Systematic Review of Environmental Burden of Disease in Canada

Final Report

Prepared for:
National Collaborating Centre for Environmental Health
400 East Tower
555 W 12th Avenue
Vancouver, BC V5Z 3X7

Prepared by*
E Risk Sciences, LLP
4647 Carter Trail
Boulder, CO 80301

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LIST OF ABBREVIATIONS

AHED	Air Health Effects Division
AQI	Air Quality Index
CEHAPE	Children's Environment and Health Action Plan
CHMS	Canadian Health Measures Survey
COPD	Chronic obstructive pulmonary disorder
CPCHE	Canadian Partnership for Children's Health and Environment
CRA	Comparative Risk Assessment
CRF	Concentration-response function
DALY	Disability-adjusted life year
DFLE	Disability-free life expectancy
EAF	Environmentally-attributable fraction
EBD	Environmental burden of disease
ETS	Environmental tobacco smoke
GBD	Global Burden of Disease
HALE	Health-adjusted life expectancy
HEIMTSA	Health and Environment Integrated Methodology and Toolbox for Scenario Assessment
HSPH	Harvard School of Public Health
HRQOL	Health-related quality of life
ICAP	Illness Costs of Air Pollution
ILO	International Labour Organization
INTARESE	Integrated Assessment of Health Risks of Environmental Stressors in Europe
MIREC	Maternal-Infant Research on Environmental Chemicals
NAPS	National air pollution surveillance
NAS	National Academy of Sciences
NCCEH	National Collaborating Centre for Environmental Health
OECD	Organisation for Economic Co-operation and Development
OMA	Ontario Medical Association
PHA	Public Health Agency of Canada
PHI	Population Health Impact
PHIRIC	Population Health Intervention Research Initiative for Canada
PM	Particulate matter
$PM_{2.5}$	Particulate matter 2.5 microns in diameter
PM_{10}	Particulate matter 10 microns in diameter
RIVM	National Institute for Public Health and the Environment
UV	Ultraviolet
U.S. EPA	United States Environmental Protection Agency
WHO	World Health Organization

1.0 Introduction

Potentially preventable illnesses and deaths resulting from exposure to environmental contaminants have been estimated to account for approximately $3.6 to $9.1 billion dollars in annual health care costs in Canada (Boyd and Genuis 2008). These statistics are driven by several primary disease categories (e.g., cardiovascular and respiratory diseases) that may be caused or exacerbated by population exposures to different environmental contaminants (e.g., indoor and outdoor air pollution, lead). Concerns have also been raised about environmental exposures and potential adverse pregnancy outcomes and childhood diseases in Canada, as well as the economic and social costs attributable to the environmental burden of childhood diseases (Bérubé 2007). According to a recently published report from the United States President's Cancer Panel, scientific evidence on individual and multiple environmental exposure effects on disease initiation and outcomes, and consequent health system and societal costs, are not being adequately integrated into national policy decisions and strategies for disease prevention, health care access, and health system reform (DHHS 2010).

The concept that the world's disease burden is attributable to a range of environmental and other (e.g., lifestyle, occupational) risk factors has been recognized for many years. Some of the earliest efforts to link specific risk factors, particularly environmental risk factors, to health outcomes initially focused on cancer as an endpoint. A landmark study by Doll and Peto (1981) estimated the percentage of avoidable cancer deaths in the United States attributable to lifestyle and environmental factors, representing one of the first attempts to quantify the relationship between risk factors and health outcomes. Since then, a number of studies have been conducted (particularly during the 1990s) that have attempted to quantify the burden of disease globally and for different regions or countries. Specifically, these studies describe public health (mortality and/or morbidity) in terms of disease burden for various categories of disease (e.g., cancer, heart disease, injuries) and risk factors. Most of these studies were not focused on environmental risk factors, however, with the exception of possible environmental pollution in developing countries. The widely cited Global Burden of Disease (GBD) study was one of the first global efforts of this kind, which evaluated premature mortality and disability from a large number of diseases and injuries due to a variety of population exposures (Murray and Lopez 1996). Health outcomes included in the 1990 GBD study were attributed to eight major risk factors (few of which were specifically related to the environment): malnutrition, poor water sanitation/personal hygiene, unsafe sex, alcohol, occupation, tobacco, hypertension, and physical activity. A subsequent 2001 GBD study targeted a greater number of risk factors, including several specifically related to environmental exposures (e.g., unsafe water, sanitation, and hygiene; urban air pollution, indoor smoke from household use of solid fuels) (Lopez et al. 2006a).

Because historical burden of disease assessments typically did not address issues specific to environmental health, additional studies have attempted to identify and quantify the environmental burden of disease (EBD) globally and for different countries or regions. These studies have generally been based on or build off of the historical burden of disease approach, and include many different definitions of the environment. The current recommended framework for EBD studies is based on a causal web structure that links environmental hazards and risk factors to disease burden (Prüss et al. 2001; Prüss-Üstün et al. 2003). Both exposure-

based and outcome-based approaches are typically used to estimate EBD, with the latter approach being used the most often due to data limitations regarding population exposure levels and quantitative dose-response relationships. The outcome-based approach involves compiling population-level health statistics data for different disease categories and determining the environmentally attributable fraction (EAF) or percentage of estimated disease burden due to environmental exposures.

In Canada, efforts are currently underway to assess the population burden of disease attributed to environmental risk factors using novel approaches and country-specific data. The World Health Organization (WHO) has also developed a country-specific EBD profile for Canada, and several published studies have estimated the EBD for specific disease categories and/or risk factors across Canada or for selected Canadian regions. Other global, regional, and national studies provide additional data that could be used to derive Canada-specific EBD estimates. Despite important data gaps and research needs, available studies provide relevant information for policymakers and health practitioners who are responsible for allocating scarce resources and designing or implementing environmental health policies to directly address specific sources of disease. This information can also be used as a teaching tool to better educate and inform the public about opportunities to reduce exposures that have been associated with particular health outcomes.

The current report presents a systematic review of EBD studies that have been conducted in Canada or are potentially relevant for the Canadian context. It is important to recognize that the available EBD studies have defined environmental risk factors in different ways, with some studies focused exclusively on those factors that lead to population exposures in the environment (e.g., air pollution, water pollution), and other studies focused on these factors in addition to exposures in the personal environment (e.g., smoking, obesity) that are more related to lifestyle choices or that include occupational exposures. Although we include all of these studies in our review for sake of completeness, we attempt to limit our discussion to EBD estimates that pertain specifically to environmental risk factors that lead to population-level exposures. Not only does this approach allow for a more "apples-to-apples" comparison among different EBD studies, but it may be more useful from a public policy perspective with respect to designing effective and targeted health intervention programs. We also provide background information on some of the original burden of disease assessments because these studies serve as the backbone or building blocks for many of the subsequent EBD studies. Important data gaps and research needs, as well as several follow-up recommendations for the National Collaborating Centre for Environmental Health (NCCEH) or other Canadian environmental or health agencies, are also provided. Specifically, the following sections include: (1) a review of the methods used to identify relevant EBD studies and supporting literature; (2) a summary of background information on the original burden of disease studies and methodologies for calculating EBD and EAFs; (3) a review and synthesis of available global, regional, national, and local EBD studies that are most relevant to Canada; and (4) a discussion of key data gaps and information needs and list of recommendations for policymakers and health practitioners in Canada.

2.0 Methods

To identify relevant data and information on EBD-related studies in Canada, we conducted a systematic search of the peer-reviewed and gray/white literature over the last 15 to 20 years. Specifically, our literature search primarily covered the time period from 1995 to 2010, although some earlier historical references are also cited. As part of our literature search, we obtained key publications related to historical burden of disease assessments and methodological approaches for estimating EBD to provide greater context regarding the origin of and basis for these studies. We also identified and summarized available global/regional and non-Canadian EBD studies and review articles, and indicated which studies or data sets are potentially relevant to the Canadian context. Finally, we identified and summarized EBD-related studies and analyses that were conducted specifically for Canada. Note that some of these latter studies do not represent actual EBD studies *per se*, but rather present input data that may be used to conduct local or national-level EBD studies in Canada. These latter studies were included for illustrative purposes only, and are not intended to provide a comprehensive survey of all available environmental data that could be used in a Canadian EBD study. In general, studies focused on demonstrating an association between a particular environmental exposure and health outcome, which is only one component of an EBD study (and does not constitute an actual EBD study), were not included in our report.

Because the many EBD studies that have been conducted to date vary significantly with respect to study design and methodology, our approach entailed summarizing the existing EBD literature in its entirety and including all identified relevant studies, regardless of whether some studies may provide more reliable or robust estimates than others. Given the current state-of-knowledge, we do not believe that it is possible to actually rank each study based on its reliability. However, we do attempt to identify which EBD studies and estimates are likely to be the most relevant for Canada (and which ones may not be particularly relevant for Canada) throughout the Results section of this report.

Our criteria for selection of papers for the review included publications that either (1) presented useful background information on historical burden of disease studies and EBD methodologies, or (2) provided EBD or EAF estimates based on global/regional or Canadian-specific studies. The former publications are summarized in the Background section below, while the latter studies are summarized in the Results section and in Appendix 1 (see Tables A1-A6). Our literature search was based primarily on the electronic resources available from the Harvard University library system. Specifically, our searches included the following types of terms: burden [and] illness (or) disease [and] Canada; burden [and] illness (or) disease [and] environment*; burden [and] illness (or) disease [and] method*. We used all the major databases and search engines, including several proprietary databases, available from the Harvard University system. These included:

- Citation Index/ISI Web of Science
- JSTOR
- National Library of Medicine/PUBMED
- MEDLINE (OvidSP)

- Google Scholar
- Google Books
- TRIP database
- EMBASE (OvidSP)
- Scirus
- Environmental Research (Harvard University resource)

We carefully reviewed and screened all papers identified from our search for relevancy. First, we reviewed the abstract or Executive Summary of the publication to determine its relevance, and then we reviewed all relevant publications in their entirety. Additionally, we reviewed the references cited in selected publications as we acquired them, to obtain additional references not identified through the original literature search.

In addition to performing a literature search using the Harvard University system, we also conducted a search of relevant Canadian, U.S., and international websites, including those of Environment Canada, Health Canada, the Public Health Agency of Canada (PHA), National Resource Canada, British Columbia Ministry of Environment, the U.S. Environmental Protection Agency (U.S. EPA), and WHO.

3.0 Background Information

3.1 Original Burden of Disease Studies

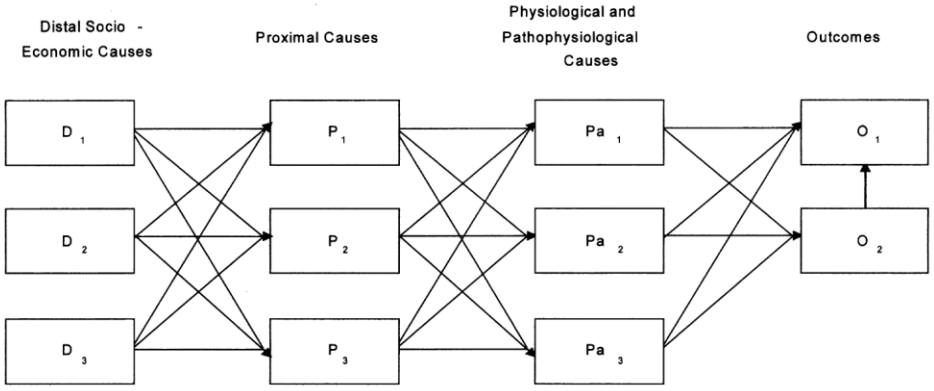
An understanding of the original burden of disease studies is important because these studies serve as the backbone or building blocks for many of the subsequent EBD studies. We therefore provide an overview of some of the key historical burden of disease studies that have been conducted in the United States and globally. This overview is not meant to provide an exhaustive summary of the burden of disease literature, but rather provides important context for the basis for and methodologies developed to support EBD studies relevant to Canada.

The landmark study by Doll and Peto (1981) was one of the first attempts to evaluate the contribution of environmental and lifestyle factors to a population's burden of disease, expressed as mortality (not morbidity). Specifically, this study estimated the percentage of avoidable cancer deaths in the United States potentially caused by these factors. Based on an extensive review of the literature, the authors concluded that 35% (range: 10-70%) and 30% (range: 25-40%) of all cancer deaths at the time were attributable to nutrition/food consumption and tobacco exposures, respectively. The authors cautioned that although the former risk factor had a higher estimated attributable fraction than the latter risk factor, there was much more uncertainty around these estimates (i.e., the largest reliably known attributable percentage was due to tobacco exposures). In this study, occupational exposures were estimated to account for approximately 4% (range: 2-8%) of all cancer deaths at the time (predominantly attributable to lung cancer), while pollution was estimated to account for approximately 2% (range: <1-5%) of all cancer deaths at the time.

With respect to a broader global perspective, the GBD study, sponsored by the World Bank in collaboration with WHO and the Harvard School of Public Health (HSPH) in 1996, represented

one of the first attempts to evaluate premature mortality and disability from a large number of diseases and injuries due to different population exposures worldwide (Murray and Lopez 1996, 1999). The GBD framework was based on the concept of a causal chain of events which linked various types of causes or exposures to specific health outcomes (Figure 1 provides a diagram of a simplified causal web for illustrative purposes). Specifically, distal social, cultural and economic factors were assumed to influence health outcomes by operating through individual behaviors or exposures (e.g., tobacco use, diet), while these proximal determinants or risk factors were believed to ultimately influence health outcomes through physiological and patho-physiological mechanisms or pathways (e.g., weight, blood pressure) (Murray and Lopez 1999). This framework also accounted for health outcomes which could lead to various impairments, functional limitations (disability), and death or changes in non-health well-being (Lopez et al. 2006b).

Figure 1. Burden of disease framework - simplified causal web linking exposures and outcomes (Source: Murray and Lopez 1999)



The purpose of the initial GBD study was to provide a comprehensive assessment of the global disease burden in 1990. The primary objectives of this study were to (1) develop internally consistent estimates of mortality from 107 major causes of death for the world and for eight geographic regions; (2) develop internally consistent estimates of the incidence, prevalence, duration, and case fatality for 483 disabling sequelae resulting from the above causes; (3) estimate the fraction of mortality and disability attributable to ten major risk factors; and (4) develop projection scenarios of mortality and disability disaggregated by age, sex, and region (Murray and Lopez 1996, 1999). A guiding principle of the GBD study was that almost all sources of health data were likely to contain useful information, and expert judgment based on the collaboration of more than 100 scientists from more than 20 countries was used to evaluate the literature and derive estimates of the burden of disease and injury attributable to identified risk factors (Murray and Lopez 1999; Lopez et al. 2006a, b).

Besides generating the first comprehensive and consistent set of estimates of mortality and morbidity, the GBD study introduced a new metric called the disability-adjusted life year (DALY) to quantify the burden of disease, which is a summary measure of population health that combines years of life lost from premature death and years of life lived in less than full health (Murray and Lopez 1999; Mathers et al. 2006a). DALYs were estimated for different categories

of disease or injuries and different risk factors based on information available at the time about causation, prevalence, exposure, and disease and injury outcomes (see Table 1).

Table 1. Estimated DALYs for 25 leading diseases and injuries by 8 major risk factors based on 1990 GBD study (Source: Murray and Lopez 1999)

Diseases or Injuries	DALYs (millions)	Risk Factors
	219.6	Malnutrition
Lower respiratory infections	112.9	
Diarrheal diseases	99.6	
	93.4	Poor water sanitation/personal hygiene
Conditions arising during perinatal period	92.3	
Unipolar major depression	50.8	
	48.7	Unsafe sex
	47.7	Alcohol
Ischemic heart disease	46.7	
Cerebrovascular disease	38.5	
Tuberculosis	38.4	
	37.9	Occupation
Measles	36.5	
	36.2	Tobacco
Road traffic accidents	34.3	
Congenital anomalies	32.9	
Malaria	31.7	
COPD	29.1	
Falls	26.7	
Iron-deficiency anemia	24.6	
Protein-energy malnutrition	20.9	
War	20.0	
	19.0	Hypertension
Self-inflicted injuries	18.9	
Tetanus	17.5	
Violence	17.4	
Alcohol use	16.6	
Drownings	15.7	
Bipolar disorder	14.2	
	13.6	Physical inactivity
Pertussis	13.4	
Osteoarthritis	13.3	
Cirrhosis of the liver	13.2	

It is noteworthy that the largest global disease burden was estimated for two risk factors that are specific to developing rather than developed countries: malnutrition and poor water/sanitation (see Table 2). This is because relatively large populations in the developing world are malnourished and have little access to safe water and sanitation. On the other hand, risk factors such as tobacco, alcohol, occupation, hypertension, physical activity, and air pollution, were estimated to account for a greater disease burden in developed regions compared to developing regions. These findings suggest that global disease burden estimates may not necessarily be relevant for country-specific assessments due to different underlying conditions and risk factors.

Table 2. Percentage of Deaths and DALYs attributable to 10 major risk factors based on 1990 GBD study (Source: Adapted from Murray and Lopez 1999)

	World		**Developed Countries**		**Developing Countries**	
	Deaths (%)	**DALYs (%)**	**Deaths (%)**	**DALYs (%)**	**Deaths (%)**	**DALYs (%)**
Malnutrition	11.7	15.9	0.0	0.0	14.9	18.0
Poor water/sanitation	5.3	6.8	0.0	0.1	6.7	7.6
Unsafe sex	2.2	3.5	0.8	2.1	2.5	3.7
Tobacco	6.0	2.6	14.5	12.1	3.7	1.4
Alcohol	1.5	3.5	1.3	9.6	1.6	2.7
Occupation	2.2	2.7	2.1	4.6	2.3	2.5
Hypertension	5.8	1.4	12.9	4.7	3.8	0.9
Physical inactivity	3.9	1.0	10.1	4.0	2.3	0.6
Illicit drugs	0.2	0.6	0.3	1.9	0.2	0.4
Air pollution	1.1	0.5	2.5	1.5	0.7	0.4

Several aspects of the 1990 GBD study were highly criticized within the scientific community, including the methods used to assess age weights and severity scores for disabilities, which were based primarily on expert opinion rather than population-based health state valuations (Lopez et al. 2006b). Additionally, the initial GBD study raised a number of methodological issues with respect to the ability to make comparable assessments of the burden of disease due to a lack of standardized methods and differences in the reliability of the underlying epidemiological studies of relative risk and population exposure levels (Murray and Lopez 1999; Lopez et al. 2006b). For example, the measures of exposure for the identified risk factors were often quite poor, and varied across risk factors, measurement approach, lag time, and reference distribution (see Table 3).

A Comparative Risk Assessment (CRA) module was therefore developed as part of the GBD study to systematically assess changes in population health which would result from modifying the population distribution of exposure to a risk factor or a group of risk factors (i.e., attributable fractions of disease due to a risk factor were calculated based on comparisons of the disease burden expected under the current estimated distribution of exposure with that expected under an alternative or "counterfactual" distribution of exposure) (Murray and Lopez 1999; Ezzati et al. 2006). This broader view allowed for the population distribution to be defined over many different levels and intensities of exposure (instead of the comparison distribution of exposure being zero) and four types of counterfactual distributions were described: theoretical minimum risk, plausible minimum risk, feasible minimum risk, and cost-effective minimum risk (Murray and Lopez 1999). To estimate attributable burden under the revised GBD framework, the following types of data were needed: (1) relative risks for each cause of death and disability as a function of exposure level, (2) the current (and past for time lagged variables) levels of exposure, (3) the counterfactual distribution of exposure, and (4) the burden of disease due to each cause of death and disability in a given population.

Table 3. Summary of methodological differences in approaches used to estimate attributable burden from 10 major risk factors based on 1990 GBD Study (Source: Murray and Lopez 1999)

Risk Factor	Type of Risk Factor		Relative Risk Controlled for Confounding	Measure of Exposure	Reference Distribution of Exposure	Time Lag from Exposure to Burden
	Exposure	Physio-logical State				
Malnutrition		0		Population less than 2 SFs weight-for-age based on extensive national surveys	Population weight-for-age higher than minus 2 SDs	Intermediate
Poor water, sanitation, and hygiene	0			Based on the theoretical fecal-oral route of transmission	Zero	Short
Unsafe sex	0			Based on the theoretical model of transmission of STDs and on contraceptive demand surveys for maternal conditions	Zero	Short to long
Alcohol (disease)		0	0	Indexed on alcohol consumption, non-hepatitis B cirrhosis, and alcohol dependence syndrome	Zero	Long
Alcohol (injury)	0			Indexed on estimate of consumption patterns based on small-scale studies	Zero	Short
Occupation (disease)	0			Registration data for EME, FSE, and LAC and constant rates for all other regions	Zero	Long
Occupation (injury)	0			Registration data for EME and constant rates for all other regions	Zero	Short
Tobacco	0		0	Indexed on lung cancer	Zero	Long
Hypertension		0		Population surveys of blood pressure	Systolic blood pressure of 110 mmHg	Long
Physical inactivity	0		0	Population surveys of activity patterns	Regular physical activity	Long
Illicit drugs	0			Small-scale studies	Zero	Short to intermediate
Air pollution	0			Monitoring systems in urban areas for most regions	WHO guidelines	Short to long

This new unified framework served as the underlying basis for the subsequent 2001 GBD study, which estimated DALYs for individual risk factors as well as the joint effects of multiple risk factors for the world and low- and middle-income versus high-income countries (see Table 4). Specific categories encompassing environmental risk factors were included in the 2001 assessment such as unsafe water, sanitation, and hygiene; urban air pollution (i.e., particulate matter [PM] with diameters between 2.5 microns [$PM_{2.5}$] and10 microns [PM_{10}]); and indoor smoke from household use of solid fuels (Ezzati et al. 2006; Lopez et al. 2006a, b; Mathers et al. 2006a).

Table 4. Percentage of deaths and DALYs attributable to risk factors based on 2001 GBD Study (Source: Adapted from Lopez et al. 2006b)

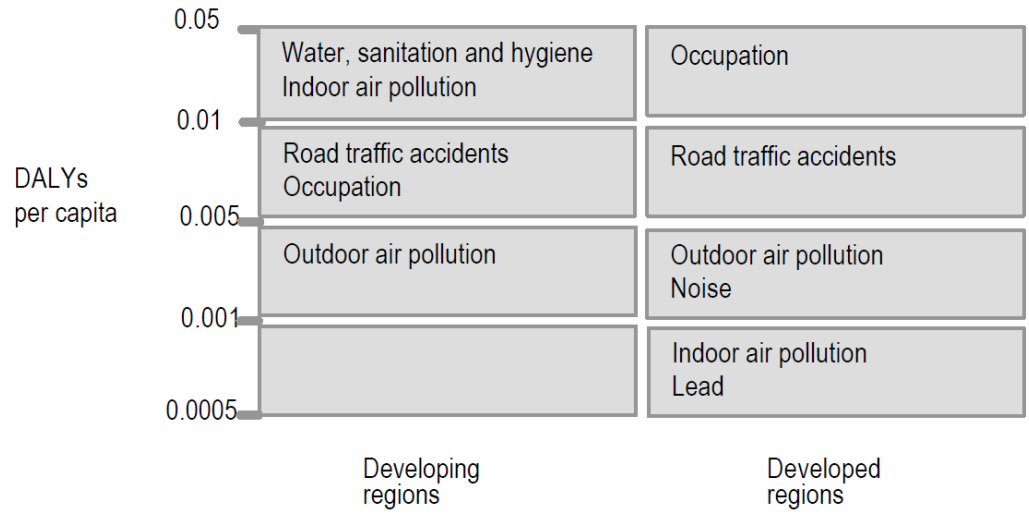
	World		**Low-and Middle-Income**		**High-Income**	
	Deaths (%)	DALYs (%)	Deaths (%)	DALYs (%)	Deaths (%)	DALYs (%)
Childhood and maternal under nutrition						
Childhood underweight	6.5	7.9	7.5	8.7	0.0	<0.1
Iron-deficiency anemia	1.1	1.6	1.3	1.7	0.1	0.5
Vitamin A deficiency	1.4	1.6	1.7	1.8	0.0	0.0
Zinc deficiency	1.5	1.8	1.8	2.0	0.0	<0.1
Other nutrition-related risk factors and physical activity						
High blood pressure	13.5	6.0	12.9	5.6	17.6	9.3
High cholesterol	6.9	3.4	6.3	3.1	10.7	6.3
Overweight and obesity	4.2	2.8	3.6	2.3	7.8	7.2
Low fruit and vegetable intake	4.7	2.4	4.8	2.4	4.2	2.7
Physical inactivity	3.4	1.8	3.2	1.6	4.8	3.2
Addictive substances						
Smoking	8.5	4.7	6.9	3.9	18.5	12.7
Alcohol use	3.4	3.6	3.9	3.6	0.3	4.4
Illicit drug use	0.4	0.6	0.4	0.6	0.5	1.4
Sexual and reproductive health						
Unsafe sex	5.1	5.3	5.8	5.8	0.4	0.6
Non-use and use of ineffective methods of contraception	0.3	0.5	0.3	0.5	0.0	<0.1
Environmental Risks						
Unsafe water, sanitation, and hygiene	2.8	3.4	3.2	3.7	<0.1	0.2
Urban air pollution	1.4	0.6	1.5	0.6	1.0	0.4
Indoor smoke from household use of solid fuels	3.2	2.7	3.7	3.0	0.0	<0.1
Other selected risks						
Contaminated injections in health care setting	0.7	0.6	0.8	0.6	<0.1	<0.1
Child sexual abuse	0.1	0.4	0.1	0.4	<0.1	0.5
All selected risk factors together	45.3	35.9	45.6	36.1	44.0	34.3

Despite the inclusion of several environmental risk factors in historical burden of disease studies, these assessments were not designed to address issues specific to environmental health. Additional efforts have therefore been undertaken since the original GBD study in response to the recognized importance of environmental (and occupational) factors as predictors of population disease. For example, in 1997, WHO and the International Labour Organization (ILO) convened a consultation comprised of an international group of scientists to review and provide suggestions for refinement of various methods proposed for the quantitative assessment

of how environmental and occupational exposures impact a population's health status (Pastides and Corvalán 1998).

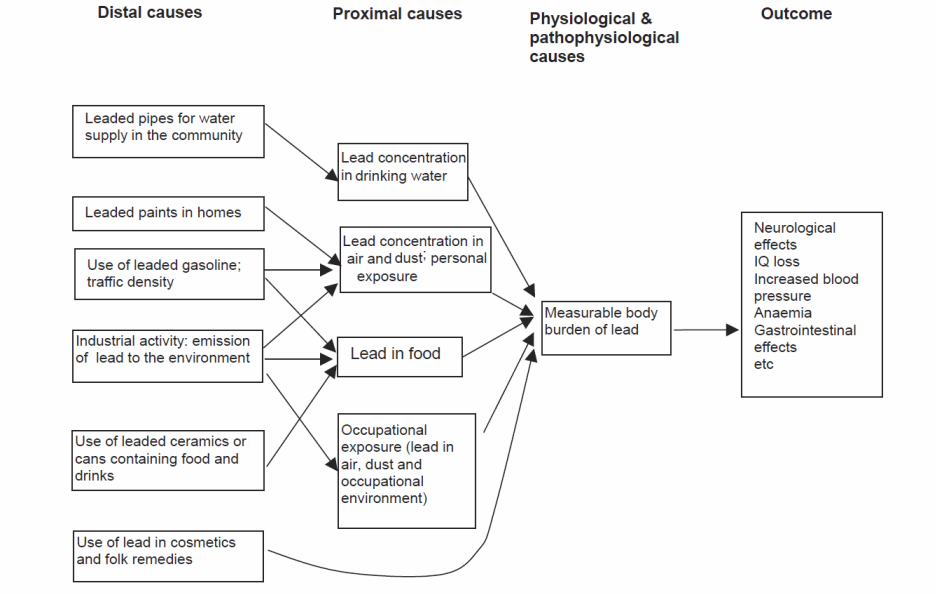
In 2000, following the 12th Annual Meeting of the International Society for Environmental Epidemiology, WHO also organized a consultation to address issues and methods related specifically to the assessment of EBD (Kay et al. 2000). The primary objectives of this workshop were to (1) provide methodological guidance on the quantitative assessment of the burden of disease from environmental risk factors at national or regional level, and (2) create a network of experts interested in developing the conceptual and practical implementation of environmental disease burden assessment. As part of the workshop, participants discussed the observed differences in orders of magnitude between least developed and most developed regions based on previous studies assessing disease burden from environmental risk factors (see Figure 2).

Figure 2. Comparison of order of magnitude of main disease burden from environmental risk factors in developed and developing regions (Source: Kay et al. 2000)



Prüss et al. (2001) also presented a number of environmental risk factors by type of hazard and relative magnitude, and similar to the GBD study, recommended a framework based on a causal web structure that links environmental hazards and risks factors to disease burden. These authors presented examples of previous EBD estimates and discussed the need for harmonized methods for quantifying the impact of environmental and occupational risk factors on disease burden. In 2003, WHO launched the EBD Series, which described a recommended methodological framework for quantitatively assessing the health impacts of environmental risk factors in general, and provided detailed guidance for assessing the health burden of specific environmental risk factors, such as lead exposures (see Figure 3). The EBD series of guides are focused on assessments of national and local populations, which are most relevant for policy-making, and the methods described in the guides and can be tailored to suit data availability (Prüss-Üstün et al. 2003).

Figure 3. Burden of disease framework for exposure to lead (Source: Fewtrell et al. 2004)



WHO's EBD methodological framework builds upon the CRA project described above, which quantified the global burden of disease from 26 risk factors having a major impact on population health, including those related to environmental and occupational health (see Table 5). Based on the results of the CRA, coupled with extensive literature reviews and standardized surveys of expert opinions, subsequent analyses have been conducted by WHO to provide more timely estimates of burden of disease from a much broader range of environmental risk factors (Prüss-Üstün and Corvalán 2006, 2007).

Table 5. Environmental and occupational risk factors included in the Comparative Risk Assessment and WHO's EBD studies (Source: Prüss-Üstün and Corvalán 2006)

Risk Factors	**Related Diseases**
Outdoor air pollution	Respiratory infections, selected cardiopulmonary diseases, lung cancer
Indoor air pollution from solid fuel use	COPD, lower respiratory infections, lung cancer
Lead	Mild mental retardation
Water, sanitation and hygiene	Diarrheal diseases, trachoma, schistosomiasis, ascariasis, trichuriasis, hookwork disease
Climate change	Diarrheal diseases, malaria, selected unintentional injuries, protein-energy malnutrition
Selected occupational factors: Injuries Noise Carcinogens Airborne particulates Ergonomic stressors	 Unintentional injuries Hearing loss Cancers Asthma, COPD Low back pain

3.2 Methodological Approaches to Estimating EBD

There are two commonly-used approaches for estimating EBD: an exposure-based approach and an outcome-based approach (Kay et al. 2000; Prüss-Üstün et al. 2003). The former approach estimates disease burden on the basis of the exposure distribution of a population, while the latter is based on the fraction of a disease burden attributable to a certain risk factor. Although these two approaches share the same underlying assumptions on a health-environment link and its quantification, they require different types or uses of data to estimate EBD. Additionally, although both approaches should yield the same estimates of EBD, this is rarely the case in practice.

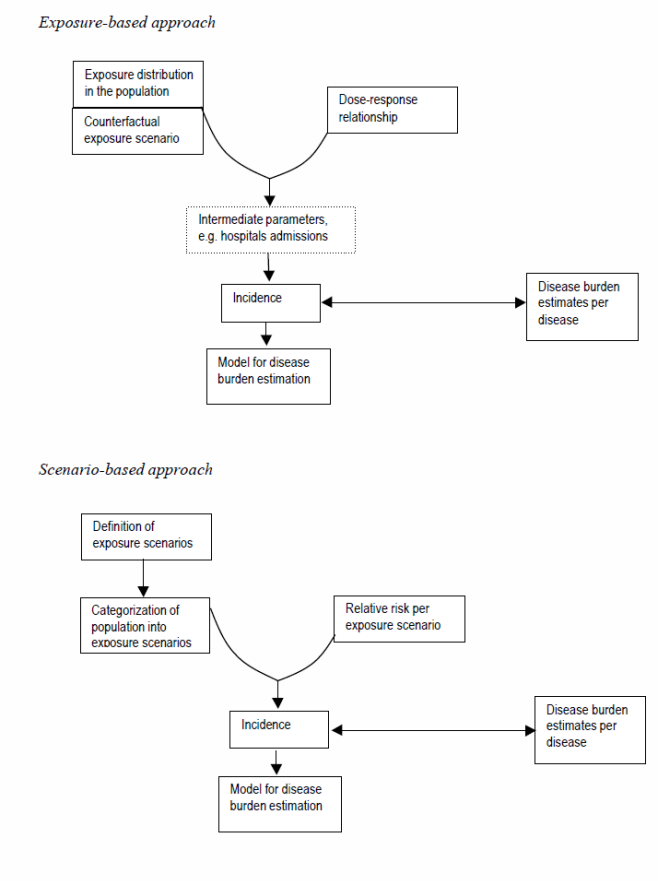
The exposure-based approach generally relies on the following types of data: (1) outcomes associated with relevant environmental risk factors, (2) population-based exposure levels, and (3) dose–response relationships for the exposures and outcomes of interest. The first step in the exposure-based approach requires identifying the specific health outcomes associated with specific environmental exposures. This is typically accomplished by reviewing the available epidemiological literature to assess whether there is evidence to suggest an association between the exposure and outcome of interest. The second step involves quantifying exposure levels in the population relative to the specific environmental chemical or stressor of interest. Exposure estimates are typically based on measured or modeled data for a population, but determining population-level exposures for multiple constituents or pathways can be challenging, and such data are often not available for use in an EBD study. The third step entails establishing dose-response relationships for a chemical or non-chemical stressor to assess the magnitude of adverse health effects associated with varying exposure levels. Quantitative dose-response relationships, which are typically based on human (epidemiology) or animal (toxicology) data, are not available for many chemicals or stressors. Under this approach, EBD is calculated by combining population-level exposure and dose-response estimates to characterize the burden of disease attributable to environmental risk factors. A simplified scenario-based approach can be used when there are complex and competing relationships between exposures, in which the study population is categorized into a number of defined exposure scenarios corresponding to a specific health risk (see Figure 4).

The outcome-based approach relies on the following kinds of data: (1) outcomes associated with relevant environmental risk factors, (2) morbidity and mortality statistics, and (3) estimates of EAF. As with the exposure-based approach, the first step in this approach requires identifying the specific health outcomes associated with specific environmental exposures based on a review of the epidemiological and toxicological literature. The second step, however, entails compiling health statistics data for each country or region to assess the annual number of deaths or illnesses by disease burden category. Such data can generally be obtained from relevant ministries and authorities that track public health. The final, and perhaps most important (and uncertain), step involves calculating the EAF for each disease category to determine what percentage of the estimated disease burden is due to environmental exposures.

Specifically, EAF calculations require knowledge of the prevalence of exposure in a population as well as an estimate of the relative risk (i.e., risk of developing disease for exposed individuals relative to non-exposed individuals in a population). Relative risk estimates are usually obtained

from epidemiological studies, only some of which may have been performed in the population of interest for estimating the disease burden. Due to data limitations, determining the EAF typically relies on a combination of the results of comparative risk assessments and expert judgment (through formal or informal expert elicitation methods) concerning the epidemiology relating exposure to outcome. Although relative risks can vary among populations due to differences in underlying causal relationships, the most important predictors of variation in disease burden is the level and type of exposure within the population and characteristics related to the population's susceptibility (e.g. age group, health status). When population-specific exposure data are unavailable, or deemed to be inadequate, EAF's from other population groups are sometimes used as proxy estimates, with or without adjustment. Uncertainty in estimates are typically reflected in lower and upper estimates or data are presented as best estimates and 95% confidence intervals.

Figure 4. Two approaches for estimating environmental disease burden (Source: Kay et al. 2000)



To date, EAFs developed by WHO (Prüss-Üstün and Corvalán 2006; 2007) and the Organisation for Economic Co-operation and Development (OECD) (Melse and de Hollander 2001) are utilized the most often in published global, regional, and national EBD studies (see Results section below). The WHO method for determining EAFs consisted of systematically reviewing the peer-reviewed literature to compile summaries of the best available evidence of population

health impacts from environmental risks for each of the 102 diseases and injuries identified from global disease statistics and classified according to the International Classification of Diseases. For each disease or injury, the identified literature was selected in the following order of priority: (1) global estimates for selected environmental risks, such as from the CRA; (2) estimates of population health impacts at the regional or national level; (3) meta-analysis or reviews on disease reduction from environmental interventions or determinants of health; and (4) individual studies of interventions and determinants of health. Using these criteria, summaries of the best available evidence were prepared and submitted to at least three experts who were asked to provide their estimates of fractions (best estimate and 95% confidence interval) attributable to the environment for one or more diseases or injuries or their groupings. These estimates therefore rely heavily on expert judgment. The OECD study utilized a similar approach to estimate EAFs in high and lower income OECD regions (i.e., relative risk estimates were obtained from the epidemiology literature where available, and exposure levels were derived from international reports or were based on published estimates).

Another outcome-based approach that differs from the WHO methodology relies on the results of population-based health surveys to estimate the health-related quality of life (HRQOL) associated with a disease or condition and its prevalence (Manuel et al. 2002). While the WHO methodology ultimately expresses outcomes in terms of DALYs, the HRQOL survey-based measure of disability is expressed as either disability-free life expectancy (DFLE) or health-adjusted life expectancy (HALE). The primary advantage of using population-based health surveys is that they directly question individuals about their preferences for particular health-states as well as their own health condition, which in theory, allows analysts to control for potential confounders with fewer assumptions when conducting country-specific EBD assessments. However, the reliance on self-reported measures of health introduces a significant element of subjectivity, and this method does not provide an estimate of the impact of various risk factors which is ultimately required for an EBD (i.e., it merely estimates the impact of changes in prevalence of particular disease states rather than providing an estimate of EAF for each risk factor).

4.0 Results

4.1 Global, Regional, and National EBD Studies

In a review of historical EBD studies and methodologies, Prüss et al. (2001) summarized nine studies conducted during the early to late 1990s (see Table 6) and selected EAF results for different environmental risk factors (see Table 7). A comparison of these studies illustrates how previous EBD assessments have varied considerably with respect to geographic scope (e.g., global, regional national), type of risk factor (e.g., environmental, lifestyle, or occupational), specific environmental risk factor (e.g., outdoor air quality, lead), methodology (e.g., exposure-based, outcome-based), and units of analysis (e.g., mortality, DALYs). These studies also show how the magnitude of disease burden is likely to differ among risk factors in different geographic areas (e.g., a greater percentage of disease burden is attributed to indoor air quality in developing countries, such as India, compared to developed countries, such as the Netherlands). Indeed, although Smith et al. (1999) estimated that the global burden of disease due to the environment was as high as 25% to 33%, with children under 5 years of age experiencing the largest

environmental burden, many of the risk factors identified in this study were related to environmental conditions experienced in developing countries (e.g., risk factors related to sanitation, clean water, and indoor biomass burning), with the portion of disease due to environmental risks decreasing with economic development. Because these studies are likely based on outdated health statistics and exposure data, and EBD estimates are largely driven by conditions in less developed countries, they are not very useful for evaluating the current EBD in Canada (but they do provide historical data which may be used to benchmark against Canada-specific estimates).

Table 6. Summary of global, regional, and national EBD studies conducted during the early to late 1990s (Source: Prüss et al. 2001)

Reference	**Level**	**Risk Factors**	**Method**	**Quantitative Estimate**	**Comparability among Risk Factors**
de Hollander et al. 1999	National Netherlands)	Several for which sufficient data were available	Exposure-based	DALYs	Yes
Smith et al. 1999	Global	Several	Outcome-based	DALYs	Yes
Smith 1999	India	Indoor air quality	Outcome-based	Mortality, DALYs	Yes
Schwela 1998	Global	Air quality	Exposure-based	Mortality	No
Murray et al. 1996	Global	Air pollution; Water supply, sanitation, and hygiene; Occupational	Outcome-based	DALYs	Yes
Peritaz et al. 1995	Regional (Canton Geneva)	Several	Exposure-based	No	No
U.S. EPA 1993	Various US regions	Several	Exposure-based	Mortality, morbidity	Qualitative
Leigh et al. 1997	United States	Occupation	Outcome-based	Mortality, morbidity	No
USAID 1990	Bangkok	Several	Exposure-based	Mortality, morbidity	Qualitative

Table 7. Summary of selected results for global, regional, and national EBD studies conducted during the early to late 1990s (Source: Prüss et al. 2001)

Risk Factor	Reference	Incidence/Year	Premature Deaths/Year	DALYs (95% CI)	% of Total Disease Burden of Study Area
Outdoor air quality (long term)	de Hollander 1999; Netherlands	22,000	8,500	75,900 (45,100 – 106,500)	3%
Outdoor air quality (short term)	de Hollander 1999; Netherlands	530,000	1,100	2,900 (1,500 – 4,700)	0.12%
Outdoor air quality	Murray 1996; Global	NR	506,800	7,254,000	0.5%
Outdoor air quality	US AID 1990; Bangkok	9-51 million restricted activity days	300 – 1,400	NR	NR
Indoor air quality	de Hollander 1999; Netherlands	780,603	540	7,610 (3,770 – 12,300)	0.31%
Indoor air quality	Smith 1999; India Global	NR	 500,000 2,000,000	 11-16M 55-83M	 4-6% 4-6%
Lead	de Hollander 1999; Netherlands	1,760	0	7,950 (980 – 18,700)	0.32%
Lead	US AID 1990; Bangkok	96k children (IQ loss 1-3pts); 32k children (IQ loss 4+ pts); 320k cases of hypertension	210 (cardiovascular disease)	NR	NR
Noise	de Hollander 1999; Netherlands	2,800,830	40	28,750 (7,370 – 53,600)	1.2%
Food	de Hollander 1999; Netherlands	1,093,000	48	4,240 (760 – 12,300	0.17%
Water, sanitation and hygiene	Murray 1996; Global	NR	2,668,000	93,392,000	6.8%
Microbiologic and infectious disease	US AID 1990; Bangkok	170k	NR	NR	NR
UV radiation	de Hollander 1999; Netherlands	2,530	20	530 (220 – 900)	0.02%
Traffic accidents	de Hollander 1999; Netherlands	42k	1,320	74,570 (52,900 – 97,500)	3%
Traffic accidents	Murray 1996; Global	14,405,000	999k	34,300,000	2.4%
Domestic accidents	de Hollander 1999; Netherlands	1,630,300	2,020	106,100 (73,400 – 163k)	4.2%
Occupation	Murray 1996; Global	NR	1,129,000	37,887,000	2.7%

4.1.1 Global/Regional EBD Studies

Since the 1990s, a number of global and regional EBD studies have been conducted that analyze data from various international sources or which are based on the original GBD studies described above (see Appendix A, Table A-1). Although these studies define environmental risk factors in different ways, we attempt to limit our discussion to EBD estimates that pertain specifically to environmental risk factors that lead to population-level exposures, rather than estimates that include personal environment (lifestyle) or occupational exposures. We also attempt to distinguish between EBD estimates for developing versus developed countries, the latter of which are the most applicable to the Canadian context (see column "Data Most Relevant to Canada" in Table A-1). Additionally, it should be noted that the vast majority of global/regional EBD studies have included a wide range of disease outcomes and risk factors, with only a few studies evaluating disease burden attributable to a specific environmental risk factor (e.g., air pollution, lead).

In a study of the environmental health impact within the OECD region in 1998, Melse and de Hollander (2001) quantified the total burden of disease attributable to environmental risk factors in low and higher-income regions (see Table 8) and for 16 disease categories (see Table 9). In this study, "environmental" was defined as any physical, chemical, or biological human made or influenced exposures, including some personal environment exposures (e.g., environmental tobacco smoke [ETS]), but other important lifestyle factors (e.g., smoking, diet) and occupational exposures were excluded. EAFs were estimated based on relative risks taken from the literature and exposure levels derived from international reports. Similar to the GBD study findings, the burden of disease was found to be considerably higher in developing countries, with non-OECD regions bearing nearly twice the burden of disease per capita compared to OECD regions. Estimated EAFs ranged from 2-5% in the OECD region and 8-12% in non-OECD countries. Within the OECD region, EAFs were estimated to range from 1.4-4.3% and 3.7-6.7% for high-income and low-income countries, respectively. EAFs varied considerably by disease category, accounting for 80-90% of diarrhea in both lower and higher income regions, and 10-20% and 5-15% of acute respiratory infections in lower income and higher income regions, respectively. Air pollution and noise were identified as important environmental causes of lost health in developed countries, primarily due to the transport and energy sectors. In this study, estimates for the higher-income OECD regions are likely to be the most relevant with respect to evaluating the EBD in Canada, although the underlying sources of data used in this study may be somewhat outdated.

Table 8. Total estimated EBD based on 1988 OECD Study (Source: Melse and de Hollander 2001)

	OECD			**Non-OECD Total**	**World Total**
	High Income	**Low Income**	**Total**		
Population in millions (%)	884 (80% OECD)	224 (20% OECD)	1,108 (19%)	4,797 (81%)	5,905 (100%)
Burden of Disease (in DALY/1000 cap)	120	190	134	258	235
Communicable diseases	8 (7%)	41 (22%)	15 (11%)	115 (44%)	96 (41%)
Non-communicable diseases	97 (81%)	115 (60%)	101 (75%)	101 (39%)	101 (43%)
Injuries	14 (12%)	34 (18%)	18 (14%)	42 (16%)	38 (16%)
Environmental Fraction	1.4-4.3%	3.7-6.7%	2.1-5%	8-12%	7.4-11%

In an effort to use a more unified framework than that used in prior assessments, Ezzati et al. (2002) estimated the contribution of 26 major risk factors to global and regional burdens of disease for 14 epidemiological subregions of the world (Canada was included along with Cuba and the United States in the Americas region). These included several specific environmental risk factors (e.g., unsafe water, sanitation, and hygiene; urban outdoor air pollution; indoor smoke from solid fuels; lead, global climate change) and many other lifestyle and occupational risk factors (e.g., vitamin deficiency, malnutrition, fruit and vegetable intake, physical activity, sexual practices, tobacco and alcohol consumption, occupational injuries and exposures). For each risk factor, expert working groups conducted a comprehensive review of published work and other sources to obtain data on the prevalence of risk factor exposure and hazard size (e.g., relative risk). Population attributable fractions (by sex) were then estimated and applied to the mortality and burden of disease (DALY) estimates from the 2000 GBD database for each risk factor and subregion. The results of this study are presented in the original publication and selected results are summarized in a review article by Briggs (2003), in which it was concluded that about 8–9% of the total disease burden globally could be attributed to pollution from environmental (broadly defined) and occupational risk factors (Table 10). The most important specific environmental risk factors identified in this analysis were indoor air pollution from solid fuels and water, sanitation, and hygiene (the health impacts from outdoor air pollution were comparatively small). However, as has been observed in other global EBD studies, risks attributable to environmental pollution were about 15–35 times greater (or more) in the developing world than in developed countries. These global estimates are therefore not particularly relevant for evaluating the EBD in Canada; instead, the region-specific estimates presented by Ezzati et al. (2002) for the Americas region are the most relevant for the Canadian context (i.e., these data suggest that approximately 3% of the total disease burden in this subregion could be attributable to environmental risk factors).

Table 9. Estimated EBD by disease category based on 1988 OECD Study (Source: Melse and de Hollander 2001)

Disease Category	**% Disease Burden Low- and Middle-Income**	**EAF (%)**	**% Disease Burden High-Income**	**EAF (%)**
Acute respiratory infections	6.6	10-20	1.4	5-15
Perinatal conditions	6.2	1-5	1.9	1-5
Diarrhea	5.7	80-90	0.3	80-90
STD/HIV	5.5	0-1	0.9	0-1
Cancer	5.1	1-5	15.0	1-5
Child cluster	4.4	5-10	0.4	1-5
Depression	4.0	1-5	6.5	1-5
Malnutrition	3.4	5-10	0.9	1-5
Ischaemic heart disease	3.3	5-20	8.8	5-15
Malaria	3.1	70-80	0.0	--
Cerebrovascular disease	2.9	1-5	4.8	1-5
Chronic respiratory disease	2.9	5-15	3.4	5-15
Road traffic accidents	2.7	5-10	4.2	5-10
Maternal conditions	2.5	1-5	0.4	1-5
Tuberculosis	2.2	5-10	0.1	5-10
Congenital anomalies	2.1	0-1	1.8	0-1

Table 10. EBD estimates for selected environmental and occupational risk factors based on WHO GBD study (Source: Briggs 2003)

Risk Factor	Deaths		DALYs	
	Number	%	Number	%
Total (all risk factors)	55,861		1,455,473	
Water, sanitation and hygiene	1730	3.1	54,158	3.7
Urban outdoor air pollution	799	1.4	6404	0.4
Indoor smoke from solid fuels	1619	2.9	38,539	2.6
Lead	234	0.4	12,926	0.9
Occupational carcinogens	118	0.2	1183	0.1
Occupational airborne particulates	356	0.6	5354	0.4
Occupational noise	0	0.0	4151	0.3
Total (pollution-related)	4856	8.7	122,715	8.4

In a follow-up analysis, Ezzati et al. (2003) estimated the burden of disease and injury attributable to the joint effects of 20 selected leading risk factors in the same subregions of the world in 2000. As above, these included several specific environmental risk factors and many other lifestyle and occupational risk factors. Approximately 47% of premature deaths and 39% of total disease burden globally were estimated to be attributable to these combined risk factors, and removal of these risks were estimated to increase global healthy life expectancy by 17% (only 6% in developed countries such as the Western Pacific). Although selected specific environmental risk factors (e.g., indoor smoke from solid fuels and unsafe water, sanitation and hygiene) were found to contribute significantly to global disease burden for some diseases (e.g., lower respiratory infections and diarrheal disease), these environmental risk factors did not appear to contribute significantly to the disease burden in more developed regions. For example, indoor smoke from solid fuels was estimated to account for only 0% and 2% of the disease burden for trachea bronchus and lung cancers and chronic obstructive pulmonary disease (COPD), respectively, in developed regions. Similarly, Danaei et al. (2005) estimated mortality from site-specific cancers attributable to nine risk factors (individually and jointly) for seven World Bank regions in 2001. These primarily included different lifestyle risk factors (e.g., smoking, alcohol use, fruit and vegetable intake, obesity, physical activity) and a couple of specific environmental risk factors (e.g., urban air pollution; indoor smoke from solid fuels), but did not include occupational exposures. Although approximately 35% of cancer deaths worldwide were estimated to be attributable to these combined risk factors, <0.5% and 1% of the disease burden was attributable to indoor smoke from household use of solid fuels and urban air pollution, respectively. Both of these studies contain potentially useful data for evaluating the likely contribution of environmental risk factors to the disease burden in Canada, but only if estimates for developed countries are used and the definition of environmental risk factor is understood.

Other global/regional EBD studies have evaluated the burden of disease attributable to specific environmental risk factors such as water, sanitation, and hygiene (Prüss et al. 2002), lead (Fewtrell et al. 2003, 2004), air pollution from fine particulates (Cohen 2005), and ultraviolet (UV) radiation (Lucas et al. 2008). These studies have found that some of these environmental risk factors continue to contribute significantly to mortality and disability in certain regions of the world, particularly among children in developing countries. However, only a fraction of the total disease burden in these studies is attributed to these environmental risk factors in the Americas region, and these latter data are the most relevant for evaluating the EBD in Canada.

In the most recent global EBD study conducted by WHO, EAFs were developed for many disease categories and environmental risk factors, which included lifestyle factors and occupational exposures, based on an updated comparative framework (Prüss-Üstün and Corvalán 2006, 2007; WHO 2009a). Attributable fractions were estimated based on a systematic literature review conducted by a survey of experts using a variant of the Delphi method, which is a formal expert elicitation method. In this study, it was estimated that 24% of the global burden of disease was attributable to environmental risk factors (broadly defined), with 34% of the global disease burden in children attributable to the environment. Note that EAF estimates in this study were higher than some prior estimates due to differences in study design (e.g., standardized surveys of expert opinions) and the inclusion of a broader range of environmental risk factors (e.g., lifestyle and occupational risks). As in prior assessments, EAFs varied widely across regions and were much greater in developing than developed countries. In the Americas subregion, 15-22% of the disease burden was attributable to environmental, lifestyle, and occupational risk factors. Although many of the identified risk factors and estimated EAFs from this study are not likely to be applicable to developed countries, such as Canada, selected data for certain disease categories and risk factors may be useful for benchmarking against Canadian-specific estimates (see Table 11).

4.1.2 EBD Studies Conducted in the United States

Additional EBD studies, focused primarily on children, have been conducted at the national level or for specific locations within the United States (see Appendix A, Table A-2). All of these studies focused exclusively on specific environmental risk factors that lead to population-level exposures (i.e., none included lifestyle factors or occupational exposures). These studies also included a range of disease outcomes and risk factors, rather than a single outcome or factor, although these were generally more limited in scope than those included in the global/regional studies.

The study by Landrigan et al. (2002) estimated the contribution of environmental pollutants to the incidence, prevalence, mortality, and costs of pediatric disease among American children (see Table 12). Specifically, the burden of disease attributable to chemical pollutants in the ambient environment was calculated for four categories of illness. Data on disease rates and costs were based primarily on national data collected by federal government agencies. EAFs for lead poisoning, asthma, and cancer were developed by three panels of experts using a Delphi process, while the EAF for neurobehavioral disorders was based on recently published findings from the National Academy of Sciences (NAS) in the United States. In this study, best estimates of EAFs were 100% for lead poisoning, 30% for asthma, 5% for cancer, and 10% for neurobehavioral disorders. EAF estimates from this study are potentially relevant for evaluating the EBD among Canadian children because they are based on extensive reviews of the available exposure and epidemiology literature and focus on disease categories that are relevant for developed countries. However, as is the case for most EBD studies, EAF estimates are highly uncertain (particularly for cancer) and are largely dependent on expert judgment. As noted by the study authors, the primary limitation of this study was a lack of etiologic research quantifying the contribution of environmental factors to causation of pediatric diseases.

Table 11. EAF Estimates developed by WHO that are potentially relevant for developed countries such as Canada (Source: Adapted from Prüss-Üstün and Corvalán 2006; 2007)

Disease or Injury	**Environmental Risk Factor or Intervention Area**	**EAF Best Estimate (95% confidence interval)**
Perinatal conditions	Mother's exposure to ETS, chemicals, air pollution	6 (2-10)[1]
Congenital anomalies	Mother's exposure to chemicals, radiation, air pollution	5% (2-10)[2]
Cardiovascular disease	Stressful workplace conditions, air pollution, ETS, lead	14% (7-23)[2]
COPD	Exposure to dusts and chemicals in the workplace, exposure to indoor and outdoor air pollution	27 (19 - 35) males[1] 9 (6 - 12) females[1]
Asthma	Indoor exposures to dust mites and fungal allergens, possible indoor smoke from solid fuels, ETS, exposure to outdoor air quality, occupational exposure to allergens	44% (26-53)[2]
Cancer (overall)	Exposure to chemicals, outdoor and indoor air pollution, ETS, ionizing radiation, UV radiation (exposures at work and other settings)	30 (6-55) lung cancer (males and females)[1] 16 (10-34) other cancer (males)[1] 13 (10-23) other cancer (females)[1]
Neuropsychiatric disorders	Occupational stress has been linked to depression, noise exposure to insomnia, exposure to chemicals to Parkinson disease, drug use and alcohol disorder to the occupational environment, posttraumatic stress disorders to natural disasters which could partially be prevented by environmental measures, epilepsy to occupational head trauma, mild mental retardation to childhood exposure to lead	13% (10-16)[2]

[1]Specific to developed countries or industrialized regions
[2]Global estimates (not separated by region)

Table 12. EAF and cost estimates for pediatric disease among American children (Source: Landrigan et al. 2002)

	EAF (%)		Costs (billion $)	
	Best Estimate	Plausible upper and lower bound	Best Estimate	Low and high estimate
Lead poisoning	100	N/A	43.4	N/A
Asthma	30	10-35	2.0	0.7-2.3
Cancer	5	5-90	332	132-663
Neurobehavioral disorders	10	5-20	9.2	4.6-18.4

The EAF and cost estimates derived by Landrigan et al. (2002) have been used in other statewide assessments in the United States to quantify the disease burden and economic costs associated with major diseases and disabilities attributable to environmental contaminants in these states. This includes EBD studies focused on children in Massachusetts (Massay and Ackerman 2003), children and adults in Washington State (Davis and Hauge 2005), and children in Minnesota (Shuler et al. 2006). The primary conclusion of these statewide studies is that preventable childhood illnesses and disabilities attributable to environmental factors are associated with large monetary costs. However, because these studies are focused on direct and indirect costs at the local (state) level, and do not provide original EAF estimates for most disease categories, they are of limited independent value in evaluating the EBD in Canada (with the potential exception of new EAF estimates provided for cardiovascular disease and birth defects).

4.1.3 EBD Studies Conducted in Europe

Several EBD studies, primarily focused on children, have also been conducted for various countries or regions in Europe (see Appendix A, Table A-3). Similar to the EBD studies conducted in the United States, these studies focused primarily on risk factors related to environmental pollution, rather than risk factors related to the personal environment (lifestyle factors) or occupational exposures. Although generally more limited in scope than those included in the global/regional studies, these studies also included a range of disease outcomes and risk factors.

For example, Valent et al. (2004) evaluated the burden of disease attributable to four environmental risk factors among children and adolescents in three subregions of Europe (see Table 13). Although most of these risk factors related to population-level exposures in the environment, the broader category of injuries was also included in this study. EBD estimates were based on a review of published studies and reports from international agencies with respect to risk factor exposures and exposure-response relationships coupled with burden of disease estimates from the WHO GBD 2001 database. The primary impetus for this work was to provide the knowledge base for development of the Children's Environment and Health Action Plan (CEHAPE) for the European Region. Among children aged 0–4 years, it was estimated that 1.8-6.4% of deaths from all diseases were attributable to outdoor air pollution, 4.6% of deaths and 3.1% DALYs from acute lower-respiratory-tract infections were attributable to indoor air pollution, and 4.4% of DALYs from mild mental retardation were a result of lead exposure. In the age-group 0–14 years, 5.3% of all deaths and 3.5% of DALYs from diarrhea were attributed to inadequate water and sanitation. Finally, in the age-group 0–19 years, 22.6% of all deaths and 19% of all DALYs were attributed to injuries. Besides varying by age group, the burden of disease was found to vary significantly by subregion, and was much higher in European subregions B and C than subregion A. This study provides potentially useful data for evaluating the EBD in Canada, particularly if comparable age groups and regions are selected for comparison, but results are limited to a small number disease outcomes and risk factors. Additionally, as acknowledged by the study authors, there is substantial uncertainty around some of the estimates (especially for outdoor air pollution) due to a lack of valid exposure data and strong evidence of exposure–response relationships.

Table 13. EBD estimates for deaths and DALYs for children in 3 age groups and 3 subregions in Europe (Source: adapted from Valent et al. 2004)

	% of Deaths from all causes			% of DALYs from all causes		
	0-4 years	**5-14 years**	**15-19 years**	**0-4 years**	**5-14 years**	**15-19 years**
Outdoor air pollution						
Subregion A	<0.1-0.8	--	--	--	--	--
Subregion B	2.4-7.5					
Subregion C	0.9-5.8					
Total	1.8-6.4					
Household solid fuel use						
Subregion A	0	--	--	0	--	--
Subregion B	6.6			5.0		
Subregion C	1.1			0.7		
Total	4.6			3.1		
Inadequate water/sanitation						
Total[1]	9.6	0.8	--	7.9	1.0	--
Lead	--	--	--		--	--
Subregion A				2.3		
Subregion B				4.5		
Subregion C				5.0		
Total				4.4		
Injuries						
Total[2]	6.0	41.2	59.9	7.3	29.8	27.1
All	21.9-26.5	42.1	59.9	22.7	30.8	27.1

[1]Data for subregions based on combined age groups (0-14 years); % deaths: 0.2 (A), 7.5 (B), and 2.4 (C); % DALYs: 0.8 (A), 5.2 (B) and 1.6 (C)

[2]Data for subregions based on combined age groups (0-19 years); % deaths: 30.2 (A), 10.7 (B), and 38.8 (C); % DALYs: 14.9 (A), 13.8 (B) and 29.1 (C)

In a separate study, Mathews and Parry (2005) evaluated the burden of disease attributed to environmental pollution for a larger number of health outcomes among children in England and Wales. In this study, EAFs (best estimates) for asthma (30%), cancer (5%), and neurodevelopmental disorders (10%) were based on the study by Landrigan et al (2002) and the results of the expert committee convened by the NAS. Although the burden of disease from lead exposure was also considered based on WHO's assessment, it is not entirely clear what EAF estimate was used in this study. Because no data were available on the EAF for allergic disease, the study authors assumed that the exposures and mechanisms involved in the etiology and exacerbation of asthma were similar to those involved in allergy, and the percent of children with skin complaints and adults with allergic rhinitis were used to infer an EAF of 3.3% for allergies. An EAF of 20% was also estimated for congenital abnormalities based on the finding that 80% of the population in the United Kingdom reside within 2 Km of a landfill site and the relative risk of a congenitally malformed baby for mothers in this region was approximately 1.01, with similar studies in Europe and Wales reporting relative risks ranging from 1.19 to 1.39 (i.e., the EAF was chosen as the mid-range value of the relative risk estimates; i.e., 1.20). An EAF of 6.3% was estimated for respiratory disease based on the proportion of children with low measured lung functions at the lowest and highest levels of exposure to $PM_{2.5}$ in a prospective cohort study of children in the United States, while an EAF of 0.8% was estimated for cardiovascular disease based on a meta-analysis of short-term associations between ambient PM concentrations and hospital admissions. Note that the key findings from this study are presented in a publication by the Health Protection Agency (2005), which is an independent body in the

United Kingdom that was established by the federal government in 2003 to protect the public from threats to their health from infectious diseases and environmental hazards. This study presents EAF estimates for several health outcomes not previously considered that are potentially useful for evaluating the EBD in Canada, but as recognized by the study authors, these estimates are based on very uncertain data and limited data sets and need to be interpreted with extreme caution.

Knol and Staatsen (2005) evaluated trends in the EBD in the Netherlands for 49 groups of diseases based on several specific environmental risk factors. Population exposures were estimated using measured and modeled data, and relative risks were obtained from recent Dutch epidemiological studies or relevant international estimates. Expert judgment was used when data were missing or uncertain. In this study, approximately 2-5% of the total disease burden (i.e., DALYs) was attributed to the effects of short-term exposure to air pollution, noise, radon, total natural UV radiation, and dampness in houses for the year 2000. The more uncertain effects of long-term exposure to PM_{10} had the greatest impact on the environment-related disease burden, and inclusion of this exposure increased the estimated EBD to slightly over 10% assuming no threshold and 3-9% assuming a reference level of 20 micrograms per cubic meter ($\mu g/m^3$). Estimated DALYs related to short-term PM_{10} exposures were also greater using international exposure-response relationships compared to Dutch values. Based on calculations made for the past (1980 or 1990) and the future (2010 or 2020), it was concluded that the disease burden related to PM_{10} exposures would likely decrease, the disease burden related to noise would likely increase, and the disease burden related radon and UV radiation would likely remain the same. Although this study provides useful information with respect to the contribution of several risk factors to total disease burden, EBD estimates are not presented separately by disease outcome or risk factor, thereby hindering the use of this study for other applications. The authors also caution that data used to derive exposure and exposure-response estimates are highly uncertain (or unknown) and EBD estimates are based largely on informal expert opinion.

It is noteworthy that there are several ongoing research studies in Europe related to the development of the next generation of EBD studies. For example, the Health and Environment Integrated Methodology and Toolbox for Scenario Assessment (HEIMTSA) and the Integrated Assessment of Health Risks of Environmental Stressors in Europe (INTARESE) bring together international multi-disciplinary teams of scientists who are collaborating on developing and applying new integrated approaches to the assessment of environmental health risks and consequences in support of various European policies (http://www.heimtsa.eu/; http://www.intarese.org/). These research efforts should provide relevant data in the future with respect to the relationship between environmental exposures and population health outcomes in Europe, and may provide a useful framework for other national or local-level EBD studies.

4.2 Canada-Specific EBD Studies

4.2.1 Canadian EBD Studies

Over the last five years, several EBD studies have been proposed or conducted at the national and local levels in Canada (see Appendix A, Table A-4). Most of these studies have focused on specific environmental risk factors that lead to population-level exposures, although ongoing efforts in Canada and WHO's country-specific estimates include factors related to the personal

environment (lifestyle factors) and occupational exposures. Some of these studies have included a range of disease outcomes and risk factors (although these are usually limited in scope), while other studies have attempted to evaluate disease burden attributable to a specific environmental risk factor (e.g., air pollution) or for a specific health outcome (e.g., cancer).

Potentially the most significant effort currently underway with respect to estimating the EBD in Canada is the Population Health Impact (PHI) of disease in Canada program, which is sponsored by the PHA (2006). This program is intended to provide summary measures of population health that combine the impact of both death and reduced functioning. Specifically, this program will assess the impact of approximately 200 diseases, injuries, and risk factors using a single, comparable indicator. The PHI builds on the WHO burden of disease studies by adapting their methods to address diseases and injuries most relevant to Canadians, applying them to Canadian data, and measuring them within a Canadian societal context. Although this program is still in its infancy, some preliminary results are available for two health outcomes: cancer and diabetes. For cancer, the PHI has determined that (in order of priority) smoking, lack of fruit and vegetables in the diet, physical inactivity, obesity, and alcohol account for nearly 35% of all cancer deaths in Canada. For diabetes, the PHI has not yet estimated attributable fractions, only preference values for particular health states associated with this disease. The PHI goes beyond current methods by using microsimulation models that integrate many diseases and risk factors simultaneously and model the interplay between them. The goal is to develop models to provide policy analysts with a broader and more realistic context that considers how diseases and risk factors overlap and interact. For example, "what-if" scenarios can examine how a change in one disease or risk factor may affect several others at the same time. This information could be used to assess various intervention options to identify which ones would provide an optimal return on population health investments. If successful, the outcome of the PHI project will provide policymakers with a useful set of integrative tools to evaluate the EBD in Canada within a larger burden of disease context. The PHI program will also likely provide valuable data for the Population Health Intervention Research Initiative for Canada (PHIRIC), which is a self-organized consortium consisting of many public Agencies across Canada focused on improving population health intervention research in Canada (Sullivan 2009; CIHR 2006).

To date, the only national estimate of EBD across all health outcomes and risk factors in Canada is provided by the WHO as part of its annual country-specific EBD profiles. Specifically, WHO (2009b) estimated that approximately 13% of all preventable diseases in Canada were related to environmental causes, which includes secondary ETS, occupational exposures and stress, and selected lifestyle factors (see Table 14). According to the WHO assessment, the disease categories that contributed the most to the total burden of disease in Canada were neuropsychiatric disorders, cardiovascular diseases, lung and other cancers, other unintentional injuries, asthma, and musculoskeletal diseases. With the exception of cardiovascular disease, the EBDs for these disease outcomes are notably higher in Canada compared to some other countries worldwide. The WHO summary provides estimates for only two environmental risk factors: outdoor air pollution (mean urban PM_{10}) and water, sanitation and hygiene (diarrhea only). Combined, these risk factors are estimated to account for approximately 7% of the total reported preventable deaths and 4% of the total reported preventable DALYs/1000 capita each year in Canada (the contribution from other risk factors is not provided). The WHO EBD estimates are based on the EAFs developed under the CRA project and applied to Canada, combined with

expert evaluation for regional exposures and WHO 2004 country health statistics (Prüss-Üstün and Corvalán 2007).

The WHO estimates are useful for understanding the EBD in Canada from a high-level perspective in that potentially important diseases and environmental risk factors are identified. However, the country profile is not very informative with respect to designing or evaluating intervention strategies in Canada because the contribution of individual (or joint) risk factors to specific disease outcomes is not specified. This level of detail is important and necessary because the WHO definition of the environment includes a wide range of risk factors (see Table 11) and the contribution of each of these risk factors to different disease categories is likely to vary considerably. The underlying exposure and dose-response data used to calculate EAFs in this assessment are also not specific to Canada, thereby increasing the uncertainty in these estimates.

Table 14. WHO country profile of EBD for Canada (source: WHO 2009)

	Deaths/year	**DALYs/1000 cap/year**
Disease Group		
Diarrhea	--	0.3
Respiratory infections	--	0.1
Lung cancer	--	1.2
Other cancers	--	2.3
Neurosphyschiatric disorders	--	2.4
Cardiovascular disease	--	2.4
COPD	--	0.4
Asthma	--	1.0
Musculoskeletal diseases	--	0.9
Road traffic injuries	--	0.4
Other unintentional injuries	--	1.2
Intentional injuries	--	0.7
Risk factor	--	
Water, sanitation and hygiene (diarrhea only)[1]		0.2
Outdoor air	2,700	0.4
Total	36,800	15

[1]According to other WHO documentation, this risk factor includes aspects of food safety (e.g., food contamination).

The only other study that provides national-level EBD estimates for Canada for several health outcomes and risk factors was conducted by Boyd and Genuis (2008), in which rough estimates of EBD were provided for respiratory disease, cardiovascular disease, cancer, and congenital affliction (see Table 15). This study relied on statistics of morbidity and mortality obtained from the Canadian Institute for Health Information, Statistics Canada, Health Canada, Canadian Lung Association, Canadian Cancer Society, and the National Cancer Institute of Canada. The authors combined these statistics of disease incidence and mortality in Canada with the EAFs calculated primarily by WHO (Prüss-Üstün and Corvalán 2006) and OECD (Melse and de Hollander 2001) for developed countries, although they also selectively reviewed EAFs from the published literature.

Specifically, with respect to respiratory disease, the primary environmental sources identified were indoor and outdoor air pollution. EAFs reported in the literature for respiratory diseases

ranged from 10-53%, depending on what was included in the definition of environmental sources (e.g., WHO's estimates include indoor and outdoor secondary ETS and occupational exposures to dust, chemicals, and smoke). Boyd and Genuis (2008) ultimately relied on the WHO EAF estimates of 10%-30% for COPD and 26%-53% for asthma to conclude that the burden of respiratory disease caused by modifiable environmental exposures includes 34,000–93,000 hospitalizations, 200,000–570,000 days in the hospital, and between 1050 and 3100 deaths each year in Canada. For cardiovascular disease, the primary environmental factors identified were air pollution and lead. WHO's best estimate for an EAF for cardiovascular disease in North America was 16% (range 7.5-23%), but this included occupational factors (including stress), while the OECD estimate ranged from 5%-15%. Based on the available data and assuming a narrower definition of the environment, Boyd and Genuis (2008) selected an EAF ranging from 7.5-15%, and estimated the environmental burden of cardiovascular disease in Canada to be 5500–11,000 deaths, 33,000–67,000 hospitalizations, and 291,000–583,000 patient-days spent in the hospital. For cancer, Boyd and Genuis (2008) relied on an EAF ranging from 5-15%, which included exposure to contaminated drinking water, asbestos, radon, air pollution, and environmental tobacco smoke. The selected EAF range was higher than the 1-5% range used by the OECD and the 2-10% range used by other researchers because, according to the study authors, known cases of environmentally-linked cancer represent at least 5% of cancer deaths in Canada (although no specific source was given for this statement). The selected EAF range was also lower than the WHO global EAF range of 19-29%, given that these estimates represented a broader definition of environmental risk factors. The estimated EAF resulted in mortality and morbidity in Canada caused by cancer attributable to adverse environmental factors ranging from 3400–10,200 deaths, 8000–24,000 new cases of cancer diagnosed, 11,000–32,000 hospitalizations, and 104,000–312,000 patient-days spent in the hospital. Finally, with respect to congenital afflictions, Boyd and Genuis (2008) relied on an EAF ranging from 2-10% based on WHO to estimate the burden of congenital affliction in Canada attributable to adverse environmental factors as 72–360 deaths, 128–640 serious congenital anomalies, 300–1500 hospitalizations, 2000–10,000 patient-days spent in the hospital, and 500–2500 low birth weight babies.

The EBD estimates from this study provide a general indication or approximation of risk and are likely to be the most relevant for Canada at this time, given the reliance on up-to-date country statistics and an initial attempt to evaluate the relevance of existing EAF estimates. However, the results of this study are largely driven by the EAFs from WHO, which in turn, are based primarily on expert judgment and consider a broad range of environmental risk factors. As was the case for the WHO (2009) country-specific EBD profile, no details are presented with respect to which specific risk factors (or set of factors) contribute the most to each of the four disease categories, and it is unclear whether (or to what extent) these EAFs are applicable to Canada. Without more detailed or country-specific information, it is not possible to determine what types of public health intervention strategies will be the most effective at different geographic scales. The study authors acknowledged these limitations and recommended that further research on the EBD in Canada should strive to refine the broad ranges of EAFs identified in this study.

Table 15. Estimates of EBD by disease category in Canada (source: Boyd and Genuis 2008)

	EAFs used to calculate EBD	**EBD (number attributable to environment)**		
Outcome		**Deaths**	**Hospitalizations**	**Days in Hospital**
Respiratory disease				
COPD	10 - 30% (WHO)	977 - 2932	25,646 - 76,938	170,611 - 511,832
Asthma	26 - 53% (WHO)	75 - 153	8060 - 16,430	28,448 - 57,989
Cardiovascular disease	7.5 - 15% (WHO, OECD)	5456 - 10,911	33,541 - 67,083	291,419 - 582,838
Cancer	5 - 15% (professional judgment)	3416 - 10,248	10,775 - 32,324	103,948 - 311,845
Congenital affliction	2 - 10% (WHO)	72 - 360	312 - 1558	1982 - 9910
Totals	NA	9996 - 24,604	78,334 - 194,333	596,409 - 1,474,414

A more focused (unpublished) EBD study related to air pollution mortality in Canada was conducted by the Air Health Effects Division (AHED) of Environment Canada (Judek et al. 2005). Specifically, AHED estimated the annual number of excess deaths due to anthopogenic sources of air pollution levels in selected Canadian cities for the period 1998-2000 using country-specific data. Non-accidental mortality counts and National Air Pollution Surveillance (NAPS) data were combined with pollutant-mortality concentration-response functions (CRFs) from local epidemiological studies (e.g., Burnett et al. 2004) to estimate the number of annual preventable deaths that could be achieved by eliminating air pollution from human sources within North America. In this study, the estimated number of annual excess deaths within eight census divisions of Canada associated with short-term and long-term exposures was 1,800 and 4,200, respectively. The authors noted that this is a conservative estimate because their base case analysis included only about one-third of the Canadian population, mainly residents of large, urban areas. The study authors also estimated that 8-10% of all-cause mortality is attributable to "human sources" of air pollution, and concluded that additional impacts on morbidity (e.g., hospital admissions) would be substantial. Although not published in a peer-reviewed journal, this study provides a good example of how to link a specific environmental risk factor (air pollution) to a specific health outcome (mortality) in Canada, using relevant population exposure and dose-response data. The primary limitation of this study with respect to designing effective intervention strategies is that it does not account for all health effects related to air pollution or consider the impact of other risk factors on mortality or morbidity.

Another more focused (unpublished) EBD study related to air pollution mortality and morbidity in Ontario was conducted by the Ontario Medical Association (OMA 2005). In June 2000, OMA presented the first version of their Illness Costs of Air Pollution (ICAP) software model and detailed findings on the health effects and economic costs of air pollution in Ontario, and these findings were updated in 2006 to include results for four major health endpoints (premature mortality, hospital admissions, emergency room visits, and minor illnesses) for the period 2005-2026. OMA's ICAP analysis relied on four principal information elements: (1) the size and characteristics of the exposed population, (2) the type and concentration of air pollution to which the population was exposed, (3) the expected health responses of the population to air pollution exposures, and (4) the economic consequences of adverse health effects caused by air pollution. Specifically, this analysis combined data on air concentrations in Ontario with CRFs taken from the literature (the exact references are not specified, but the report states that the analysis preferentially relied on cohort-based relative risk epidemiological studies as opposed to time-

series analyses). In this study, approximately 5,800 annual deaths, 16,000 total hospital admissions, 60,000 emergency room visits, and 29 million minor illnesses were associated with smog and air pollution exposures in Ontario in 2005 (see Table 16). OMA predicted that these health impacts would increase in the future if air pollution was not controlled in the region. Although not published in a peer-reviewed journal and limited in scope with respect to other risk factors and disease outcomes, this study also provides a good example of how to conduct a local EBD analysis using site-specific data where available.

Table 16. Provincial health damage summary for four example years (Source: OMA 2005)

Outcome	**Example Years**			
	2000 (original model)	**2005**	**2015**	**2026**
Premature deaths	1,925	5,829	7,436	10,061
Hospital admissions	9,807	16,807	20,067	24,587
Emergency room visits	45,250	59,696	71,548	87,963
Minor illnesses	46,445,663	29,292,100	31,962,200	38,549,300

4.2.2 Studies Conducted in Canada Related to Specific Environmental Risk Factors and/or Health Outcomes

Additional studies have been conducted in Canada that assess certain aspects of an EBD evaluation, such as establishing a concentration or dose-response relationship for a specific environmental risk factor (e.g., air pollution) or qualitatively assessing the link between environmental exposures and a specific health endpoint (e.g., cancer). Although these studies do not represent an actual or complete EBD assessment *per se*, they provide useful supporting data and that can be used in more quantitative EBD evaluations. Examples of some of these types of studies are provided below for illustrative purposes.

For example, a number of epidemiological studies have evaluated the relationship between different health outcomes (e.g., cardiac and respiratory hospitalizations, premature mortality) and population exposures to air pollution in specific cities or areas of Canada (see Appendix A, Table A-5). Collectively, these studies show that exposure to air pollutants such as PM are associated with a variety of health outcomes, and many of these studies provide CRFs that could be used (along with additional exposure modeling and health statistics data) to support a Canada-specific EBD analysis related to air pollution as an environmental risk factor. These kinds of studies were relied on by Judek et al. (2005) and OMA (2005) to estimate the EBD attributable to air pollution in Canada and Ontario, respectively. As part of their attempt to develop an alternative multi-pollutant, no-threshold air quality index (AQI) for Canada, Stieb et al (2005) also discuss applying locally-derived CRFs (which are most relevant to exposure levels, exposure mixes, and population characteristics) to the estimation of public health burden in Canadian cities.

Several other studies have examined different types of cancer in Canada and have attempted to qualitatively relate the observed incidence of cancer to a range of environmental causes (see Appendix A, Table A-6). For example, the Conservation Council of New Brunswick (CCNB, 2008) recently sponsored a study of the environmental contribution to cancer incidence and

prevalence in New Brunswick (Milewski and Liu 2009a, b). In their first assessment, Milewski and Liu (2009a) reported that lung cancer incidence rates from 1991-2005 among males and females in Saint John were consistently and significantly higher than rates reported in other regions, and that occupational exposures and air pollution were key risk factors for lung cancer in Saint John. However, the authors' conclusions concerning environmental exposures and risks were based primarily on evidence from the occupational literature, which may not be applicable to the general population. To obtain more useful data, the study authors recommended conducting an epidemiological study to determine the cause of high lung cancer rates in Saint John as well as detailed individual– and community- level epidemiological studies to assess the rise in prostate and breast cancer rates in other regions. In a follow-up study, which expanded the areas under consideration, Milewski and Liu (2009b) summarized potential environmental and occupational exposures in different areas in New Brunswick, and reported a qualitative association between communities having higher overall rates of cancer and greater industrial activity and/or potential for environmental contamination (see Table 17). Although these studies provide useful health statistics data at the local level, they do not provide the necessary data with respect to population exposures and quantitative relationships between cancer outcomes and potential risk factors to support an EBD analysis for this region.

Table 17: Potential environmental and occupational exposure to known and probable carcinogens in 14 urban and rural areas in New Brunswick (1989-2005) (Source: Milewski and Liu 2009b)

Community/Area	Arsenic	Benzene and VOCs	Cadmium	Formaldehyde	Lead	Nickel	Pesticides	PAHs	Radon	Wood Dust
Base Gagetown Area							√			√
Bathhurst	√	√	√	√	√					√
Belledune Area	√		√		√			√		√
Caraquet										
Dalhousie	√	√	√		√	√	√	√		√
Drummond-Denmark Area							√			√
Edmundston		√								√
Fredricton										√
Harvey Area	√								√	√
Miramichi		√		√						√
Minto Area	√	√						√		√
Moncton										
St. John	√	√			√	√		√		√
Upper Miramichi Area		√					√			√

Similarly, a Workshop on Environmental Exposures and Cancer was convened by Cancer Care Ontario on April 25-26, 2001 (Kreiger et al. 2003). The goal of the workshop was to identify potential environmental contributors to cancer and develop consensus on priorities and recommendations regarding surveillance, research, and prevention (see Table 18). One of the research priorities identified by the workshop panel was to conduct literature reviews to determine the strength of the evidence regarding specific environmental exposures and cancer. Although no additional information is available concerning follow-up activities from this workshop, data collected in the recommended areas would be useful for developing quantitative estimates of the environmentally-attributable fraction of observed cancer outcomes in Canada.

Table 18: Environmental exposures and recommendations to Cancer Care Ontario (adapted from Kreiger et al. 2003)

Environmental Exposure	Surveillance	Research	Cancer Prevention Activity
UV light	Collect additional data on sun-protective behaviors and outcomes; monitor temporal trends in UV index		Work with Health Canada, Environment Canada, the Canadian Cancer Society, school boards and municipalities to administer sun safety programs
Environmental tobacco smoke	Support existing surveillance initiatives at the local, regional, and national levels	Conduct surveillance using exposure biomarkers (e.g., cotinine) in the general population	Advocate for banning environmental tobacco smoke in the workplace; communicate successful initiatives to partners and stakeholders
Polycyclic aromatic hydrocarbons (PAHs)	Estimate the number of cancer deaths attributable to PAHs	Develop improved exposure estimates	Advocate for cleaner engines
Asbestos	*Identify buildings with asbestos*		Support workers and businesses in building trades to reduce exposures
Water disinfection byproducts	Monitor water quality at water treatment plants		Advocate for improvements to the Ontario Drinking Water Surveillance Program
Radon	*Require radon testing prior to sale or rental*		Communicate cancer risks from radon exposure to partners, stakeholders, and the public
Pesticides	Monitor pesticide use		Communicate risks of pesticide exposure to partners, stakeholders, and the public
Dump site contaminants	*Implement monitoring programs at "dump sites"*	Link point-source exposures from dump sites to cancer registry data	
Heavy metals	Work with the Ministry of Labour and other groups to monitor industrial exposures		

4.2.3 Other Programs and Workshops in Canada Related to Health and the Environment

On September 26 - 27, 2006, the PHIRIC held a workshop in Banff, Alberta to start a more strategic, coordinated, and ambitious conversation about research to improve population health interventions in Canada (http://www.cihr-irsc.gc.ca/e/33515.html). Although this group has not directly developed any Canadian EBD studies, it appears to be developing programs that will build on the WHO EBD studies. For example, PHIRIC has developed a strategic plan and identified issues of high priority, including increasing funding for population health intervention research; increasing the capacity of researchers and policy-makers, respectively, to conduct and

use population health intervention research through training opportunities and programs; developing communication and networking capacity to increase the number of universities, researchers, practitioners, networks and other organizations within and outside the health sector that see the value in and that support increasing intervention research at the population level; and developing the field of population health research and evaluation through monitoring the quality and quantity of research, and increasing collaboration and synergy where possible.

On February 12, 2007, the McLaughlin Centre for Population Health Risk Assessment within the Institute of Population Health at the University of Ottawa hosted a one-day workshop to review the current methods used to estimate EBD and to explore these applications in the Canadian context (http://www.mclaughlincentre.ca/events/EDB_WS.shtml). The next steps that were identified at the conclusion of the workshop included generating a summary report (available on the website); initiating a EBD program for Canada building on WHO guidance; specifying methods, obtaining data, and conducting analyses; interpreting the results of these analyses; and examining policy implications. No follow-up information is available from this workshop.

On November 26-28, 2007, the Vulnerable Populations Office of the Safe Environments Programme of Health Canada organized a workshop in Ottawa related to children's health and the environment (http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/childrens_workshop-enfants_atelier/index-eng.php). This workshop identified the following developmental windows associated with age-specific susceptibility to environmental exposures: (1) preconception, (2) embryonic and fetal period, (3) neonatal period, (4) first three years of life, (5) preschool and primary-school age, and (6) adolescence. No follow-up information is available from this workshop.

On February 5-6, 2008, the Vulnerable Populations Office of the Safe Environments Programme of Health Canada organized a workshop in Ottawa on seniors' health and the environment (http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/seniors_workshop-aines_atelier/index-eng.php). The purpose of the workshop was to improve understanding of the nature and extent of the relationship between the environment and the health of seniors, as distinct from other populations, and to identify major areas for action. The workshop identified five factors which contribute most to seniors' vulnerabilities to environmental risks including: physiological changes during the process of aging; living arrangements, quality of the housing environment, especially indoor air quality and housing location; socio-economic status; body burden of environmental contaminants and historical environmental exposures; and, seniors' level of awareness on environmental health issues and environmental health literacy.

5.0 Discussion

A number of global, regional, and national burden of disease and EBD studies have been conducted over the last 20 or more years, and these studies provide useful background information and supporting data for evaluating the current burden of disease attributable to the environment in Canada. Collectively, these studies suggest that the total EBD for high-income, developed countries may range from as low as 1-5% to as high as 15-22%, depending on how EBD is calculated and defined. In studies utilizing an outcome-based approach, estimated EAFs

also vary significantly by disease category and risk factor. The primary disease categories identified or evaluated in EBD studies for developed countries include acute and chronic respiratory diseases, cardiovascular disease, diarrhea, neurobehavioral disorders, cancer, and congenital afflictions. The primary specific environmental risk factors associated with these and other diseases in developed countries include ambient air pollution, indoor smoke from solid fuels, poor water and sanitation, and inorganic lead exposure. For those studies utilizing a broader definition of the environment, lifestyle factors (e.g., smoking, alcohol consumption) and occupational exposures are key risk factors associated with disease burden in developed countries. Although many other possible risk factors may be important for Canada and elsewhere, the potential contribution of these risk factors to disease burden has not yet been explored in existing global/regional, country-wide, or localized EBD studies.

The wide disparity in published EBD estimates is due primarily to the use of different methodological approaches, data sets, and units of analysis as well as the inclusion of different disease categories and environmental risk factors. For example, historical estimates have utilized either an exposure-based (or scenario-based) or outcome-based approach that may include only mortality or both mortality and morbidity effects. As mentioned, the definition of the environment has also varied considerably among EBD studies, ranging from assessments that consider only chemical exposures in the general environment to those that consider many broader lifestyle factors (e.g., smoking, alcohol consumption, physical inactivity, excess weight and obesity), viruses and bacteria, sunlight, medications and medical procedures (e.g., radiation), and occupational exposures. Additionally, nearly all EBD studies have relied heavily on consultations with experts to draw conclusions about the total burden of disease attributable to environmental factors, and differences or biases in expert judgment can have a significant impact on study results. For these and other reasons (e.g., different assumptions or baseline estimates), it is difficult to compare results across existing EBD studies, even at the global level. The National Institute for Public Health and the Environment (RIVM) in the Netherlands is currently evaluating which types of factors contribute the most to differences in global EBD estimates (Knol and Staatsen 2005).

It is also important to recognize that there are very few primary studies from the literature that have attempted to independently estimate EAFs for different disease categories. Instead, most EBD studies rely on the EAF estimates calculated previously by WHO (Prüss-Üstün and Corvalán 2006, 2007), OECD (Melse and de Hollander 2001) and/or Landrigan et al. (2002) and combine these estimates with country-specific mortality and/or morbidity statistics to calculate the EBD for a particular country or region. For many diseases, WHO has developed separate EAF estimates for developed versus developing countries, but these estimates still include a number of environmental risk factors that most regulators or policymakers would consider as "personal environment" or "occupational" as opposed to "environmental" in the sense of the ambient environment. The OECD estimates, which include a narrower definition of the environment, are potentially more relevant for developed countries such as Canada, but these estimates are based on statistics for Europe and could therefore include regional subtleties of exposure and risk attributable to specific characteristics of that population. Landrigan et al. (2002) developed EAF estimates for children in the United States attributed solely to chemical exposures, which are also potentially relevant for Canada, but these estimates (like those by WHO and OECD) are highly uncertain due to significant data gaps with respect to population

exposures and dose-response relationships and are based primarily on expert judgment. To date, no Canada-specific EAFs have been derived.

WHO has developed a country-wide EBD estimate for Canada of 13%, which is partially attributed to exposure to ambient air pollution and water, sanitation, and hygiene. The WHO estimates were based on a recent comparative framework which was designed to better facilitate comparisons across studies, regions, and disease outcomes. Using the WHO estimates and other published data to quantify the EBD in Canada, Boyd and Genuis (2008) assumed preliminary EAFs ranging from 10-30% for COPD, 26-53% for asthma, 7.5-15% for cardiovascular disease, 5-15% for cancer, and 2-10% for congenital afflictions. These authors also estimated that environmental exposures in Canada account for approximately $3.6 to $9.1 billion dollars in annual health care costs due to potentially preventable illnesses and deaths. However, these authors cautioned that quantifying the EBD in Canada is challenging due to scientific uncertainties and data constraints, which led to their inclusion of only four disease categories and reliance on EAFs generated by other studies.

Although the available literature suggests that significant health gains could be achieved by reducing or eliminating selected environmental exposures in Canada (and elsewhere), it is often difficult to apply these findings to the design of effective public health intervention strategies. In particular, because regions and communities across Canada may be affected differently depending on their environment and exposure, health intervention strategies aimed at reducing EBD are likely to be most effective at the local or provincial level, rather than the global or national level. Such efforts require a better understanding of environmental exposures and subsequent health-related outcomes at these different geographic scales. As indicated above, most EBD assessments, including those conducted by WHO (2009) and Boyd and Genuis (2008) for Canada, rely on a combination of EAFs from the primary literature and published country-wide statistics for specific health outcomes. However, the health outcome statistics themselves are influenced by environmental exposures and reflect an implicit environmental contribution. For example, if there were 1,000 observed deaths from heart attacks in a particular study area or region, applying an EAF of 10% from the literature would result in the conclusion that 100 of these deaths were attributed to the environment. But, if in fact, a more detailed analysis of the exposure-response relationship between air pollution and health outcome data in the study area or region showed that 200 of the 1,000 heart attack deaths were attributable to air pollution exposure, this would lead to an EAF of 20%, which would be a more appropriate local or region-specific estimate. Because it is difficult to gauge the relevance of EAFs from the literature with respect to population exposures or dose-response relationships at different geographic scales, it may be more appropriate to view the general studies from the literature as providing suggestive rather than definitive evidence of the EBD, particularly in developed countries such as Canada.

Excluding lifestyle and occupational risk factors, the strongest evidence based on the available data for Canada and other developed countries relates to mortality and morbidity effects (e.g., respiratory disease, asthma) attributed to air pollution (e.g., PM). Specifically, ambient air monitoring data are available for many countries or regions and the concentration-response relationship for PM exposures and adverse health effects has been quantified in a number of epidemiology studies, including many conducted in Canada. The consistent finding among virtually all EBD studies that air pollution is a major environmental risk factor suggests that

public health interventions aimed at reducing air pollution exposures are likely to have a notable impact on reducing the EBD in Canada. However, it is important to recognize that although the evidence with respect to air pollution and illness represents a situation where good methods have been employed and reasonable estimates have been made of the health impact, this does not mean that air pollution is the most important factor contributing to environmental health impacts and there are many regional differences with respect to air quality in Canada that would influence this relationship. The health impact of many other environmental factors is simply not known at this time, and could be greater or less than indoor and outdoor air pollution. In particular, some studies have concluded that environmental chemicals are having a measurable impact on the population burden of disease in Canada, but these findings are typically based on qualitative assessments or studies of occupational exposures, and there is currently little quantitative data available to support this relationship. As discussed below, additional research and efforts are needed in several areas that would help fill key data gaps and ensure the design of effective intervention strategies aimed at reducing the EBD in Canada.

5.1 Data Gaps and Research Needs

The comparability of risk factor contributions to EBD has historically been hindered by the lack of standardization of methods (e.g., choice of risk factors, summary measures of population health, alternative scenarios) and by differences in the reliability of the underlying epidemiological studies of relative risk and population exposure levels (i.e., causation). The WHO CRA project was developed to try to address some of these issues, and WHO recently proposed an EBD framework that they recommend be adopted by other researchers to ensure that estimates are both reliable and comparable.

Despite these potential improvements in study design, there remains significant data gaps in the underlying science on population-level exposures and cause and effect relationships for many environmental risk factors and health outcomes. This paucity of data has led many researchers to conclude that the true burden of disease attributed to the environment has been severely underestimated (DHHS 2010; Boyd and Genuis 2008; Landrigan et al 2002; Prüss et al. 2001). Data limitations have also perpetuated the continual reliance on EBD and EAF estimates from a few underlying studies, which themselves rely heavily on expert judgment. Additionally, most existing EBD estimates are based on global risk factors, and few data or estimates are available on potentially more relevant risk factors at the national, regional, or local levels. Because of these limitations, there remains significant uncertainty in current EBD estimates and their applicability to specific countries or regions, and only limited efforts have been made to characterize the full extent of this uncertainty.

Some of the more important data gaps related to EBD assessments include the following:

- *Lack of well-defined or relevant environmental risk factors or disease categories.* Although this issue has been raised in many EBD assessments, including the original Doll and Peto (1981) study, there is still no consensus in the published literature about how to define "environmental risk." While some EBD studies, such as those conducted by Melse and de Hollander (2001) and Landrigan et al. (2002) have relied on a relatively narrow definition (e.g., chemical exposures in the environment), other studies, such as those conducted by Prüss-Üstün and Corvalán (2006, 2007), have relied on very broad

definitions (e.g., all sources of environmental pollution, occupational exposures and stress, and lifestyle factors). There can also be significant differences in the definition of individual risk factors or disease outcomes, depending on geographic location and time period, and each risk factor or disease category may be comprised of multiple components. For example, the commonly cited risk factor "water, sanitation and hygiene" may be predominantly influenced by microbial water contamination and poor living conditions in developed countries, whereas it may be more representative of food contamination and foodborne illnesses in developed countries. Similarly, the risk factor "indoor air pollution" may be driven by biomass burning in developed countries, but may be associated primarily with secondary environmental tobacco smoke in developed countries. At present, most EBD studies do not adequately define or disaggregate individual risk factors by these underlying sources, and many disease categories are overly broad (e.g., all cancers), making it difficult to design targeted intervention strategies to address these sources of exposure and risk. Ideally, EBD studies should identify and evaluate those risk factors and health outcomes most relevant to the geographical scale and time period of interest. Differentiating among underlying causes and health effects will become even more important as greater emphasis is placed on evaluating aggregate and cumulative exposures and risks among population groups (U.S. EPA 2007).

- *Inadequate data on population-level exposures for different risk factors, subpopulations, and geographical scales.* Although EBD studies require an understanding of population-level exposures, robust estimates of exposure as they relate to population health are often missing for the risk factors, subgroups, or regions of interest (Ezzati et al 2006; Briggs 2003). This includes inadequate environmental monitoring data for different regions in Canada, as well as very limited biomonitoring data for the general Canadian population (Bérubé 2007; Neumann 2005, 2006). In order to help fill this data gap, Health Canada (2007) is currently collaborating with Statistics Canada to add a biomonitoring component to the Canadian Health Measures Survey (CHMS), which will measure human levels of environmental chemicals in a sample that represents the overall Canadian population (i.e., the study targets 5,000 Canadians between the ages of six and 79 years). Biomonitoring measurements provide health-relevant assessments of exposure because they provide direct measurements of people's exposure to toxic substances from all environmental sources by measuring the substances or their metabolites in human specimens, such as blood or urine. Additionally, data are lacking on combined exposures to multiple environmental risk factors as well as early life-stage exposures that may occur during key windows of vulnerability (DHHS 2010). In response, the Maternal-Infant Research on Environmental Chemicals (MIREC) study was recently launched by Health Canada (2010) in conjunction with their academic and clinical research collaborators, which complements the CHMS study by measuring biological markers of environmental exposure. Specifically, this is a national five-year research study aimed at recruiting and following about 2,000 women through pregnancy and up to eight weeks after birth in order to assess maternal and infant exposures to environmental chemicals, heavy metals, and tobacco smoke.

- *Limited data on causation, relative risks, and dose-response relationship for many risk factors and disease outcomes.* Perhaps one of the most significant data gaps facing EBD studies is the lack of understanding with respect to whether a risk factor or multiple risk factors cause a disease outcome, whether there are thresholds for different disease outcomes, and the quantitative relationship between environmental exposure levels and adverse health impacts (O'Connell and Hurley 2009; Briggs 2003; Landrigan et al. 2002; Prüss et al. 2001). This data gap has caused many EBD studies to focus on a limited number of disease categories for which there is reliable evidence of an environmental etiology (Boyd and Genuis 2008; Ezzati et al. 2006). In EBD assessments, relative risks and dose-response functions are typically based on epidemiological studies conducted in the general population, such as those discussed above for air pollution, or may be based on expert judgment. However, these types of studies are much easier to conduct for air pollution than other risk factors because ambient air pollutant exposures are well defined and can easily be quantified in a population, and observed health effects often occur soon after an exposure. Other risk factors, such as chemicals in prepared foods or consumer products, nanoparticles, or releases from nuclear power plants, are much more difficult to measure in the population, and there may be a significant lag time between exposures and health effects. Dose-response relationships derived from toxicological data or occupational cohorts are generally viewed as less relevant to the general population, but these data could potentially be used together with a plausible mechanistic understanding of the biological pathways leading to health outcomes relevant in humans.

- *Lack of longitudinal studies and environmental surveillance programs*. To date, no large-scale studies have been conducted in Canada that track the disease burden and potential risk factors among large population groups over time, and the environmental surveillance of many diseases and risk factors is lacking, particularly for children (Bérubé 2007; Boyd and Genuis 2008). These types of studies are needed to identify potential relationships and interactions between risks factors and disease outcomes and provide comprehensive health statistics data. The National Children's Study (http://www.nationalchildrensstudy.gov/Pages/default.aspx) and Framingham Heart Study (http://www.framinghamheartstudy.org/) are examples of large-scale longitudinal studies that have been conducted or are currently underway in the United States. The Canadian Partnership for Children's Health and Environment (CPCHE) is a multi-sectoral collaboration of twelve organizations in Canada that have also been working together on a vision and strategy to protect children's health from environmental pollution and toxic chemicals (http://www.healthyenvironmentforkids.ca/).

- *Limited attempts to address the full range of uncertainty in EBD and EAF estimates.* In all EBD studies, there are significant sources of uncertainty related to exposures, relative risks, dose-response relationships, health statistics data, DALY estimates, and other factors, especially when extrapolating from one population group to another (Mathers et al. 2006b; Danaei 2005; Valent et al. 2004; Prüss et al. 2003; Ezzati et al. 2002). Most EBD studies only attempt to account for data and model uncertainties in a very general way, such as providing a range of EBD or EAF estimates. Although more recent studies by WHO attempt to account for statistical uncertainty of some parameters and input data

uncertainty by calculating best estimates and 95% confidence intervals (Mathers et al. 2006b), it is not always clear what methodology was used to develop confidence intervals or other measures of uncertainty. Additionally, few EBD studies provide a complete characterization (either qualitatively or quantitatively) of the uncertainty in EBD estimates. For example, ambient air pollution has been identified as a key environmental risk factor in existing EBD studies, but most studies focus on short-term exposures and relatively little is known (or discussed) about long-term exposures (Knol and Staatsen 2005). More complete uncertainty and sensitivity analyses, such as using probabilistic techniques or other methods, would help to identify input parameters or assumptions contributing most to the uncertainty in EBD estimates and could be used to better characterize or reduce these uncertainties.

- *Extensive use of expert judgment.* Due to significant data limitations, the majority of published EBD studies have relied heavily on expert judgment to estimate EAFs for different risk factors and disease outcomes. While formalized methods for systematically eliciting and evaluating expert opinion (expert elicitation) methods have improved in recent years, including rigorous procedures followed by WHO during the 2001 global EBD assessment, there are still concerns related to calibration of experts and the impact of potential biases. As acknowledged by some EBD study authors, expert elicitation processes are necessarily speculative and the outcomes depend on the underlying assumptions and beliefs of the consensus panel (Landrigan et al. 2002). In addition, some historical EBD studies, or those conducted at more local levels, have relied on more ad hoc methods in developing expert panels and eliciting expert opinion rather than following a formal expert elicitation process (Knol 2010). To be credible, the reliance on expert judgment should follow rigorous and standardized methodologies, and key assumptions, data gaps, and areas of uncertainty should be clearly presented along with the results from these studies (O'Connell and Hurley 2009). Additionally, as more quantitative data become available to support EBD assessments, prior estimates that are based on expert judgment should be revisited and revised, if applicable.

- *Need for improved and innovative methodologies.* Most EBD studies that calculate EAFs do not fully account for the fact that environmental contaminants may interact with each other and that all avoidable causes of cancer are not known. However, some innovative approaches have been taken to try to evaluate the joint interaction of multiple risk factors on disease burden (Ezzati et al. 2003; 2006). Current EBD methodologies also do not account for the possibility that various exposures, whether individual, simultaneous, sequential, or cumulative over a lifetime, may not be simply additive or that there are critical periods of exposure (e.g., prenatal and early life, puberty) when individuals may be particularly susceptible to damage from environmental contaminants. Gene-environment interactions may also contribute more to cancer risk than either environmental sources or genetic susceptibility alone. The development and use of improved and innovative methodologies, such as high-throughput screening techniques, will likely allow the consideration of these types of factors in the future (DHHS 2010).

It is important to recognize that although there remain significant data gaps with respect to better quantification of the EBD in Canada and elsewhere, the available studies conducted to date and summarized in this report provide valuable information for scientists and policymakers that can be used to make decisions about potential public health intervention strategies or future research efforts. That is, while an understanding of and appreciation for existing data gaps is important, data uncertainties and limitations should not necessarily prevent potentially protective actions from being taken to try to reduce public exposures and health risks. Greater attention toward filling these data gaps, however, will improve the ability to identify the most important risk factors and to develop targeted health intervention programs that will ultimately be most effective at reducing population risks.

5.2 Recommendations

Based on our review of the available literature, we offer several recommendations to improve the current level of knowledge with respect to the EBD in Canada and to assist policymakers and health practitioners in Canada in their efforts to design and prioritize among effective public health intervention strategies. Although most of these recommendations will require ongoing and longer-term research efforts, we also provide some tangible short-term recommendations based on the current state-of-knowledge, which may change or evolve as new information becomes available in the future.

1. To the extent possible, choices about which environmental risk factors and disease outcomes to target should be based on national, regional, or local EBD estimates in Canada (and should not be based on global estimates). In particular, studies that include site-specific data on population-level exposures and associated health outcomes will be the most beneficial for developing targeted intervention strategies. The studies conducted by Judek et al. (2005) and OMA (2005), although unpublished and not peer-reviewed, provide good examples of how local datasets can be used to characterize the contribution of a specific environmental risk factor (e.g., air pollution) to selected health outcomes (e.g., mortality, morbidity) in Canada. These types of studies should be expanded to include other disease outcomes and environmental risk factors and/or extended to other Canadian regions. EBD estimates derived for other developed countries can possibly be used to design health intervention strategies in Canada, but at a minimum, some attempt should be made to compare or account for potential differences in population-level exposures or susceptibilities that might affect the generalizability of these estimates.

2. It is of critical importance that future EBD studies in Canada should be based on a consistent framework that relies on the same types of data and information sources and adequately characterizes the uncertainty in EBD estimates. Although WHO has developed a general unified EBD framework that is useful for cross-country comparisons, it is not clear whether this framework is specific enough to yield the types of data needed for developing targeted intervention strategies in countries like Canada. Canada-specific EBD studies should also strive to utilize an exposure-based approach (which links estimates of population-level exposures with evidence-based exposure–risk information), because this approach will generally be the most informative with respect to environmental health decision-making for health outcomes related to many different risk

factors (e.g., cancer, cardiovascular disease) (Prüss et al. 2001, 2002). That is, this approach can be used to reflect regional or local environmental exposures (rather than assuming a single attributable fraction for all locations), from which potential health gains achieved under alternative interventions or scenarios can be assessed. If an outcome-based approach is used, attempts should be made to derive EAFs that are most applicable to Canada at the national, regional, or local levels. The development of Canadian-specific EAFs could be based on formal expert elicitation methods (with standardized protocols for the study design and selection of experts), but informal or ad hoc expert panels should not be used. Regardless of the approach, all underlying sources of data should be critically evaluated with respect to their relevancy and level of certainty. In particular, the applicability of available exposure and dose-response relationships to the study population of interest needs to be thoroughly evaluated, and underlying assumptions, data gaps, and uncertainties need to be made explicit and clearly communicated to policymakers, health practitioners, and the public.

3. Perhaps of greatest importance, more research needs to be conducted to fill the key data gaps identified in the prior section to help facilitate Canada-specific EBD studies in the future and to ensure the design of optimal intervention strategies. Because these data gaps are interrelated, ideally progress needs to be made across all of them to effectively advance EBD estimates for Canada. In particular, better data are needed on population exposure levels at different geographic scales as well as dose-response relationships linking specific environmental risk factors and disease outcomes. The former data gap can be filled through more aggressive environmental and biomonitoring programs, while the latter data gap will require additional (more localized) epidemiology and community-based studies. Additional long-term research will ultimately be needed to address more complex issues, such as the impact of multiple exposures on disease burden, the effect of early life-stage exposures, and gene-environment interactions. In the interim, formal expert elicitation studies that are specific to Canada could be used to fill some of these data gaps and to define appropriate ranges of uncertainty in current estimates.

4. Although the greatest emphasis should be placed on method and data development of EBD as a whole, this will require a longer time horizon, and policymakers must decide where to invest in public health intervention programs in the interim. The best available information to date, although clearly limited in scope, suggests that addressing population exposures to air pollution should be a top priority for consideration for current health intervention strategies in Canada. Specifically, the available hard evidence suggests that, until additional data are collected on other risk factors, the greatest gains in public health for countries like Canada are likely to be achieved by reducing population exposures to selected air pollutants. This conclusion is based on the extensive population exposure data for PM and other air pollutants, multiple CRFs linking air pollutant exposures to specific health outcomes, and relevant health statistics data in Canada and elsewhere. The wealth of supporting data available for air pollution (relative to other environmental risk factors) increases confidence that interventions aimed at reducing such exposures will have a measureable positive impact on public health. It is important to recognize, however, that additional research is still needed to determine the most effective air pollution intervention strategies for different regions. Stieb et al. (2005) provide an

example of how to incorporate an analysis of the public health burden of air pollution into policy analysis and risk communication by developing an easy-to-understand AQI that can be used in different areas of Canada. It is also important to be aware that the nature and source of air quality issues may change over time due to various public health policies or environmental conditions (e.g., indoor smoking bans, restrictions on wood-burning stoves and fireplaces, effects of climate change, increased production of biofuels), which will influence what types of health intervention strategies should be considered and implemented.

5. The Canadian government should develop an explicit strategy for evaluating and prioritizing among other environmental risk factors and disease outcomes in Canada. That is, until the same (or comparable) types of supporting data that are available for air pollution become available for other risk factors, decisions will need to be made about whether or to what extent to target these potential risks. Even without firm quantitative evidence, a precautionary approach may be warranted for dealing with potential environmental exposures and risks, as has been recommended by the United States President's Cancer Panel (DHHS 2010). However, in light of limited resources and competing interests, policymakers and health practitioners in Canada will ultimately need to decide where best to focus their resources and efforts. To help facilitate such decisions, a strategy (perhaps based on a decision-analytic framework) needs to be developed that considers and weighs current scientific knowledge about potentially relevant risk factors (e.g., drinking water contaminants, pesticides, endocrine disrupters, microbial toxins, nanoparticles, releases from nuclear power plants) with respect to their levels of exposure in the population and dose-response relationship as well as degree of public concern and ease of control.

In summary, the most effective EBD estimates for informing public health policy in Canada will require synthesizing and integrating methods and data across disciplines. Ultimately, in order for EBD studies to become more useful for prioritizing across risks and designing effective intervention strategies, they need to link multiple risk factors to multiple health outcomes in an integrated, dynamic framework that reflects site-specific population exposures as they relate to site-specific population-level health outcomes. Recent and ongoing efforts in Canada, including the PHI project and PHIRIC consortium, appear to be promising venues for providing useful data on the relationship between environmental exposures and health outcomes in different regions in Canada. Although these efforts will likely require significant upfront resources, such investments in public health will ultimately result in long-term gains with respect to reduced disease burden and associated health-related costs.

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APPENDIX A

Table A-1. Summary of Selected Global/Regional EBD Studies

Reference	Purpose	Approach	Methods	Results	Conclusions	Data Most Relevant to Canada	Strengths	Limitations
Melse and de Hollander 2001	Estimate the burden of disease attributable to the environment within the OECD region in 1998	outcome-based	Estimated burden of disease using data from World Health Report 1999 (adjusted for region differences) and expressed in terms of health loss (DALYs) and costs; EAFs estimated for 16 diseases based on relative risks taken from the literature and exposure levels derived from international reports	Total burden of disease attributed to environmental factors ranged from 2-5% for OECD regions (1.4-4.3% high income, 3.7-6.7% lower income) and 8-12% for non-OECD countries; estimated EAFs varied significantly by disease category	Environmental fraction of the burden of disease varied by OECD/non-OECD and high/low income areas	Data presented for high-income OECD region	Uncertainty in estimates reflected in lower and upper EAF estimates	EAFs based on potentially outdated sources of data
Ezzati et al. 2002	Estimate the burden of disease attributable to 26 major risk factors using a unified framework for 14 geographical regions of the world in 2000	outcome-based	Estimated risk factor exposure and hazard size based on review of published work and other sources by expert working groups; calculated individual population attributable fractions and applied these to the mortality and burden of disease (DALY) estimates from the WHO GBD database	Global burden of disease attributed to environmental and occupational risk factors ranged from 8–9% (Briggs 2000); burden of disease attributed to environmental risk factors for the Americas subregion (includes Canada) ranged from 3–4%	Substantial proportions of global disease burden are attributable to these 26 major risks; developing countries suffer most or all of the burden due to many of the leading risks	Data presented for the Americas subregion	Uncertainty analysis was conducted; included certain risk factors not previously considered (e.g., climate change)	Lack of direct exposure data for many risk factors, resulting in significant uncertainty in EAFs
Ezzati et al. 2003	Estimate the burden of disease attributable to the joint effects of 20 leading risk factors in 14 geographical regions of the world in 2000	outcome-based	Estimated risk factor exposure and hazard size based on prior estimates for individual risk factors; calculated joint population attributable fractions and applied these to mortality and burden of disease (DALY) estimates; gains in HALE also estimated	Estimated 47% of premature deaths and 39% of total disease burden globally were attributable to joint effects of 20 risk factors; removal of these risks estimated to increase global healthy life expectancy by 9.3 years or 17% (4.4 years or 6% in developed countries of Western Pacific)	Joint risks from 20 risk factors contributed to considerable loss of healthy life in different regions of the world; even populations with high healthy life expectancy could benefit considerably from risk reduction	Data presented for developed regions	Uncertainty and sensitivity analysis was conducted	Only a small fraction of some important diseases was attributable to the risk factors considered here (including for environmental risk factors)
Danaei et al. 2005	Estimate worldwide and regional mortality from site-specific cancers attributable to 9 risk factors (individually and jointly) for 7 World Bank regions in 2001	outcome-based	Estimated risk factor exposure and relative risks based on review of published studies and other sources by expert working groups; calculated individual and joint population attributable fractions and applied these to regional site-specific cancer mortality from the WHO GBD database	Estimated 35% of cancer deaths worldwide (37% for high income countries) were attributable to 9 risk factors; 0.5% and 1% of cancer deaths worldwide (0% and 1% for high income countries) were attributable to indoor smoke from household use of solid fuels and urban air pollution, respectively	Primary prevention through lifestyle and environmental interventions remains the main way to reduce the burden of cancers	Data presented for high-income countries	Uncertainty and sensitivity analysis was conducted	Some risk factors excluded because of the limitations of deriving detailed exposure estimates from existing data

Reference	Purpose	Approach	Methods	Results	Conclusions	Data Most Relevant to Canada	Strengths	Limitations
Pruss et al. 2002	Estimate the burden of disease attributable to water, sanitation, and hygiene for 14 geographical regions of the world in 2000	scenario-based	Derived typical exposure scenarios for populations in each region and matched six typical scenarios with relative risk information obtained from the literature to estimate mortality and burden of disease (DALY)	Estimated 4% of all deaths and 5.7% of the total disease burden attributable to water, sanitation, and hygiene worldwide (only a fraction of the total disease burden is due to American region); about 90% of this disease burden occurs in children younger than 5 years old	Water, sanitation, and hygiene are major causes of mortality and disability worldwide; its effects are mainly concentrated in developing countries	Data presented for American region	Combines exposure with evidence-based exposure–risk information (previous GBD study relied on expert judgment of attributable fractions)	Because of data gaps and the difficulties in combining the various sources of uncertainty, did not estimate an error margin for the overall results
Fewtrell et al. 2003, 2004	Estimate the burden of disease attributable to lead exposure for 14 geographical regions of the world in 2000	exposure-based	Estimated population exposure distributions based on blood lead levels and combined with estimates of disease rates for various health outcomes (e.g., distribution of IQ points lost for children)	Estimated 1% of the global burden of disease attributable to mild mental retardation and cardiovascular outcomes resulting from exposure to lead	Lead in the environment is still a major risk factor; its effects are mainly concentrated in developing countries (especially in areas where leaded gasoline is heavily used)	Data presented for the Americas subregion	Uncertainty analysis conducted by using combinations of higher and lower input parameters	Limited information on regional differences in IQ distributions and the exposure-response relationship for lead
Cohen 2005	Estimate the burden of disease attributable to urban ambient air pollution for 14 geographical regions of the world in 2000	exposure-based	Estimated concentrations of inhalable particles (PM_{10}) and converted estimates to fine particles ($PM_{2.5}$); attributable numbers of deaths and years of life lost were based on relative risk estimates from the literature	Estimated 3%, 5%, and 1% of mortality from cardiopulmonary disease, cancer of the trachea/bronchus/lung, and acute respiratory infections in children <5 years old worldwide attributable to fine particulate air pollution ($PM_{2.5}$)	The impact of outdoor air pollution on the burden of disease in the world's cities is large but varies considerably by region; this burden occurs predominantly in developing countries	N/A (global estimates presented)	Sensitivity analysis conducted to assess model uncertainty (e.g., concentration-response function)	Data uncertainties hinder the extrapolation of results to smaller geographic areas (e.g., specific countries or cities)
Lucas et al. 2008	Estimate the burden of disease attributable to UV radiation exposure for 14 geographical regions of the world in 2000	outcome-based and exposure-based	Estimated population attributable fraction from published epidemiology literature and directly applied estimates to disease burdens calculated in the WHO GBD database for 2 diseases; developed population-level exposure–disease relationships and used these to calculate disease incidence and mortality and disease burden (DALY) for 7 diseases	Estimated 0.1% of the total global disease burden attributable to UV radiation	UVR exposure is a minor contributor to the world's disease burden	N/A (global estimates presented)	Uncertainty in estimates reflected in lower and upper estimates	Limited data available for estimating incidence or prevalence of some outcomes and past exposures

Reference	Purpose	Approach	Methods	Results	Conclusions	Data Most Relevant to Canada	Strengths	Limitations
Pruss-Ustun and Corvalan 2006, 2007	Estimate the burden of disease attributable to the environment for 14 geographical regions of the world in 2002	outcome-based	Estimated attributable fractions based on systematic literature review conducted by a survey of experts (using variant of the Delphi method) and applied estimates to mortality and disease burden (DALY) estimates from WHO GBD database	Estimated 24% of global burden of disease (36% for children) attributable to environmental risk factors; 17% of disease burden in developed countries and 15-22% of disease burden in the Americas subregion attributable to environmental risk factors	EAFs varied widely across regions and children carried a disproportionate share of the disease burden, with much of this burden in developing countries	Data presented for developed countries and the Americas subregion	Greater coverage of risk factors than prior studies; uncertainty in estimates reflected as best estimate and 95% CI	Data uncertainties are large and lack of evidence for some environmental risks

Table A-2. Summary of Selected EBD Studies Conducted in the United States

Reference	Purpose	Approach	Methods	Results	Conclusions	Strengths	Limitations
Landrigan et al. 2002	Estimate contribution of environmental pollutants to the incidence, prevalence, mortality, and costs of pediatric disease among American children in 1997	outcome-based	Disease burden for each illness category attributable to toxins in the environment estimated based on panel of experts or data from NAS	Estimated burden of disease attributable to environmental factors of 100% for lead poisoning, 30% for asthma (range 10-35%), 5% for cancer (range 2-10%), and 10% for neurobehavioral disorders (range 5-20%)	Estimates are likely to be low because consider only 4 categories of illness, incorporate conservative assumptions, do not consider late complications, and do not include estimated costs of pain	Uncertainty in estimates reflected in lower and upper EAF estimates	Lack of etiologic research quantifying contribution of environmental factors to causation of pediatric diseases
Massey and Ackerman (2003)	Estimate costs associated with five health outcomes attributable to environmental exposures among children in Massachusetts in 1997-2002	outcome-based	Reviewed state-specific incidence and prevalence data for each disorder; EAF and cost estimates based primarily on prior published analysis by Landrigan et al (2002)	Used prior EAF estimates of 5-90% for cancer, 10-35% for asthma, 5-20% for neurobehavioral disorders, and 100% for lead; no reliable estimates of EAF were available for birth defects; total estimated costs ranged from $56-337 million for direct costs and $1.1-1.6 billion for direct costs plus lost future income for children in Massachusetts	Preventable childhood illnesses and disabilities attributable to environmental factors are associated with large monetary costs in Massachusetts	Provides some state-specific incidence data	EAFs are based on prior study (no new analysis) for all health outcomes

Reference	Purpose	Approach	Methods	Results	Conclusions	Strengths	Limitations
Davies and Hauge 2005	Estimate costs associated with six diseases and disabilities attributable to environmental exposures among children and adults in Washington State in 2004	outcome-based	Reviewed state-specific incidence and prevalence data for each disorder; with the exception of cardiovascular disease and birth defects, EAF and cost estimates based primarily on prior published analysis by Landrigan et al (2002) and Massey and Ackerman (2003)	Used prior EAF estimates of 30% (10-35%) for asthma, 5% (2-10%) for cancer, 100% for lead, and 10% (5-20%) for neurobehavioral disorders; estimated burden of disease attributed to environmental factors of 7.5% (5-10%) and 30% (5-10%) for cardiovascular disease and 2.5% (2.5-5%) for birth defects; best estimate of total costs for direct costs was $310 million (children) and $782 million (adults/children) and for indirect costs was $1.6 billion (children) and $1.9 billion (adults/children) in Washington State	There are likely to be very significant direct health care costs and indirect costs associated with diseases and disabilities attributable to environmental contaminants in Washington State; a significant proportion of the estimated costs can be attributed to lead exposure	Provides estimates for cardiovascular disease and birth defects based on literature	EAFs are based on prior study (no new analysis) for most health outcomes
Shuler et al. 2006	Estimate costs associated with five health outcomes attributable to environmental exposures among children in Minnesota	outcome-based	Reviewed state-specific incidence and prevalence data for each disorder; with the exception of birth defects, EAFs based on prior published analysis by Landrigan et al (2002) and updated methods adopted from Massey and Ackerman (2003) and Davies and Hauge (2005)	Used prior EAF estimates of 30% (10-35%) for asthma, 5% (2-10%) for cancer, 100% for lead, 5% (5-10%) for birth defects, and 10% (5-20%) for neurobehavioral disorders; best estimate of total costs $1.6 billion for children in Minnesota	Environmental contributors to childhood disease are largely preventable and policies should be implemented that reduce or eliminate some of the key environmental contributors	Provides estimates for birth defects based on literature	EAFs are based on prior study (no new analysis) for most health outcomes

Table A-3. Summary of Selected EBD Studies Conducted in Europe

Reference	Purpose	Approach	Methods	Results	Conclusions	Strengths	Limitations
Valent et al. 2004	Estimate the burden of disease attributable to four environmental risk factors among children and adolescents for 3 age groups and 3 subregions in Europe in 2001	exposure-based and scenario-based	Estimated risk factor exposure and exposure-response relation based on review of published studies and reports from international agencies; calculated individual population attributable fractions and applied these to the mortality and burden of disease (DALY) estimates from the WHO GBD database (or calculated directly)	Among children aged 0–4 years, estimated EAFs ranging from 1.8-6.4% (deaths only) for all diseases attributable to outdoor air pollution, 4.6% (deaths) and 3.1% (DALYs) for acute lower-respiratory-tract infections attributable to indoor air pollution, and 4.4% (DALYs) for mild mental retardation resulting from lead exposure; in the age-group 0–14 years, 5.3% (deaths) and 3.5% (DALYs) for diarrhea attributable to inadequate water and sanitation; in the age-group 0–19 years, 22.6% (deaths) and 19% (DALYs) attributed to injuries	The burden of disease varied by significantly by age and subregion (i.e., higher in European subregions B and C than subregion A), indicating the need for targeted action	Uncertainty in estimates reflected in lower and upper EAF estimate; sensitivity analysis conducted to address uncertainties in the estimates of exposures and dose–response relations	Lack of valid exposure data and strong evidence of exposure–response relations; substantial uncertainty around some of the estimates, especially for outdoor air pollution
Matthews and Parry (2005); Health Protection Agency (2005)	Estimate the burden of disease attributable to environmental pollution for various health outcomes among children and adults in England and Wales in 1998-2003	outcome-based	Reviewed country-specific incidence and prevalence data for each disorder; EAF estimates based on prior published analyses by WHO and Landrigan et al (2002) or calculated from exposure and relative risk data from the literature	Used prior EAF estimates of 30% for asthma, 5% for cancer, and 10% for neurobehavioral disorders (lead unclear); estimated burden of disease attributed to environmental factors of 3-3.3% for allergy, 20% for congenital abnormalities, 6.3% for respiratory disease, and 0.8% for cardiovascular disease	Study summarizes information available in the literature and represents first step in the process of quantifying the possible burden of disease from environmental pollution	Considers broader range of health outcomes	EAFs are based on prior study (no new analysis) for some health outcomes, EAFs are based on very uncertain data and limited data sets

Reference	Purpose	Approach	Methods	Results	Conclusions	Strengths	Limitations
Knol and Staatsen (2005)	Estimate the burden of disease attributable to five environmental risk factors for 49 groups of diseases in the Netherlands for the years 1980, 2000 and 2020	exposure-based	Reviewed country-specific incidence and prevalence data for each disorder; estimated population exposures based on measured and modeled data and relative risks from recent Dutch epidemiological studies or relevant international estimates (relied on or expert judgment when data missing or uncertain); health impacts measured in DALYs	Estimated that 2-5% of the disease burden was attributed to the effects of (short-term) exposure to air pollution, noise, radon, total natural UV and dampness in houses for the year 2000	The effects of long-term exposure to PM_{10} had the greatest influence on the environment-related disease burden	An uncertainty analysis was conducted to assess the effects of different assumptions	Data are uncertain and not all environmental-health relationships are known

Table A-4. Summary of Canadian EBD Studies

Reference	Purpose	Approach	Methods	Results	Conclusions	Strengths	Limitations
PHA (2006)	To develop national estimates of population health that combine the impact of both death and reduced functioning and describe risk factors	Microsimulation models building on the exposure-based approach	Building on WHO efforts, developing microsimulation models that integrate many diseases and risk factors simultaneously and model the interplay between them	Results available only for preventive measures related to diabetes and cancer	Not much information available yet but should provide a robust approach for evaluating burden of disease by risk factor	Best opportunity for Canada-specific estimates of risk factors related to health outcomes	Little data or information available to date
WHO (2009)	To estimate the impact of specific risk factors on prevalence and incidence of disease	outcome-based	Follows the WHO-established methodology	Environmentally-attributable fraction (EAF) of 13%	There are opportunities to reduce environmental sources of chronic diseases	Canada-specific	Not clear on what basis the 13% determination was made
Boyd and Genius (2008)	To estimate the environmental burden of disease (EBD) in Canada for respiratory disease, cardiovascular disease, cancer, and congenital affliction	outcome-based	They use the environmentally attributable fractions (EAFs) developed by the WHO, EAFs developed by other researchers, and data from Canadian public health institutions to provide an initial estimate of the environmental burden of disease in Canada for four major categories of disease	Results indicate that: 10,000–25,000 deaths; 78,000–194,000 hospitalizations; 600,000–1.5 million days spent in hospital; 1.1 million–1.8 million restricted activity days for asthma sufferers; 8000–24,000 new cases of cancer; 500–2500 low birth weight babies; and between $3.6 billion and $9.1 billion in costs occur in Canada each year	The burden of illness in Canada resulting from adverse environmental exposures is significant	Uses Canada-specific statistics for health outcomes	Relies largely on WHO estimates for EAFs; outcome statistics are specific to Canada but risk factors and exposure-response functions are not
Judek et al. (2005)	To estimate the number of excess deaths in Canada due to air pollution	exposure-based	NAPS data for the years 1998 to 2000 pollutant-mortality CRFs from epidemiological studies	The annual excess number of deaths associated with short-term exposure was estimated to be 1,800 + 700. The annual excess number of deaths associated with long-term exposure was estimated to be 4,200 + 2,000	It may take five years or more to realize these preventable deaths following reductions in air pollution levels, but that efforts should be taken to reduce exposures	Robust statistical analysis	Dose-response for long-term effects based on US-based epidemiological studies; only one risk factor (air pollution); only one endpoint (mortality)
Ontario Medical Association (2006)	To estimate morbidity and mortality in Ontario associated with air pollution for the period 2005 - 2026	exposure-based	Incidence and prevalence statistics combined with dose-response functions	5,800 deaths annually due to smog-related premature mortality; 16,000 total hospital admissions (cardiovascular illness); 60,000 emergency room visits and over 29 million minor illnesses	There are opportunities to reduce the health impacts of air pollution	Robust statistical analysis; morbidity and mortality included	Only one risk factor (air pollution) and only for Ontario

Table A-5. Epidemiological Studies in Canada Related to Air Pollution as an Environmental Risk Factor

Reference	Purpose	Methods	Results	Conclusions
Brook et al. (2007)	Evaluate the association between NO_2 and nonaccidental mortality across 10 Canadian cities	Single and two-pollutant time series models for acute effects	NO_2 is strongly associated with nonaccidental mortality, particularly during warmer seasons, and NO_2 is correlated with many other constituents (e.g., VOCs, etc.)	The strong effect of NO_2 makes it an excellent indicator for the "true" causal agent
Burnett et al. (1997a)	Evaluate the association between ozone and hospitalization for respiratory diseases in 16 Canadian cities	Regress daily hospital admissions against the high hour concentration of ozone recorded 1 day previous to the date of admission, controlling for SO_2, NO_2, CO, soiling index, and dew point temperature	Positive association for the April to December period but not in the winter months. The relative risk for a 30 ppb increase in ozone varied from 1.043 ($P < 0.0001$) to 1.024 ($P = 0.0258$)	Actual environmental exposures to ozone contribute to hospital admissions for respiratory ailments
Burnett et al. (1997b)	Explore the role that ambient air pollution plays in exacerbating cardiac disease	Relate daily fluctuations in admissions to 134 hospitals for congestive heart failure in the elderly to daily variations in ambient concentrations of CO, NO_2, SO_2, ozone, and the coefficient of haze for 1981-1991	CO concentration recorded on the day of admission displayed the strongest and most consistent association with hospitalization rates	CO was the strongest predictor of hospitalization rates among the air pollutants examined and was least sensitive to covariate adjustment
Burnett et al. (1998)	Explore the role of ambient levels of CO in the exacerbation of heart problems in individuals with both cardiac and other diseases	Compare daily variations in CO levels and daily fluctuations in nonaccidental mortality in metropolitan Toronto for the 15-year period 1980-1994	Statistically significant positive associations were observed between daily fluctuations in mortality and ambient levels of a complex mixture of pollutants, explained primarily by total suspended particles and CO	Statistically significant positive associations were observed between CO and mortality in all seasons, age, and disease groupings examined. CO should be considered as a potential public health risk to urban populations at current ambient exposure levels

Reference	Purpose	Methods	Results	Conclusions
Burnett et al. (2000)	Explore the relative toxicity of the chemical and physical components of the complex mixture found in typical urban air pollution	Model the association between particulate- and gas-phase components of urban air pollution and daily mortality in eight cities	Positive and statistically significant associations were observed between daily variations in both gas- and particulate-phase pollution and daily fluctuations in mortality rates	The authors recommend that measurements of elemental and organic carbon be undertaken in Canadian urban environments to examine their potential effects on human health
Burnett et al. (2004)	Model the association between daily variations in ambient concentrations of NO_2 and mortality in 12 of Canada's largest cities, using a 19-yr time-series analysis (1981–1999)	Parametric time-series statistical models	Increase in the 3-d moving average of NO_2 concentrations (population-weighted study mean = 22.4 ppb) was associated with a 2.25% increase in the daily nonaccidental mortality rate; insensitive to adjustment for other parameters	As NO_2 emissions arise primarily from vehicular/combustion sources, reducing combustion will result in public health benefits
Coyle et al. (2003)	Explore the benefits of reducing exposure to sulfate and at what concentrations	Decision analytic model using Monte Carlo techniques using Pope et al. for concentration-response	A one-unit reduction in sulfate air pollution would yield a mean annual increase in Quality-Adjusted Life Years (QALYs) of 20,960, with gains being greater for individuals with lower educational status and for males compared to females	Based on a tentative threshold for the value of health benefits, analysis suggests that an investment in Canada of over $1 billion per annum would be an efficient use of resources if it could be demonstrated that this would reduce sulfate concentrations by 1 $\mu g/m^3$
Goldberg et al. (2001a)	To evaluate the association between ground-level ozone concentrations and mortality	Regression of the logarithm of daily counts of cause-specific mortality on mean levels of ozone, after accounting for seasonal and subseasonal fluctuations in the mortality time series, non-Poisson dispersion, and weather variables	For an increase in the 3-day running mean concentration of ozone of 21.3 $\mu g/m^3$, the percentage of increase in daily deaths in the warm season: nonaccidental deaths, 3.3% (95% CI: 1.7, 5.0); cancer, 3.9% (95% CI: 1.0, 6.91); cardiovascular, 2.5% (95% CI 0.5 - 5); respiratory 6.6% (95% CI 1.8 - 11.8)	These results were independent of the effects of other pollutants and were consistent with a log-linear response function

Reference	Purpose	Methods	Results	Conclusions
Goldberg et al. (2001b)	To determine whether variations in concentrations of particles in the ambient air of Montreal, Quebec, during the period 1984 to 1993, were associated with daily variations in nonaccidental mortality	Regressed the logarithm of daily counts of nonaccidental mortality on daily mean levels of particulates, accounting for seasonal and subseasonal fluctuations in the mortality time series, non-Poisson dispersion, weather variables, and gaseous pollutants	Found evidence of associations between daily nonaccidental deaths and most measures of particulate air pollution	Provides further evidence of a linear association between measures of particulate and nonaccidental death, and that any threshold effect, should it exist, would be found at lower levels of air pollution than those found in Montreal
Goldberg et al. (2001c)	Determine whether variations in concentrations of particulates in the ambient air of Montreal, Quebec, during the period 1984 to 1993, were associated with daily variations in cause-specific daily mortality	Regressed logarithm of daily counts of cause-specific mortality on daily mean levels for the above measures of particulates, accounting for seasonal and subseasonal fluctuations in mortality time series, non-Poisson dispersion, weather variables, and gaseous pollutants	Positive and statistically significant associations between daily measures of ambient particle mass and sulfate mass and the deaths from respiratory diseases and diabetes	Associations were consistent with linear relationships
Liu et al. (2007)	To evaluate association between gaseous ambient air pollution and adverse pregnancy outcomes	Association between preterm birth, low birth weight, and intrauterine growth retardation (iUCR) among singleton live births and ambient concentrations of SO_2, NO_2, CO, and ozone in Vancouver, Canada, for 1985 - 1998	Low birth weight associated with exposure to SO_2 during the first month of pregnancy (OR = 1.11, 95% CI, 1.01-1.22, for a 5.0 ppb increase). Preterm birth associated with exposure to SO_2 {OR - 1.09. 95% CI, 1.01-1.19. for a 5.0 ppb increase) and to CO {OR - 1.08 95% CI, 1.01 -1.15 for a 1 ppb increase)	Relatively low concentrations of gaseous air pollutants are associated with adverse effects on birth outcomes in populations experiencing diverse air pollution profiles

Reference	Purpose	Methods	Results	Conclusions
Stieb et al. (2000)	To explore the association between air pollution, aeroallergens, and cardiorespiratory emergency room visits (n=19,821) in St. John, Canada	Generalized additive models of multipollutant, multiaeroallergen exposures and emergency room visits	In the final year-round multipollutant models, a 21% increase in emergency room visits was associated with ozone and sulfates. For the single pollutant models, $PM_{2.5}$ was most statistically significant	The authors report a significant influence of the air pollution mix on cardiac and respiratory emergency room visits
Stieb et al. (2002)	To examine relationship between air pollution and disability days for 1994-1999 in Toronto	A model of disability days in the two weeks prior to being interviewed for the National Population Health Survey in Canada paired with air pollution, weather, personal characteristics, etc.	After controlling for personal and other factors, only carbon monoxide and $PM_{2.5}$ were statistically significantly associated with disability days	While results are suggestive of significant effects of the urban air pollution mix at relatively low ambient concentrations, the precise contribution of individual pollutants could not be determined
Stieb et al. (2005)	To develop an integrated, multipollutant model of AQI to develop an increased awareness of the burden of illness of air pollution	Concentration-response based Burnett et al. (2000) to develop models of simultaneous effects of five pollutants; combined with monitoring data	Calculated AQI retrospectively and compared to individual pollutant AQI (moderate correlation); argued that this approach is more consistent and informative	The development of an alternative AQI has been used to illustrate several issues related to quantifying the public health burden attributable to air pollution
Villeneuve et al. (2003)	To evaluate the relationship between daily levels of particulate and gaseous phase pollutants and mortality (n=550,000 individuals) between 1986 and 1999 at different socioeconomic levels ("environmental justice")	The percent change in all-cause, cardiovascular, respiratory, and cancer daily mortality was calculated in relation to short-term changes in levels of a number of particulate and gaseous pollutants using time-series models	Daily mean PM_{10} concentrations were associated with premature mortality, not $PM_{2.5}$. SO_2 and NO_2 were also associated with premature mortality	For NO_2, CO, and SO_2, there was some suggestion of increased risk of all-cause and cardiovascular mortality at lower levels of socioeconomic status, although due to the small number of deaths within each socioeconomic strata, the results are not conclusive

Reference	Purpose	Methods	Results	Conclusions
Yang et al. (2003)	To evaluate the impact of ozone concentrations on daily hospital admissions for children < 3 years and adults > 65 years in the greater Vancouver area 1986 - 1998	Bidirectional case-crossover analysis was used to evaluate associations between ambient ozone and respiratory hospitalizations controlling for other pollutants, personal characteristics, weather, etc.	Odds ratios for hospital admission of 1.22 (95% CI: 1.15–1.30) for children and 1.13 (1.09–1.18) for the elderly, respectively, were found, based on an increment in exposure corresponding to the 4-day average interquartile range for ozone	Ambient ozone is positively associated with respiratory hospital admission of young children and the elderly in Vancouver

Table A-6. Studies Conducted in Canada Related to Cancer as a Health Outcome Attributed to the Environment

Reference	Approach	Purpose	Methods	Results	Conclusions
Milewski and Liu (2009a)	Qualitative correlation	To examine cancer incidence rates in the three largest cities in New Brunswick (Moncton, St. John and Fredericton) and to identify environmental contributors to cancer incidence.	Incidence data obtained from the New Brunswick Cancer Registry and national data from the National Cancer Registry. Risk factors based on qualitative analysis of literature together with occupation and labor statistics from New Brunswick using qualitative methods	Cancer incidence of certain cancer shown in the literature to be associated with particular occupations are increased, and those occupations and potential exposures do exist in these communities	Efforts should be taken to reduce exposures to industrial chemicals and air pollution. Better community-level cancer surveillance and biomonitoring should be conducted.
Milewski and Liu (2009b)	Qualitative correlation	To examine cancer incidence rates in 14 urban and rural areas in New Brunswick and to identify environmental contributors to cancer incidence	Incidence data obtained from the New Brunswick Cancer Registry and national data from the National Cancer Registry. Risk factors based on qualitative analysis of literature together with occupation and labor statistics from New Brunswick using qualitative methods	Cancer incidence of certain cancer shown in the literature to be associated with particular occupations are increased, and those occupations and potential exposures do exist in these communities	Efforts should be taken to reduce exposures to industrial chemicals and air pollution. Better community-level cancer surveillance and biomonitoring should be conducted
Kreiger et al. (2003)	Expert panel	To identify opportunities for cancer prevention research and surveillance with respect to environmental exposures	The Cancer Care Organization of Ontario convened an expert panel and administered a survey to the expert panel	The panel identified numerous contaminants and constituents and made generic recommendations concerning opportunities for surveillance and prevention	Observations and recommendations were not very specific in nature