or the specific health status of people living at receptor locations near the proposed facility. See recommendations for elements of project health risk analysis above in this scenario.

SCENARIO 3: ASSESSING THE HEALTH IMPACTS ON A SMALL NEIGHBOURHOOD OF EMISSIONS FROM AN INDUSTRIAL PLANT THAT IS APPLYING FOR A PERMIT TO MODIFY ITS PROCESS TECHNOLOGY

This scenario is a variant of Scenario 2. It is relevant because of the smaller population size of the potentially impacted group and the implications for estimating numbers of potential morbidity or mortality outcomes. In this case, the same health risk assessment procedure could be followed as in Scenario 2, but the process would stop with the risk estimate itself (i.e., estimating a fraction of a premature death per year in the exposed population is not helpful to the assessment – the risk still increases). This is only possible on the basis of general probability. If combustion particles are involved, it can be argued that increases in PM_{10} can be assumed to have an effect, though not demonstrable in a small population. Reductions in PM_{10} (as by closure of beehive burners for example) could be assumed to reduce the risk of respiratory outcomes on the basis of general probability. A very detailed health study would have to be planned to demonstrate (in Smithers for example) that significant reductions in some health outcomes had been achieved by the reductions in PM_{10}.

In this situation, the Panel does not recommend applying a formulaic estimate of health outcomes using CRFs, as would be more reasonable for a large exposed population. Estimated risks using a CRF approach would be useful inputs, but the individual circumstances of the exposed population would need to taken into account – and the implications of a (presumably) small change in risk of certain health effects explained very carefully to those who would be potentially exposed. Ambient air quality in the neighbourhood relative to other communities and the relative risks implied by the difference may be more meaningful to the affected population.

SCENARIO 4: ASSESSING THE RELATIVE IMPACTS ON HEALTH OF EMISSIONS FROM AN URBAN REGIONAL TRANSPORTATION SYSTEM IN ALTERNATIVE DEVELOPMENT SCENARIOS

See 2.2.5 for the specific issue of diesel vehicle emissions and 2.3.1 for general traffic-related emissions. Estimates can be made of the predicted outcome of changes in PM (PM_{10} or $PM_{2.5}$) derived from diesel traffic or vehicular traffic in general, incorporating an exposure metric based on residential proximity to the road that would be used. Differences in emissions between the alternative development scenarios need to be defined, and those need to be translated into differences in impacts on ambient concentrations as a function of distance from the transportation corridor. The alternatives might comprise, for example, comparison of a rapid transit system with the equivalent

flow of diesel buses that would be displaced. Specific traffic-related concentration-response factors from studies such as those summarized in Appendix 3 could be used. Once the differences in exposure were estimated, the corresponding number of health outcomes for each scenario could be estimated.

Recent studies from New York City (see Lena et al., 2002) illustrate the type of methodology that can be used to estimate exposure. Lena et al. showed that in an area of the South Bronx in New York City, sidewalk concentrations of elemental carbon (as a measure of diesel exhaust particulate) were on average nearly three times as high as at a control monitoring site.

Locally measured sidewalk or residence concentrations of $PM_{2.5}$ (ideally, speciated for markers of diesel exhaust or other traffic-related markers) should be used to estimate exposure. Application of Equation 1 with the basic $PM_{2.5}$ CRFs would provide an estimate of the low end of the range of possible effects, since diesel exhaust particulate has been shown to be more potent than other types of PM. A reliable independent CRF for diesel exhaust PM is not yet available, although as noted earlier, Hoek et al. (2002) have shown a statistically significant association between mortality and living near a major road (with estimated but not reliably quantified PM data). As noted in Chapter 2, the Harvard Six-Cities Study has found a robust association in time series analysis between traffic-related $PM_{2.5}$ and mortality across the study cities (CRF = 3%/10 $\mu g/m^3$, 24-hour average). Note as observed earlier that the latter finding for traffic-related $PM_{2.5}$ is twice the effect of $PM_{2.5}$ generally. Thus, if exposure due to living near a roadway is higher than indicated by the ambient monitoring network, and the potency of traffic-related PM is approximately twice that of general PM, the impact on residents near a busy roadway could be 2-3 times that of the general urban population.

The potential impact on lung cancer incidence can be estimated for the Greater Vancouver region assuming that diesel exhaust is responsible for about 1 $\mu g/m^3$ of $PM_{2.5}$ on average across the region (as has been estimated in studies for GVRD). The California risk factor for diesel particulate matter (DPM) is 300 per million of exposed population per $\mu g/m^3$ of DPM, assuming that the exposure is over a 70-year lifetime. Applying this risk factor to the over-65 population of Greater Vancouver (about 220,000) and assuming that this sub-population has been exposed to 1 $\mu g/m^3$ of DPM for their lives to-date results in an estimated 66 new cases of lung cancer per year ($1 \times 300 \times 10^{-6} \times 220{,}000$) attributable to the exposure to DPM. This is a very rough estimate. This number could be compared with an estimate based on the ACS lung cancer mortality rate CRF for $PM_{2.5}$ from Table 5 (8%/10 $\mu g/m^3$) and the current lung cancer mortality rate for Greater Vancouver of about 1,100/y (assumed to be 50% of the BC total of about 2,200 per year). A 1 $\mu g/m^3$ increment due to DPM would correspond to about 9 additional lung cancer deaths/y relative to a situation in which there were no DPM present ($0.08/10 \times 1{,}100 = 9$). These two results should be considered to represent the range of uncertainty, remembering that one is new cases of lung cancer and the other is incremental lung cancer deaths (the two statistics are about equal currently in BC).

SCENARIO 5: ASSESSING THE COST TO HEALTH CARE SYSTEMS OF DISEASES RELATED TO AIR POLLUTION FOR PLANNING AT THE REGIONAL OR PROVINCIAL LEVEL.

This scenario is far more complicated than the previous applications of the estimation methodology. The use of a simple estimating tool like Equation (1) is only a part of the picture of the impact of air pollutants on health care costs. Impacts of air pollution on public health are an element of the determination of the burden of disease that provincial, state, national and international health economics analyses address. The literature on health economics is extensive and beyond the purview of the Panel's mandate. The issue of how to incorporate welfare (damage) costs in the context of expenditure analysis adds to the complexity of such analysis.

In this situation, the long-term response of populations to air pollution should be the basis for estimating its contribution to health care costs. The objective of this scenario would be to estimate the attributable contribution of air pollution or specific pollutants to mortality or morbidity in the province or state (or region). Concentration-response factors from, for example, the ACS large cohort study would be most relevant (see Table 5 and Pope et al., 2002) in combination with the morbidity CRFs from the AQVM database. Since no thresholds of response to pollutants have been discovered at current ambient levels, the total current number of air pollution-related disease cases could be estimated using such a suite of concentration-response factors. Equation (1) would need to be applied for all communities in which air quality monitoring data were available, since exposure varies from community to community. It should be recognised, however, that the information provided in this report is of limited value in estimating absolute damage to public health – the benefits of incremental improvements in air quality would be more amenable to using the dose-response framework outlined in this report.

Estimates based on Equation (1) and similar algorithms should be made with caution remembering the advice given in Chapter 3 not to rely on the CRF database to estimate absolute impacts. Estimating the avoided health effects of changes in policy and the associated improvement in air quality could be estimated reasonably accurately, but estimating the absolute cost of air pollution to the health care system is fraught with uncertainty. This is especially so because of the predominance of morbidity in the actual system expenditure costs (compared with mortality and other social welfare damage costs).

In principle, this question should not be restricted to direct costs to health care systems, since the overall social costs to the exposed public are what should be considered. This means that CRFs for even 'minor' morbidity and inconvenience factors should be included. The costs of asthma emergency visits (both direct and indirect) as well as medication costs can be computed, as can the costs of hospital admissions for pneumonia or a heart attack. None of these is comprehensive, however, and such outcomes as increased school absences due to respiratory episodes that are associated with ozone levels, are usually not considered, though it should be possible to put a dollar figure on the social disruption that such events must occasion. A critical question is whether disease prevalence (of asthma or of coronary arterial disease) is affected by air pollutants. If such were shown to be the case, the economic implications would be very large.

5. Conclusions and Recommendations

The Panel reached the following conclusions and associated recommendations.

1. Levels of some air pollutants, particularly $PM_{2.5}$ and its wood smoke component, and ozone, in British Columbia are at levels which, on the basis of comparisons with international data, would be predicted to be causing adverse health effects. Since population-level (as compared with individual or panel-level) thresholds for adverse effects have not been shown to exist in the cases of particulate pollution and ozone, current air quality objectives should not be interpreted as bright lines between 'safe' and 'unsafe' levels.

   The field of air pollution management, with its attendant politics, is driven by the demonstrated adverse health effects of a number of pollutants to which people are currently exposed. When proposed developments will increase exposure to pollutants, prospective public health protection requires that, if possible, adequate safety margins are embodied in proposals; this task has been made more difficult by the absence of demonstrated exposure thresholds and by the fact that exposures to a number of air pollutants are already in the range that has been shown to cause adverse health effects. The literature also indicates that health improvements are associated with air quality improvements (from studies of situations in which air quality changed dramatically as a result of substantial changes in emissions over short periods of time).

   Recommendation: It needs to be recognised that any improvement in air quality for PM or ozone would result in fewer negative health impacts. In the Panel's opinion, also required is a stringent approach to proposals that would entail any increase in public exposure levels to these two pollutants.

2. Based on studies carried out in the Lower Fraser Valley, it appears that the increased risk of dying prematurely due to exposure to air pollution is comparable to some common risks, within broad uncertainty. This may also be the case elsewhere in BC.

   For example, using concentration-response factors from studies elsewhere, the daily risk of dying for people 65 and older is increased by about 4% at an ambient PM_{10} level of 50 $\mu g/m^3$ (i.e., a high pollution day) compared with that at 10 $\mu g/m^3$ (i.e., a relatively low pollution day). The estimated uncertainty range of the increased risk is roughly 0.8% to 4.4% (i.e., a factor of 5 lower to a factor of 1.1 higher). Over a long-term exposure, the analogous risk for the over-65 population would be about 4% excess risk of premature death for living in a community with a long-term average PM_{10} concentration of 20 $\mu g/m^3$ compared with one at 10 $\mu g/m^3$.

   Recommendation: Communicating exposure-response information in a risk context is essential. It is important that affected communities understand that risk increases with level of exposure – risk of health effects is very low at the lowest ambient concentrations in BC and increases proportionally to ambient concentrations of PM and ozone.

3. Study size, as defined as the number of outcomes multiplied by years of monitoring data, is a determining factor in deciding whether new, local studies of air pollution impacts on health should be considered. Preliminary data supplied in the report

indicate that outside of the Lower Mainland of BC and the Puget Sound area of Washington, the population of the smaller communities is a limiting factor. This means that many years of monitoring and health data are necessary to provide statistically reliable results for mortality – fewer years for morbidity studies. For example, a reliable study of relationships between air pollution and mortality in a community of 100,000 people might require 10-15 years of data, and perhaps 3-5 years of data for a morbidity outcome such as emergency room visits.

Recommendation: Consider the feasibility of pooling health and monitoring data across a number of communities if new health studies are desirable. Pooling requires careful characterisation of potential differences across communities in exposure and other variables.

4. Studies of farm workers in the Fraser Valley, and of asthmatic schoolchildren in Port Alberni, for example, have provided important assurances that generalisations from studies done elsewhere are reliable. The available local study results can be transferred to similar communities in BC. If pollutant mixes and exposure patterns for a community lacking study data are very different, however, there is no choice but to carry out a community-specific study and provide the necessary exposure monitoring.

   Qualitative estimates of the potential incidence of effects can be made based on available data, but the only way of determining whether current levels in some areas of British Columbia are of major concern is to fund the research needed to investigate possible effects. A major deficiency in reliably estimating air pollution-related health effects in Interior BC communities is the predominance of resource sector emissions in the pollutant mix in these communities, especially wood smoke (including the residential space heating contribution to the latter). At present, these types of sources impact neither the major BC coastal cities nor most of the US and European cities for which much of the work on air pollution effects has been conducted. Kelowna is probably the only major BC Interior city that is not significantly affected by wood smoke and thus has a comparable source mix to the Lower Mainland and Victoria. Communities in northeast BC are impacted by emissions from both oil & gas and forest products sectors and are likely to have unique air pollution exposure patterns requiring special study.

   Recommendation: Carry out community-specific health studies where comparison with results from similar communities is not feasible. Adequate exposure monitoring would need to be provided. A specific example of such a study that has a good chance of providing reliable information is a pooled study of hospital admissions (or other effects) in Interior BC communities that are currently monitored for PM_{10} (preferably adding $PM_{2.5}$ monitoring where feasible). Comparison of communities with significant wood smoke exposure with similar communities with low wood smoke exposure would be valuable. The Candidate communities are Fort St. John, Quesnel, Houston, Williams Lake and Prince George for the wood smoke-impacted group (pooled population: 117,000). Communities in the Kootenay region (e.g., Cranbrook and Nelson) might also be included. The Fort St. John-Taylor-Chetwynd area with its oil & gas component may also be a candidate for a special effects study, although the population is

small and spread out, so that the likelihood of successfully finding statistically significant results is small.

5. Growing evidence of traffic-related impacts in urban areas suggests that the proximity of these emissions to populated areas causes high exposures relative to typical ambient monitoring sites – and associated health effects. Traffic-related PM may be more potent in observed health effects than general PM, and concentrations of PM near roadways may be considerably higher than at locations away from roadways. The combined effect of potency and concentration might increase risk of effects in residents living near a roadway by 1.5-2 times that of the general population (based on European studies).

   Recommendation: Careful study of the results of traffic studies elsewhere should be undertaken to determine likely impacts in BC's major urban centres. Expanded exposure monitoring for PM (and its components) along roadways, such as is currently being conducted in Vancouver in a limited way, is necessary to determine whether to expect similar effects impacts as have been observed elsewhere (in US and European cities). The impacts can be quantified using available concentration-response data.

6. Continuing economic estimates of the costs of current levels of air pollution, both direct to the health care system, and indirect to society as a whole, are required.

   Recommendation: The concentration-response factor databases recommended in this report can be used in conjunction with local monitoring data as the basis for providing a preliminary estimate of air pollution-related disease outcomes in any such economic analysis. The benefits of reducing pollution levels by specified amounts should be estimated, rather than the absolute total impact values.

7. Proposed new facilities that have significant emissions known to cause adverse health effects require critical health risk assessments before approval. The examination of such assessments should be, in the Panel's opinion, in the public domain.

   Recommendation: Include specific health risk analysis in all major project assessments. Such analysis should take into account the specific demographics and health status of exposed populations and should apply the effects estimation methodology outlined in this report where feasible using estimated population-weighted incremental exposure.

8. Concentration-response factors for PM_{10} (or $PM_{2.5}$) exposure and mortality and lung cancer are recommended by the Panel for use in BC and the PNW – with respect to urban populations. The Panel's preferences are provided for short-term and long-term exposures. For these populations, the morbidity concentration-response factors from established Canadian databases, such as those in the Air Quality Valuation Model (AQVM) or Illness Costs of Air Pollution (ICAP) model, are acceptable as starting points.

   Recommendation: Since the available BC and PNW mortality and morbidity studies are quite limited, the feasibility of carrying out meta-analysis to compile a region-specific concentration-response factor database for application to this region is questionable. As the most practical approach, the Panel recommends

using the most up-to-date version of the AQVM CRF database for morbidity CRFs.

Recommendation: Carry out screening-level analysis of potential health effects impacts in smaller BC communities using the data produced from the analysis of the available local and other relevant studies (summarised in the report) to estimate the possible incidence of air pollution-related impacts. This will help in assessing priorities for mitigation in impacted airsheds and for identifying areas where health risk studies might be considered.

Recommendation: Where reliable health study data are not available for a smaller community, ambient air quality can be used as a reasonable measure of relative risk of health effects in that community in comparison with similar communities in which observations have been made – or even in comparison with larger communities with appropriate recognition of the potential uncertainty in such comparisons.

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Part I: Methods and Methodologic Issues (Authors: J. M. Samet, F. Dominici, S. L Zeger, J. Schwartz & D.W. Dockery)

Part II: Morbidity, Mortality and Air Pollution in the United States (Authors: J.M. Samet, S.L. Zeger, F. Dominici, F. Curriero, I. Coursac, D.W. Dockery, J. Schwartz & A. Zanobetti)

Revised results as of summer 2002 may be found on the HEI website at: http://www.healtheffects.org/news.htm#NMMAPS and at the principal investigators' website: http://biosun01.biostat.jhsph.edu/~fdominic/research.html.

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APPENDICES

Glossary

Term	Meaning
ACS	American Cancer Society
allergen	substance that induces an allergic response
APHEA	*Air Pollution and Health: European Approach*, a study sponsored by the European Commission
atopy	hereditary disposition to disease
AQVM	Air Quality Valuation Model (Health Canada)
banded neutrophils	Describing the appearance of certain cells that have been stained for examination under a microscope; may originate in blood, bone marrow, etc.
BHR	bronchial hyperresponsiveness
bronchiolitis	Inflammation of the bronchioles (intermediate airways in the lung)
Bronchodilator	Any drug or instrument used to increase the diameter of the pulmonary air passages
cerebrovascular	pertaining to the blood supply to the brain
CI	Statistical confidence interval, typically 95% unless otherwise noted; a 95% CI would indicate that the probability of the true result occurring in the range by random chance is less than 5%
COPD	chronic obstructive pulmonary disease
CVD	cardio-vascular disease
cytokines	hormone-like substances produced by cells of the immune system
EC	elemental carbon
ecologic study	In context, epidemiological study that uses overall population ambient exposure and disease response statistics rather than individual characteristics. See footnote 2.
eNO	Exhaled Nitric Oxide; can be evaluated as an indicator of inflammation of the lungs
ETS	environmental tobacco smoke; sidestream smoke inhaled by non-smokers rather than the mainstream smoke inhaled by a smoker
FEV1	forced expiratory volume in one second (in a lung function test)
fibrinogen	A soluble blood plasma protein that combines with thrombin to form fibrin, an insoluble protein critical to the blood-clotting process
FVC	forced vital capacity (total expiratory volume in a lung function test)
ICAP	Illness Costs of Air Pollution model (Ontario Medical Association)
leukocyte cell	A type of blood cell
morbidity	illness, disease state
mortality	death

Term	Meaning
myocardial ischemia	Diminution of blood supply to the myocardium (the muscular tissue of the heart) due to obstruction or constriction of the inflow of arterial blood
NMMAPS	*National Morbidity, Mortality and Air Pollution Study* – carried out for the Health Effects Institute by Johns Hopkins University for 90 US urban areas
OC	organic carbon
OR	odds ratio; statistical probability of outcome relative to a control
PEFR	peak expiratory flow rate (in a lung function test)
RAD	restricted activity day; day on which illness causes less than normal level of personal activity (absence from work or school, etc.)
RR	risk ratio or relative risk of an outcome (of a test population compared with control population)
tachycardia	Excessive rapidity of the heart's action

Appendix 1: Terms of reference (partial)
Health and Air Quality 2002 - Phase 1

Background:

Our understanding of the relationship between air pollution and human health is rapidly changing. The rapidly changing research in this field requires periodic updates if policy makers, air quality managers and front-line public health staff are to incorporate the best 'state of the science ' evidence in making decisions and communicating with the public on air quality issues. There is a need to provide the foundation for near-term application of recommended methods to assess the relationship between air pollution and human health in the context of BC communities, as well as development of plans to address gaps (eg. local data or research) preventing application of optimal methods. This need extends to international jurisdictions which may share a B.C. airshed, such as Whatcom County/Puget Sound.

Environment Canada, Pacific and Yukon Region, is currently undertaking a range of benefit cost analyses to determine an 'optimal' air quality standard for the GVRD and the lower Fraser Valley. This project will rely on the Air Quality Valuation Model for much of the benefit analysis with the recognition that more recent information on health effects of air pollution may be available to refine the benefit estimates. Benefit and cost analysis of emission reductions are also planned for other regions of the province.

Health effects are the prime drivers of economic benefits. In turn, health benefits are driven by concentration/response relationship, meaning the total benefits are very sensitive to the equations chosen to quantify these relationships. A wide scope of studies provides evidence on the nature and impact of the concentration/response relationships.

The overall project is intended to provide the foundation for near-term applications of recommended risk assessment methods which could be used to estimate the impacts of air pollution on human health in British Columbia and international portions of shared airsheds, as well as the development of plans to address gaps (e.g. local data or research) preventing application of optimal methods. This proposal for "Phase 1" is intended to provide a framework or context within which previous bodies of work can be viewed in relation to each other (e.g. Vedal report of 1994, GVRD AQ cost/benefit work, recent PM and ozone science assessment work, methods used for evaluation of the ramifications of Sumas 2 power proposal, and the recently completed Brauer report). It is intended to describe the range of methods currently available, and provide guidance on appropriate methods and data needs for different population and geographic scales– regional populations of 1-2 million, cities of 100,000, communities of 10,000, and the project/neighbourhood scale, including recommendations for methods to be used in the context currently available data in BC, and recommendations for improving underlying data to support application of better methods in future. An anticipated (but not yet funded or guaranteed) Phase 2 project would extend this study to the Washington portion of the Lower Fraser Valley Airshed, and apply one (or some) of these methods to a community or communities as described above.

STATEMENT OF WORK

Without limiting the general scope of work, the selected consultant shall carry out all work as outlined in the general steps below. The proponent is invited to identify alternative approaches or additional steps in completing this project.

1. Provide an up to date summary of the research literature on the relationship between common air pollutants (e.g. NOx, SOx, CO, O_3, PM (10, 2.5, other)) and effects on human health, both individually or in mixtures. The health outcomes of interest include mortality as well as the full spectrum of morbidity. This summary must include, but not limited to:

   - An emphasis on evidence for effects of air pollutants at or reasonably close to the ambient concentrations currently encountered in British Columbia. As well, the summary should provide details on the sources or mixture of air pollutants in various studies and advice on which studies (or set of studies) may be most relevant to estimating health impacts in BC communities (or subsets of BC communities).

   - A discussion of areas where there is general agreement in the scientific community as well as areas where there are differences in the interpretation of existing studies. Key areas of uncertainty in the research literature should be described, including the apparent opinion among some researchers that there are limits to how much further the ambient concentrations of pollutants, such as ozone, can be reduced.

   - An examination, in a BC context, of the conclusion of the expert panel of the Royal Society that the weight of evidence is that cohort studies such as those by Pope et al. (1995), Dockery et al. (1993) and Abbey et al.(1999) are the most relevant for a full accounting of the long-term cumulative effect of exposure. Any more recent studies providing evidence for or against the use of cohort studies (as a basis for developing concentrations response relationships) should also be reviewed.

   - An assessment, given the above, of which type of studies provide the most reliable bases for developing concentration/response relationships in BC communities.

   - An examination of the conclusion of the above-mentioned expert panel that there is some sensitivity in statistical response to socio-economic characteristics of the affected population. These findings should be assessed, and their relevance to BC ascertained, given the composition of the provincial populations.

   - An assessment of the importance of the sulphur content of particulates in the BC context, based on current knowledge of local particulate chemistry.

   - An assessment of the relative importance of $PM_{2.5}$ versus PM10 in the concentration response relationship, including advice on which of these parameters is the best indicator of health risks in BC.

- An examination of any recent literature on the relationship between mortality statistics and life expectancy, particularly with respect to the so-called harvesting effect. This is to include a recommendation of how the life expectancy results should be incorporated into benefit and risk analysis.

- A review of recent literature on thresholds for health effects, providing advice on quantification of the magnitude and uncertainty of response effects moving along the lower levels of the concentration gradient.

- A review of recent findings on the effects of air pollution on birth defects and health of newborns and advice on the applicability of these results in the BC context.

2. A review of risk assessment methods that have been or could be used to estimate the impacts of air pollution on human health in British Columbia. This review must include, but not limited to:

    - A spectrum from generalizing from published studies to use of locally specific data. Methods based on generalizing the results of published studies to other communities (e.g., using dose-response relationships, or relating ambient concentration)changes to changes in risk for various health outcomes) should be discussed in the context of other risk assessments that are used to guide environmental decision making.

    - A discussion and advice on:
        1) Parameters where values from the scientific literature can be applied to specific BC communities or groups of communities, and
        2) Parameters where provincial, regional, or local data should be used.

    - Comments on the strengths and weaknesses of the methods above and recommendations on which methods would be useful in estimating the health impacts of air pollution in British Columbia, based on current and likely future data availability.

    - A discussion on which methods are best suited to estimating impacts at the provincial, regional, city, town, or neighbourhood level, and what level of uncertainty should be expected at each scale. This is to include a discussion on which methods would be useful in estimating health impacts of air pollution in British Columbia from specific sources, such as power plants, transportation, and wood burning. It is also to include a discussion and methodology to address the effect of actual exposure (e.g. time spent indoors, in a vehicle, etc.)

    - An identification of gaps in health or environmental data that are important sources of uncertainty in estimating health impacts of air pollution along with recommendations on how they can be addressed and reduced, identifying differences appropriate to different population sizes and geographic scales.

- Practical advice on when and how to apply risk assessment methods to estimating mortality and morbidity from air pollution, identifying differences appropriate to different population sizes and geographic scales.

3. A report, complete with an Executive Summary, of the above. The primary audiences of the report are the medical health officers, other medical practitioners, and air quality managers at the provincial and local/regional level. The secondary audiences would be public service executives (especially those in the Environmental and Health fields), elected representatives (local and provincial), interested stakeholders (e.g. business and environmental groups), and the general public.

The funding agencies will arrange for a peer review of a draft version of the document. Nominations for peer reviewers will be solicited from inside and outside the funding agencies. Peer reviewers comments will be provided to the authors prior to completion of the penultimate draft version. The penultimate draft version of the report will be circulated to external health and environment stakeholders. This may include but is not limited to Health Officers' Council, Lower Fraser Valley Air Quality Advisory Committee, the NWAPA Advisory Council, BC and Washington Lung Associations, David Suzuki Foundation, Sierra Legal Defence Fund, and the Heart and Stroke Foundation. These comments will be provided to the authors prior to completion of the final report.

Appendix 2: List of questions (final revision):

A. General Statement:

The outcome will be an up to date review/summary of literature which air quality managers could use to estimate health effects (morbidity/mortality) in British Columbia and the Pacific Northwest of the common air pollutants (e.g. NOx, SOx, CO, O3, PM (2.5, 10) individually or as a mixture.

1. What are the lines of evidence demonstrating health effects from air pollutants at or close to ambient concentrations encountered in BC or in the Pacific Northwest from different sources such as wood burning, industrial or general transportation? Which among these studies can be generalized in BC communities? Which effects on health can be attributed to individual pollutants or groups of pollutants listed above?

2. How strong is the evidence of health effects from air pollutants at various concentrations? What is the lowest concentration at which we have a strong evidence of health effects from different air pollutants? What are the weaknesses and strengths of time series vs. cohort studies in estimating health impacts from air pollution within the BC context?

3. Is there any evidence that socio-economic status affects the risk to developing health effects from air pollution? Is this evidence relevant to or applicable in the BC context?

4. What is the importance of SOx alone or as a precursor to particle sulphate formation, in producing health effects in BC relative to other air pollutants? Is there any evidence that reducing SOx levels in BC will result in reduction of health effects?

5. What is the relative importance of size fraction, chemical composition, and source (e.g., diesel particles, wood smoke) of PM in the concentration response relationship? Which are the appropriate indicators or measures of PM related health risk in BC?

6. Is there any harvesting effect evident in studies on air pollution and mortality? Is this effect the same or different for different causes of death? What is the best estimate of how much reduction in life-expectancy results from air pollution related diseases?

7. Are there any studies that link air pollution to birth defects or the health of newborns? How strong is the evidence (i.e., are these outcomes accepted as being caused by or attributable to air pollution)? How applicable is this evidence to BC?

8. Who are the vulnerable populations at risk from health effects of air pollutants? Can the effects be generalized to different vulnerable populations? What are the limitations of applying generalized health impacts to various populations?

9. What is the evidence respecting co-pollutants acting as surrogates for PM exposure?

B. General statement:

What are the health impact estimation methods that should be used to estimate the impacts of air pollution on human health in British Columbia and the Pacific Northwest? Impact assessment methods include those that involve direct measurement of health outcomes in exposed populations (e.g. epidemiologic studies) and in direct estimation (e.g. using concentration effect estimates found in one community and applying them in another community).

1. What are the strengths and weaknesses of the different air pollution-health effects methods? Which among these methods should be used to estimate health impacts of air pollution in BC?

2. What is the best method to generalize results from studies in one community to another on concentration-effect relationships or to relate ambient concentrations to changes in risks for various health outcomes?

3. a. What quantitative relationships between air pollutants and health outcomes from the scientific literature can be applied to BC communities or subsets of BC communities?

   b. When should local data be used in lieu of values from the scientific literature? When is it appropriate to use local, regional or provincial level data?

4. Which among the health effects estimation methods should be used to provide appropriate estimates of health impacts from air pollution from different sources (e.g., power plants, transportation and wood burning) in BC? What is the best methodology to estimate the effect of actual exposure (e.g., morbidity from cardiovascular diseases, mortality from respiratory diseases, hospitalization, exacerbations of asthma)?

5. What are the gaps in health environmental data that are significant sources of uncertainty in estimating health impacts of air pollution in BC? Identify the sources, and how to address and reduce these uncertainties within the BC context. Can we reduce the uncertainty in estimates of air pollution related health effects in BC by using relationships found in BC

rather than those from the general literature? How much uncertainty is associated with estimates made using different sources of data?

6. When is it appropriate, when is it not appropriate to apply certain estimation methods (i.e., what are the limitations) and how do we best apply these methods to estimate mortality and morbidity from air pollution related diseases to different populations sizes and geographical scales (e.g., are the same methods appropriate for provincial, regional, airshed, community and neighbourhood level estimate of risks)? Can these methods be applied to estimating impacts from specific sources, a class of sources or only to overall ambient levels of air pollution?

7. What is the best estimation method to use to determine the short-term exposure to high levels of air pollution? What time period is most appropriate for estimating the various health impacts of air pollution? (e.g., daily, weekly, monthly, annually)?

Appendix 3: Traffic-related impacts

Summary of studies: literature regarding health effects of traffic-related air pollution (Appendix 3 references at end of table)

Asthma				
Location	Exposure	Outcome	Finding	Reference
Birmingham, UK	Living near major road, traffic density	Asthma hospital admissions, children >5 yrs	+	(Edwards et al. 1994)
Sweden	Estimated NO2 exposure (dispersion model)	Wheezy bronchitis hospital admissions, children 4-48 months	+ for girls only	(Pershagen et al. 1995)
UK	Presence of motorway in electoral ward	MD diagnosed asthma, 13-14 yrs	-	(Waldron et al. 1995)
London	Distance to nearest road/major road, traffic volume within 150m	Asthma hospital admissions, 5-14 yrs	-	(Wilkinson et al. 1999)
Dresden	Measured NO2, SO2, CO, O3, benzene at 200 gridpoints	Asthmatic symptoms, 5-7, 9-11 yrs (ISAAC)	+ cough, bronchitis -atopy, bronchial hyperresponsiveness	(Hirsch et al. 1999)
California	Traffic flow in 550 ft buffer	Asthma diagnosis, asthma medical visits	- asthma diagnosis +medical visits for asthmatics	(English et al. 1999)
Nottingham	Traffic flow in 1 km2 grids	Wheeze prevalence in children 4-16 yrs	-	(Venn et al. 2000)
Atlanta	Pre/post/during traffic reduction measures associated with 1996 Olympics	Asthma acute care visits and hospitalizations for children 1-16 yrs	22% decrease in peak weekday morning traffic counts; 11 - 44% decrease in asthma acute care events	(Friedman et al. 2001)

New York State	Distance to major State route, % of trucks within 200 / 500m buffer of home, VMT within 200m / 500m buffer of home	Asthma hospitalization 0-14 years	+ High VMT tertile within 200m of home + High % of trucks within 200m of home	(Lin et al. 2002)
Toronto	GIS-based exposure model – PM2.5 based on traffic volumes, distance, vehicle types, emissions factors	asthma, bronchitis, COPD, pneumonia, URTI hospital admissions	+	(Buckeridge et al. 2002)
The Netherlands	For schools within 300m of	Respiratory symptoms	+	(Oosterlee et al. 1996)
The Netherlands		Respiratory symptoms		(van Vliet et al. 1997)
The Netherlands		Lung Function		(Brunekreef et al. 1997)
The Netherlands	GIS-based individual estimates of NO2, PM2.5, “soot”	Asthma, Respiratory and allergic symptoms, Respiratory infections	+ Asthma (2 years) + Respiratory Infections	(Brauer et al. 2002)
Munich	GIS-based individual estimates of NO2, PM2.5, “soot”			(Gehring et al. 2002)

Mortality				
Location	Exposure	Outcome	Finding	Reference
Amsterdam	Time series stratified by proximity to major road	Mortality		(Roemer and van Wijnen 2001)
6 US Cities	Prospective Cohort – PM stratified by source factors	Mortality	10 µg/m3 increase in PM2.5: + Mobile sources: 3.4% increase in daily mortality [95% confidence interval (CI), 1.7-5.2%], + Coal combustion: 1.1% increase [CI, 0.3-2.0%). - Crustal particles	(Laden et al. 2000)
Switzerland, Austria, France	Time series/Impact Assessment Estimated contribution of motor vehicles to ambient PM concentrations	Mortality		(Kunzli et al. 2000)
The Netherlands	Regional, Urban and Traffic (within 50m of major road, 100m of freeway) components of exposure	Prospective cohort - mortality	+ traffic component had higher OR than urban air or regional background	(Hoek et al. 2002)
16 U.S cities (NMMAPS)	Percentage of PM emissions from highway diesel vehicles	Regression of PM:hospitalization coefficients on external data (traffic, etc.)		(Janssen et al. 2002)

Cancer				
Location	Exposure	Outcome	Finding	Reference
Stockholm	Dispersion models estimates of NO2 (traffic) and SO2 (residential heating)	Lung Cancer	+ for NO2	(Bellander et al. 2001); (Nyberg et al. 2000)
Denmark	Dispersion model estimates of NO2 and benzene	Leukemia, CNS tumors, lymphomas, all cancers	+ for lymphoma - for all other outcomes	(Raaschou-Nielsen et al. 2001)

Birth outcomes				
Location	Exposure	Outcome	Finding	Reference
Los Angeles	CO, PM10, NO2, O3 concentrations during pregnancy (restricted to population within 2-mile radius of monitoring site)	Preterm birth	+ PM10 + CO	(Ritz et al. 2000)
Los Angeles	CO concentrations during pregnancy (restricted to population within 2-mile radius of monitoring site)	Low birth weight	+	(Ritz and Yu 1999)
Los Angeles	Distance weighted traffic density	Low birth weight, preterm birth	+ preterm birth	Wilhem and Ritz, 2003

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Appendix 4: Summary of health effects literature for wood smoke

A number of epidemiological studies have evaluated respiratory symptoms and/or lung function in children of North American communities where wood burning is prevalent. In these communities, elevated levels of ambient air pollution are seasonal (3-8 months depending upon the climate) and variable. PM_{10} concentrations as high as 800 $\mu g/m^3$ have been measured in these communities, although peak levels (24-hour averages) of 200 – 400 $\mu g/m^3$ are more common (Heumann, Foster, et al., 1991) (Larson and Koenig, 1994) (Vedal, 1993). As wood smoke is generally emitted outdoors and since people spend most of their time indoors, indoor penetration is an important variable for exposure assessment. It is estimated that approximately 70% of outdoor wood smoke particulate penetrates indoors (Anuszewski et al., 1998).

Several early studies focused on the presence of a wood burning stove in the home as a risk factor since wood stoves, especially older varieties, can emit smoke directly into the home (Larson and Koenig, 1994). While these earlier studies strongly suggest that there are adverse impacts associated with wood smoke exposure, their crude exposure assessment precludes more specific conclusions. These studies suggest associations between wood stove use and increased risk of respiratory illness and increased respiratory symptom reporting in children (Dockery, Spengler, et al., 1987) (Honicky, Akpom, et al., 1983) (Honicky, Osborne, et al., 1985) (Tuthill, 1984) (Butterfield, LaCava, et al., 1989). In a significant improvement from these earlier studies, indoor particulate levels were measured in a case-control study of 45, 1-24 month old children hospitalized with an acute lower respiratory illness (ALRI). Matched pair analysis revealed an increased risk of ALRI for children living in households that cooked with any wood or had indoor particle concentrations above 65 $\mu g/m^3$. The indoor particle concentration was positively correlated with cooking and heating with wood but not with other sources of combustion emissions (Robin, 1996). In the only study to date to evaluate impacts of wood burning on adult asthma, Ostro and colleagues measured symptoms in a panel of 164 asthmatics and found exposure to indoor combustion sources, including wood stoves, to be associated with increased asthma exacerbation (Ostro, Lipsett, et al., 1994).

Several other studies have evaluated health outcomes in communities where wood smoke is a major source of ambient particulate. Heumann studied lung function of 410 children in high and low exposure areas of Oregon where wood smoke accounts for as much as 80% of the winter period particulate. PM_{10} ranged from approximately 50 – 250 $\mu g/m^3$ in the high exposure area and 20 – 75 $\mu g/m^3$ in the low exposure area. Lung function decreased during the wood burning season for the children in the high exposure area, but not in the low exposure area (Heumann and Foster, et al., 1991). Two studies in Montana associated acute changes in lung function in a study of 375 children with increased levels of particulates. 24-hour averages ranged from 43-80 $\mu g/m^3$ and 14-38 $\mu g/m^3$ for PM_{10} and $PM_{2.5}$, respectively (Johnson, Gideon, et al., 1990), of which approximately 68% of the particulate was attributed to wood smoke (Koenig et al., 1993).

A questionnaire study of respiratory symptoms compared residents of high (mean $PM_{2.5}$ of 55 $\mu g/m^3$) and low (33 $\mu g/m^3$) wood smoke pollution areas of Seattle. Although, no significant differences were observed between the high and low exposure areas when all age groups were combined, there were statistically significant higher levels of congestion and wheezing in 1-5 year olds from the high pollution area. This finding supports those

of other studies which suggest that young children are particularly susceptible to adverse effects of wood smoke (Browning, Koenig et al., 1990).

A more comprehensive study in the same high exposure Seattle area (where 80% of the particulates are from wood smoke) (Larson and Koenig, 1994; Norris et al., 1999) measured significant lung function decrements in the asthmatic subjects, in association with increased wood smoke exposure. The highest (night time 12-hour average) $PM_{2.5}$ level measured during the study period was approximately 195 $\mu g/m^3$ (Koenig, Larson, et al., 1993). A companion study found a significant association between PM_{10} levels and asthma emergency room visits throughout Seattle (Schwartz and Slater, et al., 1993). The mean PM_{10} level during the 1-year study period was 30 $\mu g/m^3$. At this concentration, PM_{10} appeared to be responsible for 12% of the asthma emergency room visits. The authors indicate that on an annual basis 60% of the fine particle mass in Seattle residential neighbourhoods is from wood burning. A recent study in Santa Clara County, California, an area where wood smoke accounts for approximately 45% of winter PM_{10}, demonstrated an association between wintertime PM_{10}, increased daily mortality and exacerbations of asthma (Lipsett, Hurley, et al., 1997).

In one of the few studies of air pollution from agricultural burning, 428 subjects with airways obstruction were surveyed for their respiratory symptoms during a 2-week period of exposure to straw and stubble combustion products. During the exposure period, 24-hour average PM_{10} levels increased from 15-40 $\mu g/m^3$ to 80-110 $\mu g/m^3$. 1-hour level of carbon monoxide and nitrogen dioxide reached 11 ppm and 110 ppb, respectively. Total volatile organic compound levels increased from 30-100 $\mu g/m^3$ before the episode to 100-460 $\mu g/m^3$ during the episode. While 37% of subjects were not bothered by smoke at all, 42% reported that symptoms (cough, wheezing, chest tightness, shortness of breath) developed or became worse due to the air pollution episode and 20% reported that they had breathing trouble. Subjects with asthma and chronic bronchitis were more likely affected (Long, Tate, et al., 1998). This study indicates that other forms of biomass air pollution, in addition to wood smoke, are associated with dome degree of impairment, and suggests that individuals with pre-existing respiratory disease are particularly susceptible.

FOREST FIRES, BRUSH FIRES, AGRICULTURAL BURNING

Several studies have also evaluated the health impacts associated with forest and bush fires. Duclos and colleagues evaluated the impact of a number of large forest fires in California on emergency room visits (Duclos, Sanderson, et al., 1990). During the approximately 2½-week period of the fires, asthma and chronic obstructive pulmonary disease visits increased by 40 and 30%, respectively. PM_{10} concentrations as high as 237 $\mu g/m^3$ were measured. During 1994, bush fires near Sydney, Australia led to elevated PM_{10} levels (maximum hourly values of approximately 250 $\mu g/m^3$) for a 7-day period; ozone levels were not elevated. Two studies of asthma emergency room visits during the bushfire smoke episode failed to detect any association with air pollution (Copper, Mira, et al., 1994, Smith, Jalaludin, et al., 1996). These results appear to conflict with results of studies conducted in North America. Possible reasons are differences in study design and

sample size as well as differences in chemical composition of the particulates and differences in the relative toxicity of the specific particle mixture.

An analysis was conducted of emergency room visits for asthma in Singapore during the 1994 "haze" episode in which forest fire emissions from neighbouring Indonesia impacted the Singapore urban area (Chew, Ooi, et al., 1995) indicates an association between PM_{10} and emergency room visits for childhood asthma. During the "haze" period, mean PM_{10} levels were 20% higher than the annual average. Reports from surveillance monitoring activities conducted during the 1997 and 1998 Southeast Asian haze episode also indicated effects of health care utilization. In Singapore, for example, there was a 30% increase in hospital attendance for "haze-related" illnesses and a time series analysis indicated a PM_{10} increase of 100 ug/m^3 was associated with 12%, 19% and 26% increases in cases of upper respiratory tract illness, asthma and rhinitis, respectively. This analysis did not observe any significant increases in hospital admissions or mortality (World Health Organization, 1998). Similar findings were also observed in Malaysia (Brauer and Hisham-Hashim, 1998, Leech, Burnett, et al., 1998). Preliminary results from an on-going study of the 107 Kuala Lumpur school children conducted found statistically significant decreases in lung function between pre-episode measurements in June-July 1996 and measurements conducted during the episode in September 1997 (Hisham-Hashim, Hashim, et al., 1998). These preliminary results suggest a measurable impact of the 1997 episode on the respiratory function of children. Only one study has evaluated the impacts of air pollution from vegetation fires on mortality. Shastry evaluated the population health effects in Malaysia of air pollution generated by a widespread series of fires that occurred mainly in Indonesia between April and November of 1997. The results show that the smoke haze from these fires had a deleterious effect on population health in Malaysia and were in general agreement with the mortality impacts associated with particles in urban air (Shastry 2000).The findings from this study complement those of studies conducted in areas where wood smoke is a major contributor to ambient particles.

A recent report of the impact of bush fires near Darwin, AU (Johnston et al., 2002) noted that in Australia one study for a link between bushfires and asthma was positive, but two others were negative; and one further study found no link between PEFR levels in children and a bushfire episode. The authors noted the limitation of all these studies that "they are necessarily based on post-hoc comparisons of asthma presentation rates after an unexpected fire event compared with an "equivalent" historical period". Continuous air monitoring in Darwin and comparison of presentations for asthma at the emergency department of the Royal Darwin Hospital form the basis for the Johnston et al. study conducted in the dry season from April 1 to October 31, 2000. After adjustments for weekly rates of influenza, there was a significant increase in asthma presentations with each 10 $\mu g/m^3$ increase in PM_{10}. Strongest effect was seen on days when PM_{10} was above 40 $\mu g/m^3$ compared to days when the level was less than 10. Total number of asthma presentations was 265, averaging 1.2 per day. Highest single day was 6 cases, and the PM_{10} was recorded as about 70 $\mu g/m^3$ on that day. Darwin has no significant source of atmospheric air pollution other than bushfires.

AGRICULTURAL BURNING

In one of the few studies of air pollution from agricultural burning, 428 subjects with airways obstruction were surveyed for their respiratory symptoms during a 2-week period of exposure to straw and stubble combustion products. During the exposure period, 24-hour average particle (PM_{10}) levels increased from 15-40 $\mu g/m^3$ to 80-110 $\mu g/m^3$. 1-hour level of carbon monoxide and nitrogen dioxide reached 11 ppm and 110 ppb, respectively. Total volatile organic compound levels increased from 30-100 $\mu g/m^3$ before the episode to 100-460 $\mu g/m^3$ during the episode. While 37% of subjects were not bothered by smoke at all, 42% reported that symptoms (cough, wheezing, chest tightness, shortness of breath) developed or became worse due to the air pollution episode and 20% reported that they had breathing trouble. Subjects with asthma and chronic bronchitis were more likely affected (Long, Tate, et al., 1998).

The association between asthma hospital admissions and the burning of rice field stubble and waste rice straw was examined in Butte County California over a 9-year period (Jacobs, Kreutzer et al. 1997). Although burning was not associated with any measurements of major air pollutants, burn acreage was significantly associated with an increased risk of asthma hospitalization. This association also showed a dose-response relationship. Rice stubble burning and the relationship with asthma was also studied in Nigata prefecture, Japan (Torigoe, Hasegawa et al. 2000). In this study measured particle concentrations were associated with asthma hospital admissions in a region where rice straw burning emissions lead to high particle concentrations during the September – October burning season. In addition, the investigators reported a significantly higher number of emergency room visits for asthma on days when rice straw burning occurred.

Cancado and colleagues examined the relationship between particulate matter components and pediatric respiratory hospital admissions in a region of Brazil where sugar cane cultivation is common (Cancado, Lara et al. 2002). Analyses were performed during both burning and non-burning seasons. The main particle components were potassium and black carbon which are both known to be generated during biomass combustion. The investigators report significantly increased risks of pediatric respiratory hospital admissions associated with the concentrations of both of these particle components. These risks were 3 times greater in the burning season relative to the non-burn season, suggesting the importance of sugar cane combustion in this association. In previous work in Brazil indirect measurements (sedimentation of particle mass) of air pollution during the sugar cane burning season were associated with the number of patients visiting hospitals for inhalation therapy for acute respiratory distress (Arbex, Bohm et al. 2000). This association displayed a dose-response relationship.

Appendix 5: Comparison of Christchurch, NZ PM_{10}-Hospitalization Data and Hospitalization Data for BC's Central Interior.

Data	Christchurch, NZ	Interior Cities, BC
Population (approx.)	333,000	117,000
Hospitals	1	14
PM_{10}		
Annual mean	25.17	20.00
Interquartile	14.8*	11.7 days/y >50 μg/m^3 (24-h)
Hospital admissions**		
Cardiac/day	6.84	4.75
Respiratory/day	10.17	5.95

* Occasional spikes >50

** Same ICD codes used for both datasets; all age groups except newborns included.

POPULATIONS –	Fort St. John	15,191
	Quesnel	8,588
	Houston	3,936
	Williams Lake	11,398
	Prince George	77,996

		117,102

Population Data from reference (2).

Christchurch Regressions: % increase per IQ of PM_{10} (14.8 μg/m^3)

Pneumonia/flu	5.32
Acute Respiratory infections	4.53
All Respiratory admissions	3.37
Cardiac conditions	1.36

Christchurch Data from reference (1).

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Particulate air pollution and hospital admissions in Christchurch, New Zealand
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2) *British Columbia Approved Accommodation 2002 Guide*: Tourism British Columbia, 2002 (for population data).

Appendix 6: PM_{10} Levels in British Columbia Airsheds (1998-2000)

Source:Air QualityManagement in British Columbia: State-of-Knowledge Report, 2002

Airshed Characterisation Based on Ambient PM_{10} Concentrations

The following analysis is based on airshed boundaries defined by regional air quality meteorologists, and ambient PM_{10} data collected between 1998-2000. For the purposes of this analysis, airsheds were classified on the following basis:

- **DEGRADED AIRSHED:** 3.6 (1% of the time) or more annual exceedances of daily mean PM_{10} concentrations of 50 $\mu g/m^3$
- **THREATENED AIRSHED:** Up to an average of 3.6 (or up to 1% of the time) mean annual number of exceedances of daily mean PM10 concentrations of 50 $\mu g/m^3$.
- **UNTHREATENED AIRSHED:** no mean annual number of exceedances of daily mean PM_{10} concentrations of 50 $\mu g/m^3$.
- **All Other Airsheds: Insufficient Data**

Within airshed types, airsheds were further ranked on the basis of annual mean 24-hr PM_{10} concentration.

Table headings:
Mean – annual mean averaged over 3 years (1998-2000). Units are in $\mu g/m^3$
Exposure/yr – Sum of daily mean concentrations above 0 $\mu g/m^3$, averaged over 3 years (1998-2000).
n>50 – number of exceedances of provincial air quality objective of 50$\mu g/m^3$ (24-hour average), average over 3 years (1998-2000).
Normalised # exceed – number of exceedances normalised over an entire year.
%exc50 – percentage frequency of exceedances of the provincial air quality objective of 50 $\mu g/m^3$, calculated as n>50 divided by # of 24-hr samples.

Airshed	# 24hr Samples	mean	Exposure /yr	n>50	Normalized # Exceed	%exc50	Airshed Type
Vernon	52.3	26	9659	4.5	30.8	8.6	Degraded
Golden	312.5	26	9426	21.5	25.1	6.9	Degraded
Grand Forks	53.3	26	9401	3.0	20.2	5.5	Degraded
Merritt	58.0	25	8964	3.7	22.5	6.2	Degraded
Revelstoke	62.0	23	8305	2.7	15.8	4.3	Degraded
Invermere	58.0	23	8273	3.0	19.4	5.3	Degraded
Nelson	56.0	23	8256	3.7	24.2	6.6	Degraded
Quesnel	TEOM/SSI	22	7974	10.7	25.2	6.9	Degraded
Castlegar	55.0	22	7945	1.7	11.2	3.1	Degraded
Williams Lake	TEOM/SSI	20	7289	3.0	9.9	2.7	Degraded
Prince George	362.0	20	7123	11.7	11.7	3.2	Degraded
Slocan	160.5	19	6926	1.5	3.7	1.0	Degraded
Creston	331.5	19	6923	10.0	10.9	3.0	Degraded
Chetwynd	61.7	19	6874	3.0	16.8	4.6	Degraded
Taylor	60.7	18	6624	2.3	14.0	3.9	Degraded

BV/Lakes	347.5	17	6167	8.4	8.9	2.4	Degraded
Penticton	56.3	16	5929	1.3	9.0	2.5	Degraded
Bear Lake	94.3	15	5640	3.7	13.7	3.8	Degraded
Victoria	54.6	15	5573	0.8	4.8	1.3	Degraded
Kamloops	363.0	14	5266	5.0	5.0	1.4	Degraded
Lower Fraser Valley	313.0	14	5172	3.7	4.3	1.5	Degraded
Valemount	53.0	12	4391	1.3	8.7	2.4	Degraded
Kelowna	362.3	15	5342	2.0	2.0	0.5	Threatened
Trail	327.0	14	5126	0.5	0.5	0.1	Threatened
Radium	118.7	14	5088	0.7	2.9	0.8	Threatened
Skookumchuck	55.3	13	4640	0.3	2.2	0.6	Threatened
Kitimat/Terrace	362.3	10	3766	1.1	1.1	0.3	Threatened
Sea to Sky	349.3	14	5023	0.0	0.0	0.0	Unthreatened
Campbell River	341.2	11	4127	0.0	0.0	0.0	Unthreatened
Cowichan Valley	358.5	11	3909	0.0	0.0	0.0	Unthreatened
Port Alberni	358.7	9	3459	0.0	0.0	0.0	Unthreatened
Powell River	358.5	9	3288	0.0	0.0	0.0	Unthreatened
PR Rupert	329.0	7	2408	0.0	0.0	0.0	Unthreatened

100 Mile	Insufficient Data
Bella Coola	Insufficient Data
Comox	Insufficient Data
Courtnay	Insufficient Data
Cranbrook	Insufficient Data
Elk Valley	Insufficient Data
Fernie	Insufficient Data
Fort Nelson	Insufficient Data
Fort St. James	Insufficient Data
Fraser Canyon	Insufficient Data
Lillooet	Insufficient Data
MacKenzie	Insufficient Data
Nakusp	Insufficient Data
Nanaimo	Insufficient Data
North Thompson	Insufficient Data
Sechelt	Insufficient Data
Shuswap	Insufficient Data
South Okanagan	Insufficient Data
Vanderhoof	Insufficient Data

11 June 2002

Appendix 7: Summary of Health Effects Concentration-Response Factors from AQVM and ICAP

The following tables for PM and ozone are updated versions of tables from the *Report of an Expert Panel to Review the Socio-Economic Models and Related Components Supporting the Development of Canada-Wide Standards for Particulate Matter and Ozone* to the Royal Society of Canada, June 2001, Tables 4, 5 and 6, pages 53-55.

The factors are current to early 2003. The current database of CRFs in AQVM has been updated and adjusted to reflect the GAM and other statistical problems. The data in the tables are likely to be adjusted further in the future – so the most up-to-date version of the AQVM CRF database should be consulted at the time of use (currently available from Health Canada, Air Health Effects Section). Note that the revisions will not make a material difference in most effect estimates. Note also that the current Panel recommends using different CRFs for PM mortality than the AQVM database below and also recommends not using the AQVM ozone mortality CRF in the BC/PNW region.

The updated data for the $PM_{2.5}$ and ozone tables were provided by Dr. David Stieb of Health Canada (personal communication, January 2003).

The table for PM_{10} is taken directly from the Royal Society of Canada report and is current to 1999. Updated CRFs for PM_{10} have not been supplied by Health Canada.

The concentration-response factors ('parameters') in the second column in the following tables are the CRFs per capita for the indicated concentration increments in Column 1.

Tables of CRFs from the ICAP database follow the AQVM tables.

Concentration-response relationships utilized in AQVM for $PM_{2.5}$

Health Event Category	Concentration-Response Parameter (Probability Weighting Applied)
Annual mortality risk per 1 $\mu g/m^3$ change in annual average $PM_{2.5}$ concentration	Low 1.58×10^{-5} (25%) Central 2.82×10^{-5} (50%) High 4.07×10^{-5} (25%)
Chronic bronchitis (CB) annual risk per 1 $\mu g/m^3$ change in annual average $PM_{2.5}$ concentration	For population 25 years and older: Low $4.13 \times 10\text{-}^{5}$ (25%) Central 8.27×10^{-5} (50%) High 1.24×10^{-4} (25%)
Respiratory hospital admissions (RHA) daily risk factors per 1 $\mu g/m^3$ change in daily average $PM_{2.5}$ concentration	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 $\mu g/m^3$ change in daily average $PM_{2.5}$ concentration	Low 7.90×10^{-9} (25%) Central 1.02×10^{-8} (50%) High 1.26×10^{-8} (25%)
Adjusted (net) emergency room visits (AERV) daily risk factors per 1 $\mu g/m^3$ change in daily average $PM_{2.5}$ concentration	Low 4.30×10^{-8} (25%) Central 5.22×10^{-8} (50%) High 6.15×10^{-8} (25%)
Asthma syptom day (ASD) daily risk factors given a 1 $\mu g/m^3$ change in daily average $PM_{2.5}$ concentration	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 $\mu g/m^3$ change in daily average $PM_{2.5}$ concentration	For population 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 $\mu g/m^3$ change in daily average $PM_{2.5}$ conc*entration*	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 $\mu g/m^3$ change in annual average $PM_{2.5}$ concentration	For population under age 20: Low 6.20×10^{-4} (25%) Central 1.65×10^{-3} (50%) High 2.69×10^{-3} (25%)

Concentration-response relationships utilized in AQVM for ozone

Health Event Category	Concentration-Response Parameter (Probability Weighting Applied)
Daily mortality risk factors given a 1 ppb change in daily high-hour ozone concentration	Low 1.28×10^{-9} (25%) Central 3.55×10^{-9} (50%) High 5.81×10^{-9} (25%)
Respiratory hospital admissions (RHAs) daily risk factors given a 1 ppb change in daily high-hour ozone concentration	Low 6.00×10^{-9} (25%) Central 1.10×10^{-8} (50%) High 1.60×10^{-8} (25%)
Adjusted (net) emergency room visits (AERVs) daily risk factor given a 1 ppb change in daily high-hour ozone concentration	Low 2.43×10^{-8} (25%) Central 4.46×10^{-8} (50%) High 6.48×10^{-8} (25%)
Asthma symptom days (ASDs) daily risk factor given a 1 ppb change in daily high-hour ozone concentration	Low 1.06×10^{-4} (33%) Central 1.88×10^{-4} (50%) High 5.20×10^{-4} (17%)
Minor restricted activity days (MRAD) daily risk factor given a 1 ppb change in daily high-hour ozone concentration	Low 1.93×10^{-5} (25%) Central 4.67×10^{-5} (50%) High 7.40×10^{-5} (25%)
Net days with acute respiratory symptoms (ARSs) daily risk factors given a 1 ppb change in daily high-hour ozone concentration	Low 5.07×10^{-5} (25%) Central 9.03×10^{-5} (50%) High 1.30×10^{-4} (25%)

Concentration-response relationships utilized in AQVM for PM_{10}

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

Source: Human Health and Environmental Benefits of Achieving Alternate CWS for Inhalable Particulates ($PM_{2.5}$, PM_{10}) and Ground Level Ozone. Final Report. Prepared by Paul De Civita, Environment Canada, David Stieb, Health Canada, Lauraine Chestnut, David Mills, Robert Rowe, Stratus Consulting. July 25, 1999. In Compendium of Benefits 99-08-17.

A similar database of CRFs exists within the Ontario Medical Association's ICAP model software. The following tables for PM_{10} and ozone are taken from Table D.5 and D.6, pages D-27ff in *Illness Costs of Air Pollution, Phase II: Estimating Health and Economic Damages*, DSS Management Consultants, July 2000. This report and the ICAP software are available on the OMA website: http://www.oma.org/phealth/smogmain.htm#program. In the main, the AQVM and ICAP CRF databases are similar, although ICAP has resolved the coefficients (by age group, etc.) somewhat more than AQVM.

Note that the CRFs are expressed as per 1 million people exposed (not per capita as in the AQVM database).

Table D.6 - Exposure/Response Functions for PM_{10}

	ILLNESS CATEGORY											
	PREMATURE MORTALITY						HOSPITAL ADMISSIONS					
	Respiratory			Cardio-vascular			Asthma			COPD		
	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3
ICD-9 Codes												
AGE-DEPENDENT RISK GROUP												
Start Age	0	18	65	0	18	65	0	18	65	0	18	65
Stop Age	17	65	99	17	65	99	17	65	99	17	65	99
PM_{10}												
LINEAR												
Central Illness Rate [1]	-	1.28E+00	8.90E+00	-	2.55E+00	1.78E+01	9.28E-01	9.84E-01	4.04E+00	-	4.11E+00	1.72E+01
Probability Weight	-	50%	50%	-	50%	50%	50%	50%	50%	-	50%	50%
Central Effect Threshold	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-
Lower Illness Rate	-	5.91E-01	5.76E+00	-	1.48E+00	1.44E+01	2.35E-01	4.42E-01	1.68E+00	-	2.59E+00	1.00E+01
Probability Weight	-	25%	25%	-	25%	25%	25%	25%	25%	-	25%	25%
Lower Threshold Value	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-
Upper Illness Rate	-	1.95E+00	1.08E+01	-	3.74E+00	2.06E+01	1.68E+00	1.48E+00	6.49E+00	-	5.55E+00	2.48E+01
Probability Weight	-	25%	25%	-	25%	25%	25%	25%	25%	-	25%	25%
Upper Threshold Value	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-

[1] All risks are expressed as the risk per 1,000,000 people exposed.

Table D.6 - Exposure/Response Functions for PM_{10} (*continued*)

	ILLNESS CATEGORY											
	HOSPITAL ADMISSIONS											
	Pneumonia			Coronary Artery Disease			Dysrhythmias			Congestive Heart Failure		
	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3
ICD-9 Codes												
AGE-DEPENDENT RISK GROUP												
Start Age	0	18	65	0	18	65	0	18	65	0	18	65
Stop Age	17	65	99	17	65	99	17	65	99	17	65	99
PM_{10}												
LINEAR												
Central Illness Rate (a)	2.96E+00	3.14E+00	1.29E+01	-	1.05E-01	2.67E+00	-	5.46E-01	1.39E+01	-	3.31E+00	8.43E+01
Probability Weight	50%	50%	50%	-	58%	55%	-	65%	63%	-	61%	56%
Central Effect Threshold (b)	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-
Lower Illness Rate (a)	9.35E-01	1.76E+00	6.69E+00	-	2.15E-02	1.49E+00	-	-	-	-	6.80E-01	4.69E+01
Probability Weight	25%	25%	25%	-	21%	25%	-	10%	12%	-	19%	25%
Lower Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-
Upper Illness Rate (a)	1.67E+01	1.48E+01	6.46E+01	-	1.91E-01	3.85E+00	-	1.45E+00	2.94E+01	-	5.61E+00	1.13E+02
Probability Weight	25%	25%	25%	-	21%	20%	-	25%	25%	-	20%	19%
Upper Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-

Table D.6 - Exposure/Response Functions for PM_{10} (*continued*)

	ILLNESS CATEGORY														
	EMERGENCY ROOM VISITS						DOCTOR'S OFFICE VISITS								
	Respiratory			Cardio-vascular			Child Bronchitis			Chronic Respiratory Disease			Chronic Bronchitis		
	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3
ICD-9 Codes															
AGE-DEPENDENT RISK GROUP															
Start Age	0	18	65	0	18	65	0	18	65	0	18	65	0	18	65
Stop Age	17	65	99	17	65	99	17	65	99	17	65	99	17	65	99
PM_{10}															
LINEAR															
Central Illness Rate (a)	1.29E+00	5.86E-01	-	-	5.51E-01	-	-	-	-	-	-	-	-	-	-
Probability Weight	50%	50%	50%	-	50%	50%	-	-	-	-	-	-	-	-	-
Central Effect Threshold (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Lower Illness Rate (a)	1.96E-01	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	25%	25%	0%	-	25%	25%	-	-	-	-	-	-	-	-	-
Lower Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Upper Illness Rate (a)	2.49E+00	9.72E-01	5.19E-01	-	9.38E-01	5.01E-01	-	-	-	-	-	-	-	-	-
Probability Weight	25%	25%	25%	-	25%	25%	-	-	-	-	-	-	-	-	-
Upper Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Table D.6 - Exposure/Response Functions for PM_{10} (*continued*)

	ILLNESS CATEGORY																	
	DOCTOR'S OFFICE VISITS									MINOR ILLNESSES								
	Child Asthma Attacks			Adult Asthma Attacks			Acute Respiratory Symptoms			Minor Restricted Activity Days			Restricted Activity Days			Asthma Symptom Days		
	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3
ICD-9 Codes																		
AGE-DEPENDENT RISK GROUP																		
Start Age	0	18	65	0	18	65	0	18	65	0	18	65	0	18	65	0	18	65
Stop Age	17	65	99	17	65	99	17	65	99	17	65	99	17	65	99	17	65	99
PM_{10}																		
LINEAR																		
Central Illness Rate (a)	-	-	-	-	-	-	-	-	-	1.50E+05	-	1.50E+05	2.30E+05	-	2.30E+05	2.20E+04	-	2.20E+04
Probability Weight	-	-	-	-	-	-	-	-	-	50%	-	50%	50%	-	50%	50%	-	50%
Central Effect Threshold (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Lower Illness Rate (a)	-	-	-	-	-	-	-	-	-	1.80E+04	-	1.80E+04	6.58E+04	-	6.58E+04	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	25%	-	25%	25%	-	25%	25%	-	25%
Lower Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Upper Illness Rate (a)	-	-	-	-	-	-	-	-	-	3.00E+05	-	3.00E+05	3.62E+05	-	3.62E+05	5.87E+04	-	5.87E+04
Probability Weight	-	-	-	-	-	-	-	-	-	25%	-	25%	25%	-	25%	25%	-	25%
Upper Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Table D.5 - Exposure/Response Functions for Ozone

	ILLNESS CATEGORY											
	PREMATURE MORTALITY						HOSPITAL ADMISSIONS					
	Respiratory			Cardio-vascular			Asthma			COPD		
	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3
ICD-9 Codes												
AGE-DEPENDENT RISK GROUP												
Start Age	0	18	65	0	18	65	0	18	65	0	18	65
Stop Age	17	65	99	17	65	99	17	65	99	17	65	99
O_3												
LINEAR												
Central Illness Rate [1]	-	-	-	-	-	-	2.37E+00	2.52E+00	1.04E+01	-	3.09E+00	1.30E+01
Probability Weight	-	-	-	-	-	-	50%	50%	50%	-	50%	50%
Central Effect Threshold	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-
Lower Illness Rate	-	-	-	-	-	-	3.55E-01	6.68E-01	2.54E+00	-	8.66E-01	3.35E+00
Probability Weight	-	-	-	-	-	-	25%	25%	25%	-	25%	25%
Lower Threshold Value	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-
Upper Illness Rate	-	-	-	-	-	-	2.35E+00	2.07E+00	9.08E+00	-	5.20E+00	2.32E+01
Probability Weight	-	-	-	-	-	-	25%	25%	25%	-	25%	25%
Upper Threshold Value	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-

[1] All risks are expressed as the risk per 1,000,000 people exposed.

Table D.5 - Exposure/Response Functions for Ozone *(continued)*

	ILLNESS CATEGORY											
	HOSPITAL ADMISSIONS											
	Pneumonia			Coronary Artery Disease			Dysrhythmias			Congestive Heart Failure		
	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3
ICD-9 Codes												
AGE-DEPENDENT RISK GROUP												
Start Age	0	18	65	0	18	65	0	18	65	0	18	65
Stop Age	17	65	99	17	65	99	17	65	99	17	65	99
O_3												
LINEAR												
Central Illness Rate (a)	2.36E+00	2.51E+00	1.03E+01	-	-	-	-	1.04E+00	2.65E+01	-	-	-
Probability Weight	50%	50%	50%	-	-	-	-	0.55	0.51	-	-	-
Central Effect Threshold (b)	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-
Lower Illness Rate (a)	7.08E-01	1.33E+00	5.07E+00	-	-	-	-	-	-	-	-	-
Probability Weight	25%	25%	25%	-	-	-	-	0.25	0.25	-	-	-
Lower Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-
Upper Illness Rate (a)	4.09E+00	3.60E+00	1.58E+01	-	-	-	-	2.80E+00	5.66E+01	-	-	-
Probability Weight	25%	25%	25%	-	-	-	-	0.2	0.24	-	-	-
Upper Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-

Table D.5 - Exposure/Response Functions for Ozone *(continued)*

	ILLNESS CATEGORY														
	EMERGENCY ROOM VISITS						DOCTOR'S OFFICE VISITS								
	Respiratory			Cardio-vascular			Child Bronchitis			Chronic Respiratory Disease			Chronic Bronchitis		
	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3
ICD-9 Codes															
AGE-DEPENDENT RISK GROUP															
Start Age	0	18	65	0	18	65	0	18	65	0	18	65	0	18	65
Stop Age	17	65	99	17	65	99	17	65	99	17	65	99	17	65	99
O_3															
LINEAR															
Central Illness Rate (a)	-	-	5.07E+02	-	-	4.20E+02	-	-	-	-	-	-	-	-	-
Probability Weight	50%	50%	50%	-	50%	50%	-	-	-	-	-	-	-	-	-
Central Effect Threshold (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Lower Illness Rate (a)	-	-	6.07E+01	-	-	5.02E+01	-	-	-	-	-	-	-	-	-
Probability Weight	25%	25%	25%	-	25%	25%	-	-	-	-	-	-	-	-	-
Lower Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Upper Illness Rate (a)	5.09E+01	1.53E+01	9.07E+02	-	1.33E+01	7.90E+02	-	-	-	-	-	-	-	-	-
Probability Weight	25%	25%	25%	-	25%	25%	-	-	-	-	-	-	-	-	-
Upper Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Table D.5 - Exposure/Response Functions for Ozone (*continued*)

	ILLNESS CATEGORY																	
	DOCTOR'S OFFICE VISITS									MINOR ILLNESSES								
	Child Asthma Attacks			Adult Asthma Attacks			Acute Respiratory Symptoms			Minor Restricted Activity Days			Restricted Activity Days			Asthma Symptom Days		
	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3	Age Group 1	Age Group 2	Age Group 3
ICD-9 Codes																		
AGE-DEPENDENT RISK GROUP																		
Start Age	0	18	65	0	18	65	0	18	65	0	18	65	0	18	65	0	18	65
Stop Age	17	65	99	17	65	99	17	65	99	17	65	99	17	65	99	17	65	99
O_3																		
LINEAR																		
Central Illness Rate (a)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Central Effect Threshold (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Lower Illness Rate (a)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Lower Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Upper Illness Rate (a)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Upper Threshold Value (b)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Probability Weight	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-