CHILDHOOD EXPOSURE TO AIR POLLUTION AND LONG-TERM HEALTH

ASSESSING THE LONG-TERM HEALTH EFFECTS OF CHILDHOOD EXPOSURE TO ADVERSE AIR QUALITY: CASE STUDY FROM HAMILTON, ONTARIO (1975 – 2005)

By

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ABSTRACT

This thesis examines the relationship between childhood exposure to air pollution and long-term health. The research is based on an earlier study (1978-1986) that examined the relationship between exposure to air quality and respiratory health for a cohort of elementary school-aged children (n = 3,202). These children resided in four distinct neighbourhoods in Hamilton, Ontario, which exhibited significant gradients in air pollution levels.

Informed by the Life Course Health Development model, a survey was developed and administered on a reconstructed cohort (n = 395). The following objectives were addressed: 1) to determine the current health status of the reconstructed cohort; 2) to assess the potential relationship between childhood exposure to air pollution and adult respiratory health; and, 3) to explore factors mediating this relationship. Data was collected for a range of variables including residential and occupational histories, socio-demographic variables, and health outcomes. The dataset was merged with data from childhood on respiratory health, exposure to air pollution, and socio-demographic variables.

Results indicate that a relatively high percentage of respondents had asthma in childhood (11%) compared to the original cohort (5.5%). In addition, prevalence rates of most health outcomes were higher than those of the Canadian population. Despite the gradient in air pollution levels, there were no significant differences in health status across neighborhoods. However, results of bi-variate and multi-variate analysis indicate possible significant associations between childhood exposure to SO_2 and hospital visits

for asthma, asthma incidence in adulthood, and ever being diagnosed with asthma for females. For males, results suggest that childhood exposure to SO_2 is not linked to respiratory health. Factors related to the macro and micro environments also play significant roles in long-term health.

This thesis made significant contributions to knowledge by suggesting that childhood exposure to SO_2 may impact long-term respiratory health for females, and may be linked to inflammatory diseases.

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CHAPTER ONE

INTRODUCTION

1.1 RESEARCH CONTEXT

The health consequences of acute episodes of air pollution are well documented; episodes such as the Meuse valley fog of 1930 in Belgium and the London fog of 1952 clearly demonstrate the potential of ambient air pollution to affect mortality and morbidity (Brunekeef and Holgate 2002). The dense fog of the Meuse Valley (December 1930) that contained high levels of industrial pollution from coal and coke burning resulted in 60 deaths over a two day period (Nemery et al. 2001). The most affected were elderly, asthmatics, those who suffered from poor health, or those with cardiac disease. In December 1952, the high levels of air pollution in London, UK gave rise to a smoke-laden fog that shrouded the capital. High levels of sulfur dioxide and smoke resulted in an increase in death rates that persisted over a period of several months. Estimates suggest that in the several months following the episode, a total of 12,000 additional attributable deaths occurred in the population (Bell and Davis 2001). The scale of the disaster of the London fog of the 1950s became a landmark in air pollution epidemiology.

Although pollution abatement initiatives have reduced acute air pollution episodes over the last half century, air pollution still occupies a central focus in global environmental issues (Seinfeld 2004). Currently, the health effects of chronic exposure to air pollution remain a focus of concern for scientists, policy-makers and the general

public. For example, a Pub Med search using 'health effects of air pollution' as key terms uncovered 2,280 English language articles published over the last ten years. While this is much lower than we would find for major health outcomes like cancer (622,288) or cardiovascular disease (417,195), it is a relatively large proportion of papers published on the relationships between environment and health more generally (8,528).

Policy makers are using the findings of this research to inform decision-making. For example, the U.S. Environmental Protection Agency (EPA) under the Clean Air Act revised the standards for particulate matter (PM) (first issued in 1971) in 1987, 1997, and 2006. These revisions were based on thousands of peer-reviewed scientific studies that focus on the effects of particle pollution on public health and welfare. This final revision led to a new standard for the 24-hour fine particle ($PM_{2.5}$) equivalent to 35 micrograms per cubic meter down from 65 micrograms per cubic meter (PM Standards Revision EPA 2006).

Research that focuses on the economic and healthcare costs of air pollution drives much of this policy work. For example, a report by the Ontario Medical Association estimates that illnesses attributed mostly to exposure to particulate matter under 10 μm in diameter (PM_{10}) and ozone cost the Province of Ontario, Canada (population approximately 11 million) about $600 million annually to the healthcare system and $560 million in direct losses to employers and employees. These losses do not include the annual costs of pain and suffering, or loss of life, estimated at $5 billion and $4 billion respectively (Ontario Medical Association 2000).

Concern over the health effects of air pollution translates to the general public. A study based in Ontario, Canada found that 54% of Ontarians believe that air pollution is already negatively affecting their health; 79% believe that an effect on their health is imminent (Wellner 2000). Gallup (2006) found that 67% of Americans reported that the quality of the environment is getting worse over time; respondents were particularly concerned about air quality.

1.2 THE EVIDENCE

Many studies that focus on relationships between air pollution and health show that exposure to air pollution is associated with increased morbidity and mortality. For example, a review of 24 studies (published between 1994 and 2003) that examined relationships between deficits in lung function growth in non-asthmatic children and exposure to air pollution (particulate matter of various sizes and sulfur dioxide) found that 75% of the reported associations were statistically significant. Other significant associations reported include exposure to air pollution and increased emergency room visits (Atkinson et al. 1999), increased demand for general practitioner consultations (Hajat et al. 2002), hospital admissions for respiratory conditions (Atkinson et al. 1999; Hruba et al. 2001), and mortality (Brunekreef and Forsberg 2005).

Age is an important factor in the relationship between air pollution and health. Epidemiological studies from Europe and the US found that exposure to particulate matter was positively associated with hospital admissions for asthma and chronic

obstructive pulmonary disease and all-cause mortality among older people (Brunekreef and Holgate 2002). Furthermore, a recent report on air quality and asthma highlighted the disproportionate impact of air pollution on populations; findings indicate that five times more children than adults die from asthma each year (US EPA 2002). Similarly, a recent report from Toronto, Ontario estimates that children experience more than 1,200 acute bronchitis episodes per year as a result of air pollution from traffic sources (Toronto Public Health 2007). Indeed, literature reveals that children bear a disproportionate share of the negative health consequences of exposure to air pollution, due to their relatively smaller lung surface, their stage of health development, and the proportion of time they spend outdoors (Neidell and Matthew 2004; Vichit-Vadakan et al. 2001). Furthermore, since children are not generally subject to lifestyle confounders (such as smoking and occupational exposures), they provide a useful focus for research (Vichit-Vadakan et al 2001).

Despite the substantial existing literature on the health impacts of childhood exposure to air pollution, studies tend to focus on short-term adverse health effects (Kerigan et al. 1986; Pinter et al. 1996; Braun-Fahrlander et al. 1997; Kramer et al. 1999; Heinrich et al. 2002). This practice does not account for the potential long-term health effects associated with childhood exposure to air pollution, especially given that some illnesses and health effects may have a long latency period. For example, research indicates that although exposure to occupational or residential asbestos is related to malignant mesothelioma, the mean latency period after which the disease is expressed is 44.6 years (Marinnacio et al 2007). In relation to asthma, a 10-year study that examined

the latency period between occupational exposure to bakery dust and asthma found that the latency period for the onset of symptoms ranged between few months and 24 years with a mean of 7.9 years (Smith 2005). Many researchers have acknowledged the need for studies that assess the long-term health impacts of childhood exposure to air pollution (Gauderman et al. 2000; Peters et al. 1999; Avol et al. 2001; Calderon-Garciduenas et al. 2003).

The scope of moving beyond the assessment of short-term to long-term health impacts of childhood exposure to air pollution necessitates longitudinal research designs with long follow-up periods. There are relatively few longitudinal studies of the health effects of childhood exposure to air pollution; furthermore, these studies are limited by follow-up periods that may be insufficient in the context of examining the relationship between air pollution and long-term health (i.e. few months to ten years). Longer follow-up periods may be necessary for the development of some chronic diseases which may in fact be associated with childhood exposure to air pollution; this hypothesis remains to be tested.

This thesis examines the long-term health effects of childhood exposure to adverse air quality using as a foundation a comprehensive examination of a cohort of over 3000 children originally studied in Hamilton, Ontario between 1978 and 1986.

1.3 BACKGROUND STUDY

In the late 1970s, David Pengelly and colleagues initiated a research program that examined the respiratory health effects of childhood exposure to air pollution in Hamilton, Ontario (Kerigan et al. 1986; Pengelly et al. 1989). Hamilton is an industrial city with a legacy of air quality issues stemming from the local economy that remains heavily dependent upon two major steel manufacturing facilities (Buzzelli et al. 2003). While levels of air pollution in Hamilton have fallen substantially over time, they were quite high during the period in which Pengelly and colleagues undertook the original research (www.cleanair.hamilton.ca). Furthermore, human exposure to air pollution across the city is not uniform given both the physiographic and industrial landscapes of Hamilton. That is, the City of Hamilton is bisected by a physiographic feature know as the Niagara escarpment, a 350 foot (107 meters) limestone shale structure (euphemistically referred to as 'the mountain') that affects both winds and climatic patterns. In addition, Hamilton's heavy manufacturing, including integrated steel facilities, is concentrated primarily in the north east end of the city on the harbourfront (see Figure 1.1).

The research undertaken by Pengelly and colleagues remains a gold standard with respect to investigations of air quality and health. First, the large size of the original sample (n = 3,202) allowed Pengelly and colleagues to undertake a robust analysis of the links between various air pollutants and children's health. The cohort of participants initiated in 1976 included all students from Grades 4, 5, and 6 attending schools randomly selected from five geographically distinct neighbourhoods across the city of

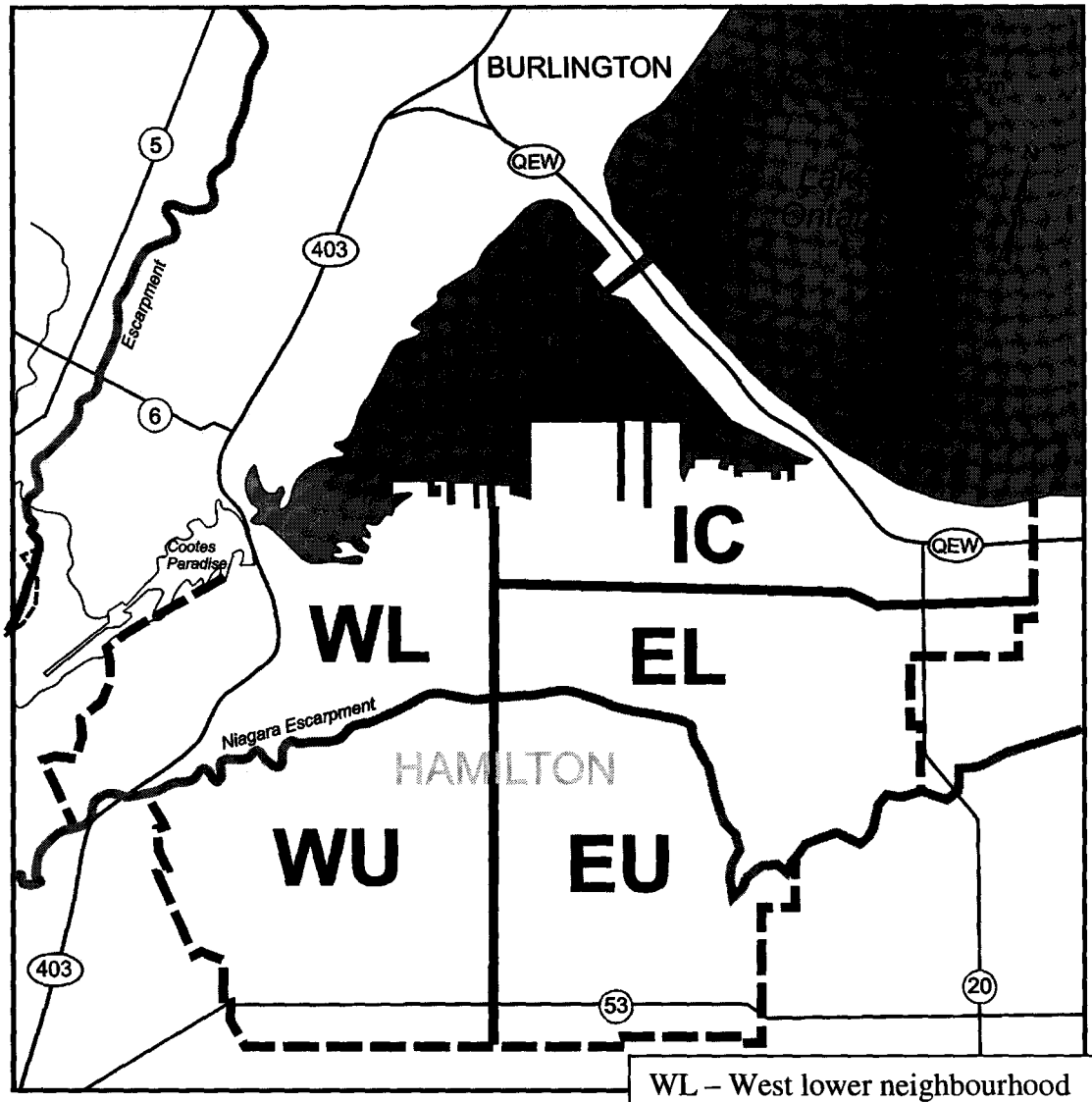

Figure 1.1 Map of study area

Hamilton (Figure 1.1) (Kerigan et al. 1986). In addition, a substantial amount of both exposure and outcome data were collected. Exposure data were obtained from a complex

network of 22 air quality monitoring stations in Hamilton measuring levels of SO_2 and particulate matter at various mass median diameters. Outcome measures (documented across five different time periods: 1978/9, 1979/80, 1980/1, 1983/4, 1985/6) included anthropometric (i.e. lung function) and self-reported (i.e. respiratory symptoms) health measures. In addition, a range of demographic (e.g. education, gender) and household (e.g. smoking status, cooking methods, family income) variables were also documented.

Analysis of the air quality monitoring data showed that the selected geographically distinct neighbourhoods exhibited a gradient in air pollution levels with the east lower neighbourhood (merged with the industrial core) having the highest levels of air pollution compared to the other three neighbourhoods. This research program found that airway obstruction increased for asthmatic children exposed to SO_2 levels above 10.6 ppb (Pengelly et al. 1989). In addition, exposure to fine particulate matter ($PM_{3.3}$) was found to be positively associated with airway obstruction. Further analysis revealed that the distribution of covariates between the four neighbourhoods was non-uniform, particularly parental smoking and gas cooking, with the highest rates being in the lower city industrial core (Kerigan et al. 1986).

The research described in this thesis is based on a longitudinal design that involves reconstructing the original cohort. The first stage of the research involved using web-based search engines to trace original cohort participants to present day. The second stage involved the development and administration of a data collection tool for the reconstructed cohort. Data collected included health, lifestyle, occupational, and residential histories of the reconstructed cohort. The final stage involved merging the old

and new datasets in order to assess the long-term health effects of childhood exposure to adverse air quality. The specific objectives of the research were:

1. to document the current health status of the reconstructed cohort with a particular emphasis on respiratory health;
2. to assess the potential relationship between childhood exposure to air pollution and adult respiratory health; and
3. to explore factors mediating this relationship

1.4 GEOGRAPHIC CONTEXT

Geographers have long recognized the role of place in shaping health (Gatrell 2002). Where one lives affects one's risk of disease or ill health. Access to basic resources such as nutritious and affordable food, clean water, clean air, decent housing, and rewarding employment is also geographically differentiated. Traditional medical geography emerged as a sub-field of geography concerned with the spatial patterning of disease and death and that of healthcare provision (Curtis and Taket 1996). The influence was mainly on the biomedical model of health. By the end of the 20th century, a new geography of health and healthcare emerged from the traditional medical geography. The new agenda focuses on human awareness, agency and creativity, particular forms of social organization, and cultural values in health experience (Curtis and Taket 1996). Emphasis is placed on the social construction of health and illness and on exploring the

role of socio-economic processes in the production of inequity in health and resource distribution (Curtis and Taket 1996).

In relation to air quality, health geographic research focuses on health impacts of air pollution in relation to spatial patterns of exposure (Madsen et al 2007; Jerrett and Finkelstein 2005; Romero et al 1999). For example, a US study found that spatial patterns of airborne toxic releases that result from socio-spatial processes (such as transportation networks and industrial activities) create a 'riskscape' that disproportionately impacts the health of populations (Cutter and Solecki 1996). In Canada, the Quebec City – Windsor corridor of Ontario and Quebec contains the most intensive point pattern of polluters that results from urbanization activities and the location of industrial facilities (Jerrett et al 1997). This gradient in air pollution is used to examine the chronic health impacts of exposure to different levels of air pollution (Jerrett and Finkelstein 2005). Similar spatial patterns in air pollution are reported in various studies from Europe and Latin America (Bayer-Oglesby et al 2005; Bakonyi et al 2004; Peacock et al 2003; Calderon-Garciduenas et al 2003). The significant gradient in air pollution for the study area in this research allows for an investigation of the long-term health impacts of childhood exposure to air pollution.

Using geography as a foundation, environment and health research searches for explanations related to factors that affect both exposure to air pollution and health outcomes. For instance, research points to the link between exposure to air pollution and socio-demographic factors such as race or income (Jerrett et al. 1997; Perlin et al. 1999; Buzzelli et al. 2003). In addition, research identifies air pollution as a major contributor

to mortality and morbidity (Sahsuvaroglu and Jerrett 2003). In examining the link between air pollution and health, this research extends the range of potential determinants of long-term respiratory health to include spatial and temporal factors related to the physical and socio-economic environments.

Informed by the Life Course Health Development (LCHD) model (Halfon and Hochstein 2002), this research investigates the relationship between exposure to air pollution and long-term health. While the main research question focuses on the health impacts of childhood exposure to air pollution, the model examines the effects of various factors on this relationship; these include residential and occupational histories, opportunities for social networking, healthcare accessibility, socioeconomic factors, lifestyle, and life events. In addition, the LCHD model integrates these geography-related factors with the micro-environment of health development where psychological, physiological, and behavioral processes impact health. A detailed discussion of the LCHD is provided in Chapter Two.

1.5 PRACTICAL IMPORTANCE OF THE RESEARCH

In 2000, air pollution-related illnesses were estimated to cost the Province of Ontario 1,900 premature deaths and over $1 billion in economic loss (Ontario Medical Association 2000). These estimates are based on 9,800 hospital admissions, 13,000 emergency room visits and 47 million sick days for employees. Moreover, a report prepared for Clean Air Hamilton estimated that five key air pollutants - nitrogen dioxide

(NO_2), ground-level ozone, inhalable particulate matter (PM_{10}), sulphur dioxide (SO_2), and carbon monoxide (CO) - contribute to about 100 premature deaths, 140 respiratory hospital admissions and 480 cardiovascular hospital admissions each year in Hamilton (Sahsuvaroglu and Jerrett 2003). While policy-makers and public health officials rely on such research findings to obtain rational and objective information (Garvin and Eyles 1997), there are several limitations associated with studies that focus on the health impacts of air pollution. For instance, although certain confounding or effect-modifying factors (such as lifestyles and housing conditions) play a role in the relationship between air pollution and health, researchers may encounter methodological or conceptual limitations in assessing these influences. Generating effective conceptual, temporal, and spatial methodologies that facilitate hypotheses building (Dunn and Kingham 1996) can potentially assist in the understanding of the relationship between air pollution and health.

In understanding the relationship between air pollution and health, it is therefore important to examine the effects of broad factors related to the social and physical environments from a temporal and spatial dimension. For instance, it is important to examine the potential role that socioeconomic status in childhood plays in impacting exposure to air pollution, which in turn affects long-term health development.

This thesis contributes to these needs in several ways. Primarily, this research will investigate the health status of the reconstructed cohort and the relationship between exposure to air pollution in childhood and adult respiratory health. In addition, the modeling component of the data analysis performed in this research will inform policy by

exploring the roles of various factors including exposure to air pollution in the development of various health outcomes, and by providing estimates of the relative contributions of various explanatory variables. The thesis also demonstrates the use of a conceptual framework that integrates the timing of exposure and the effects of diverse factors and determinants on health development.

1.6 ORGANIZATION OF THE THESIS

This thesis is organized into six chapters. In the next chapter, current evidence on the relationship between childhood exposure to adverse air quality and health is reviewed and the theoretical perspective that informs the research is discussed in detail. Chapter Three details the methodology used in this research. The chapter focuses on the method used to trace original participants, the epidemiological survey development, a description of the data collection protocol, definition of variables, and the analytical techniques adopted. Results of this research are presented in the two following chapters. Chapter Four describes the profile of the reconstructed cohort in relation to health status and socioeconomic variables. It describes the prevalence of health outcomes with a particular emphasis on respiratory health. In addition, Chapter Four examines bi-variate relationships between health outcomes and various exposure variables, including exposure to air pollution. Chapter Five contains the results of logistic regression modeling of health outcomes in relation to predictor variables, in order to explore the role of childhood exposure to air pollution as a determinant of long-term health. The final

chapter is devoted to the conclusions drawn from this research. It also addresses the substantive, methodological and theoretical contributions of the research as well as the limitations. The thesis concludes with a discussion of future directions for research.

CHAPTER TWO

LITERATURE REVIEW

2.1 INTRODUCTION

This chapter reviews relevant literature examining the relationship between childhood exposure to air pollution and health. The chapter begins with a discussion of developments in the geography of health and the conceptual framework that guides this research. The following section reviews the empirical literature on the relationship between childhood exposure to air pollution and health, including methods for assessing exposure and health outcomes in childhood. The chapter concludes with a summary of key findings.

2.2 GEOGRAPHIES OF HEALTH

By the end of the twentieth century, 'geographies of health' had evolved from traditional medical geography. The new agenda focused on examining place effects on health, human agency or actions, beliefs about health and behavior, the social construction of health and illness, and the role of socio-economic processes in the production of inequity in health and resource distribution (Curtis and Taket 1996). Three main themes were identified from this shift (Kearns and Moon 2002). First, 'place' emerged as a framework for understanding health. Second, researchers adopted

theoretical perspectives that highlighted issues of difference. Third, geographies of health became more critical and policy-relevant.

The renewed role for place that emerged in the new geographies of health explores health-related characteristics of places. Place is recognized as an arena where people's health is influenced in both positive and negative ways (Kearns 1993). Health geographic research that explores the role of place in impacting health can be classified into three main types (Kearns and Moon, 2002). Place-specific or 'locality' research focuses on community responses to threats to health. An example of this type of research is a study based in Oakville, Ontario that examined how residents that reside near a petroleum refinery negotiated odour and how this impacts symptom reporting (Luginaah et al. 2002). Research that focuses on 'landscapes' examines the 'meanings' of specific landscapes from a mainly cultural perspective. The goal is to understand how people negotiate their health status based on specific meanings. For example, a study based in an urban industrial neighbourhood in Hamilton, Canada examined differences between expert and community perceptions of risks from air pollution (Elliott et al. 1999). The third group of research used quantitative means to focus on place effects on health, mainly employing multilevel modeling, GIS, and spatial analysis techniques. For example, Perlin et al. (1999) uses GIS to examine relationships among the location of Toxic Release Inventory (TRI) facilities in the US, their total annual air emissions, and socio-demographic characteristics of residents that live nearby. The study found that more African Americans live in proximity to TRI facilities than non African-Americans.

Although limited in capturing realities of place effects on health, quantitative techniques are useful in addressing questions of health equality / inequality.

The second theme related to the new geographies of health focuses on the adoption of theoretical perspectives concerned with issues of difference. Social theories are used to develop conceptual frameworks able to derive relationships between diverse factors and health, and to conceptualize observations (Litva and Eyles 1995). With the use of theories, the new geographies of health increasingly focused on issues of distribution and equity (Kearns and Moon, 2002). For instance, a study that examines environmental equity in Canada found that the Quebec City – Windsor corridor contains the most intensive pattern of air pollution emissions (Jerrett et al. 1997). Based on a structuration perspective, the study found that this pattern of air emissions is linked to Ontario urbanization, the geographic location of manufacturing facilities, dwelling value, and socioeconomic characteristics (such as household income, manufacturing employment, and population size). Overall, researchers suggested that the inequitable distribution of resources in populations may give rise to differentials in the health status of populations. In examining environmental equity in relation to air pollution, other research documents associations between exposure to air pollution and social or demographic factors such as income, race, and age (Cutter and Solecki 1996). For example, as discussed earlier a US study found that more African Americans live in proximity to Toxic Release Inventory (TRI) facilities than non African-Americans (Perlin et al. 1999). The study also found that the percentage of residents living in poverty increased as the number of TRI facilities within 2 miles of the residence increased. These

findings suggest that environmental equity in relation to exposure to air pollution may constrain individuals of lower socio-economic status to live in high exposure areas while unaware of the associated negative health impacts (Buzzelli et al. 2003).

Implications of research findings that relate to equity and distribution have an effect on public health policy and planning (Dummer 2008). The new geographies of health apply critical perspectives to focus on policy-relevance, inequities, social justice, gradients in health status, and resource distribution. These critical perspectives can lead to activism capable of generating a social justice agenda and transformative politics (Kearns and Moon, 2002). This direction towards criticality and activism is the third main theme in the new geographies of health.

As a result of this shift, health geographic research contributed to new understandings related to health. Findings highlighted the role of social determinants in impacting health (House et al. 1988; Mermot and Feeney 1996; Cattell 2001) and led to a broadening of the understanding of health determinants. This new understanding of health determinants contributed to the development of new models of health. The following section discusses the evolution of models of health.

2.3 MODELS OF HEALTH

Paralleling the shift in the 'geographies of health', new models of health evolved from the traditional biomedical model. In Canada, the first of these models was based on the Lalonde report entitled 'A New Perspective on the Health of Canadians' (Lalonde,

1974). The report proposed that the main determinants of health are 'life-style', 'environment', 'human biology', and 'health care organization'. The health promotion model evolved and later became an integral part of the health care system (Ottawa Charter 1986). Although the range of health determinants was broadened from traditional definitions of health (that focused on human biology, disease, and healthcare), this model ignored the role of social context in the development of lifestyle changes (Evans and Stoddart 1996). With the integration of social theories, health research helped in shedding light on this limitation. By the end of the twentieth century, a new conceptual model for health 'the population health perspective' developed.

Developed in the 1990s by the Canadian Institute for Advanced Research (CIAR), the population health framework integrates the links between health and four key determinants of health: social environment, physical environment, genetic endowment, and individual response (behavior). It illustrates the impacts of these determinants on disease, health and function, healthcare, well-being, and prosperity. A review of the population health literature indicates that a large number of studies adopted this framework to conceptualize health (Hayes, 1999). As a result, literature guided by the population health perspective suggested evidence of the influence of socially mediated factors on individual biology through effects on the immune, endocrine, and central nervous systems (Hayes and Dunn 1998). Other suggested mechanisms focus on exposures to environmental quality as a mediator in the link between social factors and health (Evans and Kantrowitz, 2002). Essentially, income and socioeconomic status influence exposure to environmental quality, such as proximity to hazardous wastes, air

and water pollution, ambient noise, housing quality, educational facilities, and work environments. In turn, these exposures impact health.

Although the population health perspective acknowledges the role of social environments in determining health, criticisms regarding this perspective emerged (Coburn et al. 1996; Poland et al. 1998; Legowski and McKay 2000; Coburn et al. 2003). Critics argued that the development of the framework is heavily based on an economic perspective that focused on reducing healthcare costs, and that the framework lacks clear recognition of the effects of the social context on health. For instance, Coburn et al. (2003) argued that the model limits analyses of the determinants of health at the macro-level and ignores issues of agency and action at the micro-level that influence health. Coburn et al. (1996) noted that the population health framework understates the roles of social classes, social movements, and community interaction and ignores the local contexts in which the health of real people is shaped. While the population health framework may be limited in examining root causes that relate to social determinants and in initiating major changes to health policies, it provides a useful framework for conceptualizing health.

More recently, researchers have suggested the importance of timing of exposures in impacting health (Hertzman and Wiens, 1996; Dixon, 2002; Morello-Frosch, 2002; Schwartz, 2004). For example, a review paper that examined literature on endocrine-related cancers suggests that susceptibility to cancer may be a result of exposures throughout the life course rather than exposures existing at or near the time of tumor detection (Birhbaum and Fenton, 2003). This increased focus on timing of exposure and

health development led to a new approach that integrates the population health perspective and health development; this led to the development of the Life Course Health Development (LCHD) framework (Halfon and Hochstein 2002). Within this model, health outcomes are determined by a combination of factors stemming from both the macro and micro-environments (Figure 2.1). The macro-environment includes health determinants related to genetics; the physical, social, family, psychological, cultural, and policy environments; as well as the health care system. The micro-environment provides an arena where the design, mechanism, and regulation of bio-behavioral processes translate information from the macro-environment into biological information that alters the functioning of biological processes. The design and process of health development consists of physiological, behavioral, and psychological systems that interact with mechanism and regulatory systems such as the nervous, immune, and endocrine systems.

Essentially, the LCHD framework that integrates elements of the population health framework with the concept of health development includes several key points. First, similar to the population health perspective, the LCHD framework recognizes the interaction between the social, environmental, psychological, and biological determinants of health outcomes. Second, the LCHD framework integrates the process of embedding where experiences over the life course are programmed into the structure and functioning of biological and behavioral systems; therefore, the timing of events and experiences over the life-course impact the development of health and disease. For example, research suggests that socioeconomic differences in the quality of early life experiences become embedded in brain activities, which conditions the host defense system (that depends on

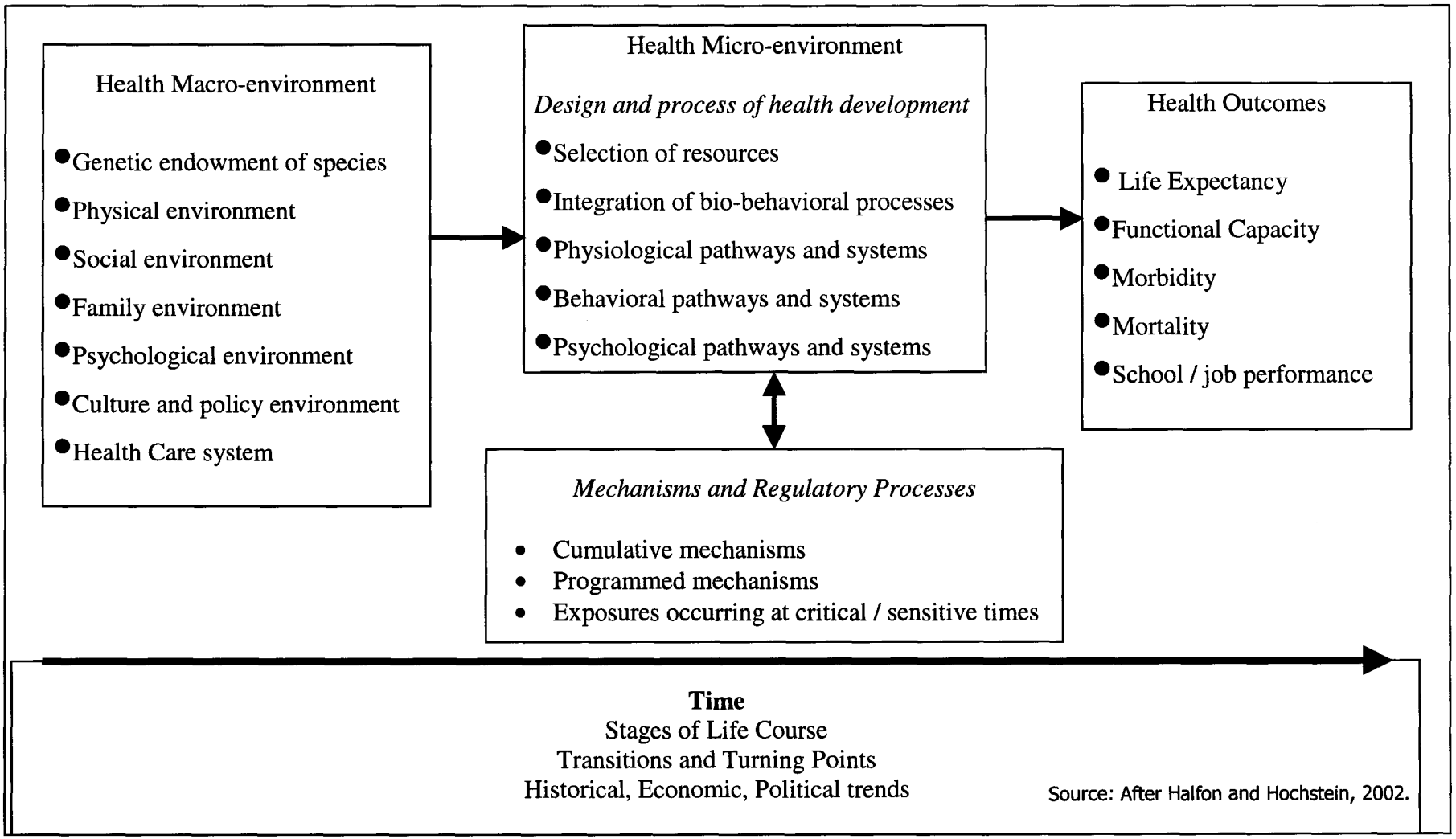

Figure 2.1 The Life Course Health Development Framework.

communication with the developing brain) and hence contributes to subsequent gradients in health status (Hertzman, 1999). Third, the LCHD framework highlights the role of risk and protective factors that balance gains in health status and growth of the human body against deterioration and loss. For example, behavior practices such as no tobacco use, moderate alcohol consumption and regular physical exercise exemplify the kind of activities needed to maintain balance in life against normal processes such as aging or against health exigencies (Breslow, 1999). While the latter two key points are implied in the population health perspective, they are explicit in the LCHD framework. As a result, the LCHD framework is a model suited to guide research that focuses on long-term health outcomes particularly since it accounts for the timing of exposures and the broad range of health determinants that impact health.

The LCHD model recognizes the importance of time and place in understanding the relationships between environments and health. Health development is influenced by the timing of exposure over the life-course that interacts with risk and protective factors which in turn lead to losses or gains in health development. Whether the timing of exposures occurs at a sensitive or critical period in a person's life-course leads to different health implications. When early exposures occur at critical or sensitive time periods, long-term or permanent change in an individual's functional system occurs (Ben-Shlomo and Kuh 2002; Wadsworth 1999). Exposures at a critical period in a person's life-course impact health, whereas exposures at a sensitive period in a person's life-course may have stronger health implications than non-sensitive or critical periods in a person's life-course (Halfon and Hochstein 2002). An example of research guided by this

perspective is a study that compares the height of English children from a range of socioeconomic backgrounds in order to assess the impact of early childhood deprivation on growth (Li et al. 2004). Height was measured at various ages. These researchers found that children living in lower socioeconomic households were 2-3 cm shorter at ages 7 and 11 than their counterparts living in higher socioeconomic status households during the same time period; these differences were statistically significant. This finding suggests that the timing of exposure plays an important role in health development and that the influence of risk and protective factors on health development also varies over the life-course.

While children are particularly susceptible to exposures in early life, opportunities and challenges that children encounter contribute to gradients in long-term health. These diverse opportunities and challenges relate to inequities in terms of environmental exposures and resource distribution, which in turn lead to inequities in health. While the LCHD framework provides the basis for integrating multi-disciplinary, longitudinal intervention strategies that improve the health of populations, these strategies are based on theoretical perspectives that examine links between gradients in exposures and health. For example, research findings that suggest health effects of prenatal or postnatal impaired growth in lung function on long-term chronic respiratory disease and potential time periods of reversible airflow obstruction (Kuh and Ben-Shlomo 1997) are based on the examination of the health effects of gradients in exposures at different time periods, particularly during the prenatal period and in childhood. Therefore, intervention strategies

are based on improving opportunities from an equity and redistributionist perspective in order to account for critical periods of health development.

The next section of this chapter is devoted to a review of the literature that addresses health impacts of childhood exposure to diverse air quality. A review of the types of exposures and outcomes investigated in the literature on air pollution and childhood exposure follows. The section concludes with a review of the evidence on the association between childhood exposure to air pollution and health.

2.4 THE RELATIONSHIP BETWEEN CHILDHOOD EXPOSURE TO AIR POLLUTION AND HEALTH

The health effects of air pollution have been the focus of a growing number of research studies since the London air quality issues of the 1950s and 1960s (Burnett et al. 2000). Research topics include examining bio-physical or psychosocial health effects related to air pollution. Air pollutants include a wide range of contaminants such as total suspended particulates, particulate matter of various diameter size, sulfur dioxide, ozone, nitrogen dioxide, acid aerosols, sulfates, and carbon monoxide. Bio-physical studies examine the relationships between exposure to various air pollutants and respiratory or other chronic health outcomes such as blood pressure changes or lung cancer. For example, Ruchirawat et al. (2007) focused on the risk of cancer in relation to exposure to chemical pollutants from urban air pollution. Other studies focused on psychosocial health impacts that result from the siting of point sources of air pollution (Luginaah et al.

2002). In examining the relationship between exposure to air pollution and health, studies focused on vulnerable sub-populations that may be most impacted by these exposures such as children or elderly. For example, the UK Childhood Cancer Study (1999) focused on the relationship between exposure to electromagnetic fields and the risk of cancer for young children.

The following section describes a systematic review of the evidence in relation to childhood exposure to air pollution and health outcome measures. The criteria for including studies in this review are guided by the overall objectives of this research and are discussed in the following section. A review of the evidence on the relationship between childhood exposure to air pollution and health follows the discussions on air pollution exposure assessment and health outcome measures.

2.4.1 REVIEW OF EVIDENCE

At the time the original Pengelly study was conducted, the cut-off size for measuring exposure to fine particulate was not recognized. The original study measured fine particulate using particulate matter under 3.3 μm in diameter ($PM_{3.3}$). Subsequently, recent literature uses $PM_{2.5}$ for fine particulate and PM_{10} for medium size particles. The existing 20-year old dataset contains exposure assessments for total suspended particulates (TSP), fine fraction particulate matter ($PM_{3.3}$) and sulfur dioxide (SO_2). The original cohort consisted to 3,202 children that attended Grades 4, 5, and 6 in randomly selected schools in four regions in Hamilton. The study design was longitudinal and collected health measures for five different sampling periods. Health outcomes included

symptomatic and anthropometric measures of respiratory health, mainly lung function growth measures. Accordingly, this review includes studies that were published since 1996 and that focused on the relationship between exposure to diverse air pollutants (mainly TSP, PM of any fraction size, and SO_2) and health effects (symptomatic, or anthropometric health measures, or both) for children.

A literature search was undertaken on MEDLINE, EMBASE, PUBMED, Science Citation Index, and CINAHL databases using the following key words: 'childhood or children' and 'air quality or air pollut$' and 'long$'. Studies that examined the health impacts of childhood exposure to particulate matter of any size and / or SO_2 were selected for this review. The review included studies that focused on asthmatic and non-asthmatic children. Furthermore, studies that assessed exposure based on a period less than eight weeks were eliminated from this review. The studies selected did not depend on the health outcomes measured and the location of the study area.

This strategy resulted in a total of 31 studies that form the basis of this review. Of these studies, five were review papers; three had a case-control embedded in a longitudinal cross-sectional design; the remainder studies were longitudinal cross-sectional or cohort designs. Two of the review papers focused on the link between air pollution and health for children in particular; the remainder did not make a distinction based on age. Appendix 2.1 presents a summary of the objectives and findings of these review papers. For the other studies that were included in this review, an examination of the geographical location of the study area, sampling and sample characteristics, research design, the air pollutants of interest, health effects, and findings related to the link

between childhood exposure to air pollution and health was performed. Thirteen studies were conducted in Europe, 7 in North America, 3 in Latin America, and 3 in the Far East (Appendix 2.2). Study duration varied between eight weeks and ten years; sample size varied between 17 and 19,090 participants. The following sections will focus on exposure and outcome assessment and the link between childhood exposure to air pollution and health.

2.4.1.1 AIR POLLUTION EXPOSURE ASSESSMENT

Most research that examines the link between air pollution and health tends to assess exposures on the basis of air pollution data collected from remote monitoring stations. Aggregate exposure measures that can be used in ecological designs have the capability of introducing ecological bias. The selection of regions with minimized within-region exposure variation and maximized between-region variation can reduce this bias (Morgenstern and Thomas 1993). However, this method does not control for potential bias that results from personal practices and choices such as the amount spent outdoors on a smoggy day. In order to improve exposure assessment, air pollution data collected from monitoring stations can be modeled to obtain individual exposure assessment measures for participants. Mathematical modeling can control for exposure variability resulting from personal, spatial, temporal, and emission sources. Although mathematical modeling can account for factors related to weather patterns and personal practices, there still remains a variance between exposure assessments and actual exposure to air pollution (Morgenstern and Thomas 1993). The use of personal monitors can be used to

obtain more accurate exposure assessment or to assess their correlations with data from remote monitors.

These diverse air pollution exposure assessment methods are reflected in the studies reviewed. Most studies (n = 24) used data from remote monitoring stations to quantify exposure to ambient air quality. Furthermore, in order to examine the effects of air pollution on health, researchers examined the health of sub-samples that are exposed to a gradient in air pollution by selecting study areas that exhibited a variance in air pollution levels. For example, the Children's Health Study included over 3,000 children between the ages of 8 and 15 that lived in 12 different communities in Southern California that exhibited gradients in air pollution levels (Gauderman et al. 2002). Gradients of 25 $\mu g/m^3$ for $PM_{2.5}$ and 50 $\mu g/m^3$ for PM_{10} were reached between the least and most polluted areas. Another example is a study that examined the health of 1,294 participants from two areas in Hong Kong that exhibit a gradient in air pollution levels (Yu et al. 2001). Specifically, the mean annual concentration of TSP in the more polluted area was 57.6 $\mu g/m^3$ compared to 44.9 $\mu g/m^3$ for the less polluted area. Furthermore, the mean annual concentration of SO_2 in the more polluted area was 22.8 ppb (parts per billion) compared to 11.8 ppb for the less polluted area.

Another method that was used to examine the health of sub-samples that are exposed to an air pollution gradient was through the selection of study areas that showed an improvement in air quality. For instance, a longitudinal cross-sectional study based in Eastern Germany examined the health of 6,959 participants in 1992, 1995, and 1998 in relation to pollution levels for three different geographical regions (Heinrich et al. 2002).

The reunification of Germany in 1990 led to changes in the industrial landscape of Eastern Germany that gave rise to lower levels of air pollution. As a result, annual TSP levels in the first study region that Heinrich et al. (2002) selected decreased from 45μg/m^3 to 29μg/m^3. Similarly, a decrease from 64μg/m^3 to 25μg/m^3 was seen in the second study region and from 79μg/m^3 to 33μg/m^3 in the third study region. The changes in the levels of SO_2 in the three study regions were larger; from 78ppb to 8ppb in the first study region, 113ppb to 9ppb in the second study region, and 84ppb to 6ppb in the third. Similarly, Kramer et al. (1999) selected six regions in Germany to examine the health of 19090 participants exposed to a gradient in air pollution levels. These regions included four from Eastern Germany and two from West Germany. In other studies, residing in urban versus non-urban regions was used to differentiate between exposure to high and low pollution levels (Peacock et al. 2003; Zhang et al. 2002; Van der Zee et al. 1999).

Although most studies examined the health of sub-samples exposed to spatial or temporal gradients in air pollution levels, one study examined the health effects of relocating from areas of high to low air pollution and from areas of low to high pollution (Avol et al. 2001). Based on an examination of the health of 110 participants from six states in the US, the study found that the rate of lung function can be altered by a large change in air pollution.

In addition to the use of data from remote monitoring stations, two studies performed exposure modeling on this data to get estimates of individual exposures (Pikhart et al. 2001; Hruba et al. 2001). The study undertaken by Hruba et al. (2001) used a combination of dispersion modeling and GIS to assess exposure to $PM_{2.5}$, PM_{10}, and

total suspended particulates (TSP). Furthermore, a US study that examined the health status of a cohort of 269 children between the ages of 9 and 11 provides evidence of the reliability in using remote air pollution levels as a proxy in exposure assessment (Linn et al. 1996). This study used data from personal air quality monitors for a sub-sample to validate exposure assessment based on data from remote air monitoring stations. The study found that daily personal exposures to PM_{10} correlates with pollutant levels measured at the remote air pollution monitoring sites.

Based on the studies reviewed, there were large differentials in exposure assessments for the various air pollutants measured. These differentials are expected given the range of geographical regions in which studies were conducted. For example, exposure to TSP ranged from an annual average of 25 $\mu g/m^3$ (Heinrich et al. 2002) to a 4-year arithmetic mean of 728 $\mu g/m^3$ (Zhang et al. 2002). Exposure to PM_{10} ranged from an annual mean of 10 $\mu g/m^3$ (Braun-Fahlander et al. 1997) to a 2-year arithmetic mean of 237 $\mu g/m^3$ (Zhang et al. 2002). In addition, exposure to $PM_{2.5}$ ranged from an annual average of 12 $\mu g/m^3$ (Ward et al. 2002) to a 2-year arithmetic mean of 150 $\mu g/m^3$ (Zhang et al. 2002). Large differentials in exposure assessments for SO_2 were also apparent; the range extended from 1.3 ppb (Peacock et al. 2003) to a daily mean of 365 ppb (Sanchez et al. 1999).

In order to control for confounders that may obscure the relationship between exposure to air pollution and health, some studies developed participant inclusion criteria that provide some covariate control. These criteria included age, symptomatic or non-symptomatic children for respiratory health, and residential location. For example, all

1,001 participants in a study that examined the link between lung function and particulate matter and SO_2 were 9 years of age (Jedrychowski et al. 1999). Another study that examined the health impacts of exposure to air pollution for children selected 1,621 participants on the basis of asthma symptoms, low lung function, and gender (females) (Roemer et al. 1999). Statistical applications were also used to account for confounders. Confounders in most cases included residential features, personal covariates such as gender and age, household and lifestyle factors, parent health history and education, weather patterns, and factors related to location such as traffic.

2.4.1.2 HEALTH OUTCOME MEASURES

In examining the link between childhood exposure to air pollution and health, studies adopted diverse methods to assess health outcomes. Based on the studies reviewed, assessment of health outcomes can be classified into three main categories. These include morbidity or mortality data from health care services, self-reported health outcomes, and anthropometric measures of health outcomes. For example, Bakonyi et al. (2004) measured health outcomes based on the daily record of outpatient attendance of children with respiratory diseases. Similarly, the 'Air pollution and Health: a European approach' (APHEA) study aggregated the daily counts of total and cause-specific deaths and hospital emergency admissions in European cities (Atkinson et al. 2001).

Most studies, however, assessed health based on self-reported symptoms of respiratory health. Symptomatic measures include self-reported data such as morning and day / night cough, wheeze, phlegm, shortness of breath, colds, nasal congestion, and sore

throat. For assessing health outcomes, the focus is to identify whether chronic conditions (of cough, phlegm, and / or wheeze) that may be related to chronic obstructive diseases exist. For example, the study based in eastern Germany was concerned (among other health measures) with the presence of chronic morning cough (Heinrich et al. 2002). Another example is the 'Four Chinese Cities Study' that assessed the persistence of chronic cough and phlegm (Zhang et al. 2002). In most cases, researchers use standard questionnaires such as the International Study on Asthma and Atopy in Children (ISAAC) to collect self-reported respiratory symptoms of cough, wheeze, and phlegm (Bayer-Oglesby et al. 2005). Others used diary symptoms recording for assessing the persistence of chronic respiratory symptoms (Desqueroux et al. 1999; Van der Zee et al. 1999; Vedal et al. 1998).

In addition to self-reported symptoms of respiratory health, researchers focus on self-reported information related to the use of health care services and respiratory illnesses or asthma diagnosis. For example, Heinrich et al. (2002) collected data related to incidences of bronchitis, otitis media, sinusitis, frequent colds, and febrile infections. Kramer et al. (1999) focused on airway diseases such as pneumonia, bronchitis, and chest colds. In addition to collecting symptomatic data on wheeze, cough and phlegm, Zhang et al. (2002) assessed the health of the participants on the basis of hospitalization as reported by participants.

Most studies that were reviewed and that collected anthropometric measures of health outcomes mainly focused on the pulmonary function of participants (Calderon-Gardiduenas et al. 2003; Peacock et al. 2003; Gauderman et al. 2002; Gauderman et al.

2000; Peters et al. 1999; Avol et al. 2001; Jedrychowski et al. 1999; Sanchez et al. 1999; Van der Zee et al. 1999; Vedal et al. 1998; Pinter et al. 1996; Linn et al. 1996; Roemer et al. 1999). Pulmonary function measures include the forced vital capacity (FVC) (maximum exhalation volume), the forced expiratory volume in one second (FEV_1), the ratio of the forced expiratory volume in one second over the forced vital capacity (FEV_1/FVC), the forced expiratory flow at 75% of FVC (FEF_{75}), the peak expiratory flow (PEF), and the maximum mid-expiratory flow (MMEF). Two studies included a significant biological component such as skin testing (Ward et al. 2002) and sampling nasal lesions, blood sampling for biomarkers such as cytokines, and performing chest x-rays (Calderon-Garciduenas et al. 2003).

As illustrated in the reviewed studies, diverse measures of health outcome assessment and exposure assessment have been adopted in search for a link between childhood exposure to air pollution and health. The following section will use the main findings of these studies to characterize the health impacts of childhood exposure to air pollution.

2.4.1.3 KEY FINDINGS

This review indicates that most literature on the health effects of air pollution relates to the short-term health effects of exposure to adverse air quality (Appendix 2.2). These include increased emergency visits (Atkinson et al. 1999), hospital admissions for respiratory conditions (Brunekreef et al. 2005; Zhang et al. 2002; Atkinson et al. 1999; Desqueyroux et el. 1999; Hruba et al. 2001), and increased prevalence of low lung

function and the exacerbation of asthma (Brunekreef et al. 2005; Ward and Ayres, 2004; Calderon-Garciduenas et al. 2003; Peacock et al. 2003; Ward et al. 2002; Gauderman et al. 2002; Pikhart et al. 2001; Avol et al. 2001; Desqueroux et al. 1999; Sanchez et al. 1999; Vedal et al. 1998; Linn et al. 1996; Roemer et al. 1999). For example, a 10-year prospective study of respiratory health in school-aged children in Southern California found an association between PM_{10} and bronchitis, and between exposure to both $PM_{2.5}$ and PM_{10} and measures of lung function (Gauderman et al. 2002). A nested study that examined the health effects of relocation on the same children found that those who relocated from high to low air pollution regions experienced an increase in lung function growth and that lung function decreased for children who relocated to communities with poorer air quality (Avol et al. 2001). Another 3-year study that examined the respiratory health of school children living in eight districts of four different Chinese cities concluded that exposure to long-term ambient particulate matter of all size fractions (PM) is positively associated with children's respiratory morbidity (wheeze, asthma, bronchitis, hospitalization for respiratory diseases, persistent cough, and persistent phlegm) (Zhang et al. 2002).

For all studies included in this review, exposure to fine particulate matter is measured by $PM_{2.5}$. In general, research indicates that exposure to fine particles is more related to daily mortality than coarse particulates (Brunekreef and Forsberg, 2005). In addition, Shwartz et al. (2000) found that fine particles have stronger acute respiratory impacts than coarse particles. Furthermore, fine particulate matter has also been associated with decrements in lung function growth (Ward and Ayres, 2004). Similar

findings were reported in the 24-cities US and Canadian study that suggests that long-term childhood exposure to fine particulates and acid particles is associated with low lung function growth and symptoms of bronchitis in childhood (Dockery et al. 1996; Raizenne et al. 1996; Spengler et al. 1996).

In relation to exposure to sulfur dioxide, research indicates that exposure to long-term ambient air pollution particularly high levels of SO_2 (Pinter et al. 1996) is positively associated with children's respiratory morbidity prevalence (Zhang et al. 2002) and infectious airway diseases such as pneumonia, bronchitis, chest colds, and tonsillitis (Kramer et al. 1999). Furthermore, the 'Four Chinese Cities' study found that sulfur dioxide (SO_2) is associated with children's respiratory symptoms such as persistent wheeze, cough, and phlegm (Zhang et al. 2002). Similarly, the 'Small area variation in air pollution and health' study found that SO_2 is associated with wheezing in the last 12 months, lifetime prevalence of wheeze, and lifetime prevalence of diagnosed asthma (Pikhart et al. 2001). In addition, research indicates that an increase in SO_2 levels is related to a decrease in lung function for asthmatic and non-asthmatic children (Sanchez et al. 1999). This is consistent with findings that indicate that exposure to SO_2 (higher than 10.6ppm) and fine fraction particulate matter ($PM_{3.3}$) is related to increased airway obstruction for children with asthma measured by FEV_1 / FVC and MMEF ratios (Pengelly et al. 1989).

Overall, the literature suggests that children bear a disproportionate share of the negative health consequences of exposure to air pollution for several reasons: their relatively smaller lung surface, their stage of health development, and the proportion of

time they spend outdoors (Neidell et al. 2004; Vichit-Vadakan et al. 2001). Furthermore, this review indicates that longitudinal studies of the health effects of childhood exposure to air pollution are limited by the relatively short follow-up periods employed (i.e. few months to ten years). Such follow-up periods may be insufficient to examined whether exposure effects on long-term health persist or whether the development of some chronic diseases are in fact associated with childhood exposure to air pollution. In order to examine the long-term health effects of childhood exposure to air pollution, longitudinal designs with long follow-up periods are needed (Avol et al. 2001; Calderon-Gardiduenas et al. 2003). In conclusion, while the literature suggests associations between childhood exposure to air pollution and respiratory health, studies are limited by the fact that none have followed children through to adulthood to examine the long-term health impacts of exposure to ambient air pollution. This remains the primary focus of this research.

2.5 RESEARCH CONTEXT

This research takes the time and place elements of the LCHD framework into account in the analysis of data obtained from the reconstructed cohort of children originally studied in the late 1970s. These children lived in four distinct neighbourhoods in Hamilton, Ontario that exhibited a gradient in relation to air pollution. In addition to exhibiting the poorest air quality, the east lower and west lower neighbourhoods had the highest proportions of: households that utilize gas cooking, participants that share a room with at least two family members, family income below $10k/yr, and high residential

mobility (Kerigan et al. 1986). In addition, the east lower neighbourhood had the highest proportions of parental smoking and parental respiratory symptoms.

Differences in socio-demographic characteristics of Hamilton neighbourhoods have been identified in more recent studies (Luginaah et al. 2001). For instance, the Central Downtown Core (CDC) and the Northeast industrial area (situated in the west lower and east lower neighbourhoods respectively) are characterized by high proportions of low income, low levels of education, and high unemployment rate. In contrast to the Northeast Industrial neighbourhood that has a low recent immigrant population and a small minority population, the CDC has high proportions of recent immigrants, residential mobility, and a high proportion of non-English or French speakers and visible minorities. The Chedoke - Kirkendall neighbourhood (situated in the southwest regions of the west lower neighbourhood) is described as one with a moderate social diversity and high income inequality, whereas the Southwest Mountain (situated in the west upper neighbourhood) is characterized by few recent immigrants, low residential mobility, high education, and relatively low income inequality.

In relation to air pollution levels, while earlier research in Hamilton found a clear pattern of socioeconomic differentiation with respect to air pollution (Pengelly et al. 1986; Jerrett et al. 1997), a 2003 study found that industrial and spatial restructuring in Hamilton may have led to a redistribution in air pollution levels, which decreased differences in air quality across neighbourhoods (Buzzelli et al. 2003). However, a fifteen-year trend of SO_2 levels between 1991 and 2006 indicates that levels of SO_2 in Hamilton are higher than those in other Ontario cities (such as Oakville, Burlington, and

Toronto) primarily due to industrial emissions that are unique to Hamilton (Clean Air Hamilton, 2007). In 2005, the annual mean SO_2 level in downtown Hamilton was the second highest in Ontario (5.3 ppb) following Sarnia (7.8 ppb) (Ministry of Ontario, 2005). $PM_{2.5}$ levels between 2003 and 2005 in downtown Hamilton were similar to those of other cities in southwestern Ontario and the Golden Horseshoe, including the Greater Toronto Area (GTA) (Ministry of Ontario, 2005).

The spatial differences in health determinants and the gradient in air pollution levels across neighbourhoods that are depicted in the study area are likely to affect health. This research examines the relative roles that childhood exposure to air pollution and determinants play in long-term health. Health determinants that are examined include factors related to the social, physical, socio-demographic, and socio-economic environments. Drawing from the LCHD framework, a conceptual model of potential determinants of long-term respiratory health is developed to guide the investigation (Figure 2.2). In this model, health determinants are categorized into two broad components that interact to impact long-term health: the macro-environment and the micro-environment of health development. The macro-environment of health development includes the physical environment, residential and occupational histories, social environment, family environment, life events, healthcare access, and demographic factors. The micro-environment of health development is an arena where factors related to the macro-environment are translated into the design of physiological (such as lung function and respiratory symptoms / illnesses in childhood), psychological (psychosocial factors such as emotional distress), and behavioral (such as lifestyle choices) processes.

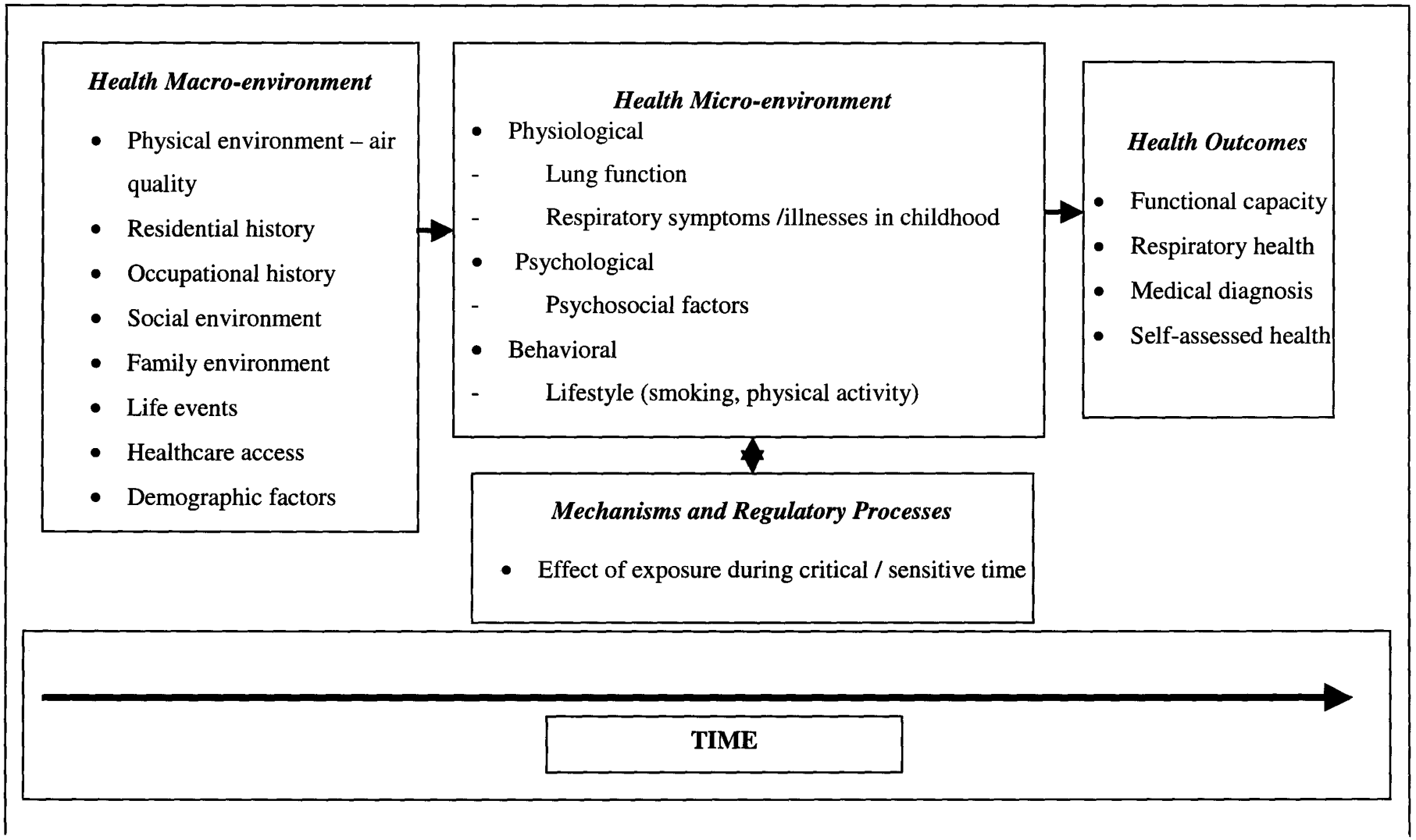

Figure 2.2 Conceptual model for studying the relationship between childhood exposure to air pollution and long-term health

The timing of exposure across the life-course influences and regulates these bio-behavioral processes.

2.6 SUMMARY

This chapter reviewed relevant literature that relates to the relationship between childhood exposure to air pollution and health. The literature review explored recent themes that emerged from the new geographies of health. These include the emergence of 'place' as a framework in understanding health, the use of theories to guide research, and the move towards a critical geography of health (Kearns and Moon, 2002). The developments of the field shed light on the importance of social determinants of health. Research findings contributed to concepts of health that evolved from the biomedical model. The chapter also included a review of studies that examined the relationship between childhood exposure to air pollution and health.

The discussion on health geography revealed an increased focus on the social determinants of health that is outlined in the conceptual framework that guides this research. The LCHD framework integrates the effects of various determinants that relate to the macro-environment (physical environment, residential history, occupational history, social environment, family environment, life events, and healthcare access) and the micro environment (physiological, psychological, and behavioral systems) of health development. In addition, the LCHD development framework accounts for the timing and place of exposures.

The review of studies that examined the link between childhood exposure to air pollution and health suggests that air pollutants have significant health impacts. However, none of the studies followed participants to adulthood to discern the long-term adult health effects of early childhood exposure to air pollution. Overall, literature findings suggest that childhood respiratory symptoms appear to be attributed to exposure to particulate matter, whereas low lung function growth and respiratory illnesses in childhood appear to be attributed to exposure to sulfur dioxide (SO_2).

The next chapter outlines the methodology used in this research. It consists of the design of the research, the development of the data collection tool, and the specification of the analytical techniques used.

CHAPTER THREE

METHODOLOGY

3.1 INTRODUCTION

This chapter is divided into three main sections. The first section describes the design of the study. In doing so, the method used to reconstruct the Hamilton Children's cohort and details of the questionnaire that was administered to the reconstructed cohort are outlined. This is followed by a section that outlines the derivation and / or definition of variables that are included in the analysis. The final section describes the analytical methods that are used to address the research objectives.

3.2 RESEARCH DESIGN

This research is a longitudinal cohort design that is based on a follow-up period of over twenty years. Data relating to the original cohort of children were collected during five distinct sampling periods between 1976 and 1986. In order to examine the link between childhood exposure to air pollution and long-term health, the original cohort of participants was traced to present day. Traced participants were contacted and a new reconstructed cohort was formed. Guided by the LCHD framework, a questionnaire was developed and subsequently administered on the reconstructed cohort (Appendix 3.6). Analysis was performed on the merged dataset that includes data collected in childhood

as well as current data. The following sections describe the cohort reconstruction and the data collection processes.

3.2.1 COHORT RECONSTRUCTION

Cohort reconstruction is limited by the availability of information that can be used to trace original participants. In addition, there are epidemiological considerations related to tracing; for instance, selection bias may be an issue if participants that are traced differ from participants that are lost to follow-up in terms of the exposure or characteristic under study (Salamone and Cauley 1997). In addressing potential bias and loss of statistical power related to follow-up in cohort studies, a number of studies have proposed acceptable follow-up rates ranging from 50% to 80% (Babbie 1973; Altman 2000). However, a recent simulation study of logistic regression modeling tested these recommendations for follow-up loss percentages ranging from 5% to 60% (Kristman et al. 2004). The study examined three different scenarios related to loss to follow-up: missing completely at random (MCAR) (i.e. independent of exposures and outcomes), missing at random (MAR) (i.e. independent of outcomes but possibly due to exposures), and missing not at random (MNAR) (i.e. dependent on health outcomes). Results indicate that as much as 60% loss to follow-up (maximum percentage in the simulations) will produce an unbiased estimate of effect provided that loss to follow-up occurred at random (MCAR or MAR) and independent of the outcome measured (Kristman et al. 2004). Loss of follow-up independent of the health outcome should not bias the measures of association and could be ignored in the analyses (Siddiqui et al. 1996).

Despite limitations and considerations in tracing research participants, international studies demonstrate that long-term follow-up of participants and re-establishing contact with participants may be feasible with the appropriate planning and persistence (Wutze et al. 2000; Rodger et al. 2001; Garcia et al. 2003). Tracing methods in the past were limited to the use of contact information, medical registries, and hospital records (Kreiger et al. 1990; Spitzer et al. 1982). However, the development of the World Wide Web has facilitated the tracing of participants in our present day mobile society that is influenced by access to the economic and social resources of a globalized world (Hampson 2001; Koo et al. 2000; Tehranifar et al. 2002). In relation to tracing success, recent research findings indicate that a combination of methods, including the use of Internet-based search engines and commercial firms is most successful for cohort reconstruction (Hampson 2001; Mertens et al. 2004; Tehranifar et al. 2002). However, the use of commercial firms for tracing participants involves financial costs of approximately $32 per person (Weinberger et al. 2002). For large sample sizes, this method may be restricted by financial limitations.

A literature review indicates that the level of tracing success varies between 39% and 99% and is dependent on the quality of information obtained at the time of initial recruitment, gender, geographical location, the length of time since last contacted (Hampson 2001), and socioeconomic factors (Mertens et al. 2004). For instance, a New York study used free nationwide telephone directories and fee-based internet services to trace over 39% of mother and daughter pairs following a 30-year no-contact period (Tehranifar et al. 2002). A two-year study used a combination of public records, personal

contacts, and an Internet-based people finder to locate over 75% of a sample of children on two Hawaiian Islands after a follow-up period of 40 years (Hampson 2001). A 25-year follow-up in Australia was able to trace 66% of research participants using a combination of tools such as phone and electoral records, health insurance information, and health records from the Hepatitis foundation (Rodger et al. 2001). Record linkage was used in Spain to trace 99% of participants after a 6 year follow-up (Garcia et al. 2003).

Given the large number of children that was sampled for the original research, resource limitations dictated the sole use of publicly available web-based search engines to trace the original cohort of children that was studied in Hamilton between 1978 and 1986, almost 20 years after the last contact. The sole use of this method for tracing also provides a unique opportunity to examine the effectiveness of this low cost tool for cohort reconstruction.

In order to provide a robust framework for data analysis, a full-scale cohort reconstruction initiative was launched to trace as many of the original research participants as possible. As a result, 3,202 participants for whom contact information existed were traced. Contact information included data collected in childhood pertaining to the participants' first and last names in childhood, residential address, residential phone number, and parental contact information. The challenge then became to trace that contact forward to the present day using publicly available web-based search engines. Participants were searched using two web-based directories: Yellow.ca and Canada 411[1]. Yellow.ca and Canada 411 are local search engines and online directories featuring over

[1] www.canada.ca; www.yellow.ca.

12 million residential listings covering all Canadian cities and towns in Canada. Using these two directories, cohort members were searched by name, childhood address, and / or phone number. The first step involved a 'reverse' search option by phone number provided in Yellow.ca (Figure 3.1). The 'reverse' option gives names and an address that correspond to the inputted phone numbers. If no match by name or residential address in childhood occurred to the phone number entered, a search by surname was initiated, followed by the available participant and parental names. A cohort member was successfully traced if the search resulted in the following: i) a match to the original address and/or phone number, ii) only one match by surname, or iii) a match to the first name or initial of male cohort members or fathers, and first and surname match for female cohort members or mothers. Initially, searching for cohort members by name was limited to the City of Hamilton; this assumed that the cohort member or their family still resided in Hamilton. For the purpose of increasing sample size, if the cohort member was not successfully traced by name, the search criteria were broadened to include searching in all of Ontario and then again to include searching in all of Canada. For each cohort member not found via Yellow.ca, the 'reverse search' by address option offered through Canada 411 was used. This option retrieves the occupant (s) name (s) and phone number when the residential address in entered in the appropriate field. A cohort member was successfully traced when the last name of the occupant at the address matched that of the cohort member.

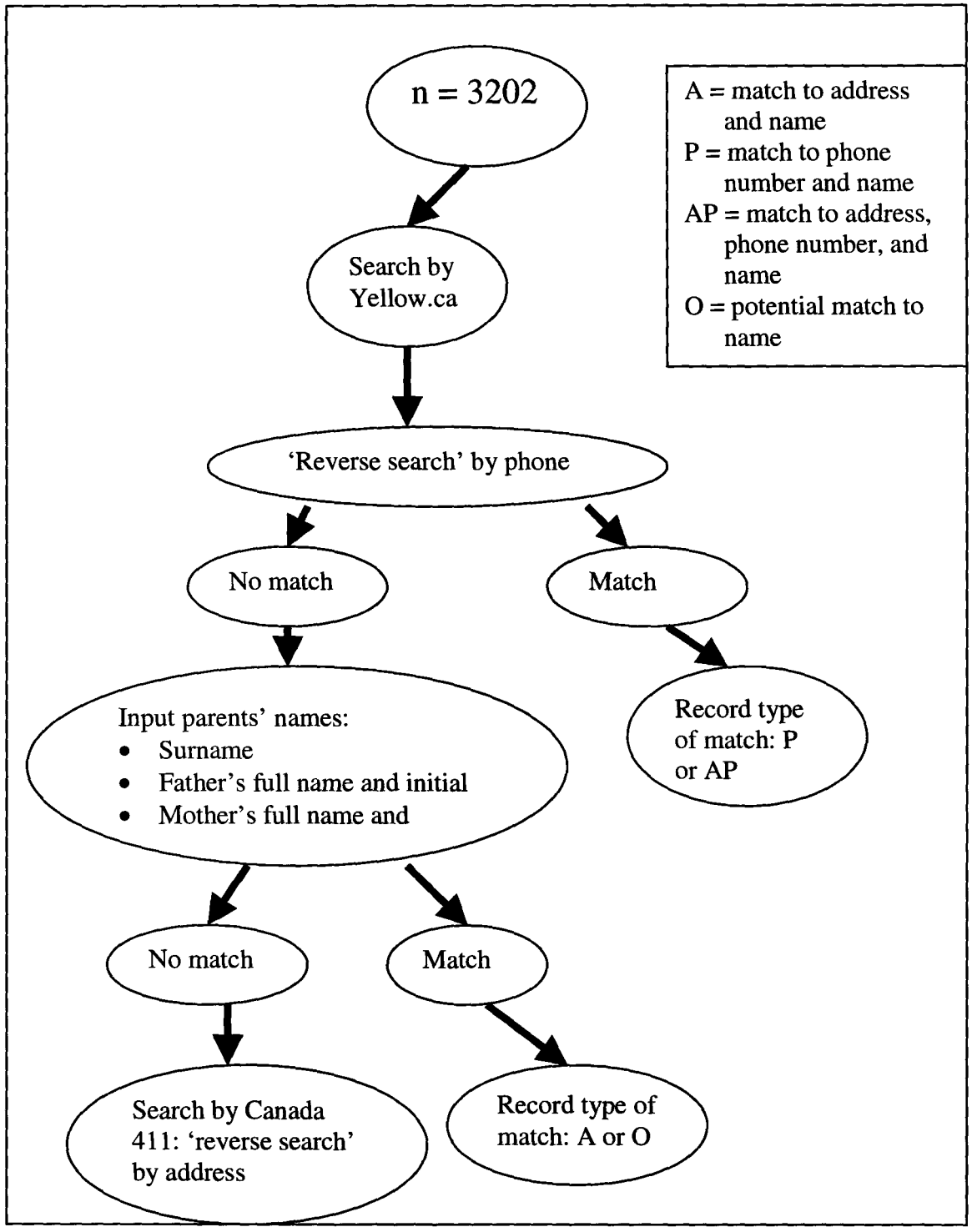

Figure 3.1 Strategy employed for tracing the participants of the 'Hamilton Child Cohort (1978 – 1986).

3.2.1.1 TRACING OUTCOME

Using this methodology, 1,374 of the original 3,202 cohort members (42.9%) were traced (Table 3.1). Several factors enhanced tracing success. First, participants for whom all contact information was available were easiest to trace. Second, female participants for whom a father's first name or initial was available were relatively easier to trace than females without a father's first name or initial, particularly since females may undergo name changes in adult life.

In order to assess sample bias, characteristics of the traced cohort were examined in relation to residential location, socioeconomic status, gender, and respiratory health (mainly asthma prevalence) in childhood. Results indicate that neighbourhood of residence in childhood of the traced participants are distributed among the four geographical quadrants with the lowest percentage of participants traced to the west lower quadrant (WL). There were statistically significant differences for tracing in relation to the four geographic quadrants ($\chi 2 = 23.86$, $p < 0.05$). Almost 50% of participants that reside in the WU neighbourhood in childhood were traced compared to 39% of those that resided in each of the WL and EL neighbourhoods (Table 3.1). This is consistent with another Hamilton study that indicated high population mobility for the Central Downtown Core (CDC) and low population mobility for the Southwest Mountain neighbourhoods, respectively situated in the WL and WU neighbourhood of the study area (Luginaah et al. 2001). In addition, the original study found relatively lower mobility for the WU (22%) and EU (19%) neighbourhoods compared to the WL (28%) and the EL (26%) neighbourhoods (Kerigan et al. 1986).

Table 3.1 Location and characteristics of the traced participants

Cohort members traced:	Neighbourhood						Overall	
	WU	EU	WL	EL	IC	Not Assigned	n	%
	Q1	Q2	Q3	Q4				
To childhood address and phone number– 'AP' (n)	138	170	96	110	12	5	531	16.6
To childhood address (n)	52	49	32	51	12	0	196	6.1
To childhood phone number (n)	76	68	38	37	12	1	232	7.2
By name (n)	113	66	88	116	21	11	415	13.0
Total Traced (n)	379	353	254	314	57	17	1374	42.9
% traced per neighbourhood	49.9	43.6	39.3	39.4		38.6	N/A	N/A
% of total traced	27.6	25.7	18.5	27		1	N/A	100
% distribution of asthma in original cohort	26.3	34.9	15.4	23.4		0	N/A	100
% distribution of asthma in traced cohort	34.6	29.6	12.3	22.3		0	N/A	100
% asthma prevalence in original cohort	6	7.5	4.1	4.3		0	N/A	5.5
% asthma prevalence in traced cohort	7.3	7	3.8	4.8		0	N/A	5.8

† WU=upper west, EU= upper east, WL = lower west, EL = lower east, IC=industrial core, 'Not Assigned' =location not coded in the original dataset.

The childhood asthma prevalence rate of traced participants (5.8%) compares well to the childhood asthma prevalence rate of the original cohort (5.5%). Further analysis revealed that tracing success is related to sex ($\chi 2 = 19.6$, $p < 0.05$) and socioeconomic status in childhood ($\chi 2 = 89.1$, $p < 0.05$). In comparison to the original cohort, the traced cohort includes less female participants (44% versus 48%) and a smaller proportion of participants of lower socioeconomic status in childhood (11% versus 21%).

Given these results, several issues need to be highlighted. First, successful tracing of 42.9% in this research is comparable to results of other reconstruction projects that use similar tracing tools and for which the mobility of the population is comparable to that of Hamilton, Ontario. For instance, a New York study successfully traced 39% of an original cohort following a 30-year period of no contact (Tehranifar et al. 2002). Second, the gradient in childhood exposure to air pollution that is depicted in the original study is comparable to that of the traced cohort, given that the latter is relatively well distributed over the four geographical neighbourhoods. Furthermore, results indicate that although a significant proportion of original participants were not traced (57.1%), loss to follow-up is related to gender, socioeconomic status in childhood, and residential location in childhood. This percentage of loss to follow-up falls within the suggested threshold of up to 60% that produces an unbiased estimate of effect provided that loss to follow-up occurs at random and independent of the outcome measured (Kristman et al. 2004). Limitations encountered during the tracing process include missing or incomplete contact information; for instance, phone information was missing for 5% of the participants and 79% did not include a father's name. In addition, since female participants are more

subject to name changes during adult life, the information available for tracing female participants was limited. Consequently, 29% of the female participants from the original cohort were lost compared to 27% of males. This is consistent with research that suggests sex as a determinant for tracing success (Hampson, 2001).

The above results indicate that follow-up contact with traced participants can potentially lead to a new reconstructed cohort, which will facilitate this longitudinal research. Before contact was initiated with participants, ethical approval was obtained from the McMaster University Research Ethics Board (MREB). This process aimed to minimize the psychological and social risks to the participants, particularly since parents of the participants were assured anonymity and confidentiality at the time of the initial study. These risks were mitigated through the use of introduction letters and phone scripts that emphasized the sole use of public search engines for tracing and the importance of this research in expanding knowledge on the relationship between childhood exposure to air pollution and long-term health.

3.2.1.2 PROTOCOL FOR ESTABLISHING CONTACT

Following the tracing process, a protocol was developed for establishing contact with participants (Figure 3.2). First, parents of the participants for whom we had contact information were contacted and informed of the study. They were sent a letter of information that informed them of the study and prepared them to expect a follow-up phone call (Appendix 3.1). The parents were then contacted via telephone and a

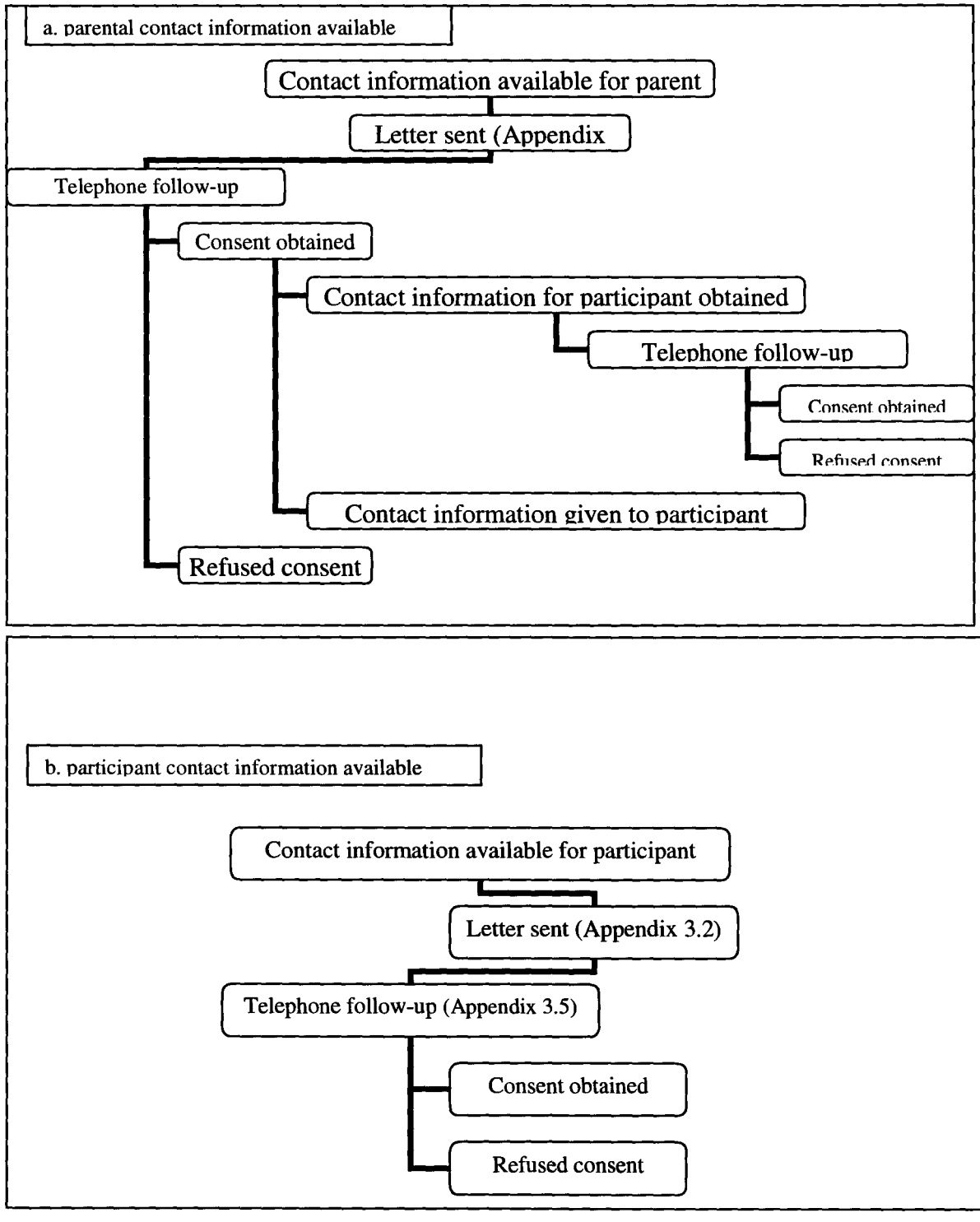
a. parental contact information available
Contact information available for parent
Letter sent (Appendix
Telephone follow-up
Consent obtained
Contact information for participant obtained
Telephone follow-up
Consent obtained
Refused consent
Contact information given to participant
Refused consent
b. participant contact information available
Contact information available for participant
Letter sent (Appendix 3.2)
Telephone follow-up (Appendix 3.5)
Consent obtained
Refused consent

Figure 3.2 Protocol for establishing contact with research participants.

telephone script was administered. The telephone script requested consent for using their old data; if consent was obtained, the script requested the original participants' contact information or provided contact information to the potential participants (Appendix 3.3). For those participants whose full contact information was retrieved during the cohort reconstruction process, direct contact was made (Appendix 3.2). Appendices 3.4 and 3.5 include the telephone scripts that were administered to the research participants. The purpose of the telephone calls was three-fold. The first purpose was to verify that the right person was reached particularly for participants that were searched by name. The second purpose was to determine the respondents' willingness to allow previous data to be used. The third purpose was to determine if traced individuals are interested in participating in this research.

Contact was established with 69% of the traced participants (n = 929). There were statistically significant differences in relation to tracing method ($\chi 2 = 95.36$, $p < 0.05$). Tracing participants by childhood address and phone number match (AP) led to the best contact results with only 9% of participants lost to follow-up compared to tracing by childhood address (10%), phone number in childhood (14%), and by name (58%). Overall, contact was established with 32% of the original cohort participants. 64% of the contacted participants consented to participate in the study (n = 595) and were mailed a questionnaire. Follow-up post cards and telephone reminders were used to enhance response rates. The reasons given for refusal included death (2.7%) (in such instances cause of death was documented), lack of interest / time (40%), and no family contact with original participant (4.5%). 66% of non-respondents were males (compared to 52% of

responders) and 5.4% had been diagnosed with asthma in childhood (compared to 11% of respondents). Overall, this contact protocol was administered from August 2005 to February 2007 and resulted in a response rate of 66% (n = 395). The reconstructed cohort represents 12% of the original cohort and 30% of the traced cohort. Table 3.2 presents characteristics of the traced, lost to follow-up, and reconstructed cohorts.

3.2.2 DATA COLLECTION

3.2.2.1 COMPONENTS OF THE DATA COLLECTION TOOL

Participants that consented to participate in this research were sent a questionnaire and a self-addressed stamped envelope. Guided by the conceptual framework, the questionnaire aimed at collecting data related to the main constructs of the model: the macro-environment, the micro-environment, and health outcomes (Appendix 3.6). The macro-environment includes the physical environment (air pollution), residential history, occupational history, social environment, family environment, life events, healthcare access, and demographic factors. Appendix 3.7 includes an itemization list of macro-environmental variables included in the questionnaire.

Data related to childhood exposure to air pollution was obtained from the original cohort dataset and will be discussed in the next section. In addition, the questionnaire collected data about second-hand exposure to smoking at home or in the workplace. Residential history was documented using a series of questions that asked about current and previous addresses, duration of residence at each address, description and age of

Table 3.2 Characteristics of the traced, lost to follow-up, and the reconstructed cohorts.

Variable (in childhood)	Traced cohort	Lost to follow-up cohort	Original cohort	Reconstructed cohort
Exposure to smoking (%)	63	69	63	55
Smoker in childhood (%)*	33	27	28	16
Family income (% below low income cut-off)*	7	19	14	16
Gas cooking (%)	14	18	16	22
Asthma (%)	6	5	6	11
Persistent morning cough (%)	5	5	5	8
Persistent day / night cough (%)	6	6	6	12
Persistent wheeze (%)	5	4	5	15
Chest illness before age 2 yrs (%)	9	9	6	6

*$p < 0.001$

property, type of heating, property ownership, residential exposure to pesticides / contaminants / insecticides, the number of people that live in the household, the availability and use of an air conditioning system, and the most frequent method used for cooking (item 6c). Moreover, two questions collected data on air duct cleaning practices and the use of air filters or purifiers (items 6b and 6f). Data was collected for up to five previous addresses.

Occupational history was documented using a range of questions related to current employment status, current employment, occupational history, type and duration of employment, as well as exposure to dust, gas, fumes and other contaminants. Item 4 consists of three components that collect information on occupational exposures (Appendix 3.7). This item was derived from a question from the American Thoracic Society that asks if respondents have 'ever been exposed to gas or chemical fumes' on the job (ATS-DLD-78-A). In order to allow for a broad range of exposures that participants' may have been exposed to, the question was rephrased to ask whether participants were exposed to 'dust', 'chemicals', or 'other contaminants' on the job. Due to the follow-up period of over 20 years, the questionnaire collected information for up to 12 previous occupations.

Data related to the social environment were collected using the Social Health Battery (SHB) (Donald and Ware 1984) (items 16 – 26). The SHB was selected for its ability to measure objective indications of levels of social resources (using the social contact subscale) and social interaction in the general population (using the group participation subscale). Data related to the family environment were collected using one item from the Ontario Health Survey that asked about marital status, as well as data related to parental morbidity and mortality. Participants were asked whether their biological mother or father were diagnosed with respiratory diseases such as chronic bronchitis, emphysema, asthma, lung cancer, and other chest conditions (Source: American Thoracic Society questionnaire). In addition, data related to parental ages or parental age at time of death and reason of death were collected (items 49 and 50).

Data on life events were collected using six items that asked about financial crises, separation or divorce, serious illnesses, and death of a spouse or close family member (items 46 a – e). Data on healthcare use and availability of medical insurance were also collected. Items 31 and 32 collected data on whether the participants' had a regular family doctor and the types of usually accessed healthcare facilities. Items 34 and 35 asked about the frequency of regular medical check-ups and the availability of any additional health insurance coverage. Demographic data were collected using items from the Ontario Health survey that asked questions about the participants' age, sex, current family income, and highest level of education completed. In addition, two items collected data on participants' ethnic background and their place of birth.

In relation to the micro-environment construct of the model, the questionnaire collected data related to psychosocial impacts and behavioral practices such as lifestyle and physical activity. Appendix 3.8 includes an itemization list related to the micro-environment construct of the model. One item collected data on the participants' height and weight. In order to examine potential psychological impacts that may play a role in long-term health development, three items collected data related to perceptions about air pollution in relation to the participants' current neighbourhood of residence. These items (items 6i–j) asked about concern over air pollution in the neighbourhood and perceptions that air pollution in the neighbourhood may lead to psychological impacts for participants or members of the household (Source: Luginaah et al. 2002). The same set of questions collected data on up to five previous residences. In addition, psychosocial impacts were documented using the General Health Questionnaire (12) (Goldberg 1970) (item 47).

This instrument was selected because of its high sensitivity (93.5%) and specificity (78.5%) for measuring elements of distress (mainly anxiety, depression, social impairment, and hypochondriasis) by focusing on changes in normal functioning rather than life-long traits. Using the Delighted-Terrible scale (Andrews 1976), one item collected data about participants' feelings about health, income, and life in general (items 48 a-c).

Data related to lifestyle were collected using questions that asked about smoking history and alcohol consumption. Current smoking and smoking in the past (including frequency) were documented (items 9 – 12). The age at which active smoking and second-hand smoking began and stopped were also documented using an item from the American Thoracic Society Survey (items 13 and 14). One item collected data related to the frequency of alcohol consumption. In addition, data related to physical activity were collected using three connecting questions that focus on time spent doing physical activities during leisure time (items 45, 45a, and 45b); this question was taken from the Joint Canada / US Survey of Health that was administered in 2002 / 2003 to residents of Canada and the US.

Health outcomes were measures using a range of questions that collected data on functional capacity, respiratory health, medical diagnosis, and self-assessed health (Appendix 3.9). Two items taken from the SF-36 questionnaire measured functional capacity over day-to-day demands and during difficult or unexpected problems (items 28 and 29). Participants were asked about current respiratory symptoms such as cough, wheeze, phlegm, and chest colds (items 37 – 44). The same questions that were

administered on the original cohort and that relate to respiratory symptoms were included in this questionnaire. In addition to measures of symptomatic respiratory health, participants were asked to report on any medical diagnoses. Participants were asked whether or not they were ever medically diagnosed with chronic bronchitis, asthma in adulthood, asthma in childhood, other chest conditions, long-term skin conditions, hay fever or allergies, arthritis or rheumatism, any respiratory problems, high blood pressure, heart disease and cancer (items 30a i – xiii). This component was guided by items from the American Thoracic Society questionnaire that focused on medical diagnosis. Furthermore, one item collected data on whether participants were diagnosed with asthma in childhood (item 31). Data were also collected for medication use and frequency (items 30c – e). Another measure of health outcome that was collected relates to hospitalization data; participants were asked whether they were ever admitted to a hospital or whether they had an emergency room visit for asthma or respiratory problems since leaving elementary school (item 34). One item measured self assessed health by asking participants to rate their health relative to other people of the same age (item 27).

3.2.2.2 DATA FROM CHILDHOOD

In examining the temporal relationship between childhood exposure to air pollution and long-term health, childhood exposure data were obtained from the original cohort dataset. Each participant was assigned an exposure value for each of total suspended particulates (TSP), fine fraction ($PM_{3.3}$), and sulfur dioxide (SO_2) for the different sampling periods between 1975 and 1986. The method for estimating exposure

is described in Pengelly et al. (1987); the researchers derived a response surface model to estimate yearly exposures. Exposure assessment data that were used in the analysis include data for TSP (1980/1, 1983/4, 1985/6), $PM_{3.3}$ (1980/1, 1981/2 1983/4, 1985/6), and SO_2 (1978/9, 1980/1, 1981/2, 1983/4). Furthermore, in exploring the relationship between exposure to air pollution in childhood and long-term health, participants were classified according to the four neighbourhoods that they resided in as children and that exhibited a gradient in air pollution.

In addition to adult health measures, data on childhood health measures were obtained from questionnaires administered by trained interviewers at five sampling periods to the principle guardian (1978/9, 1979/80, 1980/1) and to the participants themselves (1983/4, 1985/6). Guided by the LCHD framework, this data corresponds to the micro-environment of health development where health experiences over the life-course are embedded into the structure and functioning of physiological pathways and systems, which in turn impacts long-term health. The questionnaires included the same items 37 to 44 that are outlined in the previous section. Responses provided information on respiratory symptoms and medical diagnosis for respiratory illnesses in childhood. Specifically, data available included existence of morning cough, persistent morning cough, day or night cough, persistent day or night cough, wheeze, persistent wheeze, and whether the participant experienced chest colds in childhood. In addition, information on whether participants experienced chest illness before the age of two, and medical diagnosis with asthma, pneumonia or other chest illness in childhood was collected.

In addition to childhood symptomatic data, anthropometric data on lung function were collected in childhood. Pulmonary function testing was performed on each child for each of the five sampling periods. Measurements were made of indicators derived from the forced vital capacity maneuver. The protocol for obtaining health measures is detailed in Kerigan et al. (1986). For the purpose of this research, the FEV_1/FVC indicator is used to assess pulmonary function in childhood. This measure was selected because it is intrinsically independent of body size (Pengelly et al. 1989).

Guided by the conceptual framework, the new and old datasets provide the necessary information to examine the long-term health effects of childhood exposure to air pollution. As a result, the new data and the old dataset were merged using the merge by ID option available in the Statistical Package for the Social Sciences (SPSS) software. This gave rise to a large dataset collected over a 30 year period that includes exposure measures to air pollutants in childhood ($PM_{3.3}$, TSP, SO_2), respiratory symptoms and illnesses in childhood and adulthood, health status in adulthood, and diverse socioeconomic, lifestyle, and socio-demographic variables over the life-course.

3.3 OPERATIONAL DEFINITION AND DERIVATION OF VARIABLES

This research investigates the long-term health effects of childhood exposure to air pollution using a wide range of potential variables that may mediate this relationship as guided by the LCHD conceptual framework (Chapter 2). These variables are categorized into the broad areas represented by the constructs of the conceptual model.

Figure 3.3 shows the variables that were used for analysis in this study mapped onto the conceptual framework.

Data used in the analysis includes continuous and categorical data. Appendices 3.10 – 3.12 present defined or derived variables that correspond to the macro-environment, micro-environment, and health outcome constructs of the conceptual model. In relation to childhood exposure to air pollution, continuous data on exposure assessment was used in the analysis. In addition, participants were grouped into two categories based on whether they were exposed to air pollutants above the median level or at or below the median level for each sampling period. This method of exposure assessment is widely used in air pollution research mainly since the median accounts for asymmetrical distributions in air pollution levels.

In order to facilitate analysis, some indices were created for certain variables. The purpose was to create one variable for each of the following factors that are examined in the conceptual framework: concern over air pollution, exposure to air pollution in childhood, respiratory symptoms in childhood, and lung function in childhood. This was done by coding and summing responses to related items, followed by dividing the sum by the number of items. For example, using responses for items 6h – 'How concerned are you about air pollution in your neighbourhood?', 6i – 'How likely is it that air pollution will lead to health problems for you?', and 6j – 'How likely is it that air pollution will lead to health problems for other members of your household?' an index was created that measures participants' concerns over air pollution in adulthood. The responses "not at all concerned" or "very unlikely" were coded as 0, "slightly concerned" or "somewhat

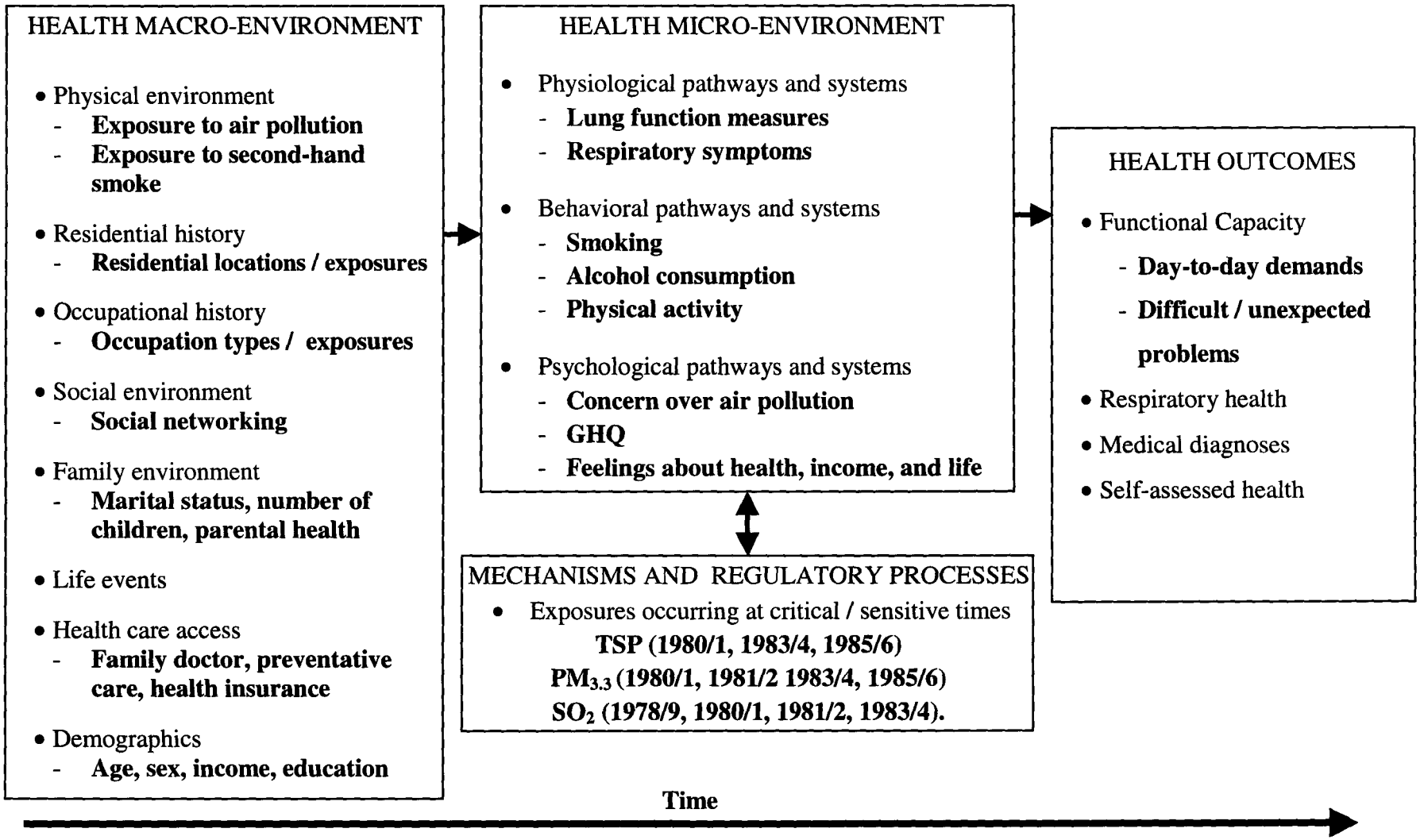

Figure 3.3 Variables mapped on the conceptual model.

unlikely" were coded as 1, "moderately concerned" or "somewhat likely" were coded as 2, and "extremely concerned" or "very likely" were coded as 3. The codes were summed and the sum was divided by three. As a result, the index measure ranged between 0 and 3, increasing as concern over air pollution increased. Similarly, for each participant, health and exposure data that were collected during the different sampling periods of the original study were used to create indices of health outcomes and exposures in childhood. The occurrence of a health outcome was coded as 1, whereas the absence of a health outcome was coded as 0. These codes were summed and the sum was divided by the number of sampling periods during which data for the health outcome were collected. Essentially, indices were created for persistent morning cough, persistent day or night cough, persistent wheeze, chest colds, and lung function using the FEV_1/FVC ratio for data that was collected between 1976 and 1986.

From the continuous exposure assessment data, indices were created to indicate the frequency of exposure to TSP, $PM_{3.3}$ and SO_2 above or below the median level in childhood. Exposure above the median level was coded as 1, whereas exposure below the median level was coded as 0. Similar to the health outcome indices, codes were summed and the sum was divided by the number of sampling periods during which exposure data were collected. Furthermore, responses to item 6a (Which best describes your heating system?) were not included in the analysis since most participants (n = 390) indicated that they use a forced air heating system.

3.4 ANALYTICAL METHODS

In addressing the objectives of this research, data were analyzed using the Statistical Package for the Social Science (SPSS – version 12). In order to characterize the sample, frequency analyses were run for all variables. The first objective of the research was to document the current health status of the reconstructed cohort with a particular emphasis on respiratory health. The merged dataset was used to perform frequency analysis for the following variables: self assessed health, functional capacity, and diverse morbidity measures (medical diagnosis, hospitalization, and respiratory health). In order to examine differences in health outcomes in relation to the four neighbourhoods of the study area, data were aggregated and analyzed for each of the four neighbourhoods, as well as for the entire sample.

Guided by the conceptual model, bi-variate analyses were performed to identify significant relationships that impact long-term health. In order to assess the potential relationship between childhood exposure to air pollution and adult respiratory health, bi-variate analyses were performed between variables that measure exposure to each of TSP, $PM_{3.3}$, and SO_2 in childhood and respiratory health outcomes in adulthood. Furthermore, bi-variate analyses were performed between exposure assessment to air pollution (in childhood) and other non-respiratory health outcomes. Consistent with the conceptual model that guides this research, long-term health may be impacted by various determinants such as lifestyle, healthcare, and the physical, family, and social environments. Hence, health outcomes were analyzed in relation to determinants that are

included in the constructs of the conceptual framework. Measures of association included the t-test for continuous data and the chi-square for categorical data.

The third objective of this research was to explore factors (e.g. residential and / or occupational history, socioeconomic /psychosocial / physiologic / behavioral factors) that mediate the relationship between childhood exposure to air pollution and long-term adult health. Logistic modeling was selected because the dependent variables are dichotomous and the independent variables include a mix of categorical and continuous independent variables. Furthermore, the relationship between the dependent variables and the independent variables is not likely to be a one-to-one function.

In this context, logistic regression is used to predict the occurrence of the health outcome from a set of explanatory variables that correspond to the conceptual framework illustrated in Figure 2.2. Logistic regression modeling was used to examine the role that childhood exposure to air pollution plays in the following health outcomes: ever diagnosed with asthma or respiratory problems, asthma in adulthood but not in childhood, hospital or ER visits for asthma or any respiratory problems since leaving elementary school, chronic bronchitis, as well as non-respiratory conditions such as hay fever / allergies, arthritis / rheumatism, high blood pressure / hypertension, any chest conditions, any long-term skin conditions, any type of cancer, fair or poor self-assessed health, and fair or poor ability to handle day-to-day demands or difficult and unexpected problems.

For each of the modeled health outcomes, variables were grouped into blocks and then entered into the model one block at a time to identify significant predictors. The first step involved conducting forward stepwise entry of variables block by block to identify

significant predictors for each of the health outcomes using a significance level of $p \leq 0.1$. Predictor variables were then entered using forward stepwise regression with a $p \leq 0.1$. One value of each categorical independent variable was chosen to be the reference category. This process resulted in a model that included significant variables that explain the health outcome. The specificity and sensitivity of the model were noted.

The next step involved performing first-order interactions of significant predictors using a backward stepwise entry with a $p \leq 0.1$ in order to determine possible significant interactions on the health outcomes. Significant variables and first-order interactions were then entered using forward stepwise entry with a $p \leq 0.1$. This gave rise to a new model that included significant predictors and significant first-order interactions, as well as new specificity and sensitivity measures. The new model was compared to the initial model that excludes first-order interactions. Interaction terms were removed if they did not improve the sensitivity of the model. This process resulted in a best-fit model for each modeled health outcome.

In order to examine the relative role that long-term exposure to air pollution and neighbourhood of residence in childhood play in long-term health, the indices of exposure to TSP, $PM_{3.3}$, and SO_2 and neighbourhood of residence in childhood were forced into the best-fit model. Therefore, for each examined health outcome, the logistic regression process gave rise to two models. The first is a best-fit model that includes statistically significant variables related to the health outcome; the second is a model that explores the relative role that childhood exposure to air pollution plays in the development of long-term health. In order to explore whether the relationship between

exposure to air pollution and long-term health is sex-related, logistic regression modeling of each health outcome was performed for females and males separately, and for both sexes combined. The statistical significance of each predictor, the relative odds for each variable and the rho-square (i.e. goodness of fit measure) are presented for each model. A rho-square of 0.2 to 0.4 is generally considered to represent a good fit (McFadden, 1974 as cited in Wrigley, 1985). In addition, not all variables in the conceptual framework came out significant in this analysis. Chapters 4 and 5 only deal with variables that were statistically significant.

3.5 SUMMARY

This chapter described the methodology required for this study design and the data collection process for this research. The longitudinal study design required that participants from the original study be traced to present day to form a new, reconstructed cohort. Cohort reconstruction consisted of a tracing methodology and a protocol for establishing contact with original research participants. A questionnaire was then administered on the reconstructed cohort.

This chapter described the questionnaire that collected data related to the major constructs of the conceptual model. These include data related to the macro-environment and micro-environment of health development, and health outcomes. Data collected in childhood during the five sampling periods of the original research were also described.

The definition and derivation of variables from the merged dataset are presented in Appendices 3.10 – 3.12.

This chapter also described the analytical methods adopted for this research. Frequency analysis is used to characterize and assess the health of the reconstructed cohort with a particular emphasis on respiratory health. Bi-variate analysis is performed to examine the relationships between childhood exposure to air pollution (TSP, $PM_{3.3}$ and SO_2) and each of respiratory health and non-respiratory health outcomes. Logistic modeling is performed to explore the relative role that various factors (mainly childhood exposure to air pollution) play on long-term health.

Chapter Four describes results of frequency analyses that characterize the reconstructed cohort in relation to each of the four neighbourhoods. The following chapter also outlines results of bi-variate relationships between childhood exposure to air pollution and long-term health outcomes, as well as between health outcomes and diverse determinants that are included in the conceptual framework that guides this research. The results of the logistic regression modeling of health outcomes are presented in Chapter Five.

CHAPTER FOUR

CHILDHOOD EXPOSURE TO AIR POLLUTION AND LONG-TERM HEALTH

4.1 INTRODUCTION

This chapter addresses the first two objectives of the research: 1. to document the current health status of the reconstructed cohort with a particular emphasis on respiratory health; 2. to assess the potential relationship between childhood exposure to air pollution and adult respiratory health. Section 4.2 describes the sample characteristics, exposure profile, and the health profile of the reconstructed cohort. Sample characteristics include the residential and socio-demographic profiles of the sample, as well as the macro-environmental (such as occupational history, social environment, family environment, life events, healthcare access, and demographic factors) and the micro-environmental (behavioural, psychological, and physiological) profiles. In examining the health profile of the reconstructed cohort, the health profile of two sub-samples will also be examined. These include a sub-sample of respondents who always resided in Hamilton (n = 214), as well as a sub-sample of respondents who always resided in their original residence from childhood (n = 33). The section concludes with a summary and discussion of descriptive results. Section 4.3 examines bi-variate relationships between childhood exposure to air pollution and long-term health. In examining these relationships, all analyses were done for both sexes combined, and separately for females and males. Furthermore, Section 4.3 presents statistically significant associations that were found between variables from the

macro-environment (residential and occupational histories, social and family environments, life events, healthcare access, and demographic factors), and micro-environment (physiological, psychological, and behavioural) constructs of the model. These include statistically significant associations that resulted from bi-variate analysis between two variables from the: i) macro-environment (macro – macro); ii) micro-environment (micro – micro); iii) macro- and micro- environments (macro - micro); iv) macro-environment and health outcomes (macro – health outcomes); v) micro-environment and health outcomes (micro – health outcomes). Analyses between variables from the macro-environment and micro-environment were undertaken in order to examine factors that may mediate the relationship between childhood exposure to air pollution and long-term health. The chapter concludes with an assessment of findings on the relationship between childhood exposure to air pollution and long-term health.

4.2 CHARACTERISTICS OF THE RECONSTRUCTED COHORT

4.2.1 RESIDENTIAL AND SOCIO-DEMOGRAPHIC PROFILES

The highest proportion of respondents (29%) resided in the west upper neighbourhood in childhood, whereas the lowest proportion (19%) resided in the west lower neighbourhood (Table 4.1). In terms of the current residential location of the cohort, 64% of respondents are current residents of the City of Hamilton and 54% have never resided outside Hamilton. Furthermore, 8% continue to reside at their childhood address. This high proportion of respondents who reside in Hamilton is not surprising

Table 4.1 Sample Characteristics (n = 395) – Residential profile					
Variable (%)	WU†	EU†	WL†	EL†	Total
Respondents living in these neighbourhoods in childhood	29	27	19	25	100
Current exposure to smoking at home / work	40	45	36	52	44
Exposure to smoking in childhood**	58	52	39	66	55
Gas cooking in childhood	15	24	23	27	22
Currently reside in Hamilton	62	71	52	66	64
Always resided in Hamilton*	61	66	50	58	60
Lived in 1 residence since 1986	7	11	7	8	9
Lived in 2 residences since 1986	27	26	18	30	26
Lived in 3 or more residences since 1986	66	63	75	62	65
Exposed to gas/dust /contaminants since 1986	92	91	95	92	92
Residential exposure > 2 years	18	31	27	19	23
Residential exposure > 5 years	85	75	81	86	82
Residential exposure > 8 years	10	20	17	11	15
Ever resided in property built prior 1950	29	21	71	42	38
Resided in property built prior to 1950 (> 5 yrs)***	17	15	52	24	25
Own air conditioner (A/C)	87	87	80	90	87
Frequency of use of A/C (always / almost always)	76	76	68	81	76

Table 4.1 continued

Variable (%)	WU†	EU†	WL†	EL†	Total
Own an air humidifier (A/H)	46	43	49	39	44
Frequency of use of A/H (always / almost always)	31	31	27	27	29
Own an air purifier (A/P)	37	35	38	28	34
Frequency of use of A/P (always / almost always)	31	27	27	23	27
Type of heating (gas / oil)	97	96	96	97	96
Air duct cleaning (rarely / do not remember)	67	67	78	66	68
Current gas cooking	41	31	41	40	38
No exposures reported	9	10	5	9	8
Exposures for more than 2 years	61	67	63	68	65
Exposures for more than 5 years	51	58	57	61	57
Exposures for more than 8 years	42	45	45	54	46

†WU – West upper neighbourhood; EU – East upper neighbourhood; WL – West lower neighbourhood; EL – East lower neighbourhood.
*$p < 0.05$; ** $p < 0.01$; ***$p < 0.001$

given that contacting participants who were traced using phone numbers and / or addresses in Hamilton in childhood led to a relatively low loss to follow-up (ranging between 9 to 14%). Table 4.1 presents a summary of data on residential history, including the number of previous residences, and residential exposures and practices. Data on exposure to smoking and gas cooking in childhood were collected from the surveys that

were administered on the original cohort between 1976 and 1986. Data from adulthood were collected from the survey that was administered on the reconstructed cohort between August 2005 and February 2007. Statistically significant differences across neighbourhoods were found for exposure to smoking in childhood, always residing in Hamilton, and residing in a property built before 1950 for more than five years (Table 4.1). 50% of respondents who lived in the west lower neighbourhood in childhood always resided in Hamilton compared to 66% of residents who lived in the east upper neighbourhood in childhood. In addition, respondents who resided in the east lower neighbourhood in childhood had the highest proportion of exposure to smoking in childhood (66%). 52% of respondents who resided in the west lower neighbourhood in childhood have resided in a property built before 1950 compared to 15% who resided in the east upper neighbourhood.

The mean age of the sample is 36 years (Table 4.2). Overall, 52% of the sample was male, with some variation across neighbourhoods. 16% were below the low-income cutoff in childhood, 98% completed high school, 85% currently own their home, 5% are currently below the low-income cutoff, and 72% are employed full-time. Although there is variation in the socio-demographic characteristics of the sample across neighbourhoods, differences are not statistically significant. However, in comparison to the general population, there are notable differences in the socioeconomic characteristics of this sample. Mainly, the population of Hamilton appears to be of relatively lower socioeconomic status, with 79% completing high school, 68% owning their homes, and 20% below the low-income cut-off (Statistics Canada, Census Data, 2001). As the

Table 4.2 Sample Characteristics (n = 395) – Socio-demographic Profile						
Variable	West Upper	East Upper	West Lower	East Lower	Total	Hamilton population
Mean age	36	36	36	36	36	39.6†
Sex (% male)	53	43	55	57	52	49
Household income in childhood (% below low-income cut-off)^	18	14	12	18	16	N/A
Completed High school (%)	98	100	99	96	98	79
Housing tenure (% homeownership)	87	84	82	84	85	68
Current household income (% below low-income cut-off)	4	8	3	6	5	20
Employed full-time (%)	79	66	68	74	72	60††

†Median age
††Employment rate
Data sources: Hamilton data from Statistics Canada, 2006 Census of Population; data on income in childhood from 1976 - 1986 survey; all other data from the 2005 – 2007 survey.

literature suggests, this difference may be attributed to loss to follow-up of participants of lower socioeconomic status, particularly those who are unemployed or less educated and who experience poor health (Lorant et al. 2006).

4.2.2 MACRO-ENVIRONMENTAL PROFILE

In relation to occupation types, 48% of respondents are professionals and 23% are manual labourers (Table 4.3). Overall, 84% reported occupational exposure to dust, 25% reported occupational exposure to contaminants, 54% reported occupational exposure to gas, and 9% did not report any occupational exposures. Although there is variation in the occupational exposure profile of the sample across neighbourhoods, differences are not statistically significant. Table 4.3 summarizes data related to occupational history, as well as the social and family environments, life events, healthcare access, and demographic factors. 74% of respondents are married, 69% have at least one parental record of asthma or respiratory problems, 10% experienced two or more stressful life events, and 92% have a regular family doctor. For each neighbourhood, at least 7% scored five or less on the social contact subscale that was collected using the Social Health Battery (SHB) (Donald and Ware 1984) (Appendix 3.10), indicating low social resources. Overall, 69% scored less than 4 on the group participation subscale. While there is variation across neighbourhoods in the macro-environmental profile of respondents, differences are only significant for the social contact subscale. 18% of respondents who resided in the west lower neighbourhood in childhood scored 5 or less on the social contact subscale compared to 7% who resided in the east lower neighbourhood in childhood.

4.2.3 MICRO-ENVIRONMENTAL PROFILE

Overall, 16% of participants smoked in childhood and 49% perform regular physical exercises (Table 4.4). For each neighbourhood, at least 13% are current smokers.

Table 4.3 Sample Characteristics (n = 395) – Occupational history, social and family environments, life events, healthcare access, and demographic factors.

Variables related to the:	Variable (%)		WU†	EU†	WL†	EL†	Total
Occupational History	Type of Employment	Clerical	7	11	12	11	11
		Manual	21	24	15	31	23
		Managerial	14	18	11	12	14
		Professional	54	39	58	42	48
		Other	4	6	5	4	5
	Exposure to dust		85	76	88	84	83
	Exposure to contaminant (s)		23	21	28	25	24
	Exposure to gas		52	57	53	54	54
	Exposure to dust, contaminants, or gas		18	15	20	18	18
	No exposures reported		9	10	5	9	8
	Exposures for more than 2 years		61	67	63	68	65
	Exposures for more than 5 years		51	58	57	61	57
	Exposures for more than 8 years		42	45	45	54	46
Social environment	Social contact subscale – score of 5 or less*		8	12	18	7	12
	Group participation subscale – score of 4 or less		70	67	63	75	69

Table 4.3 continued

Variables related to the:	Variable (%)	WU†	EU†	WL†	EL†	Total
Family Environment	Married / common-law	72	71	79	78	74
	Parental record of asthma / respiratory problems	69	71	73	61	69
Life events	Experienced 2 or more stressful life events	11	9	9	9	10
Healthcare access	Have a regular family doctor	90	94	88	94	92
	Availability of additional health insurance coverage	71	65	72	71	70
	Regular medical check-ups without a specific problem	57	63	60	58	59
Demographic factors	Born in Canada	97	93	95	95	95

†WU – West upper neighbourhood; EU – East upper neighbourhood; WL – West lower neighbourhood; EL – East lower neighbourhood. All data from 2005 – 2007 survey.
*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

Table 4.4 Sample Characteristics (n = 395) – Behavioural, physiological, and psychological profiles.

Variables related to:	Variable (%)	WU†	EU†	WL†	EL†	Total
Behavioural	Smoked in childhood (at least 1 per day for more than 6 months) ††	17	12	13	18	16
	Current smoker*	22	16	13	29	21
	Ever smoked on daily basis	37	28	31	41	34
	Regular alcohol consumption	11	8	11	14	11
	Physical activity (3 or more / week for > 30 minutes	52	42	51	50	49
Psychological	Concern over air pollution** (moderate or extreme concern)	42	65	41	64	54
	Emotional distress – score of 4 or more on the GHQ scale	8	11	17	11	11
	Feelings about health (score ≤ 3)	6	5	8	9	7
	Feelings about income (score ≤ 3)	7	18	13	14	13
	Feelings about life in general (score ≤ 3)	4	2	5	1	3
Physiological	BMI ≥ 25*	56	69	68	78	67
	Asthma in childhood††	14	12	7	10	11
	Persistent morning cough in childhood††	7	12	4	9	8

Table 4.4 continued						
Variables related to:	Variable (%)	WU†	EU†	WL†	EL†	Total
Physiological	Persistent day / night cough in childhood††	11	20	5	11	12
	Persistent wheeze in childhood††	16	19	12	9	15
	Chest illness / colds before 2 year (bronchitis or pneumonia) ††	4	5	7	7	6
	Other severe chest illness in childhood††	13	11	12	16	13
	Index for pulmonary function (always above the median) ††	29	27	31	26	28

†WU – West upper neighbourhood; EU – East upper neighbourhood; WL – West lower neighbourhood; EL – East lower neighbourhood.
*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.
Data sources: †† Survey administered between 1976 – 1986; all other data from 2005 – 2007 survey.

In relation to psychological factors, overall 11% scored 4 or more on the General Health Questionnaire scale, 7% felt mostly dissatisfied, unhappy or terrible about their health, 13% felt mostly dissatisfied, unhappy or terrible about their income, and 3% felt mostly dissatisfied, unhappy or terrible about life in general. For each neighbourhood at least 41% are concerned about air pollution. In terms of physiological factors and respiratory symptoms in childhood, overall 11% were diagnosed with asthma in childhood, 8% had persistent morning cough, 12% had persistent day or night cough, 15% had persistent

wheeze, 6% had chest illnesses or colds before the age of two, 13% had other severe chest illnesses, and 28% had pulmonary function measures below the median level for all sampling periods. For each neighbourhood, at least 56% have a body mass index greater or equal to 25, representing the cut-off point for classifying individuals as being overweight (World Health Organization, 2005). Statistically significant differences were found across neighbourhoods for current smoking, concern over air pollution, and a body mass index equal to or greater than 25. Respondents who resided in the east lower neighbourhood in childhood had the highest proportion of current smoking (29%) and a body mass index equal to or greater than 25 (78%). Respondents who resided in the east upper neighbourhood in childhood were most concerned over air pollution (65%) compared to respondents who resided in the west lower neighbourhood in childhood who were least concerned over air pollution (41%).

4.2.4 EXPOSURE PROFILE

As indicated in Table 4.5, exposure assessment data are based on three sampling periods for TSP and four sampling periods for each of $PM_{3.3}$ and SO_2. Air pollution levels ranged from 31 to 75 $\mu g/m^3$ for TSP, from 34 to 68 $\mu g/m^3$ for $PM_{3.3}$, and from 5 to 17 ppb for SO_2 in the study area. Under Canada's Clean Air Act of 1973, national Ambient Air Quality Objectives (NAAQOs) were established in 1976. The annual maximum desirable level for TSP and SO_2 are set at 60 ($\mu g/m^3$) and 11 ppb respectively (Health Canada, 1998). Although responsibility for developing and reviewing the NAAQOs shifted to the CEPA (Canadian Environmental Protection Act) Federal / Provincial

Table 4.5 Exposure assessment for the sample in relation to ambient air pollutants.

VARIABLES	Minimum	Maximum	Median	% above median					% always above median
				WU	EU	WL	EL	T	
TSP ($\mu g/m^3$) (1980 / 1981)*	30.5	74.5	51.4	53	19	78	61	51	17
TSP ($\mu g/m^3$) (1983 / 1984)*	33.8	69.3	49.5	22	38	80	65	47	
TSP ($\mu g/m^3$) (1985 / 1986)	40	67.3	45.7	23	55	100	69	47	
$PM_{3.3}$ ($\mu g/m^3$) (1980 / 1981)	34.3	57.4	45.5	69	11	100	31	50	24
$PM_{3.3}$ ($\mu g/m^3$) (1981 / 1982)	39.3	62.8	45.5	29	28	100	48	46	
$PM_{3.3}$ ($\mu g/m^3$) (1983 / 1984)*	44.8	68.9	52.2	50	15	54	80	48	
$PM_{3.3}$ ($\mu g/m^3$) (1985 / 1986)	38.3	62.6	43.2	30	24	100	72	50	
SO_2 (ppb) (1978 / 1979)*	5.4	11.1	6.75	39	25	46	75	44	10
SO_2 (ppb) (1980 / 1981)*	8.5	17.3	10.6	52	19	81	49	48	
SO_2 (ppb) (1981 / 1982)	8.4	13.1	9.7	0.9	64	13	81	41	
SO_2 (ppb) (1983 / 1984)*	7.8	12.7	11.7	30	44	70	58	47	

WU –west upper, EU – east upper, WL – west lower, EL – east lower, T – total
*$p < 0.001$

Working Group on Air Quality Objectives and Guidelines (WGAQOG), the numerical values of the objectives have not been revised since 1976 (Greater Vancouver Regional District, 2005). However, in 2001, fine particulate matter ($PM_{2.5}$) was identified as a priority substance for the development of Canada Wide Standards and the 24 hour standard was set at 30 $\mu g/m^3$, to be attained by 2010 (Canada Wide Standards 2001). Table 4.5 shows that for each sampling period, while the median levels of TSP are below the NAAQ objectives, the maximum levels of TSP exceed that limit. For $PM_{3.3}$, for all sampling periods, the minimum levels exceed 30$\mu g/m^3$. For SO_2, the median level for the sampling period 1983 / 84 and the maximum levels of SO_2 for all sampling periods exceed the NAAQ objectives. Overall, the percentage of participants who were always exposed above the median level to TSP, $PM_{3.3}$, and SO_2 for all sampling periods is 17%, 24%, and 10% respectively. As expected, there were differences in childhood exposure to air pollution across the four geographical neighbourhoods. Using the chi-square test, statistically significant associations were found between neighbourhood of residence in childhood and exposure to TSP, $PM_{3.3}$, and SO_2.

4.2.5 HEALTH PROFILE

In comparison to the Hamilton population, a relatively high proportion of the sample (15%) reported fair or poor health (Table 4.6). In comparison, 14% of the City of Hamilton population, 11% of the Province of Ontario, and 11% of the Canadian population reported fair or poor health (Statistics Canada, 2006). In examining the respiratory health of the cohort, 20% have been diagnosed with asthma and 11% were

Table 4.6 Current health status (n = 395).

Health Outcome (%)		WU	EU	WL	EL	Total	Always resided in Hamilton	Resides at original address in childhood	Hamilton population	Canadian population
Self-reported health (fair / poor)		15	14	11	19	15	16	12	14†	11†
Asthma in adulthood		23	20	15	20	20	18	18	8†	8†
Asthma in childhood		14	12	7	10	11	10	12	12†	12†
Asthma in adulthood not in childhood		16	10	11	15	13	14	12	-------	-------
Self-reported persistent symptoms	Morning cough	8	9	7	12	9	10	9	-------	-------
	Day / night cough	5	10	8	12	9	9	6	-------	-------
	Phlegm	4	7	7	11	7	6	9	-------	-------
	Wheeze	16	16	13	12	15	15	9	-------	-------
Hospital / ER visits for asthma or respiratory problems since leaving elementary school*		2	11	9	15	9	6	9	-------	-------
High blood pressure		7	11	13	9	10	9	3	-------	7††
Cancer		0	1	1	3	1	1	3	-------	2††
Arthritis		11	9	9	12	10	10	3	-------	9†
Heart disease		0	2	3	0	1	1	0	-------	3††
Chronic bronchitis		3	11	7	10	7	7	9	-------	4††
Hay fever / allergies		43	48	40	37	42	36	24	-------	18††

* $p < 0.05$; WU – west upper; EU – east upper; WL – west lower; EL – east lower. Data sources: † Statistics Canada (2006); †† National Population Health Survey (2004 - 5). All other data from 2005 – 2007 survey.

diagnosed with asthma in childhood. Data based on the City of Hamilton Health Unit indicate a much lower prevalence rate of asthma for the Hamilton population (Hamilton District Health Council 2003) (8%) and a higher prevalence rate of asthma in childhood (12%) (Canadian Community Health Survey 2005). Furthermore, 13% of the overall sample was diagnosed with asthma in adulthood but not in childhood. The highest incidence of asthma in adulthood occurs among participants who resided in the WU neighbourhood (16%) followed by the EL neighbourhood (15%). In relation to respiratory health symptoms, participants who resided in the EL neighbourhood in childhood had the highest prevalence of persistent morning cough (12%), persistent day or night cough (12%), persistent phlegm (11%), and having hospital or emergency room visits for asthma or respiratory problems since leaving elementary school (15%). Participants who resided in the east and west upper neighbourhoods had the highest prevalence of persistent wheeze. While there were no statistically significant differences across the four neighbourhoods for the prevalence of respiratory health outcomes, the relationship between neighbourhood of residence in childhood and having hospital or ER visits for asthma or respiratory health problems since leaving elementary school was statistically significant. 15% of participants who resided in the east lower neighbourhood had hospital or emergency room visits for asthma or respiratory problems compared to 7% of participants who resided in the other three neighbourhoods combined.

The rates of non-respiratory conditions were in most cases higher than those reported in the Canadian population. Of importance is the relatively high prevalence of hay fever or allergies in the sample (42%), particularly since a statistically significant

association was found between diagnosis with hay fever / allergies and ever diagnosed with asthma in the sample ($\chi 2 = 40.72$, $p = 0.000$). 74% of participants ever diagnosed with asthma were also diagnosed with hay fever / allergies. However, while there was variation across neighbourhoods in the prevalence of non-respiratory health outcomes, differences were not statistically significant.

Results indicate that the prevalence rates of respiratory and non-respiratory health outcomes in the sub-sample of respondents who always resided in Hamilton (n = 214) are similar to that of the entire sample except for diagnosis with hay fever or allergies (Table 4.6). The prevalence of hay fever or allergies in the sub-sample of respondents that always resided in Hamilton is 36% compared to 42% in the entire sample. In addition, prevalence rates of respiratory health outcomes in the sub-sample of respondents that continue to reside in their childhood address (n = 33) are similar to those of the entire sample except for the prevalence of persistent wheeze (9% in the sub-sample compared to 15% in the entire sample). However, due to sample size, the prevalence rates of chronic health outcomes in the sub-sample of respondents that continue to reside in their childhood address cannot be compared against those of the entire sample.

4.2.6 SUMMARY

Although individuals of low socioeconomic status who experience poor health may have been lost to follow-up (as suggested by the literature), the prevalence rates of respiratory and non-respiratory health outcomes are in most cases higher than those of the

Hamilton and Canadian populations. The proportions of the sample who completed high school, who own their property, who fall below the low-income cut-off, and who are employed full-time are higher than those of the Hamilton population (98% versus 79%; 85% versus 68%; 5% versus 16%; 72% versus 60% respectively) indicating that the socioeconomic profile of the sample is relatively high in comparison to that of the general population. Relative to the Canadian population, higher prevalence rates were found in the sample for asthma in adulthood (20% versus 8%), high blood pressure (10% versus 7%), arthritis (10% versus 9%), chronic bronchitis (7% versus 4%), and hay fever or allergies (42% versus 18%). The relatively high prevalence rates of asthma incidence in adulthood and hay fever or allergies are particularly notable. This may reflect the notion that original cohort members who were diagnosed with asthma were likely to be interested in participating in this study, particularly since original cohort members who were contacted were made aware of the objectives of this research. The high prevalence of asthma is beneficial for this research since analysis will be based on a relatively good representation of asthma cases (n = 80) in the sample.

In examining variations in macro- and micro-environmental factors across neighbourhoods, findings suggest that residing in the east lower neighbourhood in childhood is related to exposure to smoking in childhood (66%), current smoking (29%), scoring 5 or less on the social contact subscale (7%), and having a body mass index equal or greater than 25 (78%). Although there are significant differences in air pollution levels across the four neighbourhoods, results indicate that the long-term health of the sample is relatively consistent across neighbourhoods. Only one statistically significant association

was found between neighbourhood of residence in childhood and having hospital or ER visits for asthma or respiratory problems since leaving elementary school.

Therefore, overall results suggest that neighbourhood of residence in childhood is related to opportunities or challenges that are different across neighbourhoods and that may impact long-term health. Opportunities or challenges include exposure to air pollution, as well as environments that promotes certain behaviours (such as smoking in childhood and current smoking), social networking, concern over air pollution, and physiological effects related to body mass index. However, in focusing on the relationship between childhood exposure to air pollution and long-term health, results of analysis at the aggregate neighbourhood level indicate that despite significant differences in childhood exposure to air pollution across neighbourhoods, the long-term health of participants is relatively consistent across neighbourhoods. This may indicate that childhood exposure effects of air pollution may dissipate over time. This suggested relationship is similar to relationships observed between smoking cessation and each of decreased mortality (Anthonisen et al. 2005; Lestra et al. 2005; Peters et al. 1995) and improved lung function (Scanlon et al. 2000).

In order to examine the relationship between childhood exposure to air pollution and long-term health at the individual level, the next section examines associations between exposure measures and health outcomes. Chapter 5 will examine the effect of factors from the macro- and micro-environments (that may result from inequitable opportunities or exposures) in mediating the relationship between childhood exposure to air pollution and long-term health.

4.3 CHILDHOOD EXPOSURE TO AIR POLLUTION AND LONG-TERM HEALTH

The second objective of this research is to examine the link between childhood exposure to air pollution and adult respiratory health outcomes. Section 4.3.1 examines the relationships between childhood exposure to air pollution and respiratory health outcomes in adulthood. This is followed by an examination of the links between childhood exposure to air pollution and non-respiratory health outcomes in adulthood in section 4.3.2. Section 4.3.3 presents and discusses statistically significant associations between variables from the macro-environment and micro-environment constructs of the model that guides this research. Significant associations between health and the macro and micro environments are summarized in section 4.3.4. A discussion and a chapter summary follow in sections 4.4 and 4.5 respectively.

4.3.1 RESPIRATORY HEALTH IMPACTS

As discussed in Section 3.4, the relationship between childhood exposure to air pollution and adult respiratory health is assessed using bi-variate analysis between exposure to air pollution variables and respiratory health outcomes outlined in Appendices 3.10 and 3.12 respectively. Air pollution assessment data are based on five sampling periods when the mean age of participants was 8 (1978 / 79), 9 (1980 / 1), 10 (1981 / 2), 13 (1983 / 4), and 15 (1985 / 6) years. Overall bi-variate analysis resulted in two statistically significant associations between childhood exposure to air pollution and long-term respiratory health (Table 4.7). Results suggest that childhood exposure to SO_2

Table 4.7 Relationship between exposure air pollution in childhood and long-term health.					
RESPIRATORY HEALTH OUTCOMES		n	%	n	%
		Exposure variable			
		Reside WL / EU / WU		Reside in EL	
Hospital or emergency room visits for asthma or respiratory since leaving elementary school*	M	10	7	6	11
	F*	11	14	9	21
	T*	21	7	15	15
		$SO_2 \leq 9.7$ ppb		$SO_2 > 9.7$ ppb	
Hospital or emergency room visits for asthma or respiratory since leaving elementary school*	M	7	6	9	12
	F	7	11	12	16
	T*	14	6	21	14
		$SO_2 \leq 11.7$ ppb		$SO_2 > 11.7$	
Asthma incidence in adulthood (but not in childhood)	M	7	7	4	6
	F*	9	12	23	27
	T*	16	9	27	17
Diagnosed with asthma or respiratory illness	M	22	22	11	15
	F*	14	18	30	35
	T	36	21	41	26

Table 4.7 continued

NON-RESPIRATORY HEALTH OUTCOMES		n	%	n	%
		Exposure variable			
		$SO_2 \leq 11.7$ ppb		$SO_2 > 11.7$	
High blood pressure or hypertension	M	7	7	9	13
	F*	5	6	14	16
	T*	12	7	23	15
Arthritis / rheumatism	M	4	4	7	10
	F	7	9	16	19
	T*	11	6	23	15

*$p < 0.05$.
All data from the 2005 – 2007 Survey.

above the median level of 9.7 ppb (measured in 1981 / 82 – mean age of sample is 10 years) is associated with hospital or emergency room visits for asthma or respiratory problems since leaving elementary school. 12% of the sample who were exposed to SO_2 above the median level of 9.7 ppb had hospital or emergency room visits for asthma or respiratory problems since leaving elementary school compared to 6% of participants who were exposed at or below the median level. Results also suggest that childhood exposure to SO_2 above the median level of 11.7 ppb (measured in 1983 / 84 – mean age

of sample is 13 years) is associated with asthma incidence in adulthood (but not in childhood). 17% of participants who were exposed to SO_2 above the median level of 11.7 ppb were diagnosed with asthma in adulthood (but not in childhood) compared to 9% of participants who were exposed at or below the median level. In addition, as discussed previously in section 4.2, a statistically significant difference across neighbourhoods was found for hospital or emergency room visits for asthma or respiratory illness. For all sampling periods, associations between exposure to TSP or to $PM_{3.3}$ and respiratory health outcomes were not statistically significant. In addition, associations between exposure to SO_2 in 1978 / 9 or 1980 / 1 and respiratory health outcomes were not statistically significant. For all sampling periods, overall analysis did not result in statistically significant associations between exposure variables to TSP, $PM_{3.3}$, or SO_2 and ever diagnosed with asthma or respiratory problems. In addition, no statistically significant associations were found between respiratory health outcomes and air pollution exposure measures in the sub-samples who always resided in Hamilton or who continue to reside in their address from childhood.

In examining relationships between childhood exposure to air pollution and long-term health, significant sex differences were found (Tables 4.7 and 4.8). As indicated, no statistically significant associations between childhood exposure to air pollution and respiratory health outcomes were found for males. For females, however, significant associations were found between childhood exposure to SO_2 above the median level of 11.7 ppb and each of asthma or respiratory illness, and asthma in adulthood but not in childhood. 35% of females who were exposed to SO_2 above the median level of 11.7 ppb

Table 4.8 Correlation coefficients between exposure to air pollution and long-term health outcomes.

Health Outcome		Exposure to:						
		SO_2 (1978/9)	SO_2 (1980/1)	SO_2 (1981/82)	SO_2 (1983/84)	$PM_{3.3}$ (1980/81)	$PM_{3.3}$ (1985/86)	TSP (1985/86)
Hospital or ER visits for asthma or respiratory problems	M F T			-0.506 3.102** 1.908	-0.329 2.661** 1.783			
Hay fever	M F T	-2.145 2.040* 0.344						
High blood pressure	M F T			0.975 2.010* 2.154**				
Arthritis	M F T	2.073* 0.167 1.385	1.162 1.687 2.059*				2.087* -0.530 0.786	
Chest conditions	M F T					-1.296 2.077* 1.294		
Long-term skin conditions	M F T							2.157* -0.159 1.398

* $p < 0.05$

** $p < 0.001$All data from the 2005 – 2007 survey.

were diagnosed with asthma or respiratory illness compared to 18% who were exposed at or below the median level (Table 4.7). Furthermore, 27% of females who were exposed to SO_2 above the median level of 11.7 ppb were diagnosed with asthma in adulthood but not in childhood compared to 12% who were exposed at or below the median level. In addition, analysis of variance led to significant associations between childhood exposure to SO_2 in 1981 / 82 and 1983 / 84 and hospital or emergency room visits for asthma or respiratory problems for females only (Table 4.9). No significant associations were found between childhood exposure to TSP and $PM_{3.3}$ and long-term respiratory health for females. In addition, no significant associations were found between childhood exposure to SO_2 in 1978 / 79 or 1980 / 81 and long-term respiratory health for females.

These relationships suggest that childhood exposure to air pollution may impact long-term respiratory health and that these health effects are sex-related. In addressing physiological plausibility, research that examined biological effects of childhood exposure to air pollution found that seemingly healthy children exposed to high levels of pollution were more likely to have abnormal nasal, radiologic (using frontal and lateral chest X-rays), spirometric, and peripheral blood findings than children who were exposed to lower levels of air pollution (Calderon-Garciduenas et al. 2003). In particular, the study found significant associations between air pollution and specific cytokines (TNFά and IL8), intertistial markings through chest x-ray, and pulmonary function measures. Clinical studies that focused on the biological mechanisms that relate to exposure to air pollution suggest that ultrafine, fine, and PM_{10} induce increased levels of C-reactive protein which may cause inflammatory and coagulation responses that affect airway

systems and increase the likelihood of serious arterial vascular thrombosis (Rucker et al. 2005). Experimental research suggests that exposure to elevated PM_{10} has the ability to alter epithelial lung cell functions to favour blood coagulation via activation of the extrinsic (resulting from damaged tissue) pathway and inhibition of fibronolysis (destruction of fibrin - a protein formed when blood clots) pathways (Gilmour et al. 2005). In addition, pro-inflammatory response and oxidant stress in alveolar macrophages (cells that are key players in the immune system) has been associated with exposure to coarse particulate matter (Becker et al. 2005). In addition, research suggests that females are more susceptible to health effects of exposure to air pollution than males due to their relatively smaller lungs and higher airflow rates, which in turn increase the dose of air pollutants (Boezen et al. 2004). Other research suggests that sex hormones play a role in female susceptibility in developing respiratory diseases (Carey et al. 2007).

In conclusion, results of this research suggest that for females, childhood exposure to SO_2 around the age of 10 and 13 years may affect long-term respiratory health. For males, childhood exposure to SO_2 is not related to long-term respiratory health. In addition, childhood exposure to TSP or $PM_{3.3}$ is not related to long-term health. However, in order to explore the relative role that childhood exposure to air pollution plays in long-term respiratory health and the effect of mediating factors on these relationship, variables from the conceptual framework as illustrated in Figure 2.2 are included in the multi-variate analysis presented in Chapter 5.

4.3.2 NON-RESPIRATORY HEALTH IMPACTS

In exploring the relationship between air pollution and non-respiratory health outcomes, significant associations were found between childhood exposure to SO_2 (above or below the median level of 11.7 ppb (when the mean age of the sample was 13 years) and high blood pressure or hypertension (Table 4.7). 15% of the sample who were exposed to SO_2 above the median level of 11.7 ppb were diagnosed with high blood pressure or hypertension compared to 7% of participants who were exposed at or below the median level. Childhood exposure to SO_2 (above or below the median level of 11.7 ppb) was also associated with arthritis or rheumatism (Table 4.7). Results indicate that 15% of the sample who were exposed to SO_2 above the median level of 11.7 ppb were diagnosed with arthritis or rheumatism in adulthood compared to 6% of participants who were exposed at or below the median level. In addition, analysis of variance led to statistically significant associations between exposure to SO_2 in 1980 / 81 (when the mean age of the sample was 9 years) and arthritis or rheumatism and between exposure to SO_2 in 1981/ 82 (when the mean age of the sample was 10 years) and high blood pressure or hypertension (Table 4.8). These results are important given research that links exposure to air pollution to pro-inflammatory response in alveolar macrophages (Becker et al. 2005) and the activation of pathways that inhibit the destruction of fibrin leading to blood clotting (Gilmour et al. 2005).

For all sampling periods, overall analysis did not result in significant associations between childhood exposures to TSP and $PM_{3.3}$ and non-respiratory health outcomes. In addition, for all sampling periods, associations between childhood exposure to SO_2 and

each of hay fever / allergies, chest conditions, and long-term skin conditions were not significant. Results also indicate that childhood exposure to SO_2 in 1978 / 9 (mean age = 8 years) or 1983 / 4 (mean age = 15) is not related to any non-respiratory health outcomes. In addition, while associations between non-respiratory health outcomes and air pollution exposure measures were not significant in the sub-samples who always resided in Hamilton or who continue to reside in their address from childhood, analysis was limited by sample size.

In exploring relationships between air pollution and non-respiratory health outcomes for females and males separately, statistically significant associations were found between exposure to SO_2 in 1978 / 79 (mean age = 8 years) and hay fever or allergies for females only (Table 4.8). Furthermore, the association between exposure to SO_2 above or below the median level of 11.7 ppb and diagnosis with high blood pressure or hypertension was only significant for females (Table 4.7). 16% of female participants exposed above the median level of 11.7 ppb (mean age = 13 years) were diagnosed with high blood pressure or hypertension compared to 6% of participants who were exposed at or below the median level (Table 4.7). Similarly, analysis of variance led to a statistically significant association between exposure to SO_2 in 1981 / 82 (mean age = 10 years) and high blood pressure / hypertension for females only. For all sampling periods, associations between childhood exposure to SO_2 and each of arthritis / rheumatism, chest conditions, and long-term skin conditions were not statistically significant for females. In relation to childhood exposure to $PM_{3.3}$, a statistically significant association was found between exposure in 1980 / 81 (mean age = 9) and chest conditions for females.

However, for all other sampling periods, associations between exposure to $PM_{3.3}$ and chest conditions was not significant. In addition, no significant associations were found between childhood exposure to TSP and non-respiratory health outcomes for females.

For males, statistically significant associations were found between exposure to SO_2 in 1978 / 79 (mean age = 8 years) and arthritis or rheumatism and between exposure to $PM_{3.3}$ in 1985 / 86 (mean age = 15 years) and arthritis or rheumatism (Table 4.8). For all other sampling periods, childhood exposure to SO_2 or $PM_{3.3}$ was not significantly associated with arthritis or rheumatism. In addition, analysis of variance indicates that exposure to TSP in 1985 / 86 (mean age = 15 years) was associated with long-term skin conditions for males. However, for all sampling periods, childhood exposure to TSP, $PM_{3.3}$, or SO_2 was not significantly associated with hay fever / allergies, high blood pressure / hypertension, and chest conditions for males. In addition, for all sampling periods, childhood exposure to SO_2 or $PM_{3.3}$ is not significantly associated with long-term skin conditions.

These relationships suggest that childhood exposure to air pollution may be related to long-term non-respiratory health. Results also suggest that non-respiratory health impacts of childhood exposure to air pollution may be sex-related. In particular, for females, results suggest that the timing and level of exposure to SO_2 may impact long-term non-respiratory health, including high blood pressure / hypertension, hay fever / allergies, and chest conditions. For males, results suggest that the timings and level of exposures to SO_2 and $PM_{3.3}$ may impact arthritis or rheumatism. In addition, results suggest that childhood exposure to TSP in 1985 / 86 (mean age = 15) may be related to

long-term skin conditions. Guided by the LCHD framework, multi-variate analysis will examine the relative role that childhood exposure to air pollution plays in the long-term development of non-respiratory health outcomes and the effect of mediating factors on the relationship between childhood exposure to air pollution and long-term health.

4.3.3 ASSOCIATIONS BETWEEN VARIABLES FROM THE MACRO AND MICRO ENVIRONMENTS

In order to explore potential factors that impact long-term health, bi-variate associations between variables from the macro-environment (residential and occupational histories, social and family environments, life events, healthcare access, and demographic factors), and micro-environment (physiological, psychological, and behavioural) constructs of the model were examined. As discussed in section 4.1, statistically significant associations resulted from bi-variate analysis between variables from the: i) macro-environment (macro – macro); ii) micro-environment (micro – micro); iii) macro- and micro- environments (macro - micro); iv) macro-environment and health outcomes (macro – health outcomes); v) and micro-environment and health outcomes (micro – health outcomes).

4.3.3.1 SIGNIFICANT ASSOCIATIONS BETWEEN VARIABLES FROM THE MACRO-ENVIRONMENT

From the macro-environment construct of the model, statistically significant associations were found between healthcare access and each of residential history and

demographic factors. Statistically significant associations were found between each of sex, property ownership, current location of residence and access to healthcare (Table 4.9). A higher proportion of respondents who are female (97% versus 87% males), who own their property (95% versus 78% that do not own their property), or who reside in the City of Hamilton (96% versus 88% that reside in Ontario, and 67% that reside outside Ontario) have a regular family doctor. Associations between variables related to residential or occupational histories or exposures and each of variables from the social or family environments, life events, and demographic factors were not significant. In addition, variables related to the social / family environments (social networking, marital status, number of children, parental health) were not significantly associated with experiencing 2 or more stressful life events, age, sex, current income, or education. Furthermore, experiencing 2 or more stressful life events was not significantly associated with age, sex, current income, or education.

4.3.3.2 SIGNIFICANT ASSOCIATIONS BETWEEN VARIABLES FROM THE MICRO-ENVIRONMENT

Significant associations were found between variables from the micro-environment construct of the conceptual model. Persistent wheeze in childhood was significantly associated with smoking in childhood and psychosocial factors (Table 4.10). In comparison to participants who did not report persistent wheeze in childhood, a higher proportion of participants who reported persistent wheeze in childhood smoked in childhood (26% versus 14%), scored 4 or more on the General Health Questionnaire

Table 4.9 Associations between variables from the macro-environment construct of the model			
		Regular family doctor	
		n	%
Sex*	Female	185	97
	Male	178	87
Property ownership*	Own	305	95
	Rent	46	78
Residential location*	Reside in Hamilton	243	96
	Reside in Ontario	97	88
	Reside outside Ontario	20	67

*p < 0.001
All data from the 2005 – 2007 survey

Table 4.10 Associations between variables from the micro-environment construct of the model				
	Persistent wheeze		No persistent wheeze	
	n	%	n	%
Smoking in childhood*	15	26	45	14
Score 4 or more on the GHQ*	11	19	34	10
Feel mostly dissatisfied / unhappy / terrible about life **	6	10	6	2

*p < 0.05; **p < 0.001
All data from the 2005 – 2007 survey.

scale (19% versus 10%), and felt mostly dissatisfied / unhappy / terrible about life (10% versus 2%). Analysis of variance suggests that psychosocial factors are also associated with persistent morning cough ($t = 1.158$, $p = 0.017$) and persistent wheeze ($t = 1.805$, $p = 0.004$) in childhood. Smoking in childhood was positively associated with each of persistent morning cough ($t = 1.915$, $p = 0.000$), persistent day or night cough ($t = 2.826$, $p = 0.000$), and persistent wheeze ($t = 3.058$, $p = 0.000$) in childhood. In addition, each of persistent morning cough, persistent day or night cough, and persistent wheeze in childhood were negatively associated with current physical activity ($t = 2.355$, $p = 0.000$; $t = 2.333$, $p = 0.000$; and $t = 1.568$, $p = 0.004$ respectively). In examining relationships between variables from the micro-environment, no significant associations were found between lung function in childhood and behavioural or psychological factors; in addition, no significant associations were found between behavioural factors (smoking in childhood, alcohol consumption, and physical activity) and psychological factors (concern over air pollution, GHQ, and feelings about health / income / life).

4.3.3.3 SIGNIFICANT ASSOCIATIONS BETWEEN VARIABLES FROM THE MACRO- AND MICRO-ENVIRONMENTS

Significant associations were also found between variables from the macro-environment and micro-environment constructs of the model. However, all of the associations seem to relate to current status irrespective of childhood exposures or experiences. For instance, experiencing two or more stressful life events was significantly associated with scoring four or more on the GHQ scale (Table 4.11). In comparison to

Table 4.11 Associations between variables from the macro-environment and micro-environment constructs of the model

		Score 4 or more on the GHQ	
		n	%
Life events**	Experience two or more stressful life events	12	32
	Experienced less than 2 stressful life events	33	9
Property Ownership**	Own	27	8
	Rent	15	25
Occupational exposure*	Exposure > 8 years	30	17
	Exposure ≤ 8 years	15	7
		Feel mostly dissatisfied / unhappy / terrible about life	
Life events**	Experience two or more stressful life events	6	16
	Experienced less than 2 stressful life events	6	2

*p < 0.01
**p < 0.001
All data from 2005 – 2007 survey.

participants who experienced less than two stressful life events, participants who experienced two or more stressful life events had higher prevalence for scoring four or more on the GHQ scale (32% versus 9%). In addition, property ownership and occupational exposures to contaminants / dust / fumes greater than 8 years in duration were significantly associated with scoring four or more on the GHQ scale (Table 4.11). 25% of participants who do not own their property scored 4 or more on the GHQ scale compared to 8% of participants who own their property. 17% of participants who were exposed to occupational contaminants for more than eight years scored 4 or more on the GHQ scale compared to 7% who were exposed to occupational contaminants for eight years or less. Furthermore, experiencing two or more stressful events was associated with scoring 3 or less on the likert scale for feelings about life. 16% of participants who experienced two or more stressful life events scored 3 or less on the likert scale for feelings about their lives compared to 12% of participants who experienced less than two stressful life events.

Association between variables from the micro-environment construct of the model were not significantly associated with variables related to childhood exposure to air pollution, residential history and exposures, the social and family environments, healthcare access, and demographic factors. In addition, occupational history and exposures less than 8 years in duration, as well as experiencing two or more stressful life events were not associated with physiological and behavioural factors.

4.3.3.4 SIGNIFICANT ASSOCIATIONS BETWEEN VARIABLES FROM THE MACRO-ENVIRONMENT AND HEALTH

In examining relationships between variables from the macro-environment and health outcomes, significant associations were found between respiratory health outcomes and current income, stressful life events, and occupational exposures to gas / dust / fumes for more than eight years. Current income was significantly associated with each of asthma or respiratory problems and hospital or ER visits for asthma or respiratory health since leaving elementary school. (Table 4.12). Participants whose current family income is below the low income cutoff had higher prevalence of asthma or respiratory problems (44% versus 24%) and hospital or ER visits for asthma or respiratory problems (25% versus 8%) than participants whose current family income is above the low-income cutoff. In addition, statistically significant associations were found between stressful life events and respiratory health outcomes (Table 4.12). Experiencing two or more stressful life events was associated with hospital visits for asthma or respiratory problems since leaving elementary school and chronic bronchitis. Participants who experienced two or more stressful life events had higher prevalence rates of hospital or emergency room visits for asthma or respiratory problems since leaving elementary school (32% versus 7%), and chronic bronchitis (16% versus 6%). Furthermore, a statistically significant association was found between occupational exposures to contaminants / dust / fumes for more than 8 years and ever being diagnosed with asthma or respiratory conditions. 28% of participants exposed to occupational dust, gas, or fumes for more than eight years were diagnosed with asthma or respiratory problems compared to 19% of participants who

Table 4.12 Associations between variables from the macro-environment and respiratory health outcomes.

Variables from macro-environment:		Respiratory health					
		Ever asthma / respiratory problems		Hospital or ER visits since elementary		Chronic bronchitis	
		n	%	n	%	n	%
Current income	Below LICO	9**	44	6*	25	2	8
	Above LICO	81	24	30	8	27	7
Stressful life events	2 or more	13	34	12***	32	6*	16
	< than 2	77	22	24	7	23	6
Occupational exposure to gas / dust / fumes (years)	More than 8	51*	28	20	11	17	9
	Less or equal to 8	39	19	16	8	12	6

LICO – Low income cut-off
*p < 0.05; ** p < 0.01; ***p < 0.001
All data from 2005 – 2007 survey.

were exposed to occupational contaminants for eight years or less. In examining relationships between variables from the macro-environment and health outcomes, associations between long-term respiratory health and variables related to residential history, the social and family environments, healthcare access, property ownership, education, employment type, and occupational exposure to dust / gas / fumes for 8 years or less were not found significant.

Significant associations were also found between variables from the macro-environment and non-respiratory health outcomes. Current income was significantly associated with high blood pressure, arthritis / rheumatism, long-term skin conditions, fair or poor self-assessed health, and fair or poor self-rated ability to handle day-to-day demands and difficult situations (Table 4.13). Property ownership was significantly associated with high blood pressure, self-assessed heath, and functional capacity (Table 4.13). In addition to significant associations between socioeconomic status (measured by current income and property ownership) and non-respiratory health, statistically significant associations were found between stressful life events and non-respiratory health outcomes (Table 4.13). Experiencing two or more stressful life events was significantly associated with arthritis / rheumatism, self-assessed health, and functional capacity. Statistically significant associations were also found between current location of residence and non-respiratory health outcomes, mainly diagnosis with high blood pressure / hypertension and hay fever or allergies (Table 4.13). In terms of residential variables, significant associations were found between self-reported residential exposure to dust / gas / contaminants and non-respiratory health outcomes. Significant associations

Table 4.13 Associations between variables from the macro-environment and non-respiratory health outcomes.

Variables from the macro-environment:		Non-respiratory health													
		High blood pressure		Arthritis / rheumatism		Hay fever / allergies		Skin conditions		Fair / poor self-assessed		Fair / poor functional capacity (day-to-day)		Fair / poor functional capacity (problems)	
		n	%	n	%	n	%	n	%	n	%	n	%	n	%
Current income	Below LICO	7**	33	6*	25	10	42	8*	33	10***	56	6**	25	8**	33
	Above LICO	32	9	35	9	157	42	57	15	50	12	39	11	58	16
Property Ownership	Own	25**	8	33	10	135	42	52	16	41**	13	31*	10	49*	15
	Rent	12	20	6	10	28	48	12	20	16	27	12	20	16	27
Stressful life events	2 or more	7	18	10*	26	14	37	10	26	14***	37	10**	26	12**	32
	< than 2	32	9	31	9	153	43	55	15	46	13	35	10	54	15
Current location of residence	Hamilton	24*	10	25	10	91**	36	42	17	42	17	25	10	40	16
	Outside Ontario	7	23	4	13	16	52	4	13	3	10	1	3	5	16

Table 4.13 continued

Variables from the macro-environment:		Non-respiratory health outcomes													
		High blood pressure		Arthritis / rheumatism		Hay fever / allergies		Skin conditions		Fair / poor self-assessed		Fair / poor functional capacity (day-to-day)		Fair / poor functional capacity (problems)	
		n	%	n	%	n	%	n		n	%	n	%	n	%
Residential exposures to gas / dust / contaminants (years)	> 2	12	13	19***	21	42	46	18	20	16	17	15	16	21	23
	≤ 2	27	9	22	7	125	41	47	16	44	15	30	10	45	15
	> 5	10	14	17***	24	34	48	14	20	12	17	9	13	14	20
	≤ 5	29	9	24	7	133	41	51	16	48	15	36	11	52	16
	> 8	9	16	14***	25	28	49	11	19	8	14	6	11	10	18
	≤ 8	30	9	27	8	139	41	54	16	52	15	39	12	56	17
Occupational exposure to gas / dust / fumes (years)	> 2	31*	12	33*	13	115	45	49	19	42	17	32	13	43	17
	≤ 2	8	6	8	6	52	37	16*	12	17	12	13	9	23	17
	> 5	30**	14	28	13	101	46	41	19	35	16	26	12	37	17
	≤ 5	9	5	13	8	66	39	24	14	24	14	19	11	29	17

LICO – Low income cut-off
*p < 0.05; ** p < 0.01; *** p < 0.001.
All data from 2005 – 2007 survey.

were found between arthritis or rheumatism and each of exposure greater than 2 years in duration, exposure greater than 5 years, and exposure greater than 8 years (Table 4.13). In addition to residential history, relationships between occupational variables and health outcomes were explored. Significant associations were found between occupational exposure to contaminants / dust / fumes for more than 2 years and each of high blood pressure or hypertension, arthritis or rheumatism, and long-term skin conditions (Table 4.13). An increase in exposure duration (greater than five years) led to an increase in the strength of the association between occupational exposures and high blood pressure or hypertension (Table 4.13).

In examining relationships between variables from the macro-environment and health outcomes, associations between non-respiratory health and each of the social and family environments, healthcare access, sex, and education were not significant. In addition, no significant associations were found between variables from the macro-environment and each of cancer or diagnosis with chest conditions.

4.3.3.5 SIGNIFICANT ASSOCIATIONS BETWEEN VARIABLES FROM THE MICRO-ENVIRONMENT AND HEALTH

Statistically significant associations were found between variables from the micro-environment and respiratory and non-respiratory health outcomes. Results suggest that lifestyle choices impact health. Current smoking was significantly associated with hospital visits for asthma or respiratory problems since leaving elementary school and fair or poor self-assessed health (Table 4.14). Current smokers had higher prevalence of

Table 4.14 Associations between variables from the micro-environment and health outcomes.

Variables from the micro-environment:		Respiratory health				Non-respiratory health							
		Hospital or ER visits for asthma or respiratory		Chronic bronchitis		Chest conditions		Fair / poor self-assessed		Fair / poor functional capacity (day-to-day)		Fair / poor functional capacity (problems)	
		n	%	n	%	n	%	n	%	n	%	n	%
Behavioural factors	Current smoker	12*	15	10	13	6	8	22*	28	11	14	17	21
	Current non-smoker	24	8	19	6	16	5	38	12	34	11	49	16
	Regular alcohol	3	7	4	9	6**	14	3	7	5	12	7	16
	Non regular alcohol	33	9	25	7	15	4	57	16	40	11	59	17
Psychological factors	≥ 4 on GHQ scale	9**	20	7*	16	5	11	16***	36	17***	38	16***	36
	< 4 on the GHQ scale	27	8	22	6	15	5	44	13	28	8	50	14

*p < 0.05; **p < 0.001; *** p < 0.001.
All data from 2005 – 2007 survey.

hospital or emergency room visits for asthma or respiratory problems since leaving elementary school (15% versus 8%) and fair or poor self-assessed health (28% versus 12%) compared to non-smokers. In addition, regular alcohol consumption was significantly associated with chest conditions (Table 4.14). 14% of participants who consume alcohol on a regular basis were diagnosed with chest conditions compared to 4% of participants who do not consume alcohol on a regular basis.

Results also indicate that psychosocial factors are associated with respiratory and non-respiratory health, mainly hospital or ER visits for asthma or respiratory problems since leaving elementary school, chronic bronchitis, fair or poor self-assessed health and functional capacity over day to day demands and during unexpected or difficult problems. Participants who scored 4 or more on the GHQ scale have higher prevalence of hospital or ER visits for asthma or respiratory problems since leaving elementary school (20% versus 8%), chronic bronchitis (16% versus 6%), fair or poor self-assessed health (36% versus 13%), and fair or poor functional capacity over day to day demands (38% versus 8%) and during unexpected or difficult problems (36% versus 14%).

In examining relationships between the micro-environment and long-term health outcomes, results suggest that physiological factors are not significantly associated with long-term health. In addition, results suggest that micro-environmental variables are not significantly associated with asthma, high blood pressure / hypertension, arthritis / rheumatism, hay fever / allergies, and long-term skin conditions.

4.3.4 HEALTH IMPACTS OF VARIABLES FROM THE MACRO-ENVIRONMENT AND MICRO-ENVIRONMENT

In exploring the link between childhood exposure to air pollution and long-term health, significant associations were found between exposure to SO_2 and long-term respiratory health. Analysis on the entire sample indicates that exposure to SO_2 in childhood is positively associated with two respiratory health outcomes: asthma incidence in adulthood, and hospital or emergency room visits for asthma or respiratory problems since leaving elementary school. Results also indicate that childhood exposure to air pollution is associated with non-respiratory health outcomes such as high blood pressure and arthritis. In examining these relationships, significant sex differences were found. For females, exposure to air pollution is associated with diagnosis for asthma or other respiratory illness, asthma incidence in adulthood, high blood pressure, hay fever, and chest conditions. For males, childhood exposure to air pollution is not associated with any respiratory health outcomes, instead associations with arthritis and long-term skin conditions were found.

In exploring relationships between variables from the macro and micro-environments, overall results suggest that long-term health is affected by residential and occupational histories, life events, and demographic factors. Results also suggest that the social and family environments, as well as healthcare access are not significantly associated with long-term health. In addition, results suggest that residential and occupational histories, life events and demographic factors are associated with psychosocial factors (scoring 4 or more on the GHQ). Furthermore, results suggest that

childhood respiratory health is associated with psychosocial factors (scoring 4 or more on the GHQ scale), and that in turn, psychosocial factors are significantly associated with several health outcomes, including hospital or ER visits for asthma or respiratory problems since leaving elementary school, chronic bronchitis, fair or poor self-assessed health, and fair or poor self-rated ability to handle day-to-day demands and difficult or unexpected situations.

Guided by the conceptual model, multi-variate analysis (Chapter 5) will examine whether variables from the macro-environment and micro-environment mediate relationships between childhood exposure to air pollution and long-term health. Multi-variate analysis will also examine the relative roles that variables from the macro-environment and micro-environment play in impacting long-term health. In exploring bi-variate relationships, some variables from the conceptual framework were not significant. These include ethnicity, lung function measured by the FEV_1/FVC ratio, and social networking. Logistic regression analysis will also indicate whether these variables play a role in long-term health outcomes.

4.4 DISCUSSION

Results suggest that neighbourhood of residence in childhood is related to exposures or opportunities that may impact long-term health. Participants who resided in the lower neighbourhoods in childhood were exposed to higher levels of air pollution than participants who resided in the upper neighbourhoods. In addition, residing in the

east lower neighbourhood in childhood was associated with exposure to smoking in childhood, current smoking, and having a body mass index equal or greater than 25. However, despite significant variations in exposure to air pollution, results indicate that most differences in long-term health outcomes are not statistically significant. Only hospital or ER visits for asthma or respiratory problems since leaving elementary school was statistically significant across neighbourhoods. Therefore, results of analysis at the neighbourhood level indicate that childhood exposure effects of air pollution may dissipate over time. However, in order to examine the relationship between childhood exposure to air pollution and long-term health at the individual level, bi-variate analysis was used to examine associations between exposure measures and health outcomes.

Results suggest that childhood exposure to SO_2 may be linked to long-term respiratory health outcomes. Specifically, significant associations were found between childhood exposure to SO_2 and asthma incidence in adulthood, as well as hospital or ER visits for asthma or respiratory problems since leaving elementary school. This is important since it contributes to the literature on the relationship between exposure to SO_2 and health. Literature has suggested relationships between childhood exposure to SO_2 and short-term respiratory health, mainly low lung function (Peacock et al. 2003), infectious airway diseases and acute daily respiratory symptoms (Pinter et al. 1996; Kramer et al. 1999; Pikhart et al. 2001; Zhang et al. 2002). In relation to findings that suggest sex-related health impacts of exposure to air pollution, findings agree with previous research that found significant sex differences between air pollution and respiratory symptoms (Yu et al. 2001) and between air pollution levels and lower

pulmonary function for females (Gauderman et al. 2002). Females are more susceptible to health effects of air pollution due to their relatively smaller lungs and higher airflow rates (Boezen et al. 2004) that lead to increased dose of air pollutants, and possibly due to hormonal roles (Carey et al. 2007).

Significant associations found between childhood exposure to air pollution and non-respiratory health outcomes (such as arthritis and high blood pressure) are important given recent research that links exposure to air pollution with inflammatory processes. Results from the literature that examined the relationship between air pollution and inflammatory diseases suggests that ultra fine particles can penetrate the epithelium and vascular walls in the body and be transported via the blood to organs where pro-inflammatory events can occur (Blomberg 2000). Consistent with these findings, Van Eden and Hogg (2002) suggest that exposure to ambient particulate matter plays a role in inducing a systemic inflammatory response that includes the release of inflammatory mediators into the circulation that stimulate the bone marrow to release leukocytes and platelets; these are the driving force of autoimmune diseases. While smoking remains a confounder in the relationship between exposure to air pollution and health, recent findings that suggest smoking as a potential trigger of immune reactions that may be associated with the development of rheumatoid arthritis or lupus (Klareskog et al 2007) may be a step in finding a relationship with a similar biological mechanism. In addition, Schwartz et al. (2006) found that an increase in exposure of 10 $\mu g/m^3$ of PM_{10} is associated with increased mortality by 22% for people with inflammatory disease such as

rheumatoid arthritis; by 28% for people with chronic obstructive pulmonary disease; by 32% for people with diabetes; and by 27% for people with congestive heart disease.

Clinical research suggests significant associations between exposure to air pollution and changes in inflammatory biomarkers. For instance, literature suggests that high concentrations of air pollutants are associated with changes in inflammatory and thrombosis markers in the body (Zeka et al. 2006). In addition, research suggests that exposure to high concentrations of air pollutants is responsible for the production and release of inflammatory cytokines by the respiratory tract epithelium (Baeza-Squiban et al. 1999) that is able to cause oxidative stress to cells which causes inflammation (MacNee and Donaldson 2003). Furthermore, clinical studies that examined the relationship between air pollutants and brain markers of inflammation found positive relationships between particulate matter and several inflammatory markers in the brain of mice (Campbell et al. 2005) and canines (Calderon-Garciduenas et al. 2003). Exposure to severe air pollution was also related to brain inflammation and Abeta42 accumulation both of which are markers that precede Alzheimer (Calderon-Garciduenas et al. 2004).

Despite findings from recent literature that suggest inflammatory effects of exposure to air pollution, there is no clinical evidence of the long-term impacts of chronic exposure to air pollution or the effects following exposure to improved air quality. This research was subject to similar limitations since data on exposure to air pollution for the duration of this research was not available. In order to deal with these limitations, analysis was undertaken on two sub-samples: participants who always resided in Hamilton and participants who continue to reside in their address from childhood.

However, results of analysis on the sub-samples were limited by sample size. Other limitations in this research include potential sample and recall bias. Therefore, although this research contributes to the evidence on the relationship between childhood exposure to air pollution and long-term health, more studies that account for longitudinal exposures are needed to examine the relationships between exposure to specific pollutants and respiratory as well as non-respiratory health.

Results of bi-variate analysis suggest that determinants of long-term health include factors related to the macro-environment (exposure to air pollution, life events, socioeconomic status, and residential and occupational histories) and micro-environment (lifestyle and psychosocial factors) constructs of the model. The next step consists of performing logistic regression modeling to explore the roles of variables in the development of respiratory and non-respiratory health outcomes. Variables which were not significant in this study include ethnicity, lung function, and social networking; logistic regression will show whether they have a role to play in the development of long-term respiratory or non-respiratory health outcomes.

4.5 SUMMARY

Analysis at the neighbourhood level indicates that inequity exists in relation to exposure to air pollution. In addition, results suggest that neighbourhood of residence in childhood is associated with smoking in childhood, always residing in Hamilton, residing in a property built before 1950, social networking, current smoking, concern over air

pollution, and having a body mass index equal or greater than 25. Despite differences in exposure to air pollution and opportunities at the neighbourhood level, results indicate that differences in long-term health across neighbourhoods are not significant. However, in examining relationships between childhood exposure to air pollution and long-term health at the individual level, significant differences were found for respiratory and non-respiratory health outcomes. Therefore, that differences in long-term health across neighbourhoods were in most cases not significant indicates that at the neighbourhood level, variables may be mediating the relationship between childhood exposure to air pollution and long-term health.

In relation to health impacts of childhood exposure to air pollution, results suggest that exposure to SO_2 may be related to asthma incidence in adulthood, and hospital or emergency room visits for asthma or respiratory problems since leaving elementary school for females. Furthermore, childhood exposure to air pollution is possibly related to non-respiratory health outcomes such as high blood pressure and arthritis. However, these long-term respiratory and non-respiratory health impacts appear to be sex differentiated. For instance, 27% of females who were exposed to SO_2 above the median level of 11.7 ppb in 1983 / 84 were diagnosed with asthma in adulthood but not in childhood compared to 12% who were exposed at or below the median level. For females, associations were also found between exposure to SO_2 in childhood and diagnosis for asthma or other respiratory illnesses, high blood pressure, hay fever, and chest conditions. For males, childhood exposure to air pollution (TSP, SO_2, and $PM_{3.3}$) is not likely associated with any respiratory health outcomes. Instead, associations with

arthritis and long-term skin conditions were found. The analyses presented in this chapter also suggest that factors from the macro and micro environment constructs of the model affect long-term health.These include factors related to the macro-environment such as life events, socioeconomic status, and occupational and residential histories, as well as factors related to the micro-environment, such as lifestyle and psychosocial factors. In addition, associations found between variables from the macro-environment (sex – healthcare, and socioeconomic status – healthcare), macro and micro-environment (psychosocial factors and each of life events, socioeconomic status, and occupational and residential histories), and micro-environment (respiratory symptoms in childhood – lifestyle, and respiratory symptoms in childhood - psychosocial factors) may also impact long-term health.

Given results that suggest health impacts of a broad range of factors, the stage is set for multi-variate modeling of the health outcomes. Logistic regression will examine the role that variables from the macro and micro-environments play in long-term health. in addition, the role that childhood exposure to air pollution plays in long-term health will be examined. Results of the modeling are described in the next chapter.

CHAPTER FIVE

DETERMINANTS OF LONG-TERM RESPIRATORY AND CHRONIC HEALTH OUTCOMES

5.1 INTRODUCTION

This chapter describes the multi-variate analysis undertaken to assess the relative role that childhood exposure to air pollution plays in the development of long-term health outcomes. The analysis consists of logistic regression modeling of health outcomes using the range of variables collected from respondents and their parents in childhood and adulthood. The outcome variables modeled include long-term respiratory health outcomes, mainly diagnosis with asthma or respiratory problems, asthma diagnosis in adulthood but not in childhood, hospital or emergency room (ER) visits for asthma or respiratory problems since leaving elementary school, and chronic bronchitis, as well as non-respiratory health outcomes such as arthritis / rheumatism, chest conditions, hay fever or allergies, high blood pressure or hypertension, long-term skin conditions, any type of cancer, self-assessed health, and functional capacity. Section 5.2 maps variables used in these analyses and describes the overall results. Section 5.3 presents modeling results of long-term respiratory health outcomes for females, males, and both sexes combined. Section 5.4 presents the modeling results of long-term non-respiratory health outcomes. The chapter concludes with a summary of results of the multi-variate analysis.

5.2 MODELING HEALTH OUTCOMES IN ADULTHOOD

Results of the bi-variate analysis described in Chapter 4 suggest that childhood exposure to air pollution may be associated with long-term health outcomes. These health outcomes appear to be sex-related and include asthma incidence in adulthood, hospital or ER visits for asthma or respiratory problems since leaving elementary school, arthritis or rheumatism, high blood pressure or hypertension, chest conditions, long-term skin conditions, and hay fever or allergies. Furthermore, results also suggest that long-term health outcomes are related to factors from the macro-environment (such as life events, residential and occupational histories, and the social environment) and the micro-environment (lifestyle choices and psychosocial factors). In order to examine the relative roles that air pollution and neighbourhood of residence in childhood play in long-term health, logistic regression modeling was used. The variables included in the multi-variate analysis were assembled into blocks that correspond to the conceptual framework illustrated in Figure 2.2. Figures 5.1 and 5.2 map variables that relate to the macro-environment, micro-environment, and health outcome constructs of the conceptual model.

As discussed in Chapter 3, two models are presented for each health outcome; a best-fit model and one that examines the relative role that long-term exposure to air pollution and neighbourhood of residence in childhood play in long-term health. In addition, logistic regression modeling of each health outcome was performed for females and males separately, and for both sexes combined. Results of each logistic regression are discussed in terms of the odds ratio, which is a measure that indicates how much more

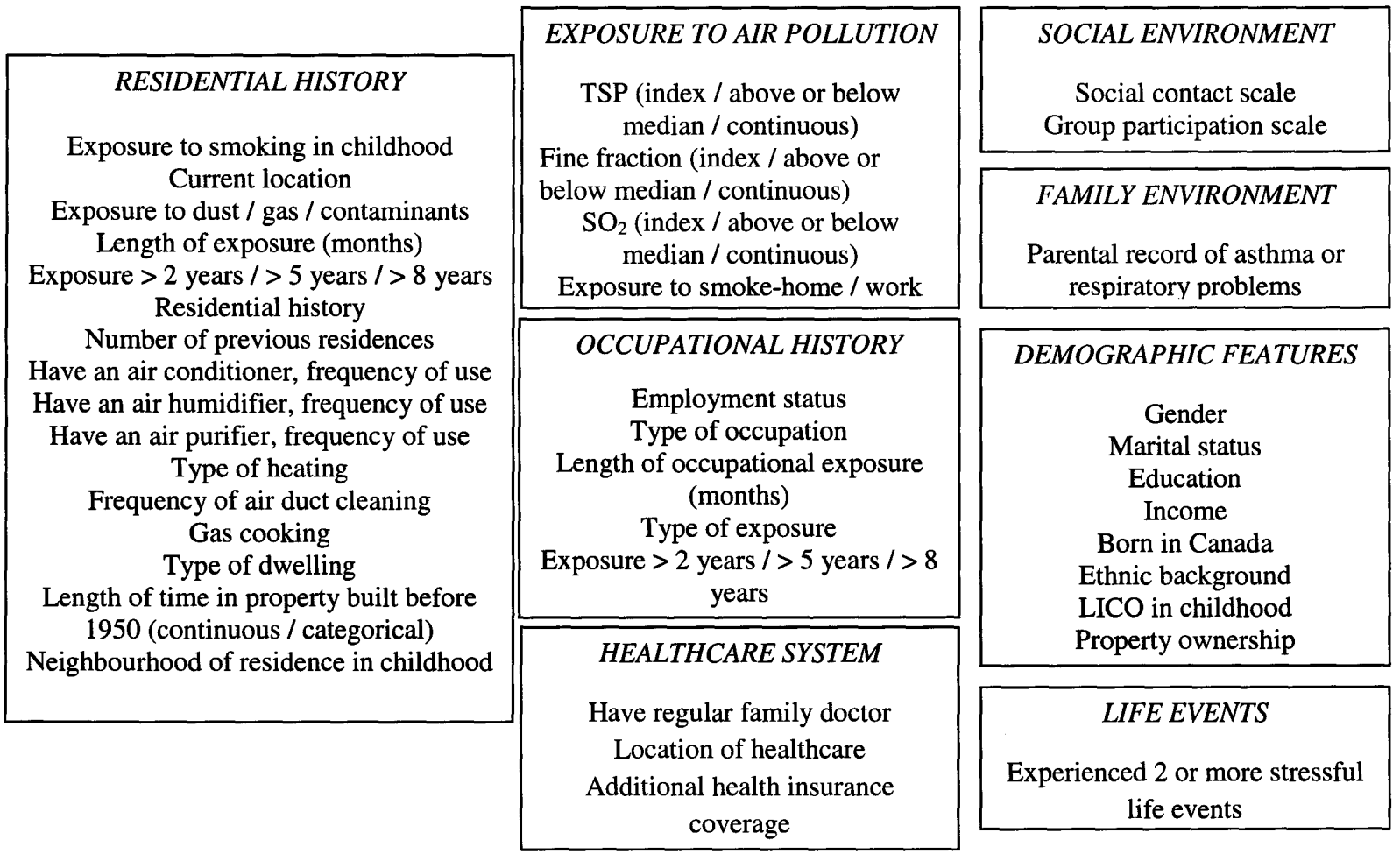

Figure 5.1 Mapping variables related to the macro-environment component of the conceptual framework.

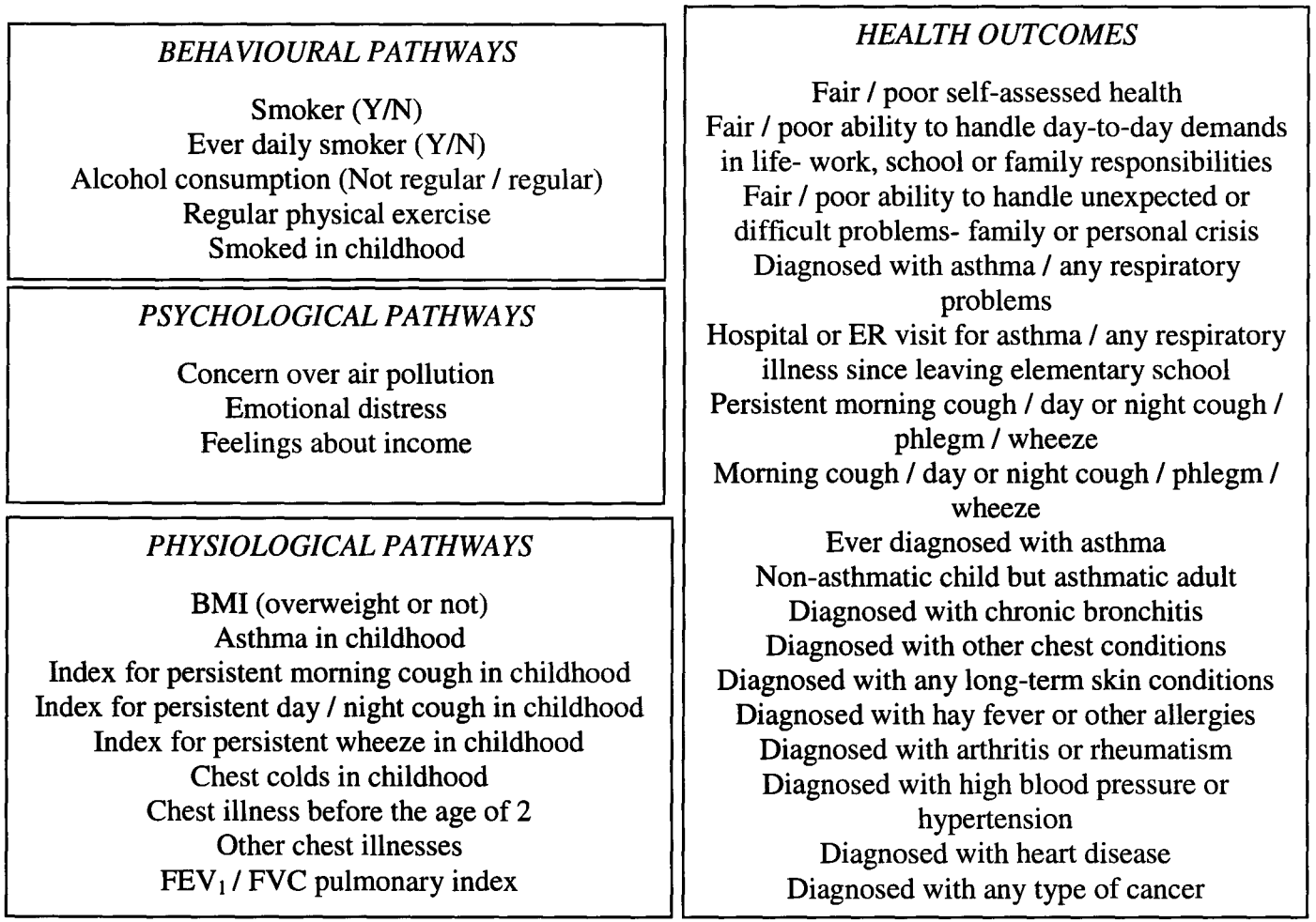

Figure 5.2 Mapping variables related to the micro-environment and health outcome constructs of the conceptual framework.

likely (or unlikely) a factor (measured by an explanatory variable) may lead to the outcome, in this case respiratory or non-respiratory health outcomes in adulthood. An odds ratio (OR) greater than one indicates an increased likelihood and an odds ratio less than one indicates a decreased likelihood. For each model, the statistical significance of each explanatory variable and the relative odds, as well as the rho-square (i.e. a goodness of fit measure) are presented. A rho-square of 0.2 - 0.4 is generally considered to represent a good fit (McFadden, 1974 cited in Wrigley, 1985). For each model, specificity and sensitivity are presented. Sensitivity is the proportion of correct predictions of cases; specificity is the proportion of correct prediction of non-cases.

The rho-square values obtained for models of respiratory and non-respiratory health outcomes ranged between 0.07 and 0.56. Models with rho-square values greater than 0.2 include asthma and respiratory problems (male model only), hospital or ER visits for asthma or respiratory problems since leaving elementary school (male, female, and combined), chronic bronchitis (female), arthritis / rheumatism (male and female), chest conditions (combined and male), long-term skin conditions (female), cancer (combined and female), and fair / poor self-assessed health (combined and female).

5.3 MODELS OF RESPIRATORY HEALTH OUTCOMES IN ADULTHOOD

5.3.1 MODELS OF ASTHMA OR ANY RESPIRATORY PROBLEMS

Results suggest that physiological factors are significant in explaining diagnosis for asthma or respiratory problems. Individuals that had asthma or chest colds in

childhood, and that reported residential exposure to gas / dust / contaminants for more than 8 years have increased odds of ever being diagnosed with asthma or any respiratory problems (Table 5.1). Surprisingly, individuals that have no parental record of asthma or respiratory problems (Appendix 3.10) have increased likelihood of ever being diagnosed with asthma or any respiratory problems (OR = 2.08). It is possible that individuals with at least one parental record of asthma or respiratory problems are exposed to preventative measures in their family environment than decrease their likelihoods of ever being diagnosed with asthma or any respiratory problems.

For females, persistent morning cough in childhood and exposure to SO_2 above 11.7 ppb in 1983 / 4 (mean age of participants = 13 years) increases the odds of asthma or respiratory problems (OR = 166.80 and OR = 2.82 respectively). For males, significant explanatory variables include asthma in childhood, residential exposure to gas / dust / contaminants for more than 8 years, no parental record of asthma or respiratory problems, and occupational exposure to gas / dust / fumes for more than 2 years (Table 5.1). The rho-square values for the models range between 0.07 and 0.33, with only the model for asthma or respiratory problems for males having a rho-square value greater than 0.2. However, large confidence intervals for explanatory variables in sex-related models indicate uncertainty about the position of the true effect, likely due to sample size.

Although rho-square values increase when variables related to long-term exposure to air pollution and neighbourhood of residence in childhood are forced into the models, results suggest that long-term exposure to air pollution in childhood does not play a significant role in ever being diagnosed with asthma or respiratory problems (Table 5.2).

Table 5.1 Logistic regression models for ever being diagnosed with asthma or any respiratory problems.

Variable (Reference)	Classification	Overall sample (n=392)		Females (n = 152)		Males (n = 203)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Asthma in childhood (Non-asthmatic)	Asthmatic	6.61***	(3.22, 13.56)			32.49***	(11.09, 95.17)
Chest cold in childhood (No)	Yes	3.32*	(1.09, 10.04)				
Residential exposure to gas / dust / contaminants (≤ 8 years)	> 8 years	1.75*	(1.04, 2.95)			3.62*	(1.17, 11.24)
Parental record of asthma /respiratory problems (at least 1)	No record	2.08**	(1.22, 3.53)			3.90**	(1.56, 9.74)
Index persistent morning cough in childhood	Increasing			166.80*	(2.11, 13210)		
Exposure to SO_2 (1983/4) (≤ 11.7 ppb)	> 11.7 ppb			2.82**	(1.3, 6.13)		
Occupational exposure to dust / gas / fumes (≤ 2 years)	> 2 years					4.33*	(1.29, 14.54)
Specificity (%)		74.3		47.7		70.4	
Sensitivity (%)		56.2		74.4		82.9	
Rho-square		0.13		0.07		0.33	

* $p < 0.05$; ** $p < 0.01$; ***$p < 0.001$.

Table 5.2 Long-term exposure to air pollution in childhood and ever diagnosed with asthma or any respiratory problems.

Variable (Reference)	Classification	Overall sample (n=391)		Females (n = 164)		Males (n = 203)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Asthma in childhood (Non-asthmatic)	Asthmatic	7.12***	(3.40, 14.98)			36.72***	(11.86, 113.65)
Chest cold in childhood (yes)	No	3.68**	(1.15, 11.75)				
Residential exposure to gas / dust / contaminants (≤ 8 years)	> 8 years	1.87*	(1.09, 3.19)			5.00**	(1.50, 16.70)
Parental record of asthma / respiratory problems (At least 1)	No record	2.05*	(1.19, 3.53)			4.36*	(1.67, 11.34)
Index persistent morning cough in childhood	Increasing			318.87*	(3.45, 29510)		
Exposure to SO_2 (1983/4) (≤ 11.7 ppb)	> 11.7 ppb			1.49	(0.44, 5.06)		
Occupational exposure to dust / gas / fumes (≤ 2 years)	> 2 years					4.13*	(1.25, 14.93)
Residence in childhood (WU)							
	East lower	0.69	(0.30, 1.58)	0.94	(0.28, 3.13)	1.06	(0.27, 4.16)
	West lower	1.55	(0.60, 3.99)	2.16	(0.55, 8.45)	0.20	(0.03, 1.38)
	East upper	0.88	(0.39, 1.94)	0.57	(0.20, 1.67)	0.95	(0.22, 4.02)
Index for TSP (Below median)	Above median	0.55	(0.20, 1.52)	0.69	(0.15, 3.20)	0.59	(0.12, 3.02)
Index for SO_2 (Below median)	Above median	2.88	(0.90, 9.30)	3.45	(0.28, 41.92)	0.56	(0.07, 4.51)
Index for $PM_{3.3}$ (Below median)	Above median	0.39	(0.10, 1.49)	0.35	(0.04, 2.79)	1.31	(0.13, 13.15)
Specificity (%)		74.8		55.0		79.6	
Sensitivity (%)		57.3		65.9		80.5	
Rho-square		0.15		0.09		0.37	

* $p < 0.05$; ** $p < 0.01$; ***$p < 0.001$.

5.3.2 MODELS OF ASTHMA IN ADULTHOOD BUT NOT IN CHILDHOOD

The best-fit model for asthma incidence in adulthood indicates that female respondents are 3.51 times more likely to develop asthma in adulthood (Table 5.3). Individuals exposed to SO_2 above the median level of 11.7 ppb in 1983 / 84 (mean age = 13 years) are more likely to develop asthma in adulthood (OR = 1.82). Results of the combined model also indicate that individuals that feel mostly dissatisfied, unhappy or terrible about their health are 7.27 times more likely to be diagnosed with asthma in adulthood compared to individuals that feel delighted / pleased / mostly satisfied / mixed about their health (Table 5.3). While feelings about health are likely to be related to current health status rather than exposures or health status in childhood, it is important to note that removing this variable from the multi-variate analysis had no effect on the significance of the other variables (sex and exposure to SO_2 above 11.7 ppb in 1983 / 4) in explaining the health outcome.

Consistent with the combined model, the sex-specific models indicate that female respondents (non-asthmatic in childhood) that feel mostly dissatisfied, unhappy or terrible about their health and that were exposed to SO_2 above the median level of 11.7 ppb in 1983 / 84 in childhood (mean age = 13 years) are 18.27 and 2.51 times more likely to be diagnosed with asthma in adulthood than female respondents that feel mostly satisfied, pleased, or delighted with their health and that were exposed to SO_2 at the median or below. For males, significant variables include residential exposure to dust / gas / contaminants for more than 8 years in duration and at least one parental record of asthma or respiratory problems (Table 5.3). Male respondents that report residential

Table 5.3 Logistic regression model for asthma incidence in adulthood.

Variable (Reference)	Classification	Overall sample (n=292)		Females (n = 142)		Males (n = 172)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Sex (Male)	Female	3.51**	(1.61, 7.63)				
Feeling about health (Delighted / pleased / mostly satisfied / mixed)	Mostly dissatisfied / unhappy / terrible	7.27**	(2.28, 23.18)	18.27*	(2.00, 167.24)		
Exposure to SO_2 in 1983 / 84 ($\leq$ 11.7 ppb)	> 11.7 ppb	1.82*	(1.01, 3.64)	2.51*	(1.04, 6.05)		
Residential exposure to gas / dust / contaminants ($\leq$ 8 years)	> 8 years					3.77*	(1.10, 12.87)
Parental record of asthma /respiratory problems (at least one)	No record					4.39**	(1.47, 13.05)
Specificity (%)			48.6		52.5		65.4
Sensitivity (%)			79.1		75.0		68.8
Rho-square			0.10		0.10		0.11

* $p < 0.05$; ** $p < 0.01$.

exposure to dust / gas / contaminants for a time period that exceeds 8 years are 3.77 times more likely to be diagnosed with asthma in adulthood. Furthermore, male respondents with no parental record of asthma or respiratory problems are 4.39 times more likely to be diagnosed with asthma in adulthood than participants that report at least one parental record of asthma or respiratory problems. As discussed in section 5.3.1, individuals with at least one parental record of asthma or respiratory problems may be exposed to preventative measures in their family environment during childhood, which decreases their likelihood of developing asthma.

When neighbourhood of residence in childhood and the indices of exposure to TSP, $PM_{3.3}$, and SO_2 are forced into the models, exposure to SO_2 above 11.7 ppb in 1983 / 84 becomes statistically insignificant in explaining asthma in adulthood in the combined and the female models. No change in the significance of all other variables from the models is observed (Table 5.4). Overall, neighbourhood of residence in childhood and long-term exposure to TSP, $PM_{3.3}$, and SO_2 in childhood are not significant in explaining asthma incidence in adulthood. Although including these variables in the multi-variate analysis led to an increase in the rho-square values of the models (Table 5.4), the large spread confidence intervals indicate uncertainty about the position of the true effect of explanatory variables.

Table 5.4 Long-Term exposure to air pollution in childhood and asthma incidence in adulthood.

Variable (Reference)	Classification	Overall sample (n=292)		Females (n = 142)		Males (n = 172)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Sex (Male)	Female	3.67**	(1.64, 8.20)				
Feeling about health (score ≥ 3)	Score < 3	6.88**	(2.09, 22.64)	17.41*	(1.79, 168.97)		
Exposure SO_2 (83/4) (≤ 11.7 ppb)	> 11.7 ppb	1.79	(0.55, 5.80)	1.49	(0.36, 6.12)		
Residential exposure to gas / dust / contaminants (< 8 years)	> 8 years					6.21**	(1.56, 24.75)
Parental record of asthma / respiratory problems (At least one)	No record					4.15*	(1.31, 13.18)
Residence in childhood (west upper)							
	East lower	0.81	(0.29, 2.30)	0.56	(0.13, 2.32)	0.85	(0.19, 3.89)
	West lower	0.62	(0.15, 2.47)	0.92	(0.18, 4.65)	0.14	(0.01, 1.79)
	East upper	0.39	(0.14, 1.12)	0.40	(0.12, 1.33)	0.12	(0.01, 1.36)
Index for TSP (below median)	Above median	0.89	(0.23, 3.52)	0.78	(0.14, 4.42)	0.89	(0.10, 7.81)
Index for SO_2 (below median)	Above median	1.18	(0.10, 13.89)	0.26	(0.02, 2.95)	0.97	(0.06, 14.82)
Index for $PM_{3.3}$ (below median)	Above median	0.39	(0.058, 2.76)	4.58	(0.22, 93.51)	0.87	(0.06, 12.10)
Specificity (%)		49.4		65.0		78.8	
Sensitivity (%)		76.7		56.3		62.5	
Rho-square		0.12		0.12		0.19	

* $p < 0.05$; ** $p < 0.01$.

5.3.3 MODELS OF HOSPITAL OR ER VISITS FOR ASTHMA OR ANY RESPIRATORY PROBLEMS SINCE LEAVING ELEMENTARY SCHOOL

Results of best-fit models indicate that neighbourhood of residence in childhood is significant in explaining hospital or ER visits for asthma or any respiratory problems since leaving elementary school (Table 5.5). Participants that resided in the east lower neighbourhood and the west lower neighbourhood in childhood are 43.60 and 16.92 times more likely to report hospital or ER visits for asthma or any respiratory problems than participants that resided in the west upper neighbourhood in childhood. Participants that resided in the east upper neighbourhood in childhood are 9.29 times more likely to report hospital or ER visits for asthma or any respiratory problems than participants that resided in the west upper neighbourhood in childhood. Significant variables also include experiencing two or more stressful life events (such as financial problems, divorce / separation, illnesses, or death of family member) (OR = 5.79), ever diagnosed with asthma in childhood (OR = 10.57), and airway obstruction in childhood (OR = 5.46) (Table 5.5). In relation to the explanatory variables that emerge for these models, research suggests plausible biologic pathways though which stress may affect asthma morbidity (Turyk et al. 2007), and lung function in childhood may affect respiratory problems (Rasmussen et al. 2002), which in turn increase the incidences of hospitalization for asthma (Schneider et al. 2008). Unexpectedly, results indicate that participants exposed to TSP in 1985 / 86 (mean age of participants = 15 years) above 45.7$\mu g/m^3$ were less likely to report hospital or ER visits for asthma or any respiratory problems since leaving elementary school (OR = 0.93). One possible explanation may be

Table 5.5 Logistic regression models for hospital or ER visits for asthma or any respiratory problems since leaving elementary school.

Variable (Reference)	Classification	Overall sample (n=312)		Females (n = 162)		Males (n = 180)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Stressful life event (less than 2)	2 or more	4.78*	(1.44, 15.88)	16.19**	(2.46 to 106.49)		
Ever asthma / respiratory problems (No)	Yes	10.57**	(4.01, 27.84)	4.64*	(1.10 to 19.66)	23.37**	(3.86 to 141.48)
Index of lung function (No airway obstruction)	Airway obstruction	5.46*	(1.50, 19.84)	7.13*	(1.06 to 47.97)		
Residence in childhood (WU)							
	East lower	43.60**	(7.02 to 270.85)				
	West lower	16.92**	(2.38 to 120.29)				
	East upper	9.29*	(1.79 to 48.11)				
Exposure to TSP (85/6) (≤ 45 µg/m^3)	> 45.7 µg/m^3	0.93*	(0.87 to 0.99)				
Exposure SO_2 (83/4) ($\leq$ 11.7 ppb)	> 11.7 ppb			4.71*	(1.53 to 25.12)		
Residential exposure to gas / dust / contaminants (Not exposed)	Exposed			10.50*	(1.55 to 71.33)		
Residential exposure to gas / dust / contaminants (< 8 years)	$\geq$ 8 years					9.22**	(1.45 to 58.56)
Smoking age - childhood (Older)	Younger			1.02**	(1.01 to 1.02)		
Residence (always in Hamilton)							
	Other in Ontario					5.51	(0.70 to 43.47)
	Other outside Ontario					39.51**	(3.56 to 438.95)
Specificity (%)			91.0		94.5		89.4
Sensitivity (%)			63.3		75.0		70
Rho-square			0.35		0.43		0.41

* $p < 0.05$; ** $p < 0.01$.

that individuals are able to detect the relatively large size of TSP particulates (approximately 50 μm in diameter) when in high levels, largely due to the fact that TSP particles tend to stick on nose and throat membranes instead of penetrating the lungs. This leads to respiratory symptoms such as coughing and sneezing that may prevent individuals from spending too much time outdoors, which in turn reduces exposure to air pollutants that are able to penetrate the lungs and that may lead to hospital or ER visits. The rho-square value of the combined best-fit model is 0.35 indicating a good fit.

When data is disaggregated by gender, results of logistic regression models point to the effect of exposure to SO_2 on the respiratory health of females. Results indicate that females exposed to SO_2 above the median level of 11.7 ppb in 1983 / 84 (mean age = 13years) have increased likelihood of hospital or ER visits for asthma or respiratory problems (Table 5.5). Females that were exposed to SO_2 above the median level of 11.7 ppb in 1983 / 84 (mean age = 13 years) were 4.71 times more likely to have hospital or ER visits for asthma or respiratory problems since leaving elementary school. Significant variables that increase likelihood of hospital or ER visits for asthma or any respiratory problems for females also include experiencing two or more stressful life events (OR = 16.19), ever being diagnosed with asthma or respiratory problems (OR = 4.64), airway obstruction in childhood (OR = 7.13), residential exposure to gas / dust / contaminants (OR = 10.50), and smoking at a young age in childhood (OR = 1.02) (Table 5.5). The rho-square value of this model is relatively high at 0.43. For males, significant variables that increase the odds of hospital or emergency room visits for asthma or any respiratory problems since leaving elementary school include residing outside Ontario

(OR = 39.51), ever diagnosed with asthma or respiratory problems (OR = 23.37), and residential exposure to gas / dust / contaminants for more than 8 years (OR = 9.22) (Table 5.5). For males, the best fit model results in an overall predictability of 88.3% and a rho-square of 0.41 (Table 5.5), however the large spread of confidence intervals indicates uncertainty about the true effect of explanatory variables.

In order to examine the role that long-term exposure to TSP, $PM_{3.3}$ and SO_2 and neighbourhood of residence in childhood play in hospital or ER visits for asthma or any respiratory problems since leaving elementary school, the indices of exposure to TSP, $PM_{3.3}$, and SO_2 and neighbourhood of residence in childhood were forced into the model. Participants that were exposed to SO_2 above the median level in childhood are 8.19 times more likely to report hospital or ER visits for asthma or respiratory problems since leaving elementary school than participants that were exposed below the median level (Table 5.6). In addition, participants that resided in the east and west lower neighbourhoods in childhood are 39.81 and 18.77 times more likely to have hospital or ER visits for asthma or respiratory problems since leaving elementary school than participants that resided in the west upper neighbourhood in childhood. Participants that reside in the east upper neighbourhood are 9.91 times more likely to have hospital or ER visits for asthma or any respiratory problems since leaving elementary school. Significant variables also include experiencing two or more stressful life events (OR = 5.99), asthma or respiratory problems (OR = 10.30), and airway obstruction (OR = 5.74) (Table 5.6). In addition, exposure to TSP above 45.7$\mu g/m^3$ in 1985 / 6 (mean age of participants = 15)

Table 5.6 Long-term exposure to air pollution in childhood and having hospital or ER visits for asthma or any respiratory problems since leaving elementary school.

Variable (Reference)	Classification	Overall sample (n=312)		Females (n = 162)		Males (n = 180)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Stressful life event (less than two)	2 or more	5.99***	(1.76, 21.48)	22.31**	(2.56 to 194.79)		
Ever asthma / respiratory problems (No)	Yes	10.30***	(3.80, 27.94)	4.59	(0.90 to 23.26)	41.79**	(4.25 to 411.33)
Index of lung function (no airway obstruction)	Airway obstruction	5.74**	(1.54, 21.41)	6.20	(0.84 to 45.71)		
Exposure to TSP (85/6) (≤ 45 $\mu g/m^3$	> 45.7 $\mu g/m^3$	0.89*	(0.79, 0.99)				
Exposure SO_2 (83/4) (≤ 11.7 ppb)	> 11.7 ppb			1.71	(0.15 to 19.23)		
Residential exposure to gas / dust / contaminants (Not exposed)	Exposed			9.59	(0.95 to 96.71)		
Smoking age - childhood (Older)	Younger			1.03*	(1.01, 1.05)		
Residential exposure to gas / dust / contaminants (< 8 years)	≥ 8 years					7.88	(0.95 to 65.40)
Residence (always in Hamilton)							
	Other in Ontario					3.35	(0.36 to 30.95)
	Other outside Ontario					42.54**	(2.97 to 645.82)
Residence in childhood (west upper)							
	East lower	39.81**	(4.93, 321.19)	8.55	(0.48 to 153.09)	13.91	(0.45 to 425.71)
	West lower	18.77**	(2.33, 150.98)	9.76	(0.22 to 427.13)	8.54	(0.17 to 431.25)
	East upper	9.91*	(1.36, 72.08)	3.58	(0.15 to 87.28)	5.50	(0.24 to 128.40)
Index for TSP exposure ($\leq$ median)	> median	2.48	(0.25, 24.83)	0.21	(0.00 to 9.27)	0.68	(0.02 to 23.87)
Index for SO_2 exposure ($\leq$ median)	> median	8.19*	(1.32, 85.70)	23.57	(0.07 to 7930)	1.07	(0.02 to 76.72)
Index for $PM_{3.3}$ exposure ($\leq$median)	> median	0.50	(0.04, 6.91)	0.22	(0.00 to 22.75)	0.25	(0.00 to 28.85)
Specificity (%)		91.3		94.5		88.8	
Sensitivity (%)		63.3		81.3		80.0	
Rho-square		0.37		0.49		0.48	

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

decreases the likelihood of hospital or ER visits for asthma or respiratory problems since leaving elementary school (OR = 0.89). The rho-square value for the model that examines the relative role of air pollution and neighbourhood of residence in childhood is 0.37 indicating a good fit.

The overall predictability and rho-square values of sex-specific models increase when neighbourhood of residence in childhood and the indices of exposure to TSP, $PM_{3.3}$, and SO_2 are forced in the analysis (Table 5.6). However, for the female model, most variables become insignificant in explaining the health outcome. Significant variables that increase the likelihood of hospital or ER visits for asthma or respiratory problems since leaving elementary school include experiencing two or more stressful life events (OR = 22.31) and smoking at a younger age in childhood (OR = 1.03). For males, significant variables include residing outside Ontario (OR = 42.54), and ever diagnosis with asthma or respiratory problems (OR = 41.79). Despite the increases in the rho-square values of the models, results indicate that long-term exposure to air pollution and neighbourhood of residence in childhood are not significant in explaining hospital or ER visits for asthma or respiratory problems since leaving elementary school. These results are surprising particularly since earlier results of logistic regression models suggest that exposure to SO_2 is an explanatory variable for respiratory health for females.

5.3.4 MODELS OF CHRONIC BRONCHITIS

Overall results suggest that behavioral factors are significant in increasing the likelihood of chronic bronchitis. Individuals that ever smoked daily are 3.02 times more

likely to be diagnosed with chronic bronchitis than individuals that never smoked (Table 5.7). In addition, individuals that scored 4 or more on the General Health Questionnaire (GHQ) are 3.57 more likely to be diagnosed with chronic bronchitis than participants that scored less than 4 on the GHQ scale. Similar to results of the previous models, results suggest that exposure to TSP in childhood is significant in decreasing the likelihood of respiratory health outcomes in adulthood. Individuals that were exposed to higher levels of TSP in 1980 / 1 are less likely to be diagnosed with chronic bronchitis than individuals that were exposed to lower levels of TSP in 1980 / 1 (OR = 0.94).

When data is disaggregated by sex, results of logistic regression models indicate an increase in the odds of chronic bronchitis for females that rarely use an air filter (OR = 124.70), that smoke (OR = 7.00), that experienced 2 or more stressful life events (OR = 13.26), and that had chest illnesses in childhood (OR = 20.63) (Table 5.7). Consistent with the combined model, exposure to TSP above the median level of 45.7$\mu g/m^3$ in 1985 / 86 decreases the likelihood of chronic bronchitis (OR = 0.04). In addition, although the rho-square value of the best-fit model of chronic bronchitis for females is 0.44 indicating a good fit, the large spread of confidence intervals indicates uncertainty about the true effect of explanatory variables. For males, results of the logistic regression model indicates that exposure to air pollution is a significant predictor of chronic bronchitis. Once again, results indicate that exposure to TSP is significant in explaining respiratory health outcomes. Individuals that were exposed to higher levels of TSP in 1980 / 81 are less likely to be diagnosed with chronic bronchitis. However, results indicate that

Table 5.7 Logistic regression models for chronic bronchitis.

Variable (Reference)	Classification	Overall sample (n=381)		Females (n = 147)		Males (n = 151)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Ever daily smoker (Non-smoker)	Smoker	3.02**	(1.35, 6.74)				
Emotional distress (Score < 4 on GHQ)	≥ 4 on GHQ	3.57*	(1.35, 9.45)				
Exposure to TSP (1980/1)	Increasing	0.94**	(0.89, 0.98)			0.88**	(0.81, 0.96)
Frequency of use of air filter (Always / almost always)	Rarely			124.70*	(3.156, 4924)		
Current smoker (Non-smoker)	Smoker			7.00*	(1.34, 36.62)		
Stressful life events (Experienced < 2)	Experienced 2 or more			13.26**	(1.95, 89.97)		
Chest illness in childhood (No)	Yes			20.63**	(2.39, 177.94)		
Exposure to TSP (≤45.7μg/m^3)	> 45.7μg/m^3			0.04*	(0.00, 0.45)		
Exposure to $PM_{3.3}$ (1985 / 6) (≤ 43.2 μg/m^3)	> 43.2 μg/m^3)					7.10*	(1.50, 33.51)
Specificity (%)			100		88.1		65.6
Sensitivity (%)			0		75.0		70.2
Rho-square			0.1		0.44		0.16

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

exposure to $PM_{3.3}$ is a significant predictor of long-term respiratory health. Male participants that were exposed to $PM_{3.3}$ above the median level of 43.2 $\mu g/m^3$ in 1985 / 6 are 7.10 times more likely to be diagnosed with chronic bronchitis than participants that were exposed below the median level (Table 5.7).

For all models of chronic bronchitis, the rho-square value increases when variables related to long-term exposure to air pollution and neighbourhood of residence in childhood are forced into the best-fit models (Table 5.8). The rho-square values of both sex-specific models of chronic bronchitis are greater than 0.2 indicating a good fit. However, the large spread of confidence intervals indicates uncertainty about the true effect of explanatory variables. In all models, long-term exposure to air pollution and neighbourhood of residence in childhood are not significant in increasing the likelihood of diagnosis with chronic bronchitis.

5.3.5 SUMMARY – CHILDHOOD EXPOSURE TO AIR POLLUTION AND LONG-TERM RESPIRATORY HEALTH

Overall, results suggest that exposure to SO_2 in childhood may impact the long-term respiratory health of females. In particular, for females, exposure to SO_2 above the median level of 11.7 in 1983 / 84 (mean age of participants = 13 years) is significant in increasing the likelihood of ever asthma or respiratory problems, asthma incidence in adulthood, and hospital or ER visits for asthma or respiratory problems since leaving elementary school. For males, results suggest that exposure to SO_2 in childhood is not significant in predicting long-term respiratory health. Instead, results of logistic

Table 5.8 Long-term exposure to air pollution in childhood and chronic bronchitis.

Variable (Reference)	Classification	Overall sample (n=381)		Females (n = 151)		Males (n = 158)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Ever daily smoker (No)	2 or more	3.28**	(1.43, 7.53)				
Emotional distress (Score < 4 on GHQ)	≥ 4 on GHQ	4.28**	(1.46, 12.53)				
Exposure to TSP (1980/1)	Increasing	0.91	(0.82, 1.01)			0.86	(0.73, 1.01)
Frequency of use of air filter (Always / almost always)	Rarely			1037*	(3.47, 30991)		
Current smoker (Non-smoker)	Smoker			9.63*	(1.42, 65.20)		
Life events (experienced < 2)	Experienced 2 or more			33.78*	(1.60, 715)		
Chest illness in childhood (No)	Yes			34.95*	(1.05, 1165.76)		
Exposure to TSP (≤45.7μg/m^3)	> 45.7μg/m^3			0.00*	(0.00, 0.63)		
Exposure to $PM_{3.3}$ (1985 / 6) (≤ 43.2 μg/m^3)	> 43.2 μg/m^3)					4.60	(0.42, 50.67)
Residence in childhood (West upper)							
	East lower	2.91	(0.65, 13.08)	2.28	(0.07, 76.53)	6E007	(0.00,8E007)
	West lower	2.66	(0.41, 17.39)	39.98	(0.07, 24225)	7E007	(0.00, 9E007)
	East upper	1.76	(0.37, 8.33)	3.80	(0.14, 101.43)	9E007	(0.00, 10E007)
Index for TSP exposure (≤ median)	> median	3.03	(0.42, 21.81)	8230.81	(0.41, 2E008)	4.37	(0.17, 112.45)
Index for SO_2 exposure (≤ median)	> median	2.99	(0.45, 20.04)	4.04	(0.03, 486.67)	.86	(0.04, 21.22)
Index for $PM_{3.3}$ exposure (≤median)	> median	0.30	(0.03, 3.28)	0.00	(0.00, 5.08)	1.99	(0.01, 789.94)
Specificity (%)			67.7		85.6		63.7
Sensitivity (%)			75		83.3		83.3
Rho-square			0.14		0.56		0.23

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

regression models indicate that exposure to $PM_{3.3}$ above the median level of 43.2μg/m^3 in 1985 / 86 (mean age of participants = 15 years) is significant in increasing the likelihood of chronic bronchitis for males. Results of logistic regression models (hospital or ER visits for asthma or any respiratory problems since leaving elementary school and chronic bronchitis) also suggest that exposure to relatively higher levels of TSP may have a protective role in long-term respiratory health. This may be related to possible protective behavioral measures undertaken by individuals exposed to high levels of TSP, particularly since total suspended particulates that include dust and large particles of up to about 50μm (similar to the size of a diameter of human hair) are possible to detect when in high concentrations. Although the size of TSP can be detected by the naked eye, TSP also includes particle material less than 50μm. Literature indicates that the fraction of PM_{10} and $PM_{2.5}$ in TSP is 40% and 24% respectively. Detecting high levels of TSP in the air may also protect individuals from exposure to smaller size particulates that may be related to long-term respiratory health. Therefore, exposure to high levels of TSP may promote less time spent outdoors, which in turn may lead to decreased likelihoods of respiratory health outcomes.

Results of logistic regression models indicate that neighbourhood of residence in childhood possibly impacts hospital or ER visits for asthma or any respiratory problems. In particular, residing in the east and west lower neighbourhoods is significant in increasing the likelihood of hospital or ER visits for asthma or respiratory problems since leaving elementary school. Therefore, in relation to hospital or ER visits, the gradient in available opportunities and distribution of resources across the four neighbourhoods in

Hamilton may have contributed to health inequity. Results indicate that neighbourhood of residence in childhood does not play a role in diagnosis for asthma or respiratory problems, asthma incidence in adulthood, or chronic bronchitis.

Significant variables that explain long-term respiratory health also include physiological (lung function, chest colds, persistent morning cough, and asthma in childhood) and behavioral (smoking) factors, psychological (emotional distress and feeling about health) factors, residential exposures (ever and greater than 8 years), occupational exposures (greater than 2 years), family environment (no parental record of asthma or respiratory problems), stressful life events, and residential history (location of residence and rare use of air filters). Results indicate that the social environment and healthcare access do not play a role in long-term respiratory health outcomes.

5.4 MODELS OF NON-RESPIRATORY HEALTH OUTCOMES IN ADULTHOOD

5.4.1 MODELS OF HAY FEVER / ALLERGIES

Overall results suggest that residential history and diagnosis with chest conditions or asthma / any respiratory problems are significant in increasing the likelihood of diagnosis with hay fever or allergies. Individuals that resided anywhere outside Hamilton are 2.43 times more likely to be diagnosed with hay fever or allergies than individuals that always resided in Hamilton (Table 5.9). In addition, individuals that are diagnosed

Table 5.9 Logistic regression models of hay fever or allergies

Variable (Reference)	Classification	Overall sample (n=395)		Females (n = 184)		Males (n = 151)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Residential history (Always resided in City of Hamilton)	Other	2.43**	(1.55, 3.83)			3.55***	(1.80, 7.00)
Diagnosed with chest conditions (No)	Yes	3.37***	(1.19, 9.51)			9.76**	(1.87, 51.08)
Ever diagnosed with asthma or respiratory problems (No)	Yes	5.74***	(3.35, 9.86)	3.67***	(1.83, 7.35)	10.49***	(4.40, 25.02)
Specificity (%)		60.1		84.6		62.6	
Sensitivity (%)		73.1		40.0		78.0	
Rho-square		0.12		0.07		0.19	

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

with chest conditions and asthma / any respiratory problems are 3.37 and 5.74 times more likely to be diagnosed with hay fever / allergies than participants that were never diagnosed with chest conditions and asthma / any respiratory problems. For females, only diagnosis with asthma / any respiratory problems is significant in increasing the likelihood of diagnosis with hay fever / allergies (OR = 3.67). For males, significant variables that increase the likelihood of hay fever / allergies include ever residing outside Hamilton (OR = 3.55), diagnosis with chest conditions (OR = 9.76), and ever diagnosed with asthma or respiratory problems (OR = 10.49). The rho-square values for best-fit models are below 0.2 indicating a low goodness of fit.

For the combined and male models of hay fever / allergies, the rho-square value increases when variables related to long-term exposure to air pollution and neighbourhood of residence in childhood are forced into the best-fit models (Table 5.10). However, only the rho-square value of the logistic regression model for males is 0.2 indicating a good fit. In addition, for all models, long-term exposure to air pollution and neighbourhood of residence in childhood are not significant in increasing the likelihood of diagnosis with hay fever or allergies. Significant variables in explaining diagnosis with hay fever / allergies remains the same as those from the best-fit models.

5.4.2 MODELS OF ARTHRITIS OR RHEUMATISM

The results of the best-fit logistic regression models for diagnosis with arthritis or rheumatism are presented in Table 5.11. Results suggest that exposure to SO_2 above 11.7

Table 5.10 Long-term exposure to air pollution in childhood and diagnosis with hay fever or allergies.

Variable (Reference)	Classification	Overall sample (n=393)		Females (n = 189)		Males (n = 204)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Residential history (Always resided in City of Hamilton)	Other	2.49***	(1.56, 3.96)			3.32**	(1.67, 6.63)
Chest condition diagnosis (No)	Yes	3.72*	(1.29, 10.74)			10.48**	(1.93, 56.83)
Ever diagnosed with asthma or respiratory problems (No)	Yes	5.61***	(3.24, 9.70)	3.67***	(1.80, 7.48)	9.94***	(4.13, 23.94)
Residence in childhood (West upper)							
	East lower	0.79	(0.39, 1.58)	0.84	(0.31, 2.29)	0.81	(0.30, 2.22)
	West lower	0.88	(0.41, 1.90)	1.01	(0.35, 2.92)	1.07	(0.35, 3.26)
	East upper	1.34	(0.69, 2.59)	1.33	(0.57, 3.11)	1.33	(0.46, 3.88)
Index for TSP exposure (≤ median)	> median	0.91	(0.38, 2.16)	0.70	(0.20, 2.43)	1.47	(0.41, 5.34)
Index for SO_2 exposure (≤ median)	> median	0.95	(0.32, 2.84)	1.75	(0.47, 6.54)	0.39	(0.09, 1.73
Index for $PM_{3.3}$ exposure (≤median)	> median	0.99	(0.38, 2.66)	1.07	(0.24, 4.87)	0.65	(0.12, 3.46)
Specificity (%)			72.8		76.9		74.2
Sensitivity (%)			61.2		44.4		67.9
Rho-square			0.13		0.07		0.20

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Table 5.11 Logistic regression models for arthritis or rheumatism.

Variable (Reference)	Classification	Overall sample (n=327)		Females (n = 184)		Males (n = 191)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Residential exposure to dust / gas / contaminants (≤ 5 years)	> 5 years	4.57***	(2.07 to 10.11)	7.24***	(2.42 to 21.67)	5.13*	(1.36, 19.28)
Stressful life events (< 2)	2 or more	3.67*	(1.33 to 10.12)			8.27**	(1.88, 36.32)
Exposure to SO_2 (1983 / 84) (≤ 11.7 ppb)	> 11.7 ppb	2.73*	(1.20 to 6.18)				
Family income in childhood (> low income cut-off)	< low income cut-off	3.84**	(1.60 to 9.25)			5.35*	(1.26, 22.68)
Physical activity (Not regular)	> 30 minutes / > 3 days			4.24**	(1.47 to 12.19)		
Current family income (> low income cut-off)	< low income cut-off			5.57*	(1.21 to 25.71)		
Air conditioner (Own)	Do not own			7.24**	(1.95 to 26.95)		
Index for cough in childhood (Not persistent)	Persistent day / night cough					167.73*	(3.07, 9155.75)
Specificity (%)			76.7		73.5		66.9
Sensitivity (%)			58.8		66.7		84.6
Rho-square			0.16		0.22		0.20

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

ppb in 1983 / 84 (mean age of participants = 13 years) is significant in increasing the odds of diagnosis with arthritis / rheumatism. Individuals exposed to SO_2 above 11.7 ppb in 1983 / 84 are 2.73 times more likely to be diagnosed with arthritis or rheumatism than individuals that were exposed below 11.7 ppb. Results also suggest that residential exposure to dust / gas / contaminants for a duration that exceeds 5 years is associated with increased odds of being diagnosed with arthritis or rheumatism (OR = 4.57). In addition, individuals that experience two or more stressful life events and that are below the low-income cutoff are 3.67 and 3.84 times more likely to be diagnosed with arthritis or rheumatism than individuals that experience less than two stressful life events and that are above the low-income cutoff.

In addressing the biological plausibility of these variables in explaining arthritis or rheumatism, research suggests biologic pathways through which exposure to air pollution induces changes in inflammatory markers in the body (Zeka et al. 2006). In addition, research indicates that psychological stress induces the release of neuroendocrine hormones, which may influence the immune system (Turyk et al. 2008).

When data is disaggregated by sex, results of logistic regression models indicate that residential exposure to dust / gas / contaminants for more than 5 years is significant in increasing the likelihood of diagnosis with arthritis / rheumatism for both males and females (Table 5.11). The logistic regression model for females indicates that individuals that perform regular physical exercise, that are currently below the low-income cutoff, and that have an air conditioner have increased odds of diagnosis with arthritis / rheumatism (OR = 4.24, 5.57, and 7.24 respectively). This is not surprising given that

regular physical exercise and the use of air conditioner to keep the environment at a constant temperature are advisable treatments for people with arthritis or rheumatism (Russell, 2007). For males, significant variables that increase the odds of diagnosis with arthritis or rheumatism include experiencing two or more stressful life events (OR = 8.27), family income in childhood below the low income cut-off (OR = 5.35), and persistent day or night cough in childhood (OR = 167.73). The rho-square values for sex-specific models are greater than 0.2 indicating a good fit. However, the large range of confidence intervals for variables such as index for cough in childhood indicates uncertainty regarding the true effect of explanatory variables.

When variables related to long-term exposure to air pollution and neighbourhood of residence in childhood are forced into the best-fit models, results indicate that exposure to SO_2 above 11.7ppb in 1983 / 84 becomes statistically insignificant in explaining diagnosis with arthritis or rheumatism (Table 5.12). In addition, low income in childhood becomes insignificant in explaining diagnosis with arthritis / rheumatism for males. Although results suggest that long-term exposure to $PM_{3.3}$ decreases the odds of arthritis / rheumatism for females, the rho-square of the new model remains at 0.22. This suggested link between exposure to $PM_{3.3}$ and arthritis / rheumatism is likely attributed to the 5% margin of error in the statistical analyses, but deserves further investigation. Results also indicate that neighbourhood of residence in childhood in relation to the four neighbourhoods and long-term exposure to TSP and SO_2 are insignificant in explaining diagnosis with arthritis or rheumatism.

Table 5.12 Long-term exposure to air pollution in childhood and diagnosis with arthritis or rheumatism.

Variable (Reference)	Classification	Overall sample (n=327)		Females (n = 184)		Males (n = 201)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Residential exposure to dust / gas / contaminants (≤ 5 years)	> 5 years	5.14***	(2.25, 11.73)	7.84***	(2.59, 23.74)	4.82*	(1.33, 17.44)
Stressful life events (< 2)	2 or more	4.20**	(1.49, 11.88)			5.25*	(1.27, 21.70)
Exposure to SO_2 (1983 / 84) (≤ 11.7 ppb)	> 11.7 ppb	3.01	(0.90, 10.04)				
Family income in childhood (> low income cut-off)	< low income cut-off	3.98**	(1.63, 9.68)			3.43	(0.89, 13.25)
Physical activity (Not regular)	> 30 minutes / ≥ 3 days			3.32*	(1.20, 9.20)		
Current family income (> low income cut-off)	< low income cut-off			4.45*	(1.08, 18.40)		
Air conditioner (Own)	Do not own			6.79**	(1.82, 25.41)		
Index for cough in childhood (Not persistent)	Persistent day / night cough					146.74*	(2.36, 9117.50)
Residence in childhood (West upper)							
	East lower	0.79	(0.25, 2.52)	0.84	(0.17, 4.30)	0.85	(0.16, 4.72)
	West lower	0.46	(0.10, 2.12)	1.17	(0.21, 6.56)	0.39	(0.05, 2.97)
	East upper	0.45	(0.13, 1.56)	0.31	(0.08, 1.28)	0.77	(0.08, 7.26)
Index for TSP exposure (≤ median)	> median	2.27	(0.47, 11.00)	4.76	(0.74, 30.71)	0.41	(0.03, 5.53)
Index for $PM_{3.3}$ exposure (≤median)	> median	0.54	(0.06, 4.84)	0.04*	(0.00, 0.53)	9.81	(0.31, 307.78)
Index for SO_2 exposure (≤median)	> median	1.04	(0.09, 11.93)	7.80	(0.75, 80.66)	1.99	(0.16, 24.72)
Specificity (%)			74.1		71.8		76.8
Sensitivity (%)			58.8		68.0		75.0
Rho-square			0.18		0.22		0.21

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

5.4.3 MODELS OF HIGH BLOOD PRESSURE OR HYPERTENSION

Results of the best-fit combined model for diagnosis with high blood pressure / hypertension indicate that exposure to SO_2 above 11.7 ppb in 1983 / 84 (mean age of participants = 13 years) is significant in increasing the odds of diagnosis with high blood pressure / hypertension (Table 5.13). Individuals exposed to SO_2 above 11.7 ppb in 1983 / 84 are 2.82 times more likely to be diagnosed with high blood pressure / hypertension than individuals that were exposed at or below 11.7 ppb. Results also suggest that owning property and a current family income below the low-income cut-off are associated with increased odds of being diagnosed with high blood pressure / hypertension (OR = 3.39 and OR = 4.01 respectively).

When data is disaggregated by sex, results of logistic regression models indicate that body mass index and feelings about income increase the likelihood of diagnosis with high blood pressure / hypertension for females (Table 5.13). Females with a body mass index greater or equal to 25 and that feel mostly dissatisfied / unhappy / terrible about their health are 4.03 and 4.21 times more likely to be diagnosed with high blood pressure / hypertension than females whose body mass index is below 25 and that feel satisfied / happy about their health. For males, significant variables that increase the odds of diagnosis with high blood pressure / hypertension include not owning property (OR = 5.25) and full-time employment (OR = 4.07). For all best-fit models, rho-square values are below 0.2.

When variables related to long-term exposure to air pollution and neighbourhood of residence in childhood are forced into the best-fit models, exposure to SO_2 above

Table 5.13 Logistic regression models for high blood pressure or hypertension.

Variable (Reference)	Classification	Overall sample (n=286)		Females (n = 184)		Males (n = 189)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Property ownership (Do not own)	Own	3.39**	(1.44, 7.98)			5.25**	(1.80, 15.35)
Current family income (> low income cut-off)	< low income cut-off	4.01*	(1.26, 12.74)				
Exposure to SO_2 (1983 / 84) ($\leq$ 11.7 ppb)	> 11.7 ppb	2.82*	(1.23, 6.47)				
Body mass index (< 25)	$\geq$ 25			4.03*	(1.09, 14.92)		
Feelings about income (Score $\geq$ 3)	Score < 3			4.21*	(1.36, 13.04)		
Employment status (Not full-time)	Full-time					4.07*	(1.18, 13.99)
Specificity (%)			46.1		47.6		79.1
Sensitivity (%)			87.1		84.2		64.7
Rho-square			0.11		0.12		0.12

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

11.7ppb in 1983 / 84 remains significant in increasing the odds for high blood pressure / hypertension (Table 5.14). Individuals that are exposed to SO_2 above 11.7 ppb in 1983 / 84 (mean age of participants = 13 years) are 4.24 times more likely to be diagnosed with high blood pressure or hypertension than participants exposed to SO_2 below 11.7 ppb. In addition, full-time employment becomes insignificant in explaining diagnosis with high blood pressure / hypertension for males. Results also indicate that neighbourhood of residence in childhood and long-term exposure to TSP, $PM_{3.3}$, and SO_2 are insignificant in explaining diagnosis with high blood pressure / hypertension. Rho-square values for all model of high blood pressure / hypertension remain below 0.2 indicating a low goodness of fit.

Therefore, while these results suggest that childhood exposure to SO_2 in 1983 / 84 is an explanatory variable for high blood pressure or hypertension, explanatory variables related to current factors interact to impact this health outcome. These explanatory variables are sex-related and include property ownership, family income, body mass index, feelings about income, and employment status.

5.4.4 MODELS OF CHEST CONDITIONS

Results of best-fit models indicate a range of variables that increase the odds of diagnosis with chest conditions. Overall, individuals that did not complete high school, consume alcohol on a regular basis, undertake regular physical exercises, feel dissatisfied / unhappy / terrible about their income, that were exposed to smoking in childhood, and that are diagnosed with chronic bronchitis or arthritis have increased odds of diagnosis

Table 5.14 Long-term exposure to air pollution in childhood and high blood pressure or hypertension.

Variable (Reference)	Classification	Overall sample (n=327)		Females (n = 184)		Males (n = 199)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Property ownership (Own)	Do not own	3.52**	(1.49, 8.30)			6.17**	(2.01, 18.89)
Current family income (> low income cut-off)	< low income cut-off	3.45*	(1.12, 10.69)				
Exposure to SO_2 (1983 / 84) (≤ 11.7 ppb)	> 11.7 ppb	4.24*	(1.19, 15.09)				
Body mass index (< 25)	≥ 25			4.02*	(1.09, 14.84)		
Feelings about income (Score ≥ 3)	Score < 3			3.19*	(1.06, 9.60)		
Employment status (Not full-time)	Full-time					2.25	(0.64, 7.95)
Residence in childhood (West upper)							
	East lower	0.73	(0.20, 2.70)	1.42	(0.24, 8.28)	0.57	(0.11, 3.06)
	West lower	1.32	(0.29, 5.89)	3.13	(0.54, 18.24)	2.89	(0.45, 18.70)
	East upper	1.30	(0.41, 4.15)	1.84	(0.40, 8.54)	0.75	(0.14, 4.15)
Index for TSP exposure (≤ median)	> median	1.78	(0.41, 7.66)	1.58	(0.19, 12.96)	0.46	(0.06, 3.40)
Index for $PM_{3.3}$ exposure (≤median)	> median	0.44	(0.05, 3.62)	0.43	(0.03, 5.68)	0.28	(0.02, 5.15)
Index for SO_2 exposure (≤median)	> median	0.31	(0.02, 3.99)	0.98	(0.11, 8.92)	2.73	(0.24, 30.82)
Specificity (%)			59.4		51.8		73.6
Sensitivity (%)			87.9		71.4		70.6
Rho-square			0.12		0.12		0.12

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

with chest conditions (Table 5.15). The rho-square value of the best-fit model for both sexes combined is 0.31 indicating a good fit. When data is disaggregated by sex, results of logistic regression modeling for females indicate that individuals that did not complete high school and that are diagnosed with chronic bronchitis or arthritis / rheumatism are 11.21, 6.52, and 4.25 times more likely to be diagnosed with chest conditions (Table 5.15). For males, significant variables that increase the odds of diagnosis with chest conditions include regular physical activity, and diagnosis with chronic bronchitis or long-term skin conditions (OR = 6.24, 18.15, and 6.21 respectively). The rho-square value for the best-fit model for females is 0.15, whereas that for males is 0.31 indicating a good fit.

The rho-square values of all model of diagnosis with chest conditions increase when neighbourhood of residence in childhood and the indices of exposure to TSP, $PM_{3.3}$, and SO_2 are forced into the best-fit models (Table 5.16). All models have a rho-square value greater than 0.2. Most significant variables from the best-fit models that explain diagnosis with chest conditions remain significant when neighbourhood of residence and indices of exposure to TSP, $PM_{3.3}$, and SO_2 are forced in the analysis (Table 5.16). However, the large spread in confidence intervals indicate uncertainty in relation to the true effect of explanatory variables. Overall, neighbourhood of residence in childhood and long-term exposure to TSP, $PM_{3.3}$, and SO_2 in childhood are not significant in explaining diagnosis with chest conditions.

Table 5.15 Logistic regression models for diagnosis with chest conditions.

Variable (Reference)	Classification	Overall sample (n=379)		Females (n = 189)		Males (n = 204)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Education (Completed high school)	Did not complete high school	8.38*	(1.27, 55.23)	11.21*	(1.68, 74.83)		
Alcohol consumption (Not regular)	Regular	5.82**	(1.67, 20.33)				
Physical activity (Not regular)	> 30 minutes / > 3 days	3.98*	(1.25, 12.70)			6.24*	(1.03, 37.74)
Feelings about income (Score ≥ 3)	Score < 3	4.40*	(1.34, 14.39)				
Exposure to smoking in childhood (No)	Yes	5.87*	(1.41, 24.25)				
Chronic bronchitis (Not diagnosed)	Diagnosed	21.72***	(6.23, 75.65)	6.52*	(1.36, 31.17)	18.15***	(3.59, 91.72)
Arthritis diagnosis (Not diagnosed)	Diagnosed	4.78*	(1.44, 15.86)	4.25*	(1.05, 17.32)		
Long-term skin conditions (Not diagnosed)	Diagnosed					6.21*	(1.31, 29.41)
Specificity (%)		85.8		80.3		91.8	
Sensitivity (%)		81.0		63.6		70.0	
Rho-square		0.31		0.15		0.31	

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Table 5.16 Long-term exposure to air pollution in childhood and diagnosis with chest conditions.

Variable (Reference)	Classification	Overall sample (n=327)		Females (n = 184)		Males (n = 204)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Education (Completed high school)	Did not complete high school	6.98	(0.90, 54.40)	13.98*	(1.37, 143.06)		
Alcohol consumption (Not regular)	Regular	4.05*	(1.07, 15.16)				
Physical activity (Not regular)	> 30 minutes / > 3 days	3.37*	(1.03, 11.07)			24.00*	(1.33, 434.21)
Feelings about income (Score ≥ 3)	Score < 3	4.87*	(1.42, 16.69)				
Exposure to smoking in childhood (No)	Yes	7.02*	(1.53, 32.26)				
Chronic bronchitis (Not diagnosed)	Diagnosed	24.37***	(6.37, 93.27)	11.50*	(1.81, 72.92)	67.27**	(5.44, 832.46)
Arthritis diagnosis (Not diagnosed)	Diagnosed	6.66**	(1.80, 24.58)	5.28*	(1.17, 23.84)		
Long-term skin conditions (Not diagnosed)	Diagnosed					8.47*	(1.10, 66.67)
Residence in childhood (West upper)							
	East lower	2.89	(0.48, 17.39)	1.40	(0.08, 23.61)	4.04	(0.30, 54.20)
	West lower	2.18	(0.32, 15.08)	3.94	(0.35, 43.87)	0.11	(0.00, 4.84)
	East upper	1.97	(0.35, 11.17)	3.10	(0.36, 26.49)	1.21	(0.05, 30.10)
Index for TSP exposure (≤ median)	> median	0.85	(0.10, 7.59)	1.35	(0.07, 28.15)	0.24	(0.01, 12.59)
Index for $PM_{3.3}$ exposure (≤median)	> median	4.64	(0.26, 81.86)	6.49	(0.13, 326.84)	23.99	(0.13, 4436.18)
Index for SO_2 exposure (≤median)	> median	0.05	(0.00, 1.11)	0.09	(0.00, 2.40)	0.02	(0.00, 1.56)
Specificity (%)			78.7		79.8		82.5
Sensitivity (%)			81.0		72.7		100
Rho-square			0.35		0.24		0.42

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

5.4.5 MODELS OF LONG-TERM SKIN CONDITIONS

Results of logistic regression models indicate that long-term skin conditions are impacted by a range of variables including occupational exposure to gas / dust / fumes for more than 2 years (OR = 1.91), current income below the low income cut-off (OR = 2.68), smoking in childhood (OR = 5.33), asthma incidence in adulthood (OR = 2.11), and diagnosis with chest conditions (OR = 3.61) (Table 5.17). However, the rho-square value of the best-fit model for both sexes combined is 0.08 indicating a poor fit. When data is disaggregated by sex, results of logistic regression modeling for females indicate that individuals exposed to occupational dust / gas / fumes for more than 2 years, that score 4 or less on the group participation scale, that experience 2 or more stressful life events, and that were diagnosed with asthma have increased odds of diagnosis with long-term skin conditions (Table 5.17). In addition, exposure to TSP above 49.5 $\mu g/m^3$ in 1983 / 84 decreases the likelihood of long-term skin conditions. The rho-square value for the female-specific model is 0.21 indicating a good fit. For males, the best-fit model includes only one variable, that is diagnosis with chest conditions (Table 5.17). Individuals that are diagnosed with chest conditions are 7.82 times more likely to be diagnosed with long-term skin conditions. The rho-square value for the male-specific best-fit model is 0.05.

The rho-square values of all models of diagnosis with long-term skin conditions increase when neighbourhood of residence in childhood and the indices of exposure to TSP, $PM_{3.3}$, and SO_2 are forced into the best-fit models (Table 5.18). Occupational exposures, current income, and exposure to TSP become insignificant in explaining this health outcome. In addition, results of combined and female-specific models indicate that

Table 5.17 Logistic regression models and long-term skin conditions.

Variable (Reference)	Classification	Overall sample (n=378)		Females (n = 149)		Males (n = 204)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Occupational exposure to gas / dust / fumes (≤ 2 years)	> 2 years	1.91*	(1.00, 3.64)	11.21*	(1.68, 74.83)		
Current income (Above low-income cutoff)	Below low income cut-off	2.68*	(1.01, 7.14)				
Smoking in childhood (Non smoker)	Smoker	5.33**	(1.43, 18.47)				
Asthma in adulthood only (Non asthmatic)	Asthmatic	2.11*	(1.03, 4.29)				
Chest conditions (Not case)	Case	3.61*	(1.32, 9.83)			7.82**	(2.10, 29.17)
Social networking (> 4 on group participation subscale)	≤ 4 on the Group Participation Subscale			0.21**	(0.08, 0.55)		
Stressful life events (< 2)	≥ 2			6.16**	(1.66, 22.86)		
Ever asthma (Not case)	Case			3.91**	(1.50, 10.17)		
Exposure to TSP (1983 /4)	≥ 49.5 $\mu g/m^3$			0.37*	(0.14, 0.98)		
Specificity (%)			43.8		73.3		97.2
Sensitivity (%)			84.1		79.3		18.5
Rho-square			0.08		0.21		0.05

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Table 5.18 Long-term exposure to air pollution in childhood and long-term skin conditions.

Variable (Reference)	Classification	Overall sample (n=378)		Females (n = 159)		Males (n = 204)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Occupational exposure to gas / dust / fumes (≤ 2 years)	> 2 years	1.82	(0.95, 3.50)				
Current income (Above low-income cutoff)	Below low income cut-off	2.58	(0.96, 6.97)				
Smoking in childhood (Non smoker)	Smoker	5.54**	(1.59, 19.33)				
Asthma in adulthood only (Non asthmatic)	Asthmatic	2.12*	(1.01, 4.42)				
Chest conditions (Not case)	Case	3.37*	(1.21, 9.35)			7.95*	(2.01, 31.44)
Social networking (> 4 on group participation subscale)	≤ 4 Group Participation Subscale			0.22**	(0.08, 0.59)		
Stressful life events (< 2)	≥ 2			4.37*	(1.07, 17.91)		
Ever asthma (Not case)	Case			4.31**	(1.50, 12.38)		
Exposure to TSP (1983 /4)	≥ 49.5 μg/m^3			0.10	(0.01, 1.02)		
Residence in childhood (WU)							
	East lower	2.57*	(1.03, 6.40)	9.22*	(1.70, 50.08)	0.80	(0.23, 2.79)
	West lower	1.15	(0.40, 3.34)	1.56	(0.30, 8.20)	0.72	(0.13, 3.86)
	East upper	1.51	(0.60, 3.78)	4.57	(0.92, 22.75)	0.58	(0.15, 2.29)
Index for TSP exposure (≤ median)	> median	0.68	(0.21, 2.14)	0.51	(0.02, 14.75)	1.95	(0.41, 9.20)
Index for $PM_{3.3}$ exposure (≤median)	> median	1.20	(0.28, 5.14)	20.47*	(1.31, 319.29)	0.13	(0.02, 1.15)
Index for SO_2 exposure (≤median)	> median	0.60	(0.16, 2.60)	0.58	(0.06, 5.87)	2.03	(0.29, 14.38)
Specificity (%)			65.4		72.9		80.2
Sensitivity (%)			68.3		86.7		33.3
Rho-square			0.10		0.27		0.08

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

neighbourhood of residence in childhood plays a role in long-term skin conditions. Individuals that resided in the east lower neighbourhood in childhood are 2.57 times more likely to be diagnosed with long-term skin conditions than individuals that resided in the west upper neighbourhood in childhood. The odds of long-term skin conditions are higher for females that resided in the east lower neighbourhood in childhood compared to those that resided in the west upper neighbourhood (OR = 9.22). In addition, results indicate that females exposed over the long-term to $PM_{3.3}$ above the median in childhood are 20.47 times more likely to be diagnosed with long-term skin conditions than females exposed over the long-term to $PM_{3.3}$ below the median. Only the female-specific model of long-term skin conditions had a rho-square value greater than 0.2 indicating a good fit. The large spread of confidence intervals indicate uncertainty of the true effect of explanatory variables.

5.4.6 MODELS OF FAIR / POOR SELF-ASSESSED HEALTH

Results of best-fit models indicate that a range of variables increase the odds of fair or poor self-assessed health. Overall, individuals exposed to smoking, that have a body mass index equal or above 25, that feel mostly dissatisfied / unhappy / terrible about their income or their life, that have current persistent cough / phlegm / wheeze, and that are diagnosed with arthritis / rheumatism or high blood pressure / hypertension have increased odds of fair or poor self-assessed health (Table 5.19). The rho-square value of the best-fit model for both sexes combined is 0.2 indicating a good fit.

When data is disaggregated by sex, results of the female-specific logistic

Table 5.19 Logistic regression models for fair or poor self-assessed health.

Variable (Reference)	Classification	Overall sample (n=392)		Females (n = 189)		Males (n = 204)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Current exposure to smoke (Not exposed)	Exposed	2.40**	(1.27, 4.56)			1.25	(0.47, 3.34)
BMI (< 25)	≥ 25	3.17**	(1.38, 7.26)	9.03**	(1.73, 47.09)		
Feel about income (Delighted /pleased /mostly satisfied /mixed)	Mostly dissatisfied / unhappy / terrible	2.22*	(1.05, 4.68)			3.02*	(1.05, 8.70)
Feel about life (Delighted /pleased /mostly satisfied /mixed)	Mostly dissatisfied / unhappy / terrible	7.84**	(2.04, 30.18)				
Persistent cough / phlegm / wheeze (No symptoms)	Symptomatic	2.18*	(1.08, 4.42)				
Arthritis/rheumatism (Not case)	Case	3.97**	(1.77, 8.90)	12.37**	(2.97, 51.47)		
High blood pressure / hypertension (Not case)	Case	3.57**	(1.60, 7.98)			0.91	(0.10, 8.35)
Emotional distress (<4 on GHQ)	≥ 4 on the GHQ			10.87**	(2.54, 46.55)	3.65*	(1.16, 11.48)
Persistent day / night cough in childhood (No symptoms)	Symptoms			768.81***	(6.40, 9.24[4])		
Current exposure to smoke x high blood pressure / hypertension (Not exposed x not case)	Exposed x case					20.50*	(1.18, 357.61)
Specificity (%)			72.2		86.7		82.1
Sensitivity (%)			75.0		75.0		51.6
Rho-square			0.20		0.33		0.15

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

regression model indicate that females with a body mass index greater or equal to 25 and that are diagnosed with arthritis / rheumatism are 9.03 and 12.37 times more likely to report fair or poor self-assessed health compared to females that have a body mass index less than 25 and that were not diagnosed with arthritis / rheumatism (Table 5.19). In addition, female participants that scored 4 or more on the General Health Questionnaire (GHQ) and that reported persistent day or night cough in childhood are 10.87 and 768.81 times more likely to report fair or poor self-assessed health than female participants that score less than 4 on the GHQ and that did not report persistent day or night cough in childhood. The rho-square value for the female-specific model is 0.33 indicating a good fit.

For males, feeling mostly dissatisfied / unhappy / terrible about ones income increases the likelihood of fair or poor self-assessed health (Table 5.19). In addition, male participants that are exposed to smoking and that are diagnosed with high blood pressure or hypertension are 20.50 times more likely to report fair or poor self-assessed health compared to individuals that are not exposed to smoking and that were never diagnosed with high blood pressure or hypertension (Table 5.19). Male participants that score 4 or more on the GHQ are 3.65 times more likely to report fair or poor self-assessed health that male participants that score less than 4 on the GHQ. The rho-square value of the male-specific model is 0.15 indicating a poor fit.

The rho-square values of all model of fair or poor self-assessed health increase when neighbourhood of residence in childhood and the indices of exposure to TSP, $PM_{3.3}$, and SO_2 are forced into the best-fit models (Table 5.20). All models that examine

Table 5.20 Long-term exposure to air pollution in childhood and fair or poor self-assessed.

Variable (Reference)	Classification	Overall sample (n=392)		Females (n = 189)		Males (n = 204)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Current exposure to smoke (Not exposed)	Exposed	2.45**	(1.27, 4.73)			1.53	(0.55, 4.30)
BMI (< 25)	≥ 25	3.59**	(1.50, 8.59)	7.92**	(1.99, 31.54)		
Feel about income (Delighted / pleased / mostly satisfied / mixed)	Mostly dissatisfied /unhappy/terrible	2.55*	(1.17, 5.57)			4.23*	(1.37, 13.09)
Feel about life (Delighted / pleased / mostly satisfied / mixed)	Mostly dissatisfied /unhappy/terrible	9.56**	(2.37, 38.58)				
Persistent cough / phlegm / wheeze (No symptoms)	Symptomatic	2.28*	(1.10, 4.75)				
Arthritis / rheumatism (Not case)	Case	4.74***	(2.04, 10.00)	6.64**	(2.12, 20.85)		
High blood pressure / hypertension (Not case)	Case	3.92**	(1.69, 9.12)			1.07	(0.1, 11.65)
Emotional distress (< 4 on GHQ)	≥ 4 on GHQ			6.82**	(2.18, 21.36)	3.90*	(1.09, 13.86)
Persistent day / night cough in childhood (No symptoms)	Symptoms			79.82*	(1.93, 3.31[3])		
Current exposure to smoke x high blood pressure / hypertension (Not exposed x not case)	Exposed x case					14.90	(0.73, 305.67)
Residence in childhood (West upper)							
	East lower	1.57	(0.58, 4.26)	2.37	(0.49, 11.58)	1.51	(0.42, 5.42)
	West lower	0.52	(0.15, 1.77)	0.94	(0.18, 5.05)	0.83	(0.16, 4.42)
	East upper	0.66	(0.25, 1.78)	1.77	(0.42, 7.53)	0.35	(0.08, 1.58)
Index for TSP exposure (≤ median)	> median	2.07	(0.56, 7.67)	0.25	(0.02, 3.05)	3.24	(0.58, 18.07)
Index for $PM_{3.3}$ exposure (≤median)	> median	0.54	(0.11, 2.78)	7.02	(0.41, 120.33)	0.19	(0.02, 1.78)
Index for SO_2 exposure (≤median)	> median	0.20*	(0.04, 0.93)	0.68	(0.08, 6.17)	0.16	(0.02, 1.33)
Specificity (%)			73.2		79.4		75.7
Sensitivity (%)			75.0		79.3		64.5
Rho-square			0.23		0.26		0.22

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

the role of neighbourhood of residence in childhood and long-term exposure to air pollution have a rho-square value greater than 0.2. Most significant variables from the best-fit models that explain fair or poor self-assessed health remain significant when neighbourhood of residence and indices of exposure to TSP, $PM_{3.3}$, and SO_2 are forced in the analysis, except for the interaction between current exposure to smoking and diagnosis with high blood pressure / hypertension (Table 5.20). In addition, results indicate that long-term exposure to SO_2 in childhood is significant in explaining fair or poor self-assessed health.

Specifically, results suggest that exposure to SO_2 decreases the odds of fair or poor self-assessed health. This finding is likely attributed to the 5% margin of error in statistical analyses. Overall, neighbourhood of residence in childhood and long-term exposure to TSP and $PM_{3.3}$ in childhood are not significant in explaining fair or poor self-assessed health. This is surprising given the differences in opportunities and challenges that are attributed to the four neighbourhoods.

In addition to the models discussed in Sections 5.3 and 5.4, logistic regression modeling was performed for diagnosis with any type of cancer, and functional capacity during day-to-day demands and during unexpected or difficult times. Results of the models are included in Appendices 5.1 to 5.6. Childhood exposures were not significant in any of these models. In addition, models for diagnosis with any type of cancer are based on a small number of cases (n = 5).

5.4.7 SUMMARY – CHILDHOOD EXPOSURE TO AIR POLLUTION AND LONG-TERM NON-RESPIRATORY HEALTH

Overall, results indicate that exposure to SO_2 above the median level of 11.7 in 1983 / 84 (mean age of participants = 13 years) may be significant in increasing the likelihood of arthritis / rheumatism and high blood pressure or hypertension. Results also suggest that long-term exposure to $PM_{3.3}$ in childhood may be significant in explaining long-term skin conditions. However, results of female-specific models suggest that long-term exposure to $PM_{3.3}$ may have a protective role in diagnosis for arthritis / rheumatism, and that exposure to TSP above 49.5$\mu g/m^3$ may protect from long-term skin conditions. While the latter suggestion may be due to behavioural patterns that result from the detection of large size particulate matter, the former suggestion is surprising and deserves further investigation.

Results of logistic regression models indicate that neighbourhood of residence in childhood may impact long-term skin conditions. In particular, residing in the east lower neighbourhood is significant in increasing the likelihood of long-term skin conditions. Despite the increase in the rho-square values of most models of non-respiratory health when variables related to long-term exposure to air pollution and the neighbourhood of residence in childhood are forced into the best-fit models, results indicate that neighbourhood of residence in childhood does not play a significant role in diagnosis for hay fever / allergies, arthritis / rheumatism, high blood pressure / hypertension, chest conditions, fair or poor self-assessed health, and fair or poor functional capacity.

Significant variables that explain long-term non-respiratory health outcomes also include family income, property ownership, employment status, exposure to smoking, physiological (body mass index, persistent day or night cough, and asthma in childhood) and behavioural (smoking in childhood and adulthood, regular alcohol consumption, and regular physical activity) factors, psychological (emotional distress and feelings about income and life) factors, residential exposures to gas / dust / contaminants (greater than 5 years) and occupational exposures to gas / dust / fumes (greater than 2 years), residential history, social environment (group participation, and social subscale), and stressful life events. Analysis suggests that healthcare access does not play a significant role in long-term non-respiratory health outcomes.

5.5 SUMMARY

Guided by the conceptual framework, logistic regression modeling was performed to explain respiratory and non-respiratory health outcomes and to examine the relative roles that exposure to air pollution in childhood and neighbourhood of residence in childhood play in long-term health. Tables 5.21, 5.22, and 5.23 present a summary of explanatory variables for long-term respiratory and non-respiratory health outcomes respectively. A number of issues arise from the results.

First, neighbourhood of residence in childhood may be significant in explaining long-term health outcomes, mainly hospital or ER visits for asthma or respiratory health since leaving elementary school and long-term skin conditions. Participants that resided

Table 5.21 Summary of statistically significant explanatory variables of long-term respiratory health.

SIGNIFICANT VARIABLES		LONG TERM RESPIRATORY HEALTH OUTCOMES											
		Ever diagnosed with asthma or respiratory problems			Asthma incidence in adulthood			Hospital or ER visits for asthma /respiratory since elementary school			Chronic Bronchitis		
		C	F	M	C	F	M	C	F	M	C	F	M
Exposure to air pollution	TSP (1980 / 81)										X^	X^	X^
	TSP (1985 / 86)							X^				X^	
	SO_2 in 1983 / 84		X		X	X			X				
	PM3.3 (1985 / 86)												X
Residential history	Rare use of air filter											X	
	Any residential exposure								X				
	Exposure > 8 years	X		X			X			X			
	Reside outside Ontario									X			
Occupational	Exposure > 2 years			X									
Family environment	No parental record of asthma or respiratory problems	X		X			X						
Life events	2 or more stressful events							X	X			X	
Physiological	Sex				X								
	Childhood asthma	X		X									
	Lung function (childhood)							X	X				
	Chest cold in childhood	X											
	Persistent morning cough	X	X										
	Chest illness in childhood											X	
	Ever asthma or respiratory problems							X	X	X			
Behavioural	Smoker in childhood								X				
	Ever daily smoker										X		
	Current smoker											X	

Table 5.21 continued

SIGNIFICANT VARIABLES		LONG TERM RESPIRATORY HEALTH OUTCOMES											
		Ever diagnosed with asthma or respiratory problems			Asthma incidence in adulthood			Hospital or ER visits for asthma / respiratory since elementary school.			Chronic bronchitis		
		C	F	M	C	F	M	C	F	M	C	F	M
	Feeling about health				X	X							
	Emotional distress										X		
Neighbourhood of residence in childhood								X					
Long-term exposure to	TSP												
	$PM_{3.3}$												
	SO_2							X					
Best-fit model	Specificity	74.3	47.7	70.4	48.6	52.5	65.4	91.0	94.5	89.4	100.0	88.1	65.6
	Sensitivity	56.2	74.4	82.9	79.1	75.0	68.8	63.3	75.0	70.0	0	75.0	70.2
	Rho-square	0.13	0.07	0.33	0.10	0.10	0.11	0.35	0.43	0.41	0.10	0.44	0.16
Model with air pollution indices	Specificity	74.8	55.0	79.6	49.4	65.0	78.8	91.3	94.5	88.8	67.7	85.6	63.7
	Sensitivity	57.3	65.9	80.5	76.7	56.3	62.5	63.3	81.3	80.0	75.0	83.3	83.3
	Rho-square	0.15	0.09	0.37	0.12	0.12	0.19	0.37	0.49	0.48	0.14	0.56	0.23

C – Combined sexes; F – Female; M – Male; ^ protective role.

Table 5.22 Summary of statistically significant explanatory variables of long-term non-respiratory health outcomes.

SIGNIFICANT VARIABLES		LONG-TERM NON-RESPIRATORY HEALTH OUTCOMES																
		Hay fever / allergies			Arthritis / rheumatism			High blood pressure / hypertension			Chest conditions			Long-term skin conditions			Any type of cancer	
		C	F	M	C	F	M	C	F	M	C	F	M	C	F	M	C	F
Exposure to air pollution	TSP (1983/4)														X^			
	SO_2 in 1983/4				X			X										
	Exposure to smoking in childhood										X							
Residential history	Own air condition					X												
	Exposure > 5 years				X	X	X											
	Exposure > 2 years																X	
	Resided anywhere outside Hamilton	X		X														
Occupation history	Exposure > 2 years													X	X			
	Length of exposure																X	X
Social	≤ 4 Group Participation Scale														X			
Demographic factors	< Low income cut-off					X		X						X				
	< Low income cut-off (childhood)				X		X											
	Not own property							X		X								
	Employed full-time									X								
	Did not complete high school										X	X						
Life events	> 2 stressful events				X		X								X			

Table 5.22 continued

SIGNIFICANT VARIABLES		Hay fever / allergies			Arthritis / rheumatism			High blood pressure			Chest conditions			Skin conditions			Cancer (Any type)	
		C	F	M	C	F	M	C	F	M	C	F	M	C	F	M	C	F
Physiologic	Persistent day/night cough (childhood)						X											
	Ever asthma or respiratory problems	X	X	X											X			
	Asthma (adulthood)													X				
	Chest conditions	X		X										X		X		
	Chronic bronchitis										X	X	X					
	Arthritis										X	X						
	Skin conditions												X					
	BMI ≥ 25								X									
Behavioural	Smoker (childhood)													X				
	Regular physical activity					X					X		X					
	Alcohol consumption (regular)										X							
Psychological	Feelings (income)								X		X							
Neighbourhood in childhood														X	X			
Long-term exposure to	TSP																	
	$PM_{3.3}$					X^									X			
	SO_2																	
Best-fit model	Specificity	60.1	84.6	62.6	76.7	73.5	66.9	46.1	47.6	79.1	85.8	80.3	91.8	43.8	73.3	97.2	98.3	98
	Sensitivity	73.1	40.0	78.0	58.8	66.7	84.6	87.1	84.2	64.7	81.0	63.6	70.0	84.1	79.3	18.5	0	33.3
	Rho-square	0.12	0.07	0.19	0.16	0.22	0.20	0.11	012	012	0.31	0.15	0.31	0.08	0.21	0.05	0.30	0.31
Model-air pollution indices	Specificity	72.8	76.9	74.2	74.1	71.8	76.8	59.4	51.8	73.6	78.8	79.8	82.5	65.4	72.9	80.2	98.6	98.8
	Sensitivity	61.2	44.4	67.9	58.8	68.0	75.0	87.9	71.4	70.6	81.0	72.7	100	68.3	86.7	33.3	75.0	100
	Rho-square	0.13	0.07	0.20	0.18	0.22	0.21	0.12	0.12	0.12	0.35	0.24	0.42	0.10	0.27	0.08	0.59	0.76

C – Combined sexes; F – Female; M – Male; ^ protective role.

Table 5.23 Summary of significant explanatory variables of fair or poor self-assessed health and functional capacity.

SIGNIFICANT VARIABLES		SELF-ASSESSED HEALTH AND FUNCTIONAL CAPACITY								
		Fair or poor self-assessed health			Fair or poor ability to handle day-to-day demands			Fair or poor ability to handle difficult or unexpected problems		
		C	F	M	C	F	M	C	F	M
Physical exposures	Current exposure to smoke	X								X
Residential history	Reside outside Hamilton but in Ontario				X					
Social environment	≤ 5 on the Social Contact Subscale							X		
Demographic factors	Did not complete high school							X	X	
Family environment	Single versus married							X		X
Physiological	Persistent day / night cough in childhood		X							
	Current persistent cough / phlegm / wheeze	X								
	Diagnosed with chest conditions							X		X
	Diagnosed with arthritis	X	X							
	Diagnosed with heart disease				X		X			
	Diagnosed with high blood pressure	X								
	BMI ≥ 25	X	X							
Psychological	Feeling about life	X								
	Feeling about income	X		X	X	X				
	Emotional distress		X	X	X	X	X			
Current exposure to smoke x high blood pressure				X						
Neighbourhood of residence in childhood										
Long-term exposure to	TSP									
	$PM_{3.3}$									
	SO_2	X^								
Best-fit model	Specificity	72.2	86.7	82.1	91.1	82.9	91.6	68.9	0	48.5
	Sensitivity	75.0	75.0	51.6	45.5	55.0	44.0	60.3	100	88.6
	Rho-square	0.20	0.33	0.15	0.16	0.10	0.13	0.09	0.02	0.11
Model with air pollution indices	Specificity	73.2	79.4	75.7	85.8	87.0	91.1	67.8	52.5	63.9
	Sensitivity	75.0	79.3	64.5	53.3	55.0	44.0	57.6	64.5	82.9
	Rho-square	0.23	0.26	0.22	0.15	0.12	0.15	0.08	0.06	0.14

C – Combined sexes; F – Female; M – Male; ^ protective role.

in the east lower neighbourhood are most likely to report these health outcomes, whereas participants that resided in the west upper neighbourhood are least likely to report hospital or emergency room visits for asthma or respiratory problems since leaving elementary school or long-term skin conditions. While results indicate that neighbourhood of residence in childhood does not play a significant role in other examined health outcomes, the increase in rho-square values when the variables related to childhood exposure to air pollution and neighbourhood of residence in childhood are forced in best-fit models of respiratory and non-respiratory health outcomes suggests that there may be factors at the neighbourhood level that impact long-term health and that were not captured in the analysis. Examples of factors that may be impacted by inequity in opportunities and resource distribution include collective social functioning and practices such as the degree of community integration, and the effect of perceived areas on the self-esteem and morale of residents (Macintyre et al. 2002).

Second, results suggest that exposure to air pollution in childhood is possibly significant in explaining respiratory and non-respiratory health in adulthood. In exploring the contribution of air pollution to long-term respiratory health, results of logistic regression models suggest that exposure to SO_2 above 11.7 ppb when the mean age of participants was 13 years, is significant in explaining asthma incidence in adulthood, ever asthma or respiratory problems for females, and hospital or ER visits for asthma or respiratory problems since leaving elementary school. In addition, results suggest that long-term exposure to SO_2 in childhood may impact hospital or ER visits for asthma or respiratory problems since leaving elementary school. These findings contribute to

evidence that suggests that sensitization to allergens at a young age is a significant factor in predicting asthma incidence in the future (Almqvist et al. 2007; Schafer et al. 2007). The study undertaken by Almqvist et al. (2007) found that young children with a family history of asthma are more likely to develop asthma at the age of five when sensitized to allergens at the age of 18 months. Similarly, Schafer et al. (2007) found that sensitization to cat and grass pollen measured at 9 years of age is a predictor of asthma and hay fever in young adults.

Results of sex-specific models suggest that the long-term health impacts of childhood exposure to air pollution may be sex-related. Exposure to SO_2 in childhood (above 11.7 ppb in 1983 / 94) explains respiratory health outcomes for females but not males. For females, exposure to SO_2 may be significant in explaining diagnosis with asthma or respiratory problems, asthma incidence in adulthood, and hospital or ER visits for asthma or respiratory problems since leaving elementary school. These findings require further investigations into the biological mechanisms in females that make them more susceptible to exposure to SO_2 around the age of 13 years. For males, however, exposure to $PM_{3.3}$ above 43.2$\mu g/m^3$ when the mean age of participants is 15 years is likely to increase the likelihood of chronic bronchitis. Findings also suggest that exposure to TSP above the median level when the mean age of participants is 9 years may be protective against chronic bronchitis for both males and females. However, exposure to TSP above the median level when the mean age of participants is 15 years may be protective against chronic bronchitis for females only. These findings are interesting given research that suggests that males are genetically more susceptible to particulate

matter than females during acute air pollution episodes and when the health outcome measured is mortality (Mage and Kreszschmar 2000). Although genetic susceptibility may be a factor, the long-term health impacts of childhood exposure to particulate matter for males may also be due to less preventative behavioural practices that young adolescent males undertake when they detect air pollution or when they experience respiratory symptoms such as coughing or runny nose that are associated with exposure to air pollution. This in turn impacts long-term respiratory health.

In terms of non-respiratory health outcomes, results of logistic regression modeling suggest that exposure to SO_2 may be related to arthritis or rheumatism, and high blood pressure or hypertension. Participants exposed to SO_2 (above 11.7 ppb in 1983 / 84) are more likely to be diagnosed with arthritis or rheumatism, and high blood pressure or hypertension. Although long-term exposure to $PM_{3.3}$ in childhood was significant in explaining long-term skin conditions for females, results also imply a protective role for long-term exposure to $PM_{3.3}$ and arthritis or rheumatism for females. These results deserve further investigation.

Thirdly, the variables retained in the models of long-term health outcomes span the range of factors included in the conceptual model developed to guide this research (Figure 2.2). For long-term respiratory health outcomes, significant explanatory variables include residential and occupational factors such as residing outside Ontario, rarely using air filters, and residential / occupational exposures to gas / dust / contaminants. In addition, experiencing stressful life events and having no parental record of asthma or respiratory problems increase the likelihood of respiratory health outcomes. The latter

factor disagrees with recent research that suggests that individuals (both males and females) with a genetic record of asthma (measured by the number of relatives with life-events of asthma) are more likely to develop asthma in young adulthood (Grassi et al. 2006). Results also indicate that physiological factors play a significant role in long-term respiratory health. In particular, odds of long-term respiratory health outcomes increase for females, participants diagnosed with asthma in childhood, and those that experience low lung function, chest colds / illnesses, and persistent morning cough in childhood. These results are supported by findings from the literature that emphasize the link between physiological pathways and asthma. For example, Toelle et al. (2004) found that childhood characteristics (airway obstruction, airway hyper-responsiveness, atopy, recent wheeze, and being female) predict asthma symptoms in adulthood. Behavioral and psychological factors also play a role in long-term respiratory health. In particular, smoking in childhood or adulthood as well as emotional distress increase the likelihood of respiratory health outcomes.

For long-term non-respiratory health outcomes, logistic regression modeling results indicate that significant variables span the range of factors included in the conceptual model. Residential and occupational histories may play a significant role in long-term non-respiratory health outcomes, such as hay fever / allergies, arthritis / rheumatism, long-term skin conditions, cancer and functional capacity. Overall, results suggest that the role of residential exposures in arthritis / rheumatism and hay fever / allergies are significant. For cancer and long-term skin conditions, occupational exposures are significant. Furthermore, participants that are exposure to second-hand

smoking at home or on the job are more likely to report fair or poor self-assessed health than participants that are not exposed to second-hand smoking.

Results also suggest that the social environment and demographic factors may play a role in long-term non-respiratory health. Significant factors include group participation, income in adulthood and childhood, property ownership, and education. Significant factors from the family environment that affect long-term non-respiratory health include marital status that impacts functional capacity in handling difficult or unexpected problems. Results also suggest that experiencing two or more stressful life events may play a significant role in arthritis / rheumatism and long-term skin conditions.

Significant predictors retained in the models emphasize the roles of physiological, behavioral, and psychological factors in long-term non-respiratory health outcomes. In terms of physiological factors, significant factors from childhood include persistent day or night cough. In adulthood, significant factors include a body mass index greater or equal to 25, current respiratory symptoms, and medical diagnosis. In particular, diagnosis with chest conditions is significant in hay fever / allergies and each of chronic bronchitis and arthritis / rheumatism are significant explanatory factors for diagnosis with chest conditions. Self-assessed health and functional capacity are possibly impacted by diagnosis with chest conditions, arthritis / rheumatism, heart disease, and high blood pressure / hypertension. In terms of behavioral factors, significant factors include smoking in childhood, regular physical activity, and regular alcohol consumption. Significant explanatory variables of long-term non-respiratory health that are retained in

the models also include psychological factors, such as feelings about income, life, and emotional distress.

Overall, results of logistic regression modeling suggest that a combination of factors interact to impact long-term health. Despite the gradient in air pollution levels across neighbourhoods, results of aggregated neighbourhood level analysis indicate that the health status of participants was relatively consistent across neighbourhoods. However, at the individual level, multi-variate analysis led to an increase in the rho-square values of models of health outcomes when indices of air pollution and neighbourhood of residence in childhood were forced into the best-fit models. Although results of logistic regression models indicate that the role of neighbourhood of residence in childhood is not significant in impacting long-term non-respiratory health, the increase in rho-square values hints to a neighbourhood-level effect that was not captured in this research, possibly related to collective social functioning and practices. In relation to childhood exposure to air pollution, overall results suggest that exposure to SO_2 in early puberty may impact long-term respiratory health outcomes (ever asthma or respiratory problems, asthma incidence in adulthood, and hospital or ER visits for asthma or respiratory problems since leaving elementary school) for females and non-respiratory health outcomes (arthritis / rheumatism and high blood pressure / hypertension) for both sexes combined. Exposure to TSP in childhood, however, may be protective against long-term respiratory health outcomes, likely due to behavioral practices that limit time spent outdoors. Factors related to the macro-environment (exposure to smoking, residential and occupational history, social and family environment, and life events) and the micro-

environment (physiological, behavioral, and psychological) are significant in explaining long-term respiratory and non-respiratory health outcomes.

CHAPTER SIX

CONCLUSIONS

6.1 INTRODUCTION

This thesis examined the relationship between childhood exposure to air pollution and long-term health. Several issues defined the scope of the research. First, the health effects of air pollution remain an issue of concern for policy-makers, scientists, and the general public. Second, the health impacts of childhood exposure to air pollution is of particular concern for the following reasons: i) children spend a lot of time outdoors, and ii) children have relatively smaller lungs that require a large intake of inhaled air, which is likely to increase the dose of pollutants (Neidell, 2004). Most research on the health effects of childhood exposure to air pollution tends to focus on short-term health outcomes. However, health outcomes of exposure to air pollution may require long latent periods before they manifest themselves (Schwartz, 2004). In light of these issues, there is a need to examine the long-term health effects of childhood exposure to air pollution.

This project responds to this need by examining the link between childhood exposure to air pollution and long-term health for a reconstructed cohort of 395 participants that resided in four different neighbourhoods in Hamilton, Ontario during childhood. These neighbourhoods exhibited a gradient in air pollution levels. Childhood exposure data was collected for total suspended particulates (TSP), particulate matter under 3.3μm in diameter ($PM_{3.3}$), and sulfur dioxide (SO_2) between 1976 and 1986. In

addition, data collected in childhood include socio-economic and demographic measures, respiratory symptoms, and lung function. Results of data analysis from childhood indicate differences in the distribution of covariates across neighbourhoods such as parental smoking and gas cooking. In addition to data from childhood, a questionnaire administered on the reconstructed cohort almost twenty years later collected data on a range of health measures including respiratory and non-respiratory health outcomes. Furthermore, guided by the Life Course Health Development framework, data was collected on various macro-environmental (residential and occupational histories, social and family environments, life events, healthcare access, and current demographic factors) and micro-environmental (physiological, behavioral, and psychological) factors that impact health. In doing so, the following objectives were addressed:

1. to document the health status of the reconstructed cohort with a particular emphasis on respiratory health;
2. to assess the potential relationship between childhood exposure to air pollution and adult respiratory health; and
3. to explore factors mediating this relationship.

6.2 SUMMARY OF FINDINGS

Results indicate significant differences across neighbourhoods for exposures to air pollution and smoking in childhood, residential history, social networking, current smoking, concern over air pollution, and having a body mass index equal or greater than

25. Air pollution levels were highest in the east lower neighbourhood followed by the west lower neighbourhood (Kerigan et al. 1986). The east lower neighbourhood also had the highest prevalence of exposure to smoking in childhood, current smoking, and having a body mass index equal or greater than 25. The east upper neighbourhood had the highest prevalence of always residing in Hamilton and concern over air pollution. Participants that resided in the west lower neighbourhood in childhood had the highest prevalence of residing in a property built before 1950 and scoring 5 or less on the social contact subscale.

6.2.1 OBJECTIVE 1: HEALTH STATUS OF THE RECONSTRUCTED COHORT

In comparison to the Canadian population, the reconstructed cohort had higher prevalence of fair or poor self-assessed health (15% versus 11%), asthma (20% versus 8%), high blood pressure / hypertension (10% versus 7%), arthritis / rheumatism (10% versus 9%), chronic bronchitis (7% versus 4%), and hay fever / allergies (42% versus 18%). Lower prevalence was calculated for asthma in childhood (11% versus 12%), cancer (1% versus 2%), and heart disease (1% versus 3%). Results also indicate that 13% of participants were diagnosed with asthma in adulthood but not in childhood and that 9% had hospital or ER visits for asthma or respiratory problems since leaving elementary school.

While differences in health status across neighbourhoods are not significant, participants that resided in the east lower neighbourhood in childhood had higher prevalence of current persistent morning cough (12%), persistent day / night cough (9%),

and persistent phlegm (7%) than participants that resided in the other three neighbourhoods in childhood (8%, 8%, and 6%) (Table 4.6). Only one significant difference across neighbourhoods was found for hospital or ER visits for asthma or respiratory problems since leaving elementary school. 15% of participants that resided in the east lower neighbourhood in childhood had hospital or ER visits for asthma or respiratory problems since leaving elementary school compared to 7% for participants that resided in the other three neighbourhoods. In terms of asthma diagnosis, prevalence of asthma in adulthood but not in childhood was highest for participants that resided in the west upper neighbourhood in childhood (16%), followed by participants that resided in the east lower neighbourhood in childhood (15%).

In relation to non-respiratory health outcomes, participants that resided in the east lower neighbourhood in childhood had higher prevalence of fair or poor self-assessed health (19%), cancer (3%), and arthritis (12%) than participants that resided in the other three neighbourhoods in childhood (13%, 1%, and 10 % respectively). In comparison, the prevalence of fair or poor self-assessed health in Hamilton is 14%, and the prevalence rates of cancer and arthritis in the Canadian population are 2% and 9% respectively (Statistics Canada, 2006). Participants that resided in the east upper neighbourhood in childhood had the highest prevalence of chronic bronchitis (11%) and hay fever / allergies, whereas participants that resided in the west lower neighbourhood in childhood had the highest prevalence of high blood pressure / hypertension (13%) and heart disease (3%).

Overall, results suggest that despite significant differences in childhood exposure to air pollution and the macro- and micro environmental profiles across neighbourhoods, there are no significant differences in the health status of participants across neighbourhoods. While these results suggest that equity and access to resources in childhood do not impact long-term health, it is important to point to the relatively high socio-economic profile of the cohort that may have resulted from loss to follow-up of participants of lower socio-economic status that experience poor health. For instance, only 10% of children of the original cohort that resided in the east lower neighbourhood are participants in this research, compared to 12%, 13%, and 15% of children that resided in the west lower, east upper, and west upper neighbourhoods. The relatively uniform health status across neighbourhoods may in fact be attributed to loss to follow-up of participants that experience poor health. Therefore, at the neighbourhood level, the absence of a gradient in health status across neighbourhoods presents a limitation for examining the effects of inequity and access to resources in childhood on long-term health. However, results of multi-variate analysis may indicate whether neighbourhood of residence in childhood has an effect on long-term health.

6.2.2 OBJECTIVE 2: RELATIONSHIP BETWEEN CHILDHOOD EXPOSURE TO AIR POLLUTION AND ADULT RESPIRATORY HEALTH

In addressing the second objective, overall analyses indicate that childhood exposure to SO_2 may be associated with long-term respiratory health, mainly asthma incidence in adulthood and hospital or emergency room visits for asthma or respiratory

problems since leaving elementary school (Table 4.6). Results also indicate that childhood exposure to SO_2 may be associated with non-respiratory health such as high blood pressure and arthritis / rheumatism. However, in examining these relationships, results indicate that these associations may be sex-related. For females, exposure to SO_2 in childhood above the median level of 11.7 ppb may be related to asthma or respiratory illness in adulthood, asthma incidence in adulthood, and high blood pressure / hypertension (Table 4.6). In addition, for females, analysis of variance led to significant associations between the following variables: i) exposure to SO_2 in 1981 / 82 (median of 9.6 ppb) and in 1983 /84 (median of 11.7 ppb) and hospital or ER visits for asthma or respiratory problems since leaving elementary school; ii) exposure to SO_2 in 1978 / 79 (median of 6.8 ppb) and hay fever / allergies; exposure to SO_2 in 1981 / 82 (median of 9.7 ppb) and high blood pressure or hypertension; and exposure to $PM_{3.3}$ in 1980 / 81(median of 46 $\mu g/m^3$) and diagnosis with chest conditions (Table 4.8).

For males, childhood exposures to TSP, $PM_{3.3}$, and SO_2 in childhood are not likely related to any respiratory health outcomes. However, in terms of non-respiratory health outcomes, diagnosis for arthritis / rheumatism may be associated with each of exposure to SO_2 in 1978 / 79 (median of 6.8 ppb) and exposure to $PM_{3.3}$ in 1985 / 86 (median of 43.2$\mu g/m^3$) (Table 4.8). Furthermore, exposure to TSP in 1985 / 86 (median of 45.7 $\mu g/m^3$) may be associated with diagnosis with long-term skin conditions.

Results that address the second objective contribute to evidence that links childhood exposure to SO_2 with respiratory health. A review of the literature discussed in section 2.4 suggests that exposure to SO_2 is related to low lung function and respiratory

illnesses in childhood, and that childhood respiratory symptoms are likely attributed to exposure to particulate matter. For instance, a study based in Germany found that exposure to SO_2 is associated with infectious airway diseases and acute respiratory morbidity (Kramer et al. 1999). In addition, the original research conducted by Pengelly et al. (1989) found that airway obstruction increased for asthmatic children exposed to SO_2 levels above 10.6 ppb. This research adds to these findings by suggesting that childhood exposure to SO_2 may also impact long-term health. However, despite research that suggests a link between childhood exposure to particulate matter and respiratory health in childhood (Peters et al. 1999; Gauderman et al. 2000; Hruba et al. 2001), results of bi-variate analysis do not indicate a link between childhood exposure to particulate matter and long-term respiratory health. In addition, results of bi-variate analysis that point to sex-related differences agree with literature that indicates stronger associations between childhood exposure to air pollution and respiratory symptoms (Yu et al. 2001) and pulmonary lung function (Gauderman et al. 2002) for females than males.

In examining bi-variate relationships between exposure to air pollution and non-respiratory health outcomes, significant sex-related associations were found between exposure to SO_2 and arthritis / rheumatism. These results are significant given recent research that focuses on the relationship between air pollutants and inflammatory diseases. For instance, Schwarz et al. (2006) found that an increase in exposure to PM_{10} is associated with increased mortality by 22% for people with inflammatory disease such as rheumatoid arthritis. While research undertaken by Swartz et al. (2006) focused on the health impacts of exposure to PM_{10}, recent research has also focused on biological

mechanisms that link exposure to air pollutants including SO_2 or inhalation of foreign particles (such as those related to smoking) to inflammation (Blomberg, 2000; Van Eden and Hogg, 2002; Klareskog et al. 2007). Clinical research suggests that exposure to air pollution is related to changes in inflammatory biomarkers in the body (Zeka et al. 2006), in the respiratory system (Baeza-Squiban et al. 1999; MacNee and Donaldson, 2003), and in the brain (Campbell et al. 2005; Calderon-Garciduenas et al. 2003; Calderon-Garciduenas et al. 2004). However, data collected for this research is self-reported and limited in its capacity to examine biological effects of childhood exposure to air pollution. Therefore, in order to move research on air pollution and health forward, future studies should focus on collecting clinical data from the reconstructed cohort that investigates relationships between childhood exposure to air pollution and biological markers.

Consistent with the LCHD framework, results of bi-variate analyses suggest that factors related to the macro and micro-environments impact long-term health. With these findings, the stage is set for addressing the third objective of this research, that is to explore various factors that mediate the relationships between childhood exposure to air pollution and respiratory health.

6.2.3 OBJECTIVE 3: FACTORS THAT MEDIATE THE RELATIONSHIP BETWEEN CHILDHOOD EXPOSURE TO AIR POLLUTION AND ADULT RESPIRATORY HEALTH

This objective was addressed using multi-variate analysis that consisted of modeling respiratory and non-respiratory health outcomes. In terms of respiratory health outcomes, models of ever diagnosed with asthma or respiratory problems for males, hospital or ER visits for asthma or respiratory problems for combined as well as sex-specific models, and chronic bronchitis for females had a rho-square value greater than 0.2 indicating a good fit.

For all long-term respiratory health outcomes, rho-square values of models increase when the indices of exposure to air pollution and neighbourhood of residence in childhood are forced into the best-fit models. While neighbourhood of residence in childhood was in most cases not a significant explanatory variable of long-term respiratory health outcomes, the increase in rho-square values suggests that there is a neighbourhood-level health effect that may have contributed to long-term health outcomes. This effect may be related to factors related to collective social behaviours and practices that are likely to result from differences in access to resources and opportunities at the neighbourhood level (Macintyre et al. 2002). Only the logistic regression model of hospital or ER visits for asthma or respiratory problems since leaving elementary school suggests that neighbourhood of residence in childhood may play a significant role in long-term respiratory health.

In relation to childhood exposure to air pollution, results of logistic regression models of respiratory health outcomes suggest that females exposed to SO_2 above 11.7 ppb in 1983 / 84 (mean age of participants = 13 years) are more likely to ever be diagnosed with asthma or respiratory problems and asthma in adulthood. However, the rho-square value of logistic regression models of ever asthma or respiratory problems and asthma incidence for females are less than 0.2 indicating the presence of other factors not included in the analysis that play a significant role in the development of these health outcomes. In addition, logistic regression models of hospital or ER visits for asthma or respiratory health suggest that females exposed to SO_2 above 11.7 ppb in 1983 / 84 (mean age of participants = 13 years) are more likely to have hospital or ER visits for asthma or respiratory problems since leaving elementary school. Results suggest that long-term exposure to SO_2 in childhood may impact hospital or ER visits for asthma or respiratory problems for both sexes combined.

Results of logistic regression models of chronic bronchitis indicate that exposure to $PM_{3.3}$ in 1985 / 86 may increase the likelihood of chronic bronchitis for males. However, exposure to TSP in 1980 / 81 may be protective against chronic bronchitis for both sexes combined and for males, and exposure to TSP in 1985 / 86 may be protective against chronic bronchitis for females. For both males and females, the rho-square values are greater than 0.2 indicating a good fit. Overall these findings suggest that exposure to high levels of TSP may promote gender-related behavioral practices that limit time spent outdoors when high air pollution levels are detected.

Consistent with the conceptual framework that guides this research, significant factors that affect long-term respiratory health include residential and occupational exposures, residential location, rare use of air filters, no parental record of asthma or respiratory problems, stressful life events, as well as physiological (lung function, chest colds, persistent morning cough in childhood, and asthma in childhood), behavioral (smoking), and psychological (emotional distress and feelings about one's health) factors.

In terms of logistic regression modeling of non-respiratory health outcomes, variables retained in the models span the range of factors included in the conceptual framework that guides this research. Models of non-respiratory health outcomes that have a rho-square value greater than 0.2 include sex-specific models of arthritis / rheumatism, combined model of chest conditions, male model of chest conditions, models of cancer, and fair or poor self-assessed health. For all long-term non-respiratory health outcomes, rho-square values of models increase when the indices of exposure to air pollution and neighbourhood of residence in childhood are forced into the best-fit models. This suggests that factors at the neighbourhood-level may play a role in long-term non-respiratory health. Results of logistic regression modeling indicate that neighbourhood of residence in childhood likely increases the odds of long-term skin conditions for both sexes combined and for females.

In examining the relationship between childhood exposure to air pollution and non-respiratory health, results indicate that exposure to SO_2 above 11.7 ppb when the mean age of participants is 13 years may increase the odds of arthritis / rheumatism and high blood pressure / hypertension. Significant factors that mediate the relationship

between childhood exposure to air pollution and long-term non-respiratory health include residential exposures, owning an air conditioner, family income in childhood and adulthood, property ownership, employment status, experiencing stressful life events, persistent day / night cough in childhood, having a body mass index greater or equal to 25, regular physical exercise, and feeling's about one's income. In addition, results of multi-variate analysis suggest that childhood exposure to air pollution does not play a significant role in fair or poor self-assessed health and fair or poor functional capacity. This is surprising given that significant differences in exposure to air pollution is likely to impact health perceptions (Elliott et al. 1999). Factors that contribute to fair / poor self-assessed health include second-hand smoking, persistent day / night cough in childhood, current respiratory symptoms, diagnosis with arthritis / rheumatism or high blood pressure / hypertension, having a body mass index equal or greater than 25, feelings about life or income, and scoring less than 4 on the GHQ. Fair or poor ability to handle day-to-day demands is impacted by residential location, diagnosis with heart disease, feelings about one's income, and scoring 4 or more on the GHQ. Fair or poor ability to handle unexpected or difficult problems is impacted by current exposure to smoking, social networking, education, single status, and diagnosis with chest conditions.

Overall, the analyses in this research indicate that a combination of factors related to the macro-environment and micro-environment contribute to long-term health. Results suggest that exposure to SO_2 in childhood may play a significant role in the development of long-term respiratory and non-respiratory health outcomes. Specifically, results suggest that long-term exposure to SO_2 in childhood may impact hospital or ER visits for

asthma or respiratory problems. Exposure to SO_2 around the age of 13 years may also impact asthma incidence in adulthood and diagnosis with asthma or respiratory problems for females. In terms of long-term non-respiratory health outcomes, results suggest that exposure to SO_2 around the age of 13 years may impact arthritis / rheumatism and high blood pressure / hypertension.

6.3 CONTRIBUTIONS OF THE RESEARCH

This longitudinal research consists of a follow-up period of approximately 25 years. Relative to longitudinal studies that focus on the health impacts of air pollution, the length of this follow-up period is unique and allows for an investigation of the long-term health impacts of childhood exposure to air pollution. Thus, this research marks a beginning in understanding the relationship between childhood exposure to air pollution and long-term health. In realizing its objectives, this research made theoretical, methodological, and substantive contributions.

An important theoretical contribution of this research is the application of a conceptual model that guides this investigation of the health impacts of childhood exposure to air pollution. The conceptual utility of this model is that it allows for the organization of health determinants into macro-environmental or micro-environmental constructs that integrate the timing of events and their effect on health across the life-course. The effectiveness of this framework is borne out by the large proportion of respiratory and non-respiratory health outcomes that it has helped explain (Chapter Five).

However, there are challenges to be addressed in the operation of this framework. While in some constructs of the model, such as healthcare access, it is relatively easy to conceptualize representative variables such as having a regular family doctor (Lambrew et al. 1996) or symptom-response measures (Baker et al. 1999), it is more difficult to conceptualize other constructs such as exposure assessment to air pollution (Morgenstren et al. 2003). For instance, in assessing exposure to air pollution, despite the availability of methods that account for personal mobility and variability (such as the use of personal monitors and biomarkers), exposure to air pollution at particular times in the life course may have more important impacts on long-term health. Therefore, conceptualizing critical or sensitive times in relation to childhood exposures to air pollution adds to the difficulties related to exposure assessment, particularly since different 'windows of exposures' to various air pollutants are likely to be related to different health outcomes. For instance, results of this research suggest that exposure to SO_2 around the age of 13 years may be critical in impacting the long-term respiratory health of females but not males. However, if similar exposures occur at different critical or sensitive times, males may also develop long-term respiratory health impacts. The difficulty remains in conceptualizing critical or sensitive times in relation to childhood exposures to air pollution.

In addition to theoretical contributions, this research presented methodological contributions by illustrating that the sole use of web-based search engines can be effective in tracing original cohort members. This is particularly important when the research question involves a temporal component and requires a longitudinal design. A

major limitation of longitudinal designs is the resources required to track a cohort over a relatively long time period (for example, from childhood to early adulthood). Longitudinal designs that maintain contact with research participants from a young age are subject to sample attrition that increases in adulthood (Wadsworth et al. 2003). Furthermore, success rates of re-establishing contact with a sample are dependent on the quality of information obtained at the time of initial recruitment, gender, geographical location, length of time since last contact (Hampson et al. 2001), and socioeconomic factors (Mertens et al. 2004).

Despite these limitations, some studies have demonstrated that long-term follow-up of participants allowing researchers to re-establish contact is feasible with appropriate planning and persistence (Wutze et al. 2000; Rodger et al. 2001; Garcia et al. 2003). Tracing methods in the past were limited to the use of contact information, medical registries, and hospital records (Spitzer et al. 1982; Kreiger et al. 1990). Although the recent development of the World Wide Web has facilitated participant tracing in recent research (Koo and Rohan 2000; Hampson 2001; Tehranifar et al. 2002), this research was unique in that it only employed publicly available web-based search engines to reconstruct an original cohort. Overall, the effectiveness of using web-based search engines in tracing research participants was validated in this research.

Another methodological contribution is the development of a questionnaire that collects information on a range of variables for each construct of the conceptual framework. In particular, an essential component of the questionnaire collects information on residential and occupational histories for 12 previous employments, as

well as the location, description, and exposures for 5 previous residences. This information is necessary in controlling for factors that mediate relationships between exposures in childhood and long-term health outcomes.

In terms of substantive contributions, an advantage of this research is the use of a dataset that includes exposure and health outcome data (including anthropometric data) collected originally when participants were young children along with new data (residential, occupational, lifestyle, and health) from adulthood. In doing so, the research has the advantage of examining the long-term health effects of air pollution with individualized estimates of exposure to various air pollutants such as TSP, SO_2, and $PM_{3.3}$ for different time periods; this allowed for an examination of the effects of exposure to air pollutants at different times across the life course, as well as exposure to air pollutants in the long-term. Substantively, this research suggests that exposure to SO_2 in childhood impacts long-term health. Results suggest that long-term exposure to SO_2 in childhood impacts hospital or ER visits for asthma or respiratory problems. In addition, exposure to SO_2 around the age of 13 years impacts asthma incidence and diagnosis with asthma or respiratory problems for females. In terms of long-term health outcomes, results suggest that exposure to SO_2 impacts arthritis / rheumatism and high blood pressure / hypertension.

Given the strength of this design, the findings of this research have several implications. First, the consistent presence of associations between SO_2 and long-term health suggest that exposure to SO_2 in childhood may interfere in certain biological mechanisms in the body that may cause the development of long-term health outcomes.

This information is useful for disciplines that focus on the biological mechanisms related to exposure to SO_2 and health. Second, results indicate that childhood exposure to SO_2 impacts long-term respiratory health outcomes for females more than males. This information is useful to public health specialists that may need to pay greater attention to sub-populations that are susceptible to air pollution. Third, since the long-term health impacts of childhood exposure to air pollution were mostly identified when exposure to SO_2 exceeded 11.7 ppb, these findings have substantive implications in guiding air quality standards. Annual means for SO_2 levels across Ontario ranged between 1.1 ppb and 7.8 ppb in 2005, but have reached concentrations as high as 30 ppb in 1971 (Ministry of Ontario, 2005). The concentration of SO_2 in Hamilton (Downtown) in 2005 was 5.3 ppb. However, the annual levels of SO_2 in some world-wide cities such as Erie (measured at Port Stanley, a rural and trans-boundary influenced site on the northern shore of Lake Erie) has exceeded 11 ppb in 2005 (Ministry of Ontario, 2005). Fourth, the associations found between childhood exposure to air pollution and arthritis or rheumatism suggest that more focus needs to be directed at the impacts of air pollution on the long-term development of inflammatory diseases.

6.4 RESEARCH LIMITATIONS

Despite these substantive contributions, this research was subject to certain limitations. Clearly, the cohort reconstruction process led to the tracing of original participants of relatively higher socioeconomic status both in childhood and adulthood.

This limitation may have an effect on the relationships between childhood exposure to air pollution and long-term health outcomes, particularly since original participants that experience poor health are likely to have not participated in this research. In addition, sample bias may have contributed to a relatively uniform health status across neighbourhoods that prevented an examination of the relationship between equity and access to resources in childhood and long-term health. Another important methodological challenge associated with the reconstruction process was the tracing of female participants, particularly since females may likely be subject to name changes in life and since almost 70% of the original participants did not include a father's name or initial. Although, this research shows that the sole use of publicly available web-based search engines is in general effective in tracing research participants and in allowing longitudinal research, there are other options available that can increase sample size and reduce bias. However, these options are subject to limitations such as time and financial requirements. Weinberger et al. (2002) report financial costs of 8,230 US dollars to trace 259 research participants of a 27 year old study.

Another methodological challenge associated with the conceptual model was how to quantify the variables that represent residential and occupational exposures, and social networking. While some variables such as income and lung function in childhood were easily measured quantitatively, others such as social networking was not easily quantifiable. The latter was quantified using proxies such as the number of friends or acquaintances, and the number of visits and social events.

Another limitation is the sole use of self-reported data in this research. If available, anthropometric measures of lung function in adulthood would have allowed us to examine the relationships between exposures to air pollutants in childhood and long-term pulmonary lung function, and between physiological measures of lung function in childhood and lung function in adulthood. In addition, the sole use of self-reported data in relation to residential and occupational exposures and the absence of data related to exposure assessment to air pollution past childhood present limitations in this research. In particular, the absence of data on exposure assessment to air pollution between 1986 and 2005 does not allow the examination of long-term health effects of chronic exposure to air pollution or whether exposure to improved air quality has a reverse effect on health.

Research limitations were also related to data collected during the original research (1975 – 1986). First, the absence of data on outdoor activities and time spent outdoors in childhood prevented examination on whether the protective effect of exposure to TSP on long-term health is related to behavioural practices. Second, in some cases, exposure assessment data were missing. Third, the definition of the four neighbourhoods in the original study may have affected results of analysis at the neighbourhood level, particularly since the east and west neighbourhoods were divided randomly. This is particularly apparent from results of recent Hamilton research that used specialized spatial techniques to examine the socio-demographic characteristics of neighbourhoods in Hamilton (Luginaah et al. 2001). Results indicate that the Central Downtown Core (CDC) and the Chedoke – Kirkendall area each have unique characteristics that are likely to impact health. However, the division of the four

neighbourhoods in the original research situated both the CDC and Chedoke - Kirkendall in the west lower neighborhood.

Overall, findings of this research are significant in promoting future studies that focus on relationships between childhood exposure to air pollution and respiratory as well as non-respiratory health outcomes. Guided by the conceptual framework, this research has enhanced knowledge on the development of long-term health, as well as provided a basis for focusing on the timing of exposures across the life course when examining health outcomes in adulthood.

6.4 FUTURE RESEARCH DIRECTIONS

Although this research enhances understanding of the relationship between childhood exposure to air pollution and health, a number of questions remain to be addressed that pertain to theory, substantive issues, and methodology. With respect to theory, it would be useful to ascertain how well the conceptual model used in this study would perform for different childhood exposures or to explain other long-term health outcomes. In this study, models of long-term health outcomes that had a relatively high predictability included hospitalization for asthma or respiratory problems, chronic bronchitis for females, arthritis / rheumatism (sex-specific), diagnosis with chest conditions, diagnosis with cancer, and fair or poor self-assessed health. A key question then is why were models of other health outcomes (such as high blood pressure and hay fever / allergies for females) not able to predict at a similar level? Further, what

predictors need to be included in addressing these long-term health outcomes? If other predictors are included, will childhood exposure to air pollution emerge as a possible predictor in the final models?

There are a number of substantive issues arising from this research that warrant further investigation. The first concerns the time period of exposure to SO_2 that has been identified in most of the associations found between childhood exposure to air pollution and long-term health. Although results suggest that childhood exposure to SO_2 above the median level of 11.7 ppb impacts long-term respiratory health, the question remains as to whether this trend is a result of the level of exposure or the age of participants or a combination of both. Furthermore, do these findings imply that males are less susceptible to exposure to SO_2 or is it possible that these results occurred because the study period that ended in 1986 did not depict an important stage of health development, that is the transition into puberty for male participants?

The second substantive issue arises from results that indicate a high prevalence rate of hay fever or allergies in our sample relative to the general population. This deserves further exploration particularly since asthma and allergies are linked in several ways; both share immunologic pathogenesis, affect lung function, and there is a co-occurrence in populations (DuBuske 1999). The rho-square values of the combined and female-specific models of hay fever / allergies are less than 0.2 indicating a poor fit. This begs the question: are there similar effects for hay fever or allergies that result from childhood exposure to air pollution as seen for asthma? If not, what are these differences and how do they result?

Finally, predictors of long-term respiratory health outcomes include various macro and micro-environmental factors. This raises important substantive questions deserving of further research: for instance, which of the residential exposures explain long-term respiratory health outcomes? What are the mechanisms that explain the role that socio-economic and psychological factors play in long-term respiratory health?

This research enhances our understanding of the relationship between childhood exposure to air pollution and long-term health. Guided by the Life Course Health Development Framework, results of this research suggest that childhood exposure to air pollution impacts long-term health. In relation to the debate on the health effects of air pollution, this research indicates that the health impacts of childhood exposure to air pollution are to a large extent, located beyond the short-term period that most research addresses.

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APPENDIX 2.1 REVIEW PAPERS THAT EXAMINED THE RELATIONSHIP BETWEEN AIR POLLUTION AND HEALTH.		
Objective	Main findings	Reference
Objective: to review the health effects of coarse and fine PM (based on 24 time-series studies) for entire population	▪ Evidence that coarse PM has an effect on daily mortality ▪ Evidence is stronger for the effect of fine particles on mortality ▪ Coarse PM and fine PM has an effect on chronic obstructive pulmonary disease, asthma and respiratory admissions ▪ Coarse PM associated with cardiovascular admissions	Brunekreef and Forsberg (2005)
Objective: to review the health effects of air pollution for children	▪ Air pollution associated with infant mortality ▪ Lung function negatively associated with pollution	Schwartz (2004)
Objective: systematic review of studies published between 1966 and 2002 on air pollution and children health (22 studies)	▪ Largest estimate health effect obtained from unselected panel versus asthmatics ▪ Health effect of $PM_{2.5}$ greater than PM_{10} on lung function ▪ Possible interaction between PM and O_3 that merits further investigation	Ward and Ayres (2004)

Appendix 2.1 continued		
Objective: review the role of air pollution in chronic obstructive pulmonary disease (COPD) (based on last 20 years – adults and children)	▪ COPD and mortality increase with increasing air pollution ▪ Long-term studies have problem in assessing mortality due to lack of specificity of cause of death ▪ For children studies: suggestion that urban air is involved in lung function development	Sunyer (2001)
Objective: systematic review of panel-studies (1987 – 1998) on the link between air pollution and health for entire population (based on 27 studies)	▪ Association between PM and all heath measures (lung function, respiratory illness, medication use, hospital admission) ▪ Ozone effect more observable on lung function tests than symptoms or medication use ▪ NO_2 effect very rare ▪ Overall, lung function impacted by all air pollutants (SO_2, NO_2, O_3, PM_{10}, $PM_{2.5}$, black smoke, TSP)	Desqueroux and Momas (1999)

APPENDIX 2.2 SUMMARY OF STUDIES PUBLISHED SINCE 1996 THAT EXAMINED THE RELATIONSHIP BETWEEN AIR POLLUTION AND HEALTH.				
STUDY FEATURES	POLLUTANT (S) OF INTEREST	HEALTH OUTCOME ASSESSMENT	MAJOR FINDINGS	REFERENCES
9 Swiss communities (1992 – 2001) Cross-sectional n = 9591, ages 6 – 8.	PM_{10}	Respiratory symptoms using the ISAAC questionnaire	▪ Declining PM associated with declining prevalence of chronic cough, bronchitis, common cold, nocturnal dry cough and conjunctivitis symptoms. ▪ PM reduction not associated with changes in prevalence of sneezing during pollen season, asthma, and hay fever.	Bayer-Oglesby et al. (2005)
Brazil Time-series (1999 – 2000) Children age: 0 - 14	PM_{10}, BS (black smoke), NO_2, O_3	Daily record of outpatient attendance	▪ Air pollution had an impact on respiratory illness ▪ Increase in 40.4 $\mu g/m^3$ associated with increase in 4.5% outpatient attendance for respiratory diseases	Bakonyi et al. (2004)

Appendix 2.2 continued				
Washington (1997 – 1999) n = 25, all asthmatic; ages 20 – 52 (adults) and 7 to 12 (children)	PM_{10}, $PM_{2.5}$, $PM_{1.0}$, coarse particles	Daily respiratory symptoms	▪ No associations for adults. ▪ For children, association for PM and cough ▪ PM_{10} and coarse fraction associated with sputum production and runny nose ▪ All size particles aggravate asthma symptoms	Mar et al. (2004)
'Southwest Metropolitan Mexico City' Cross-sectional longitudinal case-control n = 27, ages 5 to 17	PM_{10}	Anthropometric measures: chest x-ray, nasal lesions, lung function	▪ Cases associated with nasal abnormalities (22% - measured by whitish-gray nasal lesions), hyperinflammation (67%), interstitial markings in chest x-ray ▪ Cases associated with decrease in lung function	Calderon – Garciduenas et al. (2003)
Southern England (1996-1997) Cross-sectional n = 134, ages 7 to 13	PM_{10}, SO_2, O_3, and SO_4^{-2}	Anthropometric (lung function); Symptomatic measures	▪ Negative associations between lung function measured by Peak expiratory flow (PEF) and each of SO_4^{-2} and PM_{10} ▪ Effects of PM_{10} stronger in asthmatic children	Peacock et al. (2003)

Appendix 2.2 continued				
Eastern Germany (1992-1999) Longitudinal n = 6959, ages 5 to 14	TSP, SO_2	Respiratory illnesses / symptoms	▪ 50 $\mu g/m^3$ decrease in TSP associated with decrease of bronchitis, sinusitis, and colds	Heinrich et al. (2002)
UK (1997) Time-series 'panel' design n = 171, aged 9.	PM_{10}, $PM_{2.5}$, SO_2	Symptoms (wheeze); lung function; skin testing	▪ No consistent pattern in identifying effects in association with pollutants ▪ No evidence that symptomatic children are more sensitive to pollutant effects	Ward et al. (2002)
'Four Chinese Cities Study (1993-1996) Prospective cohort n = 7621, ages 5-16	$PM_{2.5}$, $PM_{10-2.5}$, PM_{10}, TSP, SO_2	Respiratory symptoms and illnesses	▪ Positive associations between morbidity (asthma, bronchitis, hospitalization) and PM of all size fractions (stronger for coarse particles) ▪ SO_2 associated with respiratory symptoms (wheeze, cough, phlegm), but weaker than PM	Zhang et al. (2002)

Appendix 2.2 continued				
'Children's Health Study' – Southern California (1986-1997) 10-year prospective n = 3035, ages 8 -15	$PM_{2.5}$, PM_{10}, O_3, NO_2, acid vapor	Physical activity Lung function ()	▪ TSP associated with low lung function – reduction of 3.4% in lung function measures (FVC, FEV_1, MMEF, FEF_{75}) ▪ $PM_{2.5}$, associated with lung function growth ▪ Association between air pollution and lower pulmonary function stronger for females	Gauderman et al. (2002)
APHEA – 'Air Pollution & Health: a European approach' (1993 – 1999) Population of 38 M – 43 M, all ages	BS, SO2, suspended particles, NO_2, O_3	Daily mortality and hospital emergency admissions	▪ For each 10 $\mu g/m^3$ increase in PM_{10}, all cause daily mortality increased by 0.6% and hospital admissions for CVD increased by 0.5% ▪ Positive association between daily mortality and air pollution levels	Atkinson et al. (2001)
Czech and Poland (1993 – 1994) Cross-sectional n = 8013, ages 7 - 10	SO_2	Wheezing, cough, asthma	▪ SO_2 levels associated with wheezing (last 12 months) ▪ SO_2 associated with lifetime wheezing and asthma	Pikhart et al. (2001)

Appendix 2.2 continued				
Thailand Study (1995-1996) Cross-sectional design n = 79, panel of children aged 8 12, and panel of adults	PM_{10}, $PM_{2.5}$	Upper and lower respiratory symptoms:	▪ Pollutants associated with upper (cold, nasal congestion) and lower respiratory symptoms (cough, phlegm, wheeze, chest tightness) ▪ Increase of 45μg/m_3 in PM_{10} associated with 50% increase in lower respiratory symptoms for adults and 30% for children	Vichit-vadakan et al. (2001)
Relocated participants of the 'Children's Health Study' (1994-1998) Cross-sectional n = 110, ages 10 to 15	PM_{10}	Lung function measures (FEV_1, FVC, MMEF, PEFR)	▪ Annual average exposure to TSP associated with differences in annual lung function growth ▪ Participants who moved to communities with higher PM showed decreased lung function	Avol et al. (2001)
Hong Kong (1994 – 1995) Cross-sectional n = 1294, ages 8 to 12	TSP, SO_2	Respiratory symptoms (cough, phlegm, wheeze)	▪ Air quality positively associated with frequent cough, frequent sputum, chronic sputum, doctor diagnosed asthma ▪ Difference among girls more marked	Yu et al. (2001)

Appendix 2.2 continued				
'Central European Study on Air Pollution & Respiratory Health' (1992 – 1996) Cross-sectional n = 719, aged 7 to 11	$PM_{2.5}$, PM_{10}, TSP	Respiratory illnesses, symptoms, hospitalization	▪ No association between long-term exposure to TSP and asthma or wheeze ▪ Air pollution associated with hospital admissions for asthma, bronchitis, pneumonia, doctor diagnosed bronchitis, and parent reported chronic phlegm	Hruba et al. (2001)
'Harvard Six City Study' (1991) Diary studies n = 1844, ages 7 to 12	$PM_{2.5}$, $PM_{2.1}$, and $PM_{10\text{-}2.5}$;	Cough, phlegm, pain, wheeze	▪ Fine particles have stronger acute respiratory effects than coarse particles	Schwartz et al. (2000)
Southern Poland (1995-1997) Cross-sectional case-control n = 1001, age 9	PM, SO_2	Lung function (FVC, FEV_1)	▪ Air pollution levels negatively associated with lung function growth.	Jedrychowski et al. (1999)

Appendix 2.2 continued				
Pollution effects of asthmatic children in Europe (PEACE) (1993 – 1994) n = 1621, symptomatic ages 6 - 12	PM10, BLACK SMOKE SO2, NO2	PEF Presents of respiratory systems and use of medication	▪ Positive association between PEF and air pollution for asthmatic children who used medication. ▪ Negative association for children selected only on cough. ▪ No consistent association for asthmatic children who did not use medication.	Roemer et al. (1999)
Chile (1996) Cross-sectional; case-control embedded n = 996, ages 6 to 12	PM_{10}, SO_2	Lung function, respiratory symptoms	▪ For participants with chronic respiratory symptoms, increase in SO_2 related to a reduction in lung function ▪ For non-symptomatic, increase in PM_{10} related to a reduction in lung function ▪ Cumulative exposure over 3 days associated with an increase of 9% for episodes of wheezing	Sanchez et al. (1999)

Appendix 2.2 continued				
Netherlands study (1992-1996) Cross-sectional with case-control n = 795, ages 7 to 11	PM_{10}, SO_2	Lung function (PEF), diary of respiratory symptoms.	▪ In symptomatic children, associations between PM_{10} and sulphates and prevalence of symptoms of lower respiratory tract and decrements in PEF ▪ In non-symptomatic, associations between PM_{10} and decrements in lung function were found.	Van der Zee et al. (1999)
East (E) and west (W) Germany (1991 – 1995) Cross sectional n = 19090, ages 7	TSP, SO_2	Airway diseases, dry cough, allergies	▪ Infectious airway diseases (bronchitis, colds, tonsillitis) and irritations of the airways (cough, wheeze) are associated with SO_2 and TSP ▪ No association for asthma	Kramer et al. (1999)
Vancouver Island, BC, Canada (1990-1992) Cross-sectional case-control n = 2200 (188 asthmatic), ages: 6 to 13	PM_{10}	Lung function (PEF) and symptom diary	▪ For entire sample, increases in PM_{10} associated with reductions in lung function and increased reporting of cough, phlegm and sore throat	Vedal et al. (1998)

Appendix 2.2 continued				
SOAP & Hit – 'Study on air pollution & health in Taiwan (1994 – 1997) Cross sectional study 5072, ages: 6 to 12	SO_2, NOx, O_3, NO_2, PM_{10}, NO	Respiratory symptoms	▪ Children in the urban communities experience more respiratory symptoms and diseases (cough, shortness of breath, nasal symptoms, sinusitis, wheezing, asthma, allergic rhinitis, bronchitis) compared to those living in rural areas ▪ No attribute of effects to specific pollutant	Chen et al. (1998)
Swiss Study (1992 – 1993) Cross-sectional n = 4470, ages: 6 to 15	PM_{10}, SO_2, NO_2, and O_3	Respiratory symptoms, asthma, wheeze	▪ Highest PM_{10} exposure led to tripling of non-specific symptoms such as cough and nocturnal cough ▪ No effect on asthma and wheeze	Braun-Fahlander et al. (1997)
Hungary – Tata Region (1993 – 1994) Longitudinal & cross-sectional n = 147, ages 9 to11	SO_2, NO	Lung function, pulse, blood pressure	▪ Significant correlation with SO_2 levels and acute daily respiratory morbidity ▪ No statistically significant relationship between SO_2 and pulmonary function	Pinter et al. (1996)

Appendix 2.2 continued				
S California – 3 communities (1992 – 1994) Cohort n = 269, ages 9-11	PM_{10}	FVC, FEV1	▪ Daily personal exposures correlated with pollutant concentrations at central sites for PM ▪ FVC decreased with increase in PM; morning to afternoon change of FEV_1 ▪ Excess asthma / allergy in children from polluted area ▪ Children may experience slight lung function changes from day-to-day air quality changes ▪ Need for longer term studies	Linn et al. (1996)

Appendix 3.1 Letter of information sent to parents of the traced cohort.

Dear _____:

Please allow me to introduce myself. My name is Caroline Haddad, and I am a PhD candidate working with Dr. Susan Elliott who directs and runs various research projects at McMaster University. One of our colleagues is Dr. David Pengelly. This name may be familiar to you since between 1978 and 1986, Dr. Pengelly along with Dr. Kerigan and Dr. Goldsmith conducted an air quality study that involved children from Hamilton elementary schools and our records indicate that your child participated in this study. We found your phone number and mailing address through Yellow.ca and Canada 411, which are electronic forms of the phonebook found on the World-Wide Web network. You may recall that at the time, you and your child completed several questionnaires that included information on respiratory symptoms, as well as several respiratory tests for your child sponsored by McMaster University.

The research conducted by Dr David Pengelly was very successful; analysis revealed that higher pollution levels for sulfur dioxide and particulate matter had a short-term effect on the respiratory health of children with asthma such as increased airway obstruction. The carefully designed study allows for follow-up longitudinal research that assesses the long-term health effects of early childhood exposure to adverse air quality. With your consent, we now propose to regain access to and use of the data previously collected and to re-establish contact with your child in order to run a follow-up longitudinal study so that we may further investigate the long-term health effects of adverse air quality. The follow-up study consists of your child completing a short survey that I can mail or e-mail to him / her.

Our researchers will be in touch with you shortly via telephone at which time they would be happy to answer any questions that you might have; they will also ask for your consent and for your child's contact information. Please be assured that you are by no way volunteering your child's involvement in this study and that your child will have

the opportunity to learn more about this study and accordingly will make his appropriate decision regarding participation. In the meantime, if you have any questions, please feel free to contact Dr. Elliott at (905) 525-9140 X23768.

Sincerely,

Caroline Haddad.
PhD Candidate
McMaster University
Hamilton, Ontario.
barakac@mcmaster.ca

Dr. David Pengelly
McMaster Institute of
Environment and Health
McMaster University
Hamilton, Ontario
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Dr. Susan J. Elliott
Professor of Geography
School of Geography and
Earth Sciences
McMaster University
Hamilton, Ontario
(905) 525 9140 ext. 23768
elliotts@mcmaster.ca

Appendix 3.2 Letter of information send to original participants that were traced by name.

Dear _____:

Please allow me to introduce myself. My name is Caroline Haddad, and I am a PhD candidate working with Dr. Susan Elliott who directs and runs various research projects at McMaster University. One of our colleagues is Dr. David Pengelly. This name may be familiar to you since between 1978 and 1986, Dr. Pengelly along with Dr. Kerigan and Dr. Goldsmith conducted an air quality study that involved children from Hamilton elementary schools and our records indicate that you participated in this study. We found your phone number and mailing address through Yellow.ca and Canada 411, which are electronic forms of the phonebook found on the World-Wide Web network. You may recall that at the time, you and your parent / guardian completed several questionnaires that included information on respiratory symptoms, as well as several respiratory tests sponsored by McMaster University.

The research conducted by Dr David Pengelly was very successful; analysis revealed that higher pollution levels for sulfur dioxide and particulate matter had a short-term effect on the respiratory health of children with asthma such as increased airway obstruction. The carefully designed study allows for follow-up longitudinal research that assesses the long-term health effects of early childhood exposure to adverse air quality. With your consent, we now propose to regain access to and use of the data previously collected and to re-establish contact with you in order to run a follow-up longitudinal study so that we may further investigate the long-term health effects of adverse air quality. The follow-up study consists of you completing a short survey that I can mail or e-mail to you.

Our researchers will be in touch with you shortly via telephone at which time they would be happy to answer any questions that you might have; they will also ask for

your consent and will verify your contact information. Please be assured that you will be given the opportunity to learn more about this study and accordingly will make your decision regarding participation. In the meantime, if you have any questions, please feel free to contact Dr. Elliott at (905) 525-9140 X23768.

Sincerely,

Caroline Haddad.
PhD Candidate
McMaster University
Hamilton, Ontario.
barakac@mcmaster.ca

Dr. David Pengelly
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Appendix 3.3 Telephone script administered on the parent's of the traced cohort.

- Hello, May I speak with Mr / Mrs _____?
- My name is _____. Recently I mailed you a letter of information about a research project on air pollution. Did you receive this letter?

- **No** - May I read the contents of the letter to you now?	- **Yes**
- Yes (Read and continue below)	**- No** - Is there a better time to call? (Note time)

- As a follow-up to that letter, I am now conducting telephone interviews on behalf of McMaster University. May I have a few minutes of your time?

- **No** - Is there another time or day that would be more convenient for me to call back?	- **Yes**
- **Yes** (Mark time / day)	- **No** - Sorry to bother you.

- Thank you. Do you have a child named _____?

- **No** - I apologize; I must have the wrong number.	- **Yes** - Does your child still reside in Canada?
	- Yes / **- No** - May I know where he resides? (Note down if no longer alive).

- Between 1979 and 1986 your child participated in an air quality study conducted in Hamilton and led by Dr. David Pengelly and Dr. Anthony Kerigan. The purpose of this study was to test for chronic exposure of children's lungs to air pollution. Do you remember your child being part of this study?
- Yes / No
- Would you like to hear more information about it?

- **Yes** - The study was a great success. Researchers found that poor air quality has short-term respiratory health effects for children with asthma. Today, however, researchers still do not know much about the long-term effects of chronic childhood exposure to poor air quality. Because of this, we would like to conduct a follow-up with the children that participated in the original research in order to study the long-term effects of air pollution from childhood. In order to re-establish contact with the families, we looked up your phone number either on Yellow.ca or Canada 411, which are electronic forms of the phonebook located on the Internet.	- **No**

- There are two questions that I need to ask. Firstly, do we have your consent to use the data collected from the previous study?

- **Yes** - Thank you; we appreciate your involvement	- **No** - I am sorry to bother you.

- Secondly, would you be willing to give me _____'s phone number or email address so that we might contact him / her for the study? Please remember that we will still ask for his / her consent to participate in this research.

- **Yes** - Thank you. I would like to assure you that this information will remain confidential. (Note information) - Thank you for your time and participation.	- **No** - Could I mail you information about the study so that you could then decide whether or not to pass it on to researchers at McMaster University?

- **Yes** - Thank you. You will receive the information shortly by mail. - Can I verify your mailing address?	- **No** - Thank you for your time and sorry to bother you.

Appendix 3.4 Telephone script administered on participants whose contact information was retrieved from their parents.

- Hello, May I speak with Mr / Mrs _____?
- My name is _____. I obtained your phone number from your Mr / Mrs _____ (senior)?
- I am conducting telephone interviews on behalf of McMaster University. May I have a few minutes of your time?

- **No** - Is there another time or day that would be more convenient for me to call back?	- **Yes**
- **Yes** (Mark time / day)	- **No** - Sorry to bother you.

↓

- Thank you. Did you attend a public elementary school in Hamilton _____?

- **No** - I apologize; I must have the wrong number.	- **Yes**

↓

- Between 1979 and 1986 you participated in an air quality study conducted in Hamilton and led by Dr. David Pengelly and Dr. Anthony Kerigan. The purpose of this study was to test for chronic exposure of children's lungs to air pollution. Do you remember being part of this study?
- Yes / No
- Would you like to hear more information about it?

- **Yes** - The study was a great success. Researchers found that poor air quality has short-term respiratory health effects for children with asthma. Today, however, researchers still do not know much about the long-term effects of chronic childhood exposure to poor air quality. Because of this, we would like to conduct a follow-up with the children that participated in	- **No**

the original research in order to study the long-term effects of air pollution from childhood.

- There are two questions that I need to ask. Firstly, do we have your consent to use the data collected from the previous study?

- **Yes** - Thank you; we appreciate your involvement	- **No** - I am sorry to bother you.

- Secondly, a team of McMaster researchers have initiated a new research project that examines the long-term effects of early childhood exposure to adverse air quality? Participation consists of filling out a questionnaire that should take less than one hour of your time. It is of great personal and societal benefit to learn more about the long-term health effects of air pollution; such knowledge can assist in the development of government policies and may assist the general public in taking preventative measures that may improve their health. Your involvement in the study is of great value since the original study contains indication of your exposure to air quality as a child. You are free to drop out of the study at any time by simple contacting the number provided on the mailed letter of information and a final copy of the report will be mailed to you if you are interested in receiving it. Would you be willing to participate in this new research study?

- **Yes** - Thank you. Can I verify your address? - Thank you for your time and participation. - Are you interested in receiving a copy of the final report? - Yes / No	- **No** - Could I mail you information about the study so that you could then decide whether you would like to participate?	
	- **Yes** - Thank you. You will receive the information shortly by mail. - Can I verify your mailing address?	- **No** - Thank you for your time and sorry to bother you.

Appendix 3.5 Telephone script administered on participants whose contact information was retrieved from publicly available web-based search engines.

- Hello, May I speak with Mr / Mrs _____?
- My name is _____. Recently I mailed you a letter of information about a research project on air pollution. Did you receive this letter?

- **No** - May I read the contents of the letter to you now?	- **Yes**
- **Yes** (Read and continue below)	- **No** - Is there a better time to call? (Note time)

- As a follow-up to that letter, I am now conducting telephone interviews on behalf of McMaster University. May I have a few minutes of your time?

- **No** - Is there another time or day that would be more convenient for me to call back?	- **Yes**
- **Yes** (Mark time / day)	- **No** - Sorry to bother you.

- Thank you. Did you attend a public elementary school in Hamilton?

- **No** - I apologize; I must have the wrong number.	- **Yes**

- Between 1979 and 1986 you participated in an air quality study conducted in Hamilton and led by Dr. David Pengelly and Dr. Anthony Kerigan. The purpose of this study was to test for chronic exposure of children's lungs to air pollution. Do you remember being part of this study?
- Yes / No
- Would you like to hear more information about it?

- **Yes**
- The study was a great success. Researchers found that poor air quality has short-term respiratory health effects for children with asthma. Today, however, researchers still do not know much about the long-term effects of chronic childhood exposure to poor air quality. Because of this, we would like to conduct a follow-up with the children that participated in the original research in order to study the long-term effects of air pollution from childhood. In order to re-establish contact with the families, we looked up your phone number either on Yellow.ca or Canada 411, which are electronic forms of the phonebook located on the Internet.

- No

- There are two questions that I need to ask. Firstly, do we have your consent to use the data collected from the previous study?

- **Yes** - Thank you; we appreciate your involvement.	- **No** - I am sorry to bother you.

- Secondly, a team of McMaster researchers have initiated a new research project that examines the long-term effects of early childhood exposure to adverse air quality? Participation consists of filling out a questionnaire that should take less than one hour of your time. It is of great personal and societal benefit to learn more about the long-term health effects of air pollution; such knowledge can assist in the development of government policies and may assist the general public in taking preventative measures that may improve their health. Your involvement in the study is of great value since the original study contains indication of your exposure to air quality as a child. You are free to drop out of the study at any time by simple contacting the number provided on the mailed letter of information and a final copy of the report will be mailed to you if you are interested in receiving it. Would you be willing to participate in this new research study?

- **Yes** - Thank you. Can I verify your address? - Thank you for your time and participation. - Are you interested in receiving a copy of the final report? - Yes / No	- **No** - Could I mail you information about the study so that you could then decide whether you would like to participate?	
	- **Yes** - Thank you. You will receive the information shortly by mail. - Can I verify your mailing address?	- **No** - Thank you for your time and sorry to bother you.

Appendix 3.6 Questionnaire administered on the reconstructed cohort.

Hamilton Children's Cohort Survey

This survey contains questions related to your working life, your daily life, your health, and your quality of life. It should take you no more than 20 minutes to complete. Your answers will help us better understand how where people live affects their health and well being. All the answers you provide to the questions will be kept strictly confidential and data will be reported in such a way that will protect your anonymity and that of all respondents. Please be assured that you have the right to refuse to answer any of the questions. We may contact you in the near future to repeat the same physiological tests that you did as a child.

When completing the survey, please follow the instructions found at the beginning of each section. **Once you have completed** the survey, please insert it along with the enclosed consent form (white copy) in the self-addressed stamped envelope provided, and drop it off at your local post office or nearest mailbox.

If you have any questions when completing the survey or would like to receive a copy of the study results, please contact Dr. Susan Elliott at (905) 525 9140 ext 23139 or elliotts@mcmaster.ca.

Thank you very much for taking the time to complete this survey.

First, we would like to ask you a few questions about your job. Please put a check mark beside the response that most accurately reflects your current status and fill in the blanks when applicable.

1. Which of the following best describes your current employment status?
   - ❑ Full-time job
   - ❑ Part-time job
   - ❑ Sick leave, maternity leave, or strike
   - ❑ Unemployed; **please go to question 5.**
   - ❑ Retired; **please go to question 5.**
   - ❑ Homemaker; **please go to question 5.**
   - ❑ Student (includes students working part-time); **please go to question 5.**
   - ❑ Other ; please specify _____

2. Which best describes your type of employment?
   - ❑ Clerical
   - ❑ Manual
   - ❑ Managerial
   - ❑ Professional
   - ❑ Other If 'other', please specify _____

3. How long have you been in this current job? Number of years____ Number of months___

4. In this job, have you ever been exposed to any of the following (Please move across rows to fill in the table below and check mark the most appropriate answer):

	Which best describes the intensity of exposure you experienced?
a) Dusty conditions for more than one year? ❑ Yes If 'yes', please specify type_____ ❑ No **If 'no', please go to b**	❑ Mild ❑ Moderate ❑ Severe
b) Gases, fumes, or chemicals? ❑ Yes If yes, please specify type_____ ❑ No **If 'no' please go to c**	❑ Mild ❑ Moderate ❑ Severe
c) Other contaminant(s) ❑ Yes If yes, please specify type _____ ❑ No **If 'no', please go to question 5 below.**	❑ Mild ❑ Moderate ❑ Severe

We would also like to know about the conditions of your employment over the past few years. Please complete the following table. Note that Job A represents the most recent job you had before your current job.

5.	Job A	Job B	Job C	Job D	Job E	Job F
a) From	___ ___ Yr Month	___ ___ Yr Month	___ ___ Yr Month	___ ___ Yr Month	___ ___ Yr Month	___ ___ Yr Month
To	___ ___ Yr Month	___ ___ Yr Month	___ ___ Yr Month	___ ___ Yr Month	___ ___ Yr Month	___ ___ Yr Month
b) Employment status	❑ Full-time ❑ Part-time ❑ Occasional	❑ Full-time ❑ Part-time ☐ Occasional	❑ Full-time ❑ Part-time ☐ Occasional	❑ Full-time ❑ Part-time ☐ Occasional	❑ Full-time ❑ Part-time ☐ Occasional	❑ Full-time ❑ Part-time ☐ Occasional
C) Type of employment	❑ Clerical ❑ Manual labor ❑ Managerial ❑ Professional ❑ Other	❑ Clerical ❑ Manual labor ❑ Managerial ❑ Professional ❑ Other	❑ Clerical ❑ Manual labor ❑ Managerial ❑ Professional ❑ Other	❑ Clerical ❑ Manual labor ❑ Managerial ❑ Professional ❑ Other	❑ Clerical ❑ Manual labor ❑ Managerial ❑ Professional ❑ Other	❑ Clerical ❑ Manual labor ❑ Managerial ❑ Professional ❑ Other
d) In this occupation, were you ever exposed to dust for more than one year? If yes, please specify the type / source of dust. i) Which describes the intensity of exposure to the dust?	❑ Yes ❑ No ______ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ______ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ______ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ______ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ______ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ______ Source / Type ❑ Mild ❑ Moderate ❑ Severe
e) In this occupation, were you ever exposed to gases, fumes, or chemical? If yes, please specify the type / source i) Which describes the intensity of exposure?	❑ Yes ❑ No ______ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ______ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ______ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ______ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ______ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ______ Source / Type ❑ Mild ❑ Moderate ❑ Severe
f) Other contaminant(s)	❑ Yes (specify type ________) ❑ No	❑ Yes (specify type ________) ❑ No	❑ Yes (specify type ________) ❑ No	❑ Yes (specify type ________) ❑ No	❑ Yes (specify type ________) ❑ No	❑ Yes (specify type ________) ❑ No

If you have had additional jobs in the past, please continue on the next page. If not, please skip to page 4.

5. (continued)	Job G	Job H	Job I	Job J	Job K	Job L
a) From	___ ____ Yr Month	___ ____ Yr Month	___ ____ Yr Month	___ ____ Yr Month	___ ____ Yr Month	___ ____ Yr Month
To	___ ____ Yr Month	___ ____ Yr Month	___ ____ Yr Month	___ ____ Yr Month	___ ____ Yr Month	___ ____ Yr Month
b) Employment status	❑ Full-time ❑ Part-time ❑ Occasional	❑ Full-time ❑ Part-time ❑ Occasional	❑ Full-time ❑ Part-time ❑ Occasional	❑ Full-time ❑ Part-time ❑ Occasional	❑ Full-time ❑ Part-time ❑ Occasional	❑ Full-time ❑ Part-time ❑ Occasional
C) Type of employment	❑ Clerical ❑ Manual labor ❑ Managerial ❑ Professional ❑ Other	❑ Clerical ❑ Manual labor ❑ Managerial ❑ Professional ❑ Other	❑ Clerical ❑ Manual labor ❑ Managerial ❑ Professional ❑ Other	❑ Clerical ❑ Manual labor ❑ Managerial ❑ Professional ❑ Other	❑ Clerical ❑ Manual labor ❑ Managerial ❑ Professional ❑ Other	❑ Clerical ❑ Manual labor ❑ Managerial ❑ Professional ❑ Other
d) In this occupation, were you ever exposed to dust for more than one year? If yes, please specify the type / source of dust. i) Which describes the intensity of exposure to the dust?	❑ Yes ❑ No ________ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ________ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ________ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ________ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ________ Source / Type ❑ Mild ❑ Moderate ❑ Severe	❑ Yes ❑ No ________ Source / Type ❑ Mild ❑ Moderate ❑ Severe
e) In this occupation, were you ever exposed to gases, fumes, or chemical? If yes, please specify the type / source i) Which describes the intensity of exposure?	❑ Yes ❑ No ________ Source / Type ❑ Mild ❑ Moderate Severe	❑ Yes ❑ No ________ Source / Type ❑ Mild ❑ Moderate Severe	❑ Yes ❑ No ________ Source / Type ❑ Mild ❑ Moderate Severe	❑ Yes ❑ No ________ Source / Type ❑ Mild ❑ Moderate Severe	❑ Yes ❑ No ________ Source / Type ❑ Mild ❑ Moderate Severe	❑ Yes ❑ No ________ Source / Type ❑ Mild ❑ Moderate Severe
f) Other contaminant(s)	❑ Yes (specify type _______) ❑ No	❑ Yes (specify type _______) ❑ No	❑ Yes (specify type _______) ❑ No	❑ Yes (specify type _______) ❑ No	❑ Yes (specify type _______) ❑ No	❑ Yes (specify type _______) ❑ No

Next, we would like to ask you a few questions about the neighbourhoods that you have lived in.
For each question, please put a check mark beside the response that most accurately reflects your answer and fill in the blanks when applicable.

6. How long have you lived at your current address?

Number of years_____ Number of months_____

6i) Do you or your spouse own □ or rent □ this property?

6ii) How many people live in your household including yourself? _____

6iii) Property description:
- ❑ Detached
- ❑ Semi-detached / Attached
- ❑ 1 or 2 bedrooms Condo / Apartment
- ❑ 3 or more bedrooms Condo / Apartment

6iv) Approximate age of property: _____ years

6v) Type of heating:
- ❑ gas
- ❑ electricity
- ❑ oil
- ❑ other; (please specify _____

6a) Which best describes your heating system?
- ❑ Forced air (ducts / vents) AND gas space heater / gas operated fireplace
- ❑ Forced air WITHOUT gas space heater / gas operated fireplace
- ❑ Not forced air WITH gas space heater
- ❑ Not forced air WITHOUT gas space heater
- ❑ Gas operated space heater only
- ❑ None of the above
- ❑ Do not know

6b) If applicable, how often do you get your air ducts cleaned?
- ❑ Yearly
- ❑ As needed
- ❑ Rarely
- ❑ Do not remember
- ❑ Not applicable

6c) Which best describes your most frequently used cooking method?
- ❑ Electricity
- ❑ Gas
- ❑ Microwave
- ❑ Electricity plus microwave
- ❑ Gas plus microwave
- ❑ Other _____

Please fill in the table below by moving across each row:

i) **Do you have any of the following?**	ii) **How often do you use it?**
6d) Air conditioner ❑ Yes ❑ No **If 'no', please go to 6e on next row.**	❑ always with hot weather ❑ almost always ❑ rarely ❑ never
6e) Air humidifier ❑ Yes ❑ No **If 'no', please go to 6f on next row.**	❑ always ❑ almost always ❑ rarely ❑ never
6f) Air filter / air purifier ❑ Yes ❑ No **If 'no', please go to 6g below.**	❑ always ❑ almost always ❑ rarely ❑ never

6g) Have you been exposed to any contaminant / pesticide / insecticide in your current place of residence?

❑ Yes Please specify _____

❑ No

6h) How concerned are you about air pollution in your neighbourhood?

❑ Not at all concerned
❑ Slightly concerned
❑ Moderately concerned
❑ Extremely concerned

6i) How likely is it that air pollution will lead to health problems for you?

❑ Very likely
❑ Somewhat likely
❑ Somewhat unlikely
❑ Very unlikely

6j) How likely is it that air pollution will lead to health problems for other members of your household?

❑ Very likely
❑ Somewhat likely
❑ Somewhat unlikely
❑ Very unlikely

7) Do you have a previous place of residence?

❑ Yes
❑ No **If your answer is 'no', please skip to question 9.**

8) Dr. Pengelly's study ended in 1986. Please describe your previous places of residence **since 1986**. Each column (Residence A, Residence B, etc.) represents a previous place of residence for you and your family.

	Residence A	Residence B	Residence C	Residence D	Residence E
8a) Duration of residence From	____ ____ Yr Month	____ ____ Yr Month	____ ____ Yr Month	____ ____ Yr Month	____ ____ Yr Month
To	____ ____ Yr Month	____ ____ Yr Month	____ ____ Yr Month	____ ____ Yr Month	____ ____ Yr Month
8b) Location City	_____	_____	_____	_____	_____
Province	_____	_____	_____	_____	_____
Postal code	_____	_____	_____	_____	_____
8c) Please describe the property	❑ Detached ❑ Semi-detached / Attached ❑ Condo / Apartment	❑ Detached ❑ Semi-detached / Attached ❑ Condo / Apartment	❑ Detached ❑ Semi-detached / Attached ❑ Condo / Apartment	❑ Detached ❑ Semi-detached / Attached ❑ Condo / Apartment	❑ Detached ❑ Semi-detached / Attached ❑ Condo / Apartment
8d) How many people lived in this household including yourself?	_____	_____	_____	_____	_____
8e) How many bedrooms did this property have?	_____	_____	_____	_____	_____
8f) What was the approximate age of the property at the time of residence?	_____years	_____years	_____years	_____years	_____years
8g) Which best describes the type of heating at this property?	❑ Gas ❑ Electricity ❑ Oil ❑ Other - (please specify _____)	❑ Gas ❑ Electricity ❑ Oil ❑ Other - (please specify _____)	❑ Gas ❑ Electricity ❑ Oil ❑ Other - (please specify _____)	❑ Gas ❑ Electricity ❑ Oil ❑ Other - (please specify _____)	❑ Gas ❑ Electricity ❑ Oil ❑ Other - (please specify _____)
	Residence A continued on next page	Residence B continued on next page	Residence C continued on next page	Residence D continued on next page	Residence E continued on next page

	Residence A	Residence B	Residence C	Residence D	Residence E
8h) Which best describes your heating system at this location?	❑ Forced air (ducts / vents) AND gas space heater / gas operated fireplace ❑ Forced air WITHOUT gas space heater / gas operated fireplace ❑ Not forced air WITH gas space heater ❑ Not forced air WITHOUT gas space heater ❑ Gas operated space heater only ❑ Neither of the above ❑ Do not know	❑ Forced air (ducts / vents) AND gas space heater / gas operated fireplace ❑ Forced air WITHOUT gas space heater / gas operated fireplace ❑ Not forced air WITH gas space heater ❑ Not forced air WITHOUT gas space heater ❑ Gas operated space heater only ❑ Neither of the above ❑ Do not know	❑ Forced air (ducts / vents) AND gas space heater / gas operated fireplace ❑ Forced air WITHOUT gas space heater / gas operated fireplace ❑ Not forced air WITH gas space heater ❑ Not forced air WITHOUT gas space heater ❑ Gas operated space heater only ❑ Neither of the above ❑ Do not know	❑ Forced air (ducts / vents) AND gas space heater / gas operated fireplace ❑ Forced air WITHOUT gas space heater / gas operated fireplace ❑ Not forced air WITH gas space heater ❑ Not forced air WITHOUT gas space heater ❑ Gas operated space heater only ❑ Neither of the above ❑ Do not know	❑ Forced air (ducts / vents) AND gas space heater / gas operated fireplace ❑ Forced air WITHOUT gas space heater / gas operated fireplace ❑ Not forced air WITH gas space heater ❑ Not forced air WITHOUT gas space heater ❑ Gas operated space heater only ❑ Neither of the above ❑ Do not know
8i) If applicable, how often did you get the air ducts cleaned?	❑ Yearly ❑ As needed ❑ Rarely ❑ Do not remember ❑ Not applicable ❑ Do not know	❑ Yearly ❑ As needed ❑ Rarely ❑ Do not remember ❑ Not applicable ❑ Do not know	❑ Yearly ❑ As needed ❑ Rarely ❑ Do not remember ❑ Not applicable ❑ Do not know	❑ Yearly ❑ As needed ❑ Rarely ❑ Do not remember ❑ Not applicable ❑ Do not know	❑ Yearly ❑ As needed ❑ Rarely ❑ Do not remember ❑ Not applicable ❑ Do not know
8j) Which best describes your most frequently used cooking method at this location?	❑ Electricity ❑ Gas ❑ Microwave ❑ Electricity plus microwave ❑ Gas plus microwave ❑ Other; (please specify ________)	❑ Electricity ❑ Gas ❑ Microwave ❑ Electricity plus microwave ❑ Gas plus microwave ❑ Other; (please specify ________)	❑ Electricity ❑ Gas ❑ Microwave ❑ Electricity plus microwave ❑ Gas plus microwave ❑ Other; (please specify ________)	❑ Electricity ❑ Gas ❑ Microwave ❑ Electricity plus microwave ❑ Gas plus microwave ❑ Other; (please specify ________)	❑ Electricity ❑ Gas ❑ Microwave ❑ Electricity plus microwave ❑ Gas plus microwave ❑ Other; (please specify ________)
	Residence A continued on next page	**Residence B continued on next page**	**Residence C continued on next page**	**Residence D continued on next page**	**Residence E continued on next page**

	Residence A	Residence B	Residence C	Residence D	Residence E
8k) Have you been exposed to any contaminant / pesticide/ at this residence?	❑ Yes; specify type _____ ☐ No	❑ Yes; specify type _____ ☐ No	❑ Yes; specify type _____ ☐ No	❑ Yes; specify type _____ ☐ No	❑ Yes; specify type _____ ☐ No
8l) How concerned were you about air pollution in this neighbourhood?	❑ Not at all concerned ❑ Slightly concerned ❑ Moderately concerned ❑ Extremely concerned	❑ Not at all concerned ❑ Slightly concerned ❑ Moderately concerned ❑ Extremely concerned	❑ Not at all concerned ❑ Slightly concerned ❑ Moderately concerned ❑ Extremely concerned	❑ Not at all concerned ❑ Slightly concerned ❑ Moderately concerned ❑ Extremely concerned	❑ Not at all concerned ❑ Slightly concerned ❑ Moderately concerned ❑ Extremely concerned
8m) How likely is it that air pollution at this residence led to health problems for you?	❑ Very likely ❑ Somewhat likely ❑ Somewhat unlikely ❑ Very unlikely	❑ Very likely ❑ Somewhat likely ❑ Somewhat unlikely ❑ Very unlikely	❑ Very likely ❑ Somewhat likely ❑ Somewhat unlikely ❑ Very unlikely	❑ Very likely ❑ Somewhat likely ❑ Somewhat unlikely ❑ Very unlikely	❑ Very likely ❑ Somewhat likely ❑ Somewhat unlikely ❑ Very unlikely
8n) How likely is it that air pollution will lead to health problems for other members of your household?	❑ Very likely ❑ Somewhat likely ❑ Somewhat unlikely ❑ Very unlikely	❑ Very likely ❑ Somewhat likely ❑ Somewhat unlikely ❑ Very unlikely	❑ Very likely ❑ Somewhat likely ❑ Somewhat unlikely ❑ Very unlikely	❑ Very likely ❑ Somewhat likely ❑ Somewhat unlikely ❑ Very unlikely	❑ Very likely ❑ Somewhat likely ❑ Somewhat unlikely ❑ Very unlikely

Now, we would like to ask you a few questions about your day to day activities. Please put a check mark beside the response that most accurately reflects your answer to each question and fill in the blanks when applicable.

9. At the present time how often do you smoke cigarettes?
- ❑ Daily
- ❑ Occasionally
- ❑ Not at all
- ❑ Refused

10. Did you ever smoke cigarettes on a daily basis?
- ❑ Yes
- ❑ No
- ❑ Refused

11. On average, about how many cigarettes a day do you now smoke?
Number of cigarettes _____.

12. How often are you exposed to tobacco smoke at home or at work?
- ❑ Daily
- ❑ Occasionally
- ❑ Not at all
- ❑ Don't know

13. a) How old were you when you first started regular-smoking? _____age in years

b) How old were you when you first started being regularly exposed to second hand smoke?
_____age in years

14. a) If you have stopped smoking completely, how old were you when you stopped?
_____age in years
❑ Not applicable

b) If you are no longer exposed to regular second-hand smoking, how old were you when that stopped? _____age in years

15. In terms of alcohol consumption, how often do you drink alcohol beverages? The word 'drink' implies one bottle or can of beer or a glass of draft, one glass of wine or a wine cooler, or one straight or mixed drink with one and a half ounces of hard liquor.
- ❑ less than once a month
- ❑ about once a month
- ❑ 2 – 3 times a month
- ❑ about once a week
- ❑ 2 – 3 times a week
- ❑ 4 – 6 times a week
- ❑ every day
- ❑ not at all
- ❑ do not know
- ❑ refused

The following section will ask questions about your interactions with your family, friends, and neighbors. Please put a check mark beside the response that most accurately reflects your current status and fill in the blanks when applicable.

16. About how many families in your neighbourhood are you well enough acquainted with, that you visit each other in your homes? _____families

17. About how many close friends do you have – people you feel at ease with and can talk with about what is on your mind? (you may include relatives.)
_____close friends

18. Over a year's time, about how often do you get together with friends or relatives, like going out together or visiting in each other's homes?	❑ Every day ❑ Several days a week ❑ 2 or 3 times a month ❑ About once a month ❑ 5 to 10 times a year ❑ Less than 5 times a year
19. During the past month, about how often have you had friends over to your home? (Do not count relatives)	❑ Every day ❑ Several days a week ❑ 2 or 3 times a month ❑ About once a month ❑ 5 to 10 times a year ❑ Less than 5 times a year
20. About how often have you visited with friends at their homes during the past month? (Do not count relatives)	❑ Every day ❑ Several days a week ❑ 2 or 3 times a month ❑ About once a month ❑ 5 to 10 times a year ❑ Less than 5 times a year
21. About how often were you on the telephone with close friends or relatives during the past month?	❑ Every day ❑ Several days a week ❑ 2 or 3 times a month ❑ About once a month ❑ 5 to 10 times a year ❑ Less than 5 times a year
22. About how often did you write/ email a letter to a friend or relative during the past month?	❑ Every day ❑ Several days a week ❑ 2 or 3 times a month ❑ About once a month ❑ 5 to 10 times a year ❑ Less than 5 times a year
23. How often have you attended a religious service during the past month?	❑ Every day ❑ Several days a week ❑ 2 or 3 times a month ❑ About once a month ❑ 5 to 10 times a year ❑ Less than 5 times a year

24. In general, how well are you getting along with other people these days?
- ❑ Better than usual
- ❑ About the same
- ❑ Not as well as usual

25. About how many voluntary groups or organizations do you belong to? – like church groups, clubs or lodges, parent groups etc ('Voluntary' implies that you want to).

_____ groups or organizations

26. How active are you in the affairs of these groups or clubs you belong to? If you belong to a great many, just count those you feel closest to. If you don't belong to any, check 4.

- ❑ Very active, attend most meetings
- ❑ Fairly active, attend fairly often
- ❑ Not active, belong but hardly ever go
- ❑ Do not belong to any groups or clubs

The following section asks you some general questions about your health. Please put a check mark beside the response that most accurately reflects your answer and fill in the blanks when applicable.

27. In general, compared to other people your age, how do you rate your health?

- ❑ Excellent
- ❑ Very good
- ❑ Good
- ❑ Fair
- ❑ Poor
- ❑ Do not know
- ❑ Refused

28. How would you rate your ability to handle the day-to-day demands in your life, like work or school or family responsibilities?

- ❑ Excellent
- ❑ Very good
- ❑ Good
- ❑ Fair
- ❑ Poor
- ❑ Do not know
- ❑ Refused

29. How would you rate your ability to handle unexpected or difficult problems, like a family or personal crisis?

- ❑ Excellent
- ❑ Very good
- ❑ Good
- ❑ Fair
- ❑ Poor
- ❑ Do not know
- ❑ Refused

Please fill in the table below by moving **across each row**:

30 a) Were you ever told by a doctor or health professional that you have:	30 b) If yes to a, do you still have this condition?	30c) Are you or were you ever on any medication for this condition?	30 d) If yes to 27 c, do you still take this medication?	30 e) Please specify the type of medication
i) Chronic Bronchitis?	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____
ii) Emphysema?	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____
iii) Asthma?	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____
iv) Lung cancer	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____
v) Other chest conditions?	❑ Yes; Please specify _____ ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____
vi) Any long-term skin conditions?	❑ Yes; Please specify _____ ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____
vii) Hay fever / other allergies?	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____
viii) Arthritis / rheumatism?	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____
ix) Any respiratory problems?	❑ Yes; Please specify _____ ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____
x) High blood pressure / hypertension?	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____
xi) Heart disease?	❑ Yes; Please specify _____ ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____
xii) Any type of cancer?	❑ Yes; Please specify _____ ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____
xiii) Other major health diagnosis?	❑ Yes; Please specify _____ ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	❑ Yes ❑ No; **go to next question** ❑ Do not know; **go to next question**	_____

31. Did you ever have asthma as a child?
- ❑ Yes
- ❑ No
- ❑ Never been told / Do not know

32. Do you have a regular family medical doctor?
- ❑ Yes
- ❑ No
- ❑ Do not know

33. Where do you go most often when you need health care?
- ☐ Family doctor
- ☐ Walk-in clinic
- ☐ Emergency room service
- ☐ Alternative care provider
- ☐ Internet
- ☐ Other, Please specify _____

34 a) Since you left elementary school, have you spent any nights as an in-patient in a hospital for asthma or any respiratory health illness?
Yes
No
Do not know

34 b) Since you left elementary school, how many times have you had emergency room visits for asthma or any respiratory health illness? Please enter a new number below.
_____ times

35) How often do you have a physical medical check-up without having a specific problem?
- ☐ more than once a year
- ☐ about once a year
- ☐ about every two years
- ☐ less often than every two years
- ☐ never

do not know

36. Do you have additional health insurance coverage such as private, government (other than what is available to the general public) or employer-paid plans?
- ☐ Yes
- ☐ No
- ☐ Do not know

37. Do you cough in the morning (not counting just clearing your throat)?
- ☐ Yes
- ☐ No; please go to question 38
- ☐ Do not know; please go to question 38

37i) Do you cough like this most mornings for as much as three months in a row each year?
☐ Yes
☐ No
☐ Do not know

38. Do you usually cough during the day or night, not counting just clearing your throat?
☐ Yes
☐ No; please go to question 39
☐ Do not know; please go to question 39

38i) Do you cough like this most days or nights for as much as three months in a row each year?
☐ Yes
☐ No; please go to question 39
☐ Do not know; please go to question 39

38ii) For how many years have you had a persistent cough? _____

39. Do you usually bring up phlegm from your chest?
☐ Yes
☐ No
☐ Do not know

39i) Do you usually bring up phlegm at all when getting up or first thing in the morning?
☐ Yes
☐ No
☐ Do not know

39ii) Do you usually bring up phlegm at all during the rest of the day or at night?
☐ Yes
☐ No
☐ Do not know

39iii) Do you usually bring up phlegm like this as much as twice a day, 4 or more days out of the week?
☐ Yes
☐ No
☐ Do not know

39 iv) Do you bring up phlegm like this on most days for 3 consecutive months or more during the year?
☐ Yes
☐ No; please go to question 40
☐ Do not know; please go to question 40

---

39 v) For how many years have you had trouble with phlegm? _____years

40) Does your chest ever sound wheezy or whistling?
☐ Yes For how many years has this been present? _____
☐ No
☐ Do no know

41. Have you ever had an attack of wheezing that has made you short of breath?
☐ Yes
☐ No; please go to question 44

i) How old where you when you had your first 'wheeze' attack? _____years

42. Have you had two or more 'wheeze' attacks?
☐ Yes
☐ No; please go to question 44

43. Have you ever required medicine or treatment for these 'wheeze' attacks?
☐ Yes
☐ No

44. When you have a cold, does it usually go to your chest?
☐ Yes
☐ No

45) In the past year, did you participate in any physical activities during your leisure time; for example: walking for exercise, gardening or yard work, swimming, bicycling, popular or social dance, home exercises, ice hockey, ice skating, in-line skating or rollerblading, jogging or running, golfing, exercise class or aerobics, downhill skiing, bowling, baseball or softball, tennis, weight-training, fishing, volleyball, basketball, soccer…
☐ Yes
☐ No; please go to question 46.

45a) How many times did you participate in any of these activities? _____/ day or week or month

45b) About how much time did you spend on average on each occasion?
☐ 1 to 15 minutes
☐ 16 to 30 minutes
☐ 31 to 60 minutes
☐ More than one hour
☐ Do not know

---

46) In the past 12 months, did you experience any of the following:

a) a major financial crisis	❑ Yes ❑ No ❑ Do not know
b) divorced or separated from your spouse or partner?	❑ Yes ❑ No ❑ Do not know
c) experienced a serious illness?	❑ Yes ❑ No ❑ Do not know
c) spouse or child experienced a serious illness?	❑ Yes ❑ No ❑ Do not know
d) spouse or partner die?	❑ Yes ❑ No ❑ Do not know
e) death of a close family member?	❑ Yes ❑ No ❑ Do not know

47. Have you recently:

a) been able to concentrate on whatever you're doing?	❑ Not at all ❑ Same as usual ❑ Less able than usual ❑ Much less able
b) lost much sleep over worry?	❑ Not at all ❑ Same as usual ❑ Less able than usual ❑ Much less able
c) felt that you are playing a useful part in things?	❑ Not at all ❑ Same as usual ❑ Less able than usual ❑ Much less able
d) felt capable of making decisions about things?	❑ Not at all ❑ Same as usual ❑ Less able than usual ❑ Much less able
e) felt constantly under strain?	❑ Not at all ❑ Same as usual ❑ Less able than usual ❑ Much less able
f) felt you couldn't overcome your difficulties?	❑ Not at all ❑ Same as usual ❑ Less able than usual ❑ Much less able
g) been able to enjoy your normal day-to-day activities?	❑ Not at all ❑ Same as usual ❑ Less able than usual ❑ Much less able
h) have been to face up to your problems?	❑ Not at all ❑ Same as usual ❑ Less able than usual

	❑ Much less able
i) been feeling unhappy and depressed?	❑ Not at all ❑ Same as usual ❑ Less able than usual ❑ Much less able
j) been losing confidence in yourself?	❑ Not at all ❑ Same as usual ❑ Less able than usual ❑ Much less able
k) been thinking of yourself as worthless?	❑ Not at all ❑ Same as usual ❑ Less able than usual ❑ Much less able
l) been feeling reasonably happy, all things considered?	❑ Not at all ❑ Same as usual ❑ Less able than usual ❑ Much less able

48) Please indicate by circling the appropriate box, the feelings you have now taking into account what has happened in the last 5 years and what you expect in the near future. How do you feel about

a) your health?	I feel:						
	7 Delighted	6 Pleased	5 Mostly Satisfied	4 Mixed (About equally Satisfied and Dissatisfied)	3 Mostly dissatisfied	2 Unhappy	1 Terrible
b) your income?	I feel:						
	7 Delighted	6 Pleased	5 Mostly Satisfied	4 Mixed (About equally Satisfied and Dissatisfied)	3 Mostly dissatisfied	2 Unhappy	1 Terrible
c) your life in general?	I feel:						
	7 Delighted	6 Pleased	5 Mostly Satisfied	4 Mixed (About equally Satisfied and Dissatisfied)	3 Mostly dissatisfied	2 Unhappy	1 Terrible

Next, we will ask you questions about the health of your biological parents. For each question, please put a check mark beside the response that most accurately reflects your answer and fill in the blanks when applicable.

49. Were either of your natural parents ever told by a doctor / health professional that they had the following conditions:

Medical Condition	Mother	Father
a) Chronic Bronchitis?	❑ Yes ❑ No ❑ Do not know	❑ Yes ❑ No ❑ Do not know
b) Emphysema?	❑ Yes ❑ No ❑ Do not know	❑ Yes ❑ No ❑ Do not know
c) Asthma?	❑ Yes ❑ No ❑ Do not know	❑ Yes ❑ No ❑ Do not know
d) Lung cancer?	❑ Yes ❑ No ❑ Do not know	❑ Yes ❑ No ❑ Do not know
e) Other chest conditions?	❑ Yes ❑ No ❑ Do not know	❑ Yes ❑ No ❑ Do not know

50. Please provide information on your biological parents:

	Mother	Father
a) Is your parent currently alive?	❑ Yes ❑ No; **please go to c** ❑ Do not know	❑ Yes ❑ No; **please go to c** ❑ Do not know
b) What is the age of your parent?	_____years of age **Please go to question 51**	_____years of age **Please go to question 51**
c) What was the age at death? Reason for death	_____years of age	_____years of age

Finally, we would like to ask a few questions about you:

51a. In what year where you born? _____

51b. What is your sex:

❑ Male
❑ Female

51c. How tall are you without shoes on? _____ m □ / cm □ / ft □ / in □

51d. How much do you weigh? _____kg □ / lb □

51e. What is your marital status?

- ❑ Single
- ❑ Married / Common-law
- ❑ Widowed
- ❑ Separated / Divorced
- ❑ Other

52a. How many people live in your household including yourself? _____

52b. Are any of your household members less than 18 years of age?

- ❑ Yes
- ❑ No

53. What is your highest level of education?

- □ No schooling
- □ Some elementary school
- □ Completed elementary school
- □ Some high school / junior high school
- □ Completed high school
- □ Some community college
- □ Some technical school (CEGEP, Collège Classique)
- □ Completed community college
- □ Completed technical school (CEGEP, Collège Classique)
- □ Some university
- □ Completed bachelor's degree (Arts, Sciences, etc..)
- □ Post graduate training: MA, MSc, MLS, MSW, etc…
- □ Post graduate training: PhD, 'doctorate'

54. Could you please tell me how much income you and all other members of your household received in the past year? Be sure to include FROM ALL SOURCES such as savings, pensions, rent, and unemployment insurance as well as wages.

- □ less than $20, 000
- □ between $20,000 and $30,000
- □ between $30,000 and $40,000
- □ between $40,000 and $50,000
- □ between $50,000 and $60,000
- □ between $60,000 and $70,000
- □ between $70,000 and $80,000
- □ between $80,000 and $100,000
- □ more than $100,000
- □ Do not know
- □ Refused

55a. To what ethnic or cultural group do you belong?

- ❑ Canadian
- ❑ Australian
- ❑ Austrian
- ❑ Bahamian
- ❑ Bangladeshi
- ❑ Black / African
- ❑ British
- ❑ Chinese
- ❑ Croatian
- ❑ Czech
- ❑ Danish
- ❑ Dutch
- ❑ English
- ❑ El Salvador
- ❑ Ethiopian
- ❑ French
- ❑ Finnish
- ❑ German
- ❑ Greek
- ❑ Guyanese
- ❑ Haitian
- ❑ Holland
- ❑ Hungarian
- ❑ Irish
- ❑ Italian
- ❑ Indian
- ❑ Israeli
- ❑ Jamaican
- ❑ Japanese
- ❑ Jewish
- ❑ Korean
- ❑ Lebanese
- ❑ Macedonian
- ❑ New Zealand
- ❑ Netherlands
- ❑ Nigerian
- ❑ Norwegian
- ❑ Pakistani
- ❑ Philippine
- ❑ Polish
- ❑ Portuguese
- ❑ Russian
- ❑ Scottish
- ❑ Serbia
- ❑ Sikh
- ❑ Somalia
- ❑ Slovakian
- Spanish
- ❑ Sri Lanka
- ❑ Swedish
- ❑ Tamil
- ❑ Trinidadian
- ❑ Ukrainian
- ❑ Vietnamese
- ❑ Yugoslavian
- ❑ Welsh
- ❑ Inuit, Métis,
- ❑ Aboriginal

- ❑ Other; please specify _____
- ❑ Do not know

55b. Were you born in Canada?

- ☐ Yes
- ☐ No
- ☐ Do not know

Thank You for Taking the Time to Complete this Survey!

ID # _____

Appendix 3.7 Itemization of variables related to the macro environment component of the survey.			
Factors related to:	Item	Variable measured	Categorizing the variable
Physical environment	12	Second-hand smoking	Daily / occasional / never
Residential history	6	Length at residence	Continuous
	6iii	Property description	Detached / Semi / Attached / 1 or 2 bedroom Condo or Apartment / 3+ bedrooms Condo
	6iv	Age of property	Continuous
	6v	Type of heating	Gas or oil / other
	6g	Exposure to contaminant/ pesticide / insecticide	Yes / No
	6ii	Number in household	Continuous
	6a	Heating system	Forced air and gas heater / Forced air without gas heater / Not forced air with gas heater / Not forced air without gas heater / Gas space heater
	6b	Air duct cleaning	Regular / Not regular
	6c	Cooking method	Gas / Other
	6d, e, f	Air conditioner, humidifier, filter / purifier	Yes / No and frequency of use
	6i	Property ownership	Own / rent
	7, 8	Residential history	For up to 4 previous residences

Appendix 3.7 continued			
Factors related to:	Item	Variable measured	Categorizing the variable
Occupational history	1	Employment status	Full time / not full-time
	2	Type of employment	Clerical / Manual / Managerial / Professional
	3	Length of employment	Open-ended continuous variable
	4a	Exposure to dust	Yes / No; If 'Yes', then categorize intensity as mild, moderate, or severe.
	4b	Exposure to gas / fume / chemicals	Same as above
	4c	Exposure to any other contaminant	Same as above
	5	Previous occupations	For up to 12 previous occupations.
Social environment	16 - 26	Social networking index	Social contact subscale (scale of 10) / Group Participation scale of 9.
Family environment	51e	Marital status	Single / not single
	52	Number of children	Continuous
	49, 50	Parental health	Parental diagnosis for respiratory diseases; age of parents / age and reason of death
Life events	46a to e	Life events	Yes or no for each of the following: financial crisis, divorce / separation, illnesses, spousal or family member death

Appendix 3.7 continued			
Factors related to:	Item	Variable measured	Categorizing the variable
Healthcare access	31	Regular family doctor	Yes or no
	32	Type of healthcare access	Family doctor , Walk-in clinic, Emergency room service, Alternative care provider, Internet, or other
	34a	Medical check-ups	Frequency of medical check-ups without specific problem
	35	Additional health insurance	Any additional health insurance coverage
Demographic Factors	51a	Date of birth	Year of birth
	51b	Sex	Male / Female
	53	Education	Completed / did not complete high school
	54	Income	Above or below low-income cutoff
	55a	Ethnicity	Canadian / Canadian and other / other
	55b	Born in Canada	Yes / no

Appendix 3.8 Itemization of variables related to the micro environment component of the survey.			
	Item	Variable measured	Categorizing the variable
Physiological	51c	Height	Continuous
	51d	Weight	Continuous
Psychosocial factors	6h, i, j	Perception to air pollution	- Concern over air pollution - Perception that air pollution will lead to health impacts
	47	Emotional distress	General Health Questionnaire (12) – measure of emotional distress
	48	Feelings about health, income, and life	Scale of 1 (terrible) to 7 (delighted)
Behavioural variables	9	Current smoking	Daily / occasionally / never
	10	Ever daily smoke	Yes / No
	11	Frequency of smoke (number of cigarettes)	Continuous
	13	Age when smoking began	Continuous
	14	Age when smoking stopped	Continuous
	15	Alcohol consumption	Categorized frequency
	45	Physical activity	Time spent doing physical activities during leisure time

Appendix 3.9 Itemization of variables related to health outcomes.			
Factors related to:	Item	Variable measured	Categorizing the variable
Functional capacity	28	Coping – day to day	Excellent / very good / good versus poor / fair
	29	Coping – hard times	Excellent / very good / good versus poor / fair
Respiratory symptoms	37, 38	Cough	- Morning cough - Persistent morning cough for more than 3 consecutive months per year - Day or night cough - Persistent day or night cough Number of years cough present
	39	Phlegm	- Morning phlegm - Day or night phlegm - Persistent phlegm for more than 3 consecutive months per year - Number of years with phlegm problems
	40 - 43	Wheeze	- Wheeze symptoms and duration Frequency of wheeze attacks (2 or more) - Age at which wheeze began Medication use
	44	Chest colds	Yes or no
	31	Asthma in childhood	Yes or no

Appendix 3.9 continued			
Factors related to:	Item	Variable measured	Categorizing the variable
Medical diagnosis	30a	Respiratory illnesses	- Chronic Bronchitis - Asthma - Emphysema - Any respiratory problems - Medication use
	30b	Other non-respiratory conditions	- Lung cancer - Chest conditions - Long-term skin conditions - Hay fever / allergies - Arthritis / rheumatism - High blood pressure / hypertension Heart disease - Any type of cancer - Other health diagnosis - Medication use
Hospitalization	34	Hospitalization for asthma or respiratory illness since leaving elementary school	- Yes / no - Number of times hospitalized since leaving elementary school
Self-assessed health	27	Self – rated health	In relation to others from the same age

Appendix 3.10 Derived variables that correspond to the macro-environment component of the conceptual framework.	
PHYSICAL ENVIRONMENT	
Exposure to air pollution	Exposure to TSP (1980/1) ($\mu g/m^3$); Continuous and categorical (above median or at / below median)
	Exposure to TSP (1983/4) ($\mu g/m^3$); Continuous and categorical (above median or at / below median)
	Exposure to TSP (1985/6) ($\mu g/m^3$); Continuous and categorical (above median or at / below median)
	Index for exposure to TSP: index was created based on categorical data for the 3 sampling periods
	Exposure to $PM_{3.3}$ (1980/1) ($\mu g/m^3$); Continuous and categorical (above median or at / below median)
	Exposure to $PM_{3.3}$ (1981/2) ($\mu g/m^3$); Continuous and categorical (above median or at / below median)
	Exposure to $PM_{3.3}$ (1983/4) ($\mu g/m^3$); Continuous and categorical (above median or at / below median)
	Exposure to $PM_{3.3}$ (1985/6) ($\mu g/m^3$); Continuous and categorical (above median or at / below median)
	Index for exposure to $PM_{3.3}$: index was created based on categorical data for the 4 sampling periods
	Exposure to SO_2 (1978/9) (ppb); Continuous and categorical (above median or at / below median)
	Exposure to SO_2 (1980/1) (ppb); Continuous and categorical (above median or at / below median)
	Exposure to SO_2 (1981/2) (ppb); Continuous and categorical (above median or at / below median)

Appendix 3.10 continued	
PHYSICAL ENVIRONMENT	
Exposure to air pollution	Exposure to SO_2 (1983/4) (ppb); Continuous and categorical (above median or at / below median)
	Index for exposure to SO_2: index was created based on categorical data for the 3 sampling periods
	Second-hand exposure to smoke at home or at work (current) – yes / no
	Daily exposure to smoking in childhood – continuous variable from 0 to 5 packs per day
	Daily exposure to smoking in childhood – yes / no
RESIDENTIAL HISTORY	
Neighbourhood of residence in childhood in relation to west upper, west lower, east upper, and east lower quadrants	
Current location: Hamilton, Other in Ontario, Other	
Residential exposures to contaminants, pesticides, or insecticides – Yes / no	
Number of previous residences – Continuous from 1 to 5	
Type of dwelling – detached / semi-detached / condo or apartment	
Length of exposure to contaminants (months) - Continuous	
Exposures (pesticides, insecticides, or contaminants) greater than 2 years – Yes / no	
Exposures (pesticides, insecticides, or contaminants) greater than 5 years – Yes / no	
Exposures (pesticides, insecticides, or contaminants) greater than 8 years – Yes / no	
Length of time in property built before 1950 (years) - Continuous	

Appendix 3.10 continued
RESIDENTIAL HISTORY
Property ownership – own versus rent
Air conditioner available – yes / no
Frequency of use of air conditioner – always, almost always, rarely, not applicable
Air humidifier available – yes / no
Frequency of use of air humidifier – always, almost always, rarely, not applicable
Air purifier / filter available – yes / no
Frequency of use of air purifier / filter – always, almost always, rarely, not applicable
Type of heating – gas or oil / other
Frequency of air duct cleaning – regular (every year) / not regular (as needed, never)
Current gas cooking – yes /no
Microwave cooking – yes / no
Gas cooking in childhood - yes / no
OCCUPATIONAL HISTORY
Current employment status – full-time / not full-time
Type of occupation – clerical / manual / managerial / professional
Length of time to occupational exposures (months) - Continuous
Type of exposure – dust / gas/ fume / chemicals / contaminants
Exposure to gas / dust / fumes / contaminants for more than 2 years – Yes / no

Appendix 3.10 continued
OCCUPATIONAL HISTORY
Exposure to gas / dust / fumes / contaminants for more than 5 years – Yes / no
Exposure to gas / dust / fumes / contaminants for more than 8 years – Yes / no
SOCIAL ENVIRONMENT
Social contact scale – good (score more than 5) / not good (score of 5 or less)
Group participation scale – good (score more than 4 / not good (score of 4 or less)
FAMILY ENVIRONMENT
Marital status – single / not single
Parental health – at least one record (maternal / paternal) of asthma or respiratory problems versus no record of asthma or respiratory problems
LIFE EVENTS
Experienced 2 or more stressful life events – Yes / no
HEALTHCARE SYSTEM
Have a regular family doctor – yes / no
Type of healthcare – traditional (family doctor, walk-in, ER) / non-traditional (alternative, internet)
Additional health insurance coverage – yes / no

Appendix 3.10 continued
DEMOGRAPHIC FACTORS
Gender – male / female
Education – completed high school / did not complete high school
Income – above the low-income cutoff / below the low-income cutoff (derived using the variables for family income and number of people in the household)
Place of birth – Canada / other
Ethnicity – Canadian / Canadian and other / other
Income in childhood – above low income cutoff / below low income cutoff (assumed form original dataset

Appendix 3.11 Derived variables that correspond to the micro-environment construct of the conceptual framework.
PHYSIOLOGICAL PATHWAYS
Asthma in childhood – yes / no
Index for persistent morning cough in childhood – yes/no based on 5 sampling periods
Index for persistent day or night cough in childhood - yes/no based on 5 sampling periods
Index for persistent wheeze in childhood - yes / no based on 5 sampling periods
Index for chest cold in childhood - yes / no based on 5 sampling periods
Chest illness before the age of two - yes / no
Other chest illnesses (bronchitis, pneumonia, other) – yes / no
Index for pulmonary function based on FEV_1/FVC data – participants were classified above or below the median for each sampling period – index was created based on the five sampling periods
Body mass index (using height and weight) – BMI below 25 / equal or above 25
PSYCHOLOGICAL PATHWAYS
Concern over air pollution – index created using items 6h, 6i, 6j
Emotional distress – score of 4 or more on the General Health Questionnaire – Yes / no
Feelings about life – delighted / pleased / mostly satisfied / mixed (score more than 3) / mostly dissatisfied / unhappy / terrible (score 3 or less)
Feelings about income - delighted / pleased / mostly satisfied / mixed (score more than 3) / mostly dissatisfied / unhappy / terrible (score 3 or less)

Appendix 3.11 continued
Feelings about health - delighted / pleased / mostly satisfied / mixed (score more than 3) / mostly dissatisfied / unhappy / terrible (score 3 or less)
BEHAVIOURAL PATHWAYS
Current smoking – yes / no
Ever daily smoker – yes / no
Alcohol consumption – regular (every day or almost every day) / not regular (other)
Regular physical exercise – regular (at least 30 minutes for 3 or more days per week) / not regular (other)
Smoking in childhood – more than one cigarette per day for more than a 6 month period – yes / no

Appendix 3.12 Variables that correspond to health outcomes in adulthood.
FUNCTIONAL CAPACITY
Over day to day events - excellent, very good, or good / fair or poor
Over difficult or unexpected problems - excellent, very good, or good/ fair or poor
MEDICAL DIAGNOSIS
Chronic bronchitis – yes / no; if yes, medication use.
Chest conditions – yes / no; if yes, medication use.
Long-term skin conditions – yes / no; if yes, medication use.
Hay fever or allergies – yes / no; if yes, medication use.
Arthritis or rheumatism - yes / no; if yes, medication use.
Asthma or respiratory problems – variables combined – yes / no; if yes, medication use.
High blood pressure or hypertension – yes / no; if yes, medication use.
Heart disease – yes / no; if yes, medication use.
Ever diagnosed with asthma – yes / no; if yes, medication use.
Diagnosed with asthma in adulthood but not in childhood – yes / no
Any type of cancer – yes / no; if yes, medication use.
HOSPITALIZATION OF EMERGENCY ROOM VISITS
Hospital or emergency room visits for asthma or respiratory illnesses since leaving elementary school – Yes / no

Appendix 3.12
RESPIRATORY SYMPTOMS
Persistent morning cough – Yes / no
Persistent day or night cough – Yes / no
Persistent phlegm – Yes / no
Persistent wheeze – Yes / no
Any respiratory symptoms reported – Yes / no
SELF-ASSESSED HEALTH
Self-rated health – excellent, very good, or good / fair or poor

Appendix 5.1 Logistic regression models for diagnosis with any type of cancer.

Variable (Reference)	Classification	Overall sample (n=360)		Females (n = 154)	
		OR	95.0% CI	OR	95.0% CI
Length of occupational exposure to gas / dust / fumes	Increasing	1.01**	(1.00, 1.02)	1.018**	(1.01, 1.03)
Residential exposure to gas / dust / contaminants (≤ 2 years)	> 2 years	9.03*	(1.02, 101.66)		
Specificity (%)			98.3		98
Sensitivity (%)			0		33.3
Rho-square			0.30		0.31

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Appendix 5.2 Long-term exposure to air pollution and diagnosis with any type of cancer.

Variable (Reference)	Classification	Overall sample (n=378)		Females (n = 159)	
		OR	95.0% CI	OR	95.0% CI
Length of occupational exposure to gas / dust / fumes	Increasing	1.03*	(1.00, 1.06)	1.08	(0.98, 1.18)
Residential exposure to gas / dust / contaminants (≤ 2 years)	> 2 years	113.80	(0.72, 1.79^{4})		
	East lower	4.66^{8}	(0.00, NL)	2.65^{16}	(0.00, NL)
	West lower	11.52	(0.00, NL)	197.84	(0.00, NL)
	East upper	9.44^{8}	(0.00, NL)	4.80^{6}	(0.00,NL)
Index for TSP exposure (≤ median)	> median	0.11	(0.00, 1.51^{3})	0.00	(0.00, 2.88^{4})
Index for $PM_{3.3}$ exposure (≤median)	> median	0.30	(0.00, 1.83^{4})	2.38^{8}	(0.00, 8^{21})
Index for SO_2 exposure (≤median)	> median	3.42	(0.01, 1.76^{3})	0.00	(0.00, 1.38^{5})
Specificity (%)			98.6		98.8
Sensitivity (%)			75.0		100
Rho-square			0.59		0.76

NL – no limit; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Appendix 5.3 Logistic regression models for fair or poor ability to handle day-to-day demands.

Variable (Reference)	Classification	Overall sample (n=392)		Females (n = 190)		Males (n = 204)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Place of residence (Always in Hamilton)			(1.27, 4.56)				
	Other in Ontario	4.46*	(0.44, 45.66)				
	Outside Ontario	10.50	(0.99, 111.16)				
Feel about income (Delighted /pleased /mostly satisfied /mixed)	Mostly dissatisfied / unhappy / terrible	2.96*	(1.30, 6.75)	3.09*	(1.05, 9.57)		
Emotional distress (<4 on GHQ)	≥ 4 on the GHQ	6.03***	(2.75, 13.21)	5.39**	(1.80, 16.12	7.72***	(2.87, 20.77)
Heart disease (Not case)	Case	39.30**	(1.74, 890.57)			14.32*	(1.05, 195.31)
Specificity (%)			91.1		82.9		91.6
Sensitivity (%)			45.5		55.0		44.0
Rho-square			0.16		0.10		0.13

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Appendix 5.4 Long-term exposure to air pollution in childhood and fair or poor ability to handle day-to-day demands.

Variable (Reference)	Classification	Overall sample (n=392)		Females (n = 189)		Males (n = 204)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Place of residence (Always in Hamilton)*							
	Other in Ontario	3.26	(0.39, 27.62)				
	Outside Ontario	7.51	(0.87, 65.13)				
Feel about income (Delighted /pleased /mostly satisfied /mixed)	Mostly dissatisfied / unhappy / terrible	2.51*	(1.10, 5.73)	3.35*	(1.03, 10.86)		
Emotional distress (<4 on GHQ)	≥ 4 on the GHQ	6.65***	(3.01, 14.71)	6.27**	(1.96, 20.00)	9.19***	(2.98, 28.35)
Heart disease (Not case)	Case	11.84	(0.97, 144.00)			14.23	(0.97, 207.83)
Residence in childhood (West upper)							
	East lower	0.68	(0.23, 1.99)	0.61	(0.12, 3.17)	0.50	(0.11, 2.37)
	West lower	0.61	(0.19, 1.98	1.16	(0.24, 5.73)	0.28	(0.05, 1.59)
	East upper	0.78	(0.27, 2.70)	0.49	(0.11, 2.20)	1.04	(0.21, 5.18)
Index for TSP exposure (≤ median)	> median	0.98	(0.24, 4.13)	0.88	(0.09, 8.29)	1.19	(0.18, 7.95)
Index for $PM_{3.3}$ exposure (≤median)	> median	1.55	(0.28, 8.57)	1.02	(0.08, 13.35)	2.28	(0.20, 26.11)
Index for SO_2 exposure (≤median)	> median	1.93	(0.42, 8.78)	3.06	(0.32, 29.55)	1.21	(0.15, 9.95)
Specificity (%)			85.8		87.0		91.1
Sensitivity (%)			53.3		55.0		44.0
Rho-square			0.15		0.12		0.15

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Appendix 5.5 Logistic regression models for fair or poor ability to handle difficult or unexpected problems.

Variable (Reference)	Classification	Overall sample (n=312)		Females (n = 190)		Males (n = 204)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Marital status (Not single)	Single	2.36*	(1.23, 4.53)			2.47*	(1.13, 5.41)
Education (Completed high school)	Did not complete high school	8.78*	(1.42, 54.26)	5.32*	(1.02, 27.88)		
Social networking (> 5 on Social Contact Scale)	≤ 5 on Social Contact Subscale	2.52*	(1.10, 5.77)				
Chest conditions (Not case)	Case	5.33**	(1.87, 15.22)			5.62*	(1.37, 23.17)
Exposure to smoking (Not exposed	Exposed					2.95**	(1.32, 6.60)
Specificity (%)			68.9		0		48.5
Sensitivity (%)			60.3		100.0		88.6
Rho-square			0.09		0.02		0.11

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

Appendix 5.6 Long-term exposure to air pollution in childhood and fair or poor ability to handle difficult or unexpected problems.

Variable (Reference)	Classification	Overall sample (n=392)		Females (n = 189)		Males (n = 204)	
		OR	95.0% CI	OR	95.0% CI	OR	95.0% CI
Marital status (Not single)	Single	1.92*	(1.06, 3.47)			2.72*	(1.21, 6.10)
Education (Completed high school)	Did not complete high school	4.94*	(1.16, 20.95)	7.11*	(1.39, 36.46)		
Social networking (> 5 on Social Contact Scale)	≥ 5 on Social Contact Subscale	2.50*	(1.17, 5.36)				
Chest conditions (Not case)	Case	4.37**	(1.67, 11.39)			5.00*	(1.19, 21.01)
Exposure to smoking (Not exposed	Exposed					3.10**	(1.36, 7.10)
Residence in childhood (West upper)							
	East lower	1.50	(0.61, 3.72)	1.11	(0.28, 4.31)	1.89	(0.53, 6.78)
	West lower	1.13	(0.41, 3.05)	1.76	(0.45, 6.90)	0.69	(0.15, 3.05)
	East upper	1.31	(0.55, 3.10)	1.07	(0.33, 3.47)	2.84	(0.75, 10.78)
Index for TSP exposure (≤ median)	> median	2.07	(0.67, 6.32)	2.46	(0.47, 12.96)	1.64	(0.36, 7.53)
Index for $PM_{3.3}$ exposure (≤median)	> median	0.90	(0.23, 3.54)	0.70	(0.10, 4.95)	2.37	(0.31, 17.93)
Index for SO_2 exposure (≤median)	> median	0.56	(0.16, 1.98)	0.89	(0.16, 4.93)	0.23	(0.04, 1.56)
Specificity (%)			67.8		52.5		63.9
Sensitivity (%)			57.6		64.5		82.9
Rho-square			0.08		0.06		0.14

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.