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University of Alberta

Atmospheric Risk Factors of Human Mortality

by

Daniel Gareth Charles Rainham 

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfilment of the requirements for the degree of Master of Science

Department of Earth and Atmospheric Sciences

Edmonton, Alberta

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Dr. Karen E. Smoyer

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Dr. Colin L. Soskolne

Dated: Oct 2, 2000

Abstract

The associations of weather (Chapter Two) and air pollution (Chapter Three) with nonaccidental mortality among the population of Metropolitan Toronto from 1980 to 1996 were examined using an ecological study design. A quasi time series approach was employed using nonparametric scatterplot smoothers and generalized additive models. Statistically significant associations were found between variations of humidex and elderly, cardiac and respiratory mortality. Moderate risk of mortality attributable to concentrations of air pollution was also evident. Relationships tended to vary by season and by synoptic situation (weather). The results suggest that mortality among the elderly and those with pre-existing health conditions varies with both weather and with concentrations of air pollution. The results also indicate the potential for interaction between air pollution and weather on warm, summer days.

Dedication

I dedicate this thesis to my beautiful,
dedicated and brilliant soulmate,
Tarah Wright.

Acknowledgements

I acknowledge the spirit, leadership, and intellect of my mentor and supervisor, Dr. Karen Smoyer. Her guidance was invaluable. Her belief in my abilities never wavered. I will certainly miss our chats and "brainstorming" sessions.

I would like to acknowledge the guidance and input from the committee, Dr. John Wilson and Dr. Colin Soskolne. Your suggestions and comments were invaluable. I am also particularly grateful for your participation on the committee so late in the process.

I would also like to thank Dr. Rick Burnett for the mortality and air pollution data. The third chapter of this thesis would not exist without it. I am also thankful to Dr. Burnett for his willingness to discuss statistical methodologies and the intricacies of federal environmental policy. Thanks are also warranted for Marc-Smith Dorion and the rest of the air pollution research group at the Environmental Health Directorate of Health Canada.

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Finally, I would like to acknowledge all of those who have bestowed their support to me over the past two years, including my family, all of the hardworking support staff from the Department of Earth and Atmospheric Sciences, Bar Tecka coffee, the Clan from Waterloo, Max's Light Cuisine, and all of those people who have had to put up with my rants on the environment and its associated issues.

Table of Contents

CHAPTER 3 (Continued)

List of Tables

List of Tables (continued)

List of Tables (continued)

List of Figures

List of Abbreviations

GAM	Generalized Additive Model
AIC	Akaike's Information Criterion
LOESS	Locally Weighted Scatterplot Smoothing
RR	Relative Risk
CV	Coefficient of Variation
CO	Carbon Monoxide
NO_2	Nitrogen Dioxide
SO_2	Sulfur Dioxide
O_3	Ozone
PM_{10}	Particulate Matter of 10 or less microns in diameter
$PM_{2.5}$	Particulate Matter of 2.5 or less microns in diameter
PPM	Parts Per Million
PPB	Parts Per Billion
SSC	Spatial Synoptic Classification
DM	Dry Moderate
DP	Dry Polar
DT	Dry Tropical
MM	Moist Moderate
MP	Moist Polar
MT	Moist Tropical
TR	Transition
CC	Cloud Cover
SLP	Sea Level Pressure

CHAPTER 1

An Introduction to Atmospheric Human Health Risks

Whoever wishes to investigate medicine properly should proceed thus: in the first place to consider the seasons of the year, and what effects each of them produces for the seasons are not alike, but differ widely both in themselves and at their changes (Hippocrates, from *Airs, Waters, Places*).[1]

We depend on the life-sustaining properties of the atmosphere. It protects us from radiation, maintains climatic equilibrium, and supplies all species with necessary elements of carbon dioxide, water, and oxygen. Recently, however, consternation over the health effects of diminishing atmospheric quality has evolved in conjunction with a belief that an increasingly human-dominated planet, provoked by a progressively destructive attitude towards nature, is altering environmental quality in an unparalleled fashion.

It has long been known that specific atmospheric processes, such as weather and longer-term climatic fluctuations, affect human health. The biometeorological literature refers to this relationship as *meteorotropism*, defined as a change in an organism that is correlated with a change in atmospheric conditions.[2] Extreme examples of this relationship can be drawn from the recent Chicago heat wave in 1995. It is alleged that high temperatures, combined with debilitating socio-economic factors, were responsible for over 500 deaths.[3] Of course, the ravages of weather on human health are largely beyond our control. However, these effects and subsequent risks may be intensified by many factors, including the

addition of air pollutant emissions from anthropogenic activities to the atmosphere. This thesis explores how potential atmospheric risk factors, such as weather extremes and air pollution, affect human mortality.

The effects of environmental chemical exposures on human health, particularly their combined or synergistic effects, remain largely unknown. In North America, Rachel Carson proposed the build-up of various chemicals and toxins in the ambient environment and their potential to affect human health in 1962.[4] Presently there are over 70 000 chemicals in use and between 500 and 1000 new ones manufactured each year.[5] Humans have emerged as a relatively new force of nature and have modified physical, chemical, and biological systems in novel ways, at faster rates, and over larger spatial scales than have non-human processes. We have embarked on a grand experiment with the planet; little is currently known of potential consequences, however. It is this author's opinion that many of the environmental changes will have serious consequences, the largest related to human health and well-being.

Human exploitation of the atmosphere, as a reservoir for the many pollutants and products of industrial processes, has allowed mainly industrialized and semi-industrialized societies to maintain unsustainable levels of consumption and production. Current societal emphasis is placed on expansion of these processes in an attempt to raise the level of industrialization on a global scale with a conscious, yet tacit understanding of the environmental consequences. This thesis will not discuss the intrinsic values and conflicts associated with this trend. The claim merely suggests that, like many environmental issues, pollution of the air is an inherent constituent of the modern social fabric and thus is not confined to just epidemiological scrutiny. Therefore the study of air pollution is also influenced by social, cultural, economic, and political perspectives.

The topic of air pollution and health effects from weather also has notable consequence for sustainability and equity. Readily transparent is the fact that the atmosphere, like so many other reservoirs, is a finite resource with a limited capacity. The constant motion of the atmosphere frees pollutants from the confines of any political boundaries and enable many emissions to concentrate in areas with specialized atmospheric weather patterns and geographical topographies. In areas with large populations and high population densities, as well as with marked microclimatic variations from urban structures and stagnant air, high pollutant concentrations combined with stable weather conditions can have quite dramatic health effects.[6]

Environmental epidemiology, an area of research in which potential relationships between human health and the environment are explored,[7] often examines the health effects of air pollution. Many epidemiologists would envisage the environment as comprising everything that is not genetic; this would include such things as diet, housing, smoking, and even exercise. With many similarities to infectious disease epidemiology, environmental epidemiology encompasses the study of more than just physical and chemical agents, however. Environmental epidemiology, rather, is the study of those factors beyond the direct control of the individual, and includes the study of exposures such as air pollution and occupational hazards, as well as psychosocial elements of environmental concern.[8]

Research on the health effects of various environmental pollutants has been difficult to conduct and causal inference problematical to ascertain.[9] The methodologies derived from recent advances in environmental epidemiology are well suited to the study of relationships between air pollution and human mortality. Although methodologic

issues still exist with respect to studies in environmental epidemiology,[i] new methods and alternative approaches to examining health/environment relationships are continually in development. More pertinent methodologic issues associated with studies of human health risk from air pollution are found elsewhere.[10]

Yet epidemiologic research on the topic of air pollution and human health remains controversial. Many air pollution studies fail to account for (i.e., control adequately) the health effects from natural atmospheric processes such as weather. Fortunately, alternative and more interdisciplinary methods have been proposed that allow more integration of diverse approaches. These methods to explore temporal relationships may prove useful in revealing potentially interactive or synergistic atmospheric factors of health risk.

1.1 Motivation

This thesis concentrates on the connection between the applied climatological and air pollution risk literature by examining the relationships between city-level conditions of atmospheric quality and human mortality in Metropolitan Toronto, Canada's largest and most urbanized city. The methodology will utilize both descriptive and quantitative approaches, including the employment of non-parametric statistical approximations, to evaluate variations in human mortality over a seventeen-year period from 1980 to 1996.

This study has two goals:

1) to consolidate methodological frameworks from investigations of human health outcomes from atmospheric risk factors, and

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[i] Environmental Health Perspectives devoted an entire issue to environmental epidemiology and pertinent methodological issues. See *Environmental Health Perspectives* 101(Suppl 4), 1993.

2) to provide comprehensive information on the epidemiology of human mortality from air pollution in an urban setting.

Achieving these goals will require the synthesis of theories and methodologies from traditionally disparate fields and specializations.

My motivation for exploring the association between mortality and the holistic components of atmospheric risk factors is three-fold. First, human health and well being are increasingly being recognized as dependent on environmental components and quality. To study these dependencies will require the development of integrated and unique methodologic approaches. For example, it is purported that even low ambient levels of air pollution in combination with foreseeable impacts from climatic change will result in both direct and indirect human and ecological health impacts, including increases in heat stress, as well as cardio-respiratory and allergic health complications.[11] Integrated forms of modeling and environmental prediction from statistical models are being used to evaluate how changes in environmental quality encourage modifications in the spatial distribution and virulence of an assortment of diseases, including hantavirus and dengue fever.[12] Although a much less complex investigation, I hope that the approach taken and results presented from this study will contribute to a more informed understanding of the inextricable link between the environment and human health.

Second, ambient concentrations of air pollution are a major contributor to premature mortality and morbidity-related hospital admissions in Canada. Even though the concentration of many air pollutants has declined over the past two decades (Figure 1.1), it is estimated that in eleven of Canada's largest cities, approximately 5 000 people per year die prematurely as a result of air pollution.[13] This number is potentially exacerbated by weather events, such as heat waves, that have been shown to contribute to considerable short-term human health

effects.[14] This study will investigate potential interactions and synergism between air pollution concentrations and weather conditions in an attempt to quantify potential resulting fluctuations in human mortality.

Third, human health effects from atmospheric risk factors are characteristically ecological and inherently complex. Hence, little effort will be devoted to the assessment of potentially causal relationships. Nevertheless, an investigation of atmospheric risk factors in human mortality affords the opportunity to investigate the utility of recently developed methodologies[ii] in environmental epidemiology. Furthermore, this study will inform enquiries into the development of experimental study designs and unite research from, traditionally, theoretically-distinct disciplines.

1.2 Consequence of the Research

This research contributes to our understanding of the atmospheric environment and human health in several ways. First, it offers estimates on the interplay between atmospheric risk factors of human mortality that will provide valuable information to public health policy officials. Mortality and, more generally, human health outcomes from air pollution and extreme weather events, is a current issue of concern for emergency health planning in Metropolitan Toronto.[15] Identification of susceptible populations and potentially harmful atmospheric conditions can be used for the development of warning systems, modifications to existing public environments, and proactive decision-making.

[ii] This research will make use of methods developed in the fields of climatology, epidemiology, and statistics. See Kalkstein's original work (1991) on estimations of mortality using synoptic climatological approaches in *Environmental Health Perspectives* 96: 124-150. See also the discussion by Schwartz on robust modern statistical approaches to evaluating health outcomes from air pollution in *The Canadian Journal of Statistics* 22: 471-487.

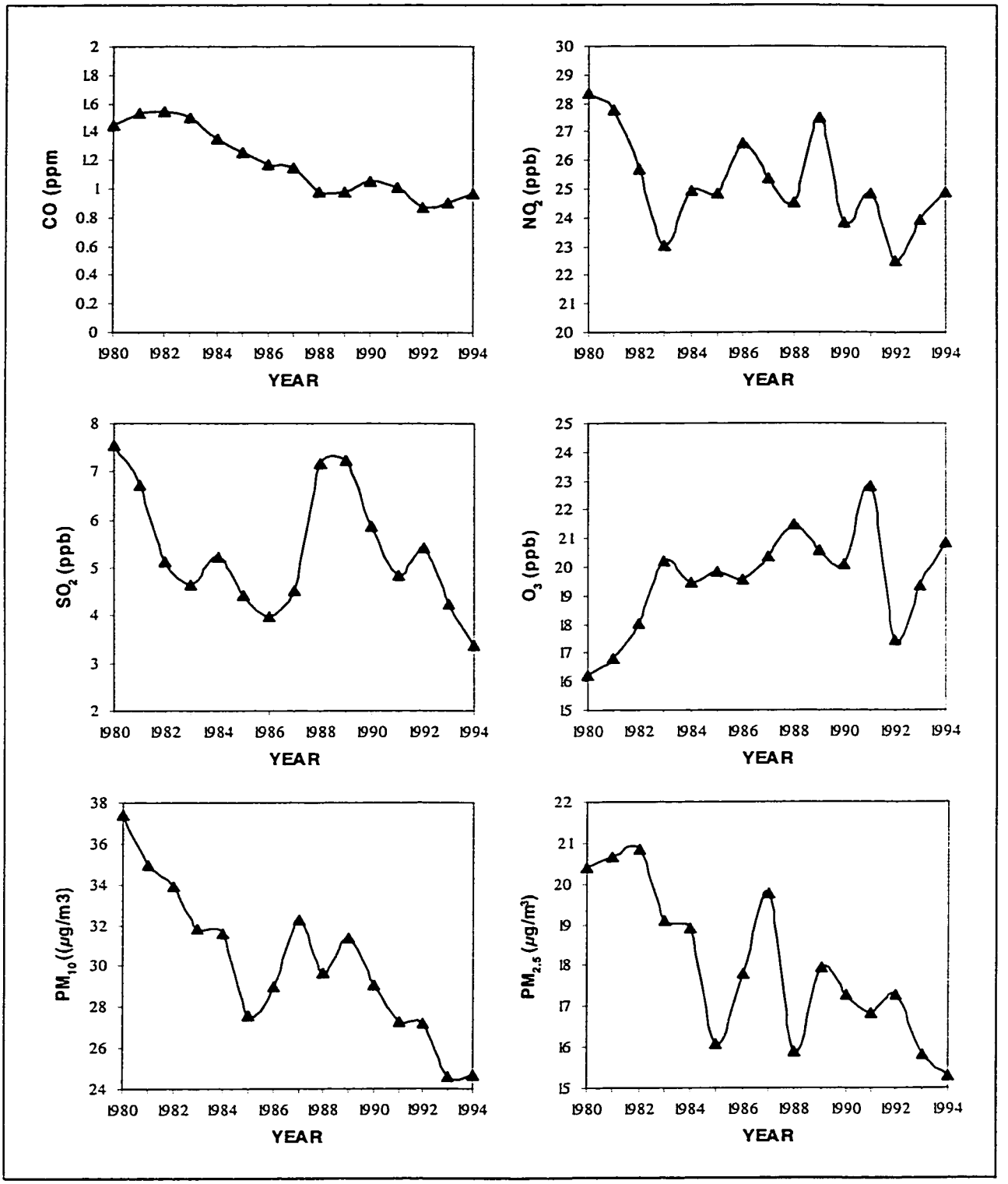

Figure 1.1. Mean annual air pollutant concentrations for Metropolitan Toronto, 1980 to 1994. Notes: CO = carbon monoxide, NO_2 = nitrogen dioxide, SO_2 = sulfur dioxide, O_3 = ozone, PM_{10} and $PM_{2.5}$ = respirable particles of less than 10 and 2.5 microns in aerodynamic diameter.

Second, little research to date has been devoted to the integration of methodologies commonly applied in public health and applied climatology. The techniques used in the study will contribute to the methodological literature in environmental epidemiology. More tangentially this study will contribute to and assist with enhancements of mortality estimates from climate change scenarios and forecast models.

In general, susceptible populations, consisting mainly of the elderly and those with pre-existing and chronic health conditions, are at most risk from ambient concentrations of air pollution combined with extreme weather events. Metropolitan Toronto is typical of many other major cities in industrialized nations that will experience an amplification of risk and growing susceptible populations due to increases in life expectancy and an ageing populace. This study will provide useful estimates of associations between holistic measures of environmental (atmospheric) quality and cumulative health outcomes of vulnerable populations. Although the research presented here is statistical in nature, it aids in the conceptualization of issues inherent to disciplines such as studies in environmental justice, health, and other important contributors to understanding the dynamics of human progress and social interaction.

1.3 Study Objectives

This research hypothesizes that 1) certain synoptic atmospheric conditions may interact with, or modify the effects of, ambient air pollution concentrations, thereby affecting human mortality risk; and that 2) allowing for potential interactions between weather and air pollution (e.g., using weather extremes) may alter mortality/pollution relationships. For example, pollution may have a greater impact on mortality during extremely hot or cold days. Thus the goal of this research is to offer a thorough epidemiological assessment of human mortality from ambient air pollution concentrations.

Figure 1.2 reveals how the relationships studied here can be theoretically conceptualized. It is readily apparent that the interrelations among weather, human activity, air pollution emissions and atmospheric concentrations, and human health, are highly complex. For example,

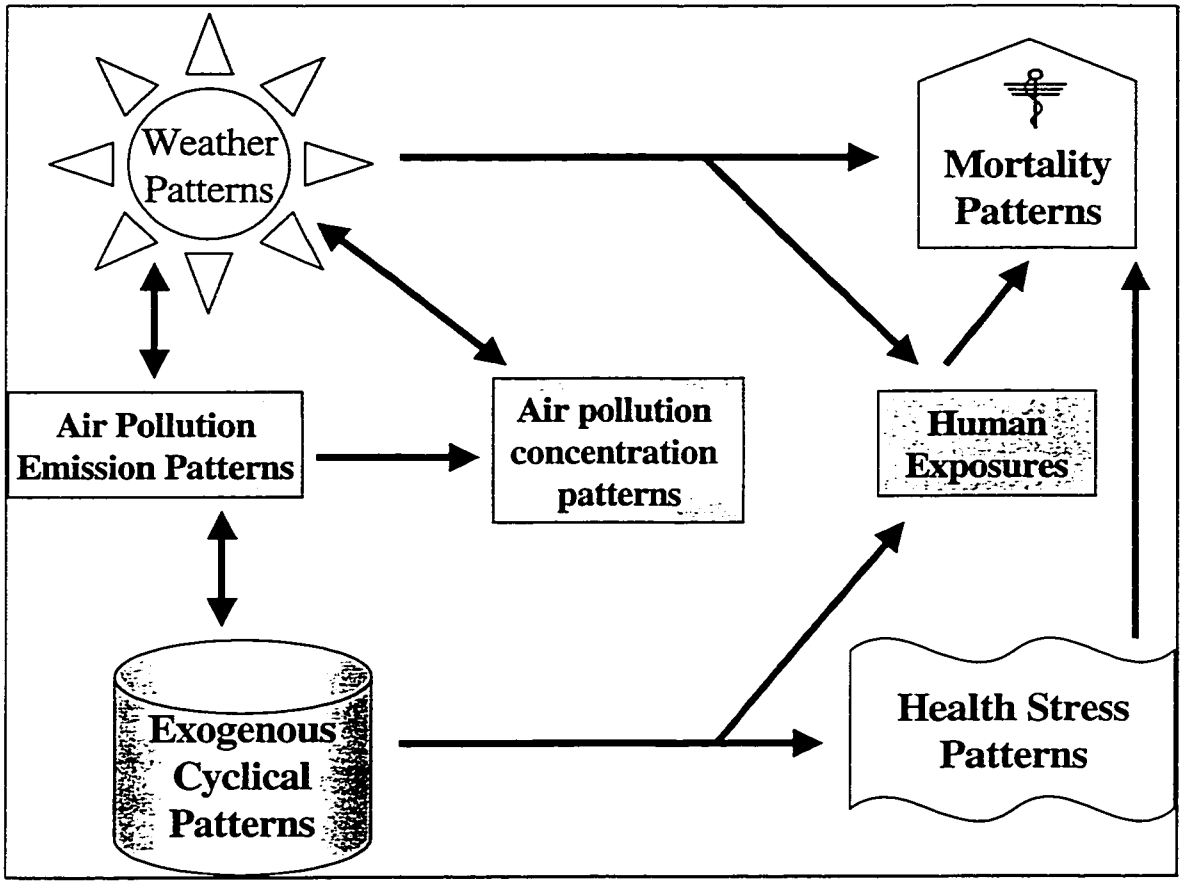

Figure 1.2. Theoretical path model of human health effects (mortality) from exposures to air pollution and weather patterns.

weather may influence human behaviour, which in turn can affect emission levels. The weather also directly affects air pollutant genesis and longevity in the atmosphere, while human health is affected by the weather, air pollution, and various exogenous factors.

Two major issues arising from this study may warrant criticism. First, confounding will be difficult to control due to the complexities previously described. To address this issue, this study will make use of

several methods from disparate disciplines, including nonparametric statistical methods. Second, studies of this nature have been criticized for conclusions generalized to individual-level effects, more commonly referred to as ecological fallacy.[16] In essence, health risks from air pollution and weather extremes affects whole populations and geographic areas rather than individuals, albeit more so among aggregations of susceptible people.[17] In this study, the level of organization will be isolated to the data aggregated to the level of a city. Thus generalizations to the individual level will not be a consideration. However, this does not limit the ability of the study to make assumptions of causality.

In order to investigate the mortality risks from air pollution this study will:

1) Describe the levels of human mortality, selected air pollutants, and weather, in addition to their associations over a seventeen-year period;
2) Compare seasonal models of air pollution, weather, and mortality using a nonparametric statistical approach;
3) Explore the potential for interaction and effect modification by weather of the air pollution-mortality relationship; and,
4) Add to existing discussions of causality and the health impact of air pollution on public health policies and climate change.

To achieve these objectives this thesis offers two stand-alone, yet complimentary papers:

1. *The relationship between humidex and mortality in Metropolitan Toronto, 1980 to 1996 (Chapter 2)*

The use of weather indices (such as the humidex) attempt to simulate the physiological effects that weather, especially temperature extremes, has on human health. Many studies of summer weather have demonstrated a threshold temperature of approximately 20° to 25°C above

which health effects increase.[18, 19] However, little effort has been devoted to the systematic study of the full range of temperatures during the summer season on populations in Canada. In particular , the patterns of mortality specific to gender, age, and cause, along with reference to on-going policy formation need to be investigated. This paper addresses these knowledge gaps.

2. *Effects of air pollution on mortality: an examination of seasonality and weather interactions (Chapter 3)*

It is generally acknowledged that both weather and ambient air pollution concentrations are causally related to variations of human mortality over the short-term.[20, 21] However, little is known about the potential for interaction, or possible synergism, across multiple pollutants and mortality outcomes. Furthermore, it is uncertain in what respects specific weather characteristics may initiate negative human health outcomes or how they may be beneficial. This paper assists to elucidate these uncertainties.

1.4 Thesis Outline and Format

This research is presented as a collection of four chapters. The remainder of this chapter will review the literature from epidemiological studies of the associations between weather, air pollution, and human mortality. The second and third chapters are comprised of two independent research papers, each with their own abstract, introduction, method, results, discussion and set of references. Chapters two and three have been written with the intent of publication. Chapter two has already been submitted to the *American Journal of Public Health* and is under review. The fourth chapter provides an overview and summary of the research, giving attention to the contribution of this research in

environmental epidemiology and public health policy, the limitations of the research, and directions for future study.

The remainder of this chapter presents a summary of the literature in two main categories:

1) the relationship between weather, as represented by various weather indices, and human mortality; and,
2) the association between human mortality and ambient outdoor concentrations of air pollution.

1.5 Weather and Human Health Relationships: A Review of the Literature

The central focus of this review is on the empirical human biometeorology literature, an area of study concerned with the statistical relationships between weather elements or types and human actions, reactions, and health.[22] In many situations the causative agent, or transfer mechanism, is not known. Nevertheless, an extensive amount of published epidemiological information exists on the short-term health effects associated with exposures to individual and combinations of weather elements. This review seeks to examine the weather/mortality association, with particular focus on the consistency of observed relationships. In ecological studies, replication of results in other populations and environments is required before an epidemiological judgement of causality can be inferred.[23] Furthermore, this review is limited to those studies that attempted to employ a clear and replicable study design, and those that minimized or controlled for the effects of confounding. Special attention has been given to impacts on mortality due to seasonality, relationships between weather and specific causes of death, and on the associations between weather extremes and mortality outcomes, including potential effects from a changing climate.

Much of the research examining the seasonality of human mortality has focused on temperature change. Bull and Morton[24] investigated the association between temperature and mortality from myocardial infarction, strokes, and pneumonia in England and Wales. Using linear regression techniques and correlation analysis, they reported a subtle monotonic decrease in deaths from - 10° to + 20°C. Above 20°C and below - 10°C the slope of the response increased dramatically, most likely due to a failure of autonomic control of body temperature. The investigators also noticed cause-specific lag times between mortality and temperature.

Subsequent studies have attempted to control for the effects of seasonality, and other time-varying confounders, using time-series approaches. Using autoregressive moving average (ARIMA) model fitting techniques, Bowie and Prothero[25] found short-term associations between temperature and mortality from ischemic heart disease. Time-series techniques are now commonplace in environmental epidemiological studies examining health relationships with environmental variables. Using a similar approach, fitting sine-cosine functions to represent seasonal variations, Douglas and Al-Sayer[26] reported significantly higher than expected all-cause mortality in winter (December to February) than in other months. Several other studies have found higher rates of mortality in winter,[27-29] owing to cloudy, damp, and snowy conditions.[30]

Several studies have reported a J or V –like relationship between specific weather elements (especially mean temperature), and mortality. Alberdi et al.[31] found a similar relationship between temperature, relative humidity and mortality in Madrid, Spain. Shumway et al.[32] observed a comparable association between mortality and temperature but not relative humidity. Finally, Yan[33] found statistically significant relationships, using multivariate techniques, between colder and cloudy conditions and mortality in Hong Kong.

Many investigations have found the potential for pre-existing physiological conditions, such as age and chronic health deficiencies, to increase susceptibility to the health effects of weather. Macy and Schneider[34] reported a statistically significant relationship between temperature extremes and mortality in the elderly, especially for men. Associations between high temperatures and mortality have also been described in high-risk subgroups such as nursing-home patients,[35] and those with coronary,[36,37] respiratory,[38] and cardiovascular diseases.[39,40]

Much research on the relationship between weather and human health has focused on the short-term effect of summer temperature extremes on human mortality, likely in light of warnings from the scientific community about potential implications from climate change. In 1972, Ellis[41] reported an excess of 500 deaths above expected mortality over a five year period in the U.S. due to excessive heat and solar radiation. Probably the most poignant research to date is the result of a heat wave in 1995, when a moist, warm air mass slowly moved its way across the United States and into south-eastern Canada. Nashold et al.[42] reported an excess of 154 hyperthermic-related deaths among Wisconsin residents during a 24 day period. However, it was the city of Chicago that experienced the brunt of the same heat wave. Whitman and Good[43] estimated an excess of 696 deaths over a seven day period by comparing actual deaths to a standardized death rate over a sixteen year period for Cook County. Using a case-control approach, Semenza et al.[44] determined that the majority of deaths during the same heat wave were among the elderly with pre-existing medical illnesses who were socially isolated, and did not have access to air conditioning. A similar investigation of U.K. cities reported an estimated 619 extra deaths, after controlling for seasonal influences and air pollution, during August of the same year (1995).[45]

The past few decades have witnessed the development of more complex models and statistical approaches to understanding atmospheric

risk factors of human mortality. One reason is probably related to a better understanding of the number and complexity of interactions among determining environmental factors. Another is associated with growing concern among scientists, especially climatologists, that significant impacts are likely a result of a global warming trend. A third reason is linked to a belief that more complex models are required to achieve a more representative approach to the study of synergism and interaction among atmospheric health-risk factors. The result is an evolution of two approaches, 1) the development of weather indexes, and 2) synoptic climatological procedures. A weather index can be described as a representative measure of human exposure to weather conditions. Indices are frequently derived from constituents of environmental conditions, human factors (e.g., age, metabolism, gender, etc.), and definitions of reaction-effect criteria.[46] For example, there are now over twenty heat stress indices available to determine health effects from a combination of atmospheric and physiological parameters.[47] In contrast, synoptic climatology attempts to mathematically or statistically derive categories of characteristic weather, based on actual weather conditions occurring within specific patterns of air flow.[48] Synoptic procedures have garnered considerable attention due to their capacity to integrate other well known atmospheric risk factors such as air pollution, and for their ability to help predict climatological patterns of potentially offensive weather conditions to human health. One study, for example, estimated mortality due to hot weather and socio-economic status across 44 U.S. cities using a synoptic classification approach.[49] Synoptic classification has also been used to forecast concentrations of air pollutants[50-52], and is currently in use to help predict weather associated with excess mortality, for use in weather-watch warning systems.[53] Although arguments exist for and against each approach, this thesis will apply both lines of investigation to study the relationships between atmospheric risks factors and human mortality.

1.6 Risk of Mortality Attributable to Air Pollution

During the past decade, a number of epidemiologic studies have reported associations between ambient concentrations of air pollution, particularly particulate pollution, and adverse health effects, even at the relatively low concentrations of pollution found in both U.S. and Canadian cities. This review focuses on the ecologic epidemiologic literature concerned with the relationships between several pollutant measures and human mortality. It is limited to studies that have taken substantial steps to reduce the effects of confounding from the influences of weather, season, and other chronologically varying factors. Although analyses of health effects from air pollution have typically employed both cross-sectional and time-series approaches, all of the studies described in this review used time-series analysis as the principle study design, where changes in mortality rates within a specific area are correlated with fluctuations of air pollution concentrations.

The most famous air pollution episode in history occurred over a two-week span in December of 1952 in London, UK. In an official report, it was estimated that approximately 4000 excess deaths were directly attributable to the effects of smoke and sulfur dioxide (Figure 1.3).[54] Although levels of air pollution were four to five times higher than present day ambient concentrations, interest in the health effects of air pollution continues because of factors such as increasing population size (i.e., larger population at risk), an aging population, and the release of more complex pollution mixtures.

Analysis of the relationship between acid aerosols and human mortality for the period 1963 to 1972 was performed for London, UK.[56] Using the Shumway technique[57] to pre-filter the mortality data of potential confounding from autocorrelation and seasonal influences, the researchers reported a strong correlation between acid aerosols and mortality, more

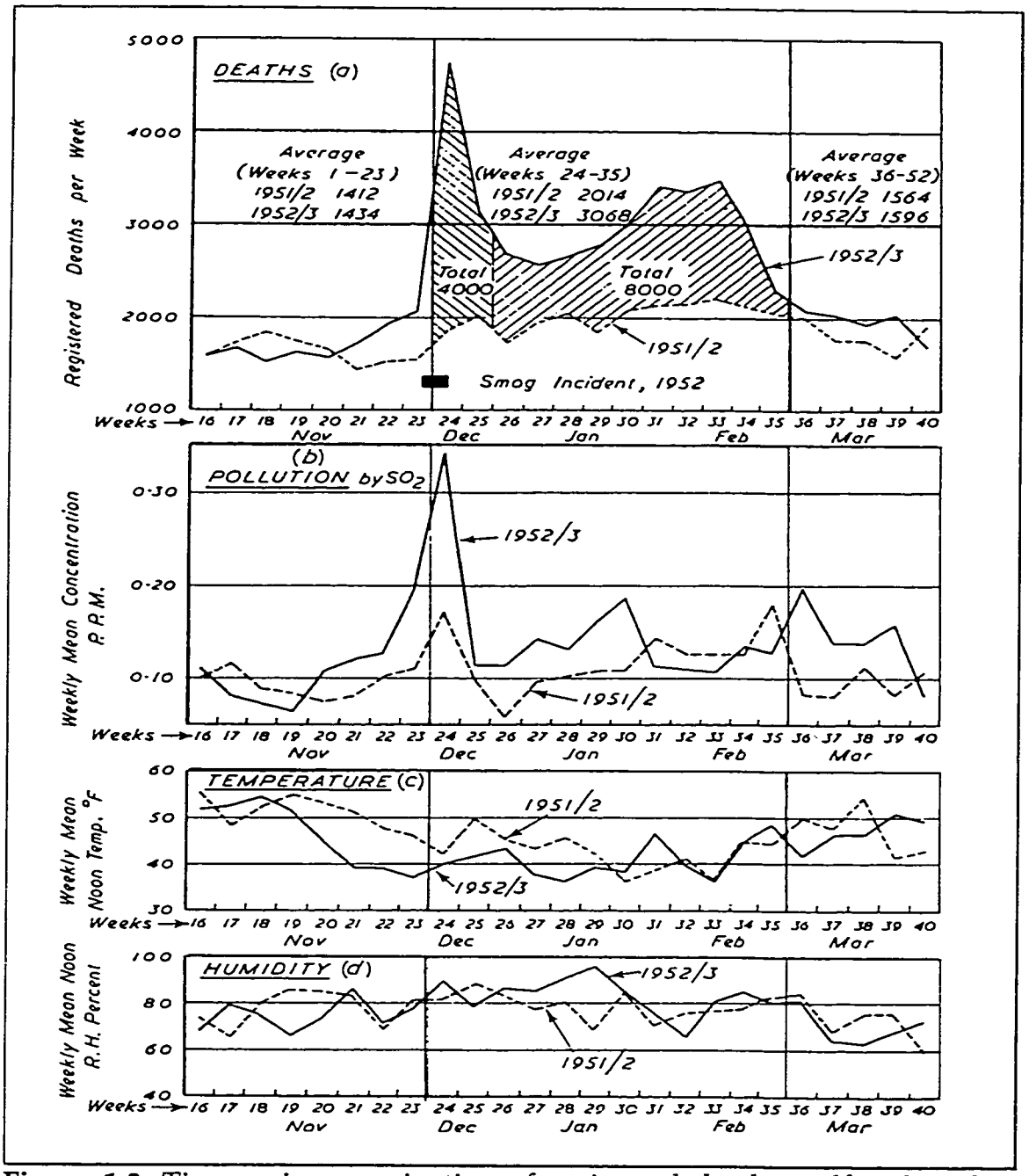

Figure 1.3. Time-series examination of registered deaths, sulfur dioxide, temperature, and humidity, in London, England, 1952.
Source: Lipfert FW. [55]

than for sulfur dioxide (SO_2) and smoke. Using cross-correlation analysis, the results also indicated a one day lag between the pollutant concentrations and greatest effects on mortality.

Kinney and Özkaynak[58] reported the relationship between total, cardiovascular, and respiratory deaths, and a pollutant mixture composed of SO_2, nitrogen dioxide (NO_2), carbon monoxide (CO), and particulates using a combination of prefiltering and multivariate regression. They controlled for the confounding effects of mean temperature and relative humidity, and for temporal cycles in the mortality data. Mortality was associated with a one-day lagged function of air pollution, resulting in a 4% explanation of the total variance in mortality.

Schwartz and Dockery[59] in their 1992 investigation of the association between total suspended particulates, SO_2, and mortality, described the potential for interactions from seasonal and weather-related influences. The same data were re-analyzed to investigate the respirable portion of the particulate mix, commonly known as PM_{10}, or those particles with an aerodynamic diameter of 10 microns or less.[60] Levels of PM_{10} of 100 micrograms per cubic metre ($\mu g/m^3$) were associated with a 16% increase in total mortality.

Data from Athens, Greece, have been analyzed for two summers (1981 and 1987), to investigate the differential and/or synergistic impacts of air pollution and high temperatures on human mortality.[14] This was the first study to specifically examine the combined effects of weather, using a weather index, and air pollution on mortality. The results derived from multiple linear regression attributed a larger portion of excess mortality to the effects of weather than of air pollution.

In a study of the association between ambient levels of particulate air pollution and daily mortality in Birmingham, Alabama, Schwartz employed the use of a nonparametric approach to filter out potential confounding from chronologically related variables and to estimate mortality risk.[61] This paper was the first to use a nonparametric time-series approach and provided a methodological template for future research. A 12% increase in total mortality attributable to particulate air pollution was

reported after controlling for the effects of dewpoint temperature and other temporal trends. Schwartz and co-workers used a similar approach for subsequent investigations in Philadelphia,[62] and Milan.[63, 64] These investigations also evaluated the effects of air pollution on cause-specific mortality and found a 25% increase in mortality from chronic lung disease, a 7 to 9% increase in cardiovascular deaths, an increase of between 10 and 13% for respiratory mortality, a 6% increase in deaths from ischemic heart disease, and an increase of between 7 and 13% for total non-accidental mortality.

Styer et al.[65] investigated the seasonality of the relationship between particulate pollution and mortality, controlling for the effects of temperature, specific humidity, and pressure over a six-year period in Salt Lake County, Utah. A positive significant particulate effect was found in spring and autumn but not in winter or summer.

Since 1995 there have been over twenty-one studies from four continents that have explicitly examined the association between ambient air pollutant mixes and daily mortality. Associations have been reported in data from various locations around the world, including South America,[66, 67] Europe,[68-71] Asia,[72] and Mexico,[73] all with varying air pollutant concentrations, weather conditions, population characteristics and public health policies. These studies provide good evidence for consistency of the association between air pollution and human mortality in the evaluation of epidemiologic causality.

As an alternative to model-based analyses of parallel time series of air pollution and mortality data, Schwartz[10] explored the use of nonparametric smoothing based on the development of generalized additive models.[74] An alternative to multiple and bivariate linear regression modeling, generalized additive models have played a useful role in time-series analyses. They provide a flexible context for controlling non-linear characteristics on potential covariates and confounders, and

they can allow for a direct test of whether relationships with air pollution show thresholds. The nonparametric approach is almost standard in modern air pollution research since the method can be applied with relative ease by those without substantial statistical knowledge. Furthermore, using a standardized approach allows for comparison across studies, another important factor in the pursuit of epidemiologic causality. Since Schwartz's introduction of the approach, more than ten studies have utilized the nonparametric procedure in the evaluation of air pollution and human mortality associations.

In Canada, most results from air pollution studies have been reported by researchers from the federal health agency, Health Canada. The majority of Canadian studies have examined associations between ambient concentrations of air pollution and morbidity. Two recent studies of air pollution and mortality relationships are worth noting. In 1998 Burnett et al.[13] reported an increased risk of premature mortality attributable to a mixture of gaseous, rather than particulate, air pollutants with statistically significant positive risks detected for eleven Canadian cities. The results of another analysis released a few months later for Metropolitan Toronto, and performed by the same group of researchers, revealed a positive and statistically significant association between ambient concentrations of CO and mortality for all seasons.[75] The results of these studies are important for three reasons. First, the relationship between air pollution and human mortality in Canada is still significant, even with extremely low ambient concentrations of air pollution. Second, even with low ambient concentrations of air pollutants, the percentage increase in mortality is similar to the findings of other studies, with much higher concentrations. This finding could imply that Canadians are more susceptible to the effects of air pollution, or more likely, that there is no pollutant/health threshold under which health effects cease to occur. Third, the largest increase in mortality in both studies was due to daily

variations of carbon monoxide. This result is atypical of many epidemiological evaluations of air pollution and human mortality in which the main causative agent is typically respirable particulate matter (PM_{10} and $PM_{2.5}$).

1.7 Statistical Approach

The purpose of this section is to briefly outline the statistical approach adopted in this thesis, based on the review of the literature, to estimate variations of nonaccidental mortality from weather and/or air pollution exposures. Much recent interest in environmental epidemiology has been focused on the examination of events that occur over time (time series). These events are modeled as the dependent variable and include such outcomes as respiratory symptoms, hospital discharge information, or even mortality. The appeal of time series modeling is evident in much of the literature, partly due to the unlikely event of derived relationships to be confounded by personal risk factors, such as smoking and eating habits, that do not vary from day to day with concentrations of air pollution. Nevertheless, it is difficult to surmise the functional form of the relationship between air pollution and any health outcomes of interest.

It is possible to argue that many epidemiologists make many assumptions in the course of employing statistical models with linearity as probably the most universal. To address any possible non-linear functional forms, introductory statistical texts generally describe methods of "linear" estimations using various transformations. For example, it would be difficult to detect health outcome thresholds from air pollution concentrations while employing the optimality properties of least squares and maximum likelihood estimators.[76] The practice of assuming linearity, and the application of transformations to study potential non-linear responses, has been characterized as "imposing global solutions on what are typically local problems."[77] In essence, the dependence of the health

outcome on weather or air pollution is quite clear; however, it is the functional form of that dependence that is in question.

Generalized additive models (GAMs), which are essentially extensions of generalized linear models[78] (please refer to McCullagh and Nelder for details), can be specified as:

$$h(\mathrm{E}y) = \sum_i S_i(X_i), \qquad (1)$$

where h is a link function, y is the outcome being predicted, E signifies the expected value, and S_i are smooth functions. Estimates of y are obtained using a backfitting algorithm and nonparametric estimations of X_i using scatterplot smoothers. An extremely detailed discussion and the nuances of this mathematical procedure can be found in Hastie and Tibshirani's text on generalized additive models.[74]

Many of the key intuitions about GAMs are from ideas on bivariate scatterplot smoothing. Smoothing is an important tool for nonparametric regression techniques and is intuitive to many epidemiologists as a generalization of the running moving averages. The local regression smoother (LOESS), developed by Cleveland and Devlin,[79] is calculated using a tri-cube weight function to provide a local estimate of a specified group of data points (window or span). The number of data points that are smoothed can be specified by the user where a larger window produces a smoother curve. For example, if all the points were selected for smoothing, the resultant fit would be characteristic of a model derived from least-squares regression techniques. Used within the framework of GAMs, the outcome (y) is assumed to depend on the additive sum of smooth and/or linear functions of the predictors. Thus GAMs can:

1) provide a flexible framework for controlling the nonlinear dependence on potential covariates; and
2) allow for a direct test of whether the mortality relationship with air pollution and/or weather shows a threshold.

Please refer to Appendix A for further details.

1.8 Summary of the Literature: Pertinent Findings

In investigations that examined the associations among weather phenomena, ambient concentrations of air pollution and human mortality, a clear picture emerged regarding risk estimates. Most studies, even those that reported comparatively low air pollution concentrations, reported increased risks of death ranging from 1 to 14%. Overall, risks were higher for studies reporting estimates from weather elements, mainly from extremes of temperature and/or changes in relative humidity and pressure. Mortality risks from air pollution tended to be much lower than from weather; however, some studies reported synergistic effects between air pollutants and weather during stressful weather events.

It is clear that mortality is associated with changes in weather, in terms of both seasonal changes and daily perturbations. Mortality typically exhibits a seasonal trend with more deaths in winter than in summer, presumably due to the incidence of infectious diseases, such as influenza and pneumonia. However, many studies that have examined weather/mortality relationships reported a U, J, or V –shaped curve, with the minimum mortality risk between 17° and 25°C, and increasing risk as temperature both rises and falls. This relationship can be confounded by factors such as access to air conditioning and public health prevention programs.

It is also quite apparent that concentrations of pollutants are inextricably linked with changes in weather patterns, most notably temperature. Many studies reported increases in ozone concentrations associated with increases in ambient temperature as well as interactions between hot weather and particulate matter. Thus there is a general concern about potential confounding between temperature, and possibly other weather elements, and air pollution in time-series analyses of

atmospheric factors and associations with human mortality. Quite surprising was the observation that many investigations of weather/mortality relationships neglected to control for the effects of air pollution, whereas all studies of air pollution/mortality controlled for the effects of weather.

Lack of understanding about the precise nature of weather/health associations makes it difficult to decide how to control for weather in statistical analyses of air pollution and mortality. Preliminary re-analysis of the data used by Burnett et al.[75] (which is presented in detail in Chapter 3) shows that suitable control for weather may depend on the type of mortality outcome (Figure 1.4). For example, the effects of confounding by weather in the total and elderly mortality data can be suitably controlled using smoothed functions of dew-point temperature. However, the data for cardiovascular and respiratory mortality seem to be influenced more by other weather parameters such as sea-level pressure and relative humidity respectively.

Synoptic, or air mass-based, approaches to studying weather/mortality relationships have provided some insight, however. Previous research has indicated the presence of oppressively hot, humid air masses responsible for a majority of excess mortality during the summer months in most of eastern United States and south-eastern Canada.[80] When these air masses are present, daily mortality is uniformly high regardless of fluctuations in ozone and particulate concentrations. Further study has revealed that air pollution and air masses may synergistically affect mortality, and that the relationships may well be city specific.[81] To investigate this phenomena further, this thesis will explore how pollution varies according to synoptic weather categories in an attempt to better understand and/or discover weather/pollutant interactions and their combined or differential impacts on human mortality.

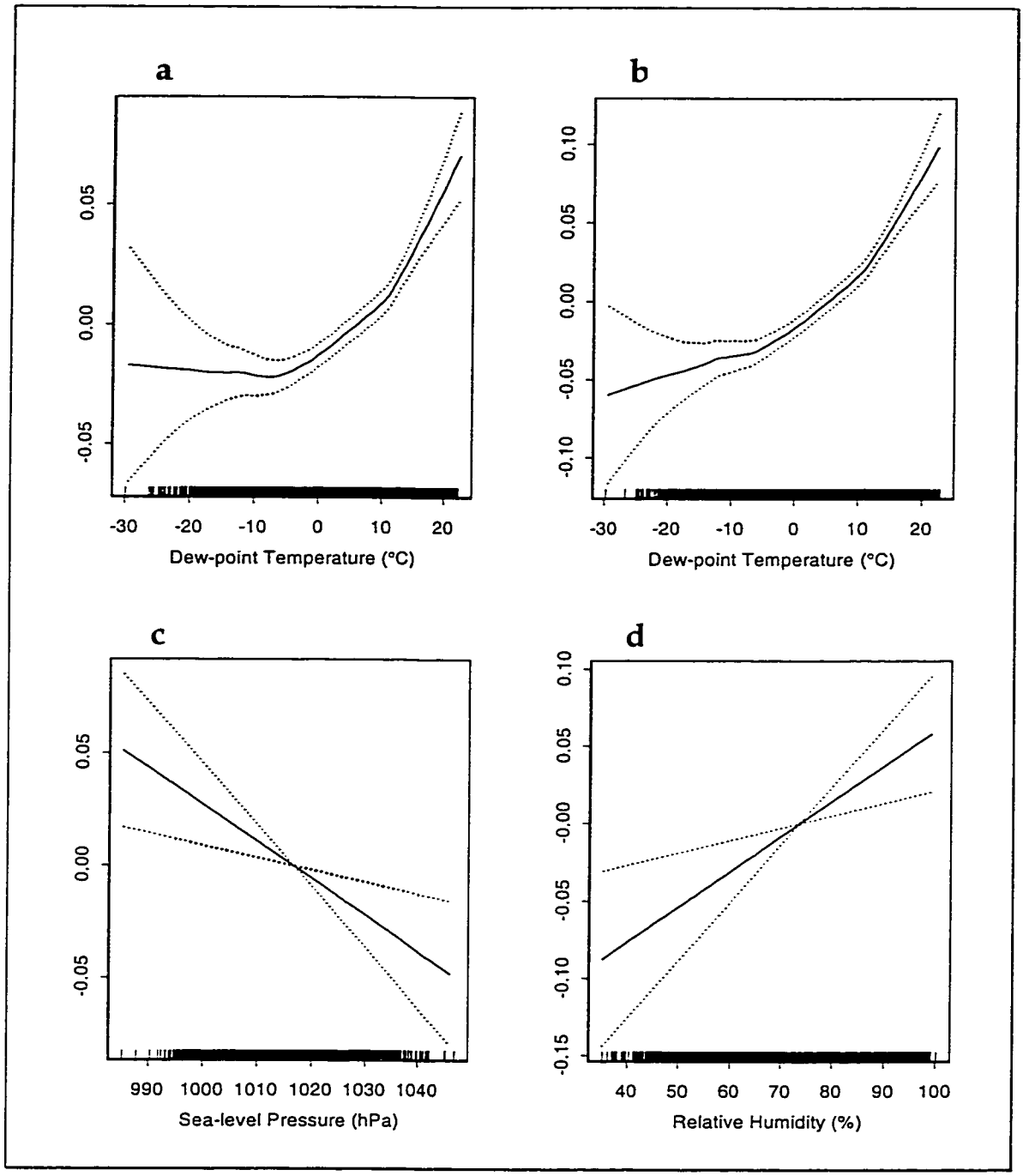

Figure 1.4. Association between (a) total mortality and smoothed same day dew-point temperature, (b) elderly mortality and smoothed same day dew-point temperature, (c) cardiac mortality and same day sea-level pressure, and (d) respiratory mortality and same day relative humidity, for Metropolitan Toronto, 1980 to 1994. Smoothed fits achieved using LOESS with a 50% span (95% confidence intervals given by dashed lines and daily observations represented by tick marks).

A number of studies reported higher mortality risks in populations characterized by extremes of age and pre-existing or chronic health conditions. These include the elderly, the very young, and those with existing cardiac, respiratory, and cerebrovascular diseases. This observation remains a key issue in the study of atmospheric risk factors of mortality and merits the question: *Is increased mortality associated with extremes of weather and/or ambient concentrations of air pollutants restricted to very frail persons for whom life expectancy is short in the absence of either risk?* This study will examine this question in more detail by examining how the risk of mortality and air pollution varies between the elderly, and those with either cardiovascular or respiratory diseases.

Much of the literature also identified potential effects of confounding and bias, from misclassification in the presence of measurement error for independent variables, in the context of multiple regression models.[82] In many of the ecological studies reviewed above, measurement error is implicit for the exposure variables, since only outdoor concentrations are available. Air pollution variables reflect measurements taken at one or more ambient outdoor monitoring stations and weather measurements are normally taken from a single station such as an airport or educational facility. In an attempt to reduce measurement error in the pollution data structure, this study utilizes air pollution data from four ambient air pollution monitoring stations at various locations in Metropolitan Toronto. In addition, the individuals responding to the pollution are at various locations in the study area, including the indoors. Residential indoor temperatures can differ significantly from outdoor levels, depending on the type of home construction and age, accessibility to air conditioners, and duration of stressful weather events. Nevertheless, previous studies have found high indoor/outdoor correlations (i.e., between 0.7 and 0.9) in both summer and winter for SO_2, SO_4 (sulfates).[83, 84] Correlations for NO_2 were more modest at between 0.4 and 0.6. Personal

exposure measurements would be required to negate the difference between indoor and outdoor exposures. Due to financial constraints, the method for this study will not include personal exposure measurements.

Previous research also identified potential bias among mortality risk estimates from confounding between independent variables, mainly caused by temporal variation and serial correlation. For example, both ozone and mortality vary in time (i.e., with season) with highest ozone concentrations in summer and highest mortality counts in winter. Failure to account for temporal influences will result in either finding a negative association or other spurious results. Errors can also occur if the relationships between the predictor and response variables is non-linear. Furthermore, many covariates can be highly correlated (e.g., ozone and temperature). Thus regressions involving both weather and pollutant variables may estimate risk of mortality from the joint effects of all correlated independent variables. The problem then becomes one of separating collinear variables that tend to be linked more strongly with each other than they are with mortality. In this study, the potential for this form of confounding will be addressed using single pollutant models, rather than regression using multiple pollutants (letting covariates compete for variance), and through utilization of generalized additive models, which have the ability to integrate non-linear covariate dependencies into the estimation of mortality risk.

Overlying these situations is the existence of lags between exposure and mortality response. Among studies that have investigated the association between weather and human mortality, effects tend to be acute. The highest correlations are normally found for lag 0 days. In other studies, examination of the autocorrelation between subsequent days of temperature, for example, and mortality sometimes reveal strongly negative serial correlation for lags of 2 to 5 days. This is referred to as a mortality displacement effect where those who died were from a

population for whom life expectancy is short in the absence of pollution. Furthermore, regression models estimates can be impacted if model selection procedures choose predictors (i.e., air pollution and/or weather variables) at different lags and that are also correlated over time.

For example, in the air pollution studies reviewed, it was unclear as to how long an acute pollution episode may exert an influence on mortality. Studies of the London Fog episode in 1952 revealed that residual adverse effects from high pollution days could persist for several additional days.[56] In contrast, it is also possible to observe shorter-term effects, even from less severe environmental stresses associated with low ambient concentrations and subtle changes in weather; however, these lower-stress events typically only affect the most frail subpopulations. In an effort to understand the short-term relationships between mortality and environmental stressors, this thesis will employ stepwise regression to consider each variable-lag combination as an independent variable. Unfortunately, this approach will not negate the potential for collinearity among independent variables, especially those with different lags. However, the model selected will provide insight into the nature of the bivariate relationships between mortality and environmental risk factors. This study will attempt to "correctly" specify the diverse lag structures among the covariates, and will attempt to reduce serial correlation, by utilizing nonparametric statistical techniques.

The previous paragraphs have examined and summarized previous epidemiologic studies of the relationship between air pollution or weather, and human health outcomes, and have described some of the more specific issues associated with study design and bias. Ultimately, however, the acceptability of any epidemiologic study is assessed on the competency of the design to address criteria for drawing inferences. The criteria proposed by Bradford Hill[85] are probably the most well-known and are presented here with specific reference to air pollution research.

1. *Temporal Relationship:* If the air pollution is believed to be the cause of death, then exposure to air pollution must have occurred before death. Stronger associations should be observed for concentrations recorded on the same day, or one or two days previous to the event;
2. *Strength of the Association*: Attempts to answer the question: How great is the risk of mortality apparently induced by air pollution? In this thesis, the risk will be expressed as the relative risk (RR) which compares the number of deaths on any day with average mortality during fluctuations of air pollution concentrations;
3. *Dose-Response Relationship:* As the dose or concentration of air pollution increases, the risk of death should also increase. The models adopted in this thesis will be able to examine this concentration-response relationship using diagnostic plots. In general, past studies have reported a linear relationship; however, some studies have noted concave upward (bathtub) or downward relationships. This author is not aware of any study reporting threshold relationships where the concentrations-response relationship increases and then levels off at some concentration of air pollution (threshold level);
4. *Consistency of the Findings:* Consistent findings in different populations, through multiple studies and in different locations, using diverse epidemiological designs and protocols, strengthens a conclusion of causation.;
5. *Specificity of the Association:* The term specificity implies that the putative cause, in this case air pollution, is responsible for inducing mortality or any specific disease. According to the literature, for example, air pollution is associated with cardiopulmonary mortality, not with accidents and homicides. This thesis will

examine the relationship between air pollution and nonaccidental, elderly, respiratory, and cardiac mortality;

6. *Biological Plausibility*: This criterion asks the question of whether the association between air pollution and mortality makes sense in terms of the current understanding of human biology. Experimental or animal studies can enhance the credibility of epidemiolgic findings by indicating mechanisms of disease. However, the literature reveals that the underlying mechanisms for mortality from air pollution are not well known, even though a causal relationship is evident; and,

7. *Control for Potential Confounding:* Is the relationship between air pollution and mortality real or is it mediated by another potentially unknown factor? Air pollutants and climatic factors covary in time and with each other. This thesis will attempt to reduce the potential for confounding by examining the relationship between air pollution and mortality one pollutant at a time. Moreover, special attention will be given to potential confounding through climatic factors by examining both the bivariate relationships with mortality, and the associations between air pollution and mortality in the context of significantly distinct climatic conditions.

The issue of causation and how it applies to the design, methods, and results of this thesis, will be revisited in Chapter Four.

The following chapters describe the relationship between weather, air pollution, and human mortality for Metropolitan Toronto. The models derived from the studies presented in this thesis will be useful in identifying the complex relationships and attributes among atmospheric variables and human mortality. The utility of the results will lie in the hands of those responsible for public health policies and among those who are able to take steps ultimately to reduce their impact on the environment.

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CHAPTER 2

The Relationship Between Humidex and Mortality in Toronto, 1980 to 1996

2.1 Abstract

This study examined the relationship between summer weather, using humidex, and daily mortality in Toronto, Canada. Regression models were used to identify risk of mortality from summer weather. Smoothing techniques were used to control for air pollution while allowing for overdispersion and autocorrelation. An increase in humidex from the 5th to the 95th percentile was associated with an increase of 3.46% (95% confidence interval = 0.35, 6.67) in non-accidental mortality among the 65 and older age group, with the largest increase of 15.94% (9.72, 22.51) found for those greater than 84 years of age. An increase from the 5th to the 95th percentile in humidex was associated with a 13.06% (7.05, 19.41) increase for cardiac mortality, while an increase of 12.72% (4.21, 21.94) was noted for ischemic heart disease. In Toronto, ambient summer weather conditions, as represented by humidex, are associated with significant increased non-accidental mortality for the elderly and populations with pre-existing cardiac conditions.

2.2 Introduction

Recent literature suggests that human mortality directly varies with fluctuations of air temperature over the short term. [1-4] Few studies have attempted to estimate independent effects of weather and air pollution,[5]

although a number have identified potential synergistic relationships.[6] In temperate environments, summer threshold temperatures have often been identified above which daily mortality increases.[7] A number of epidemiological studies indicate that these effects vary according to location,[8] climate,[9] and risk of susceptibility,[10] however, few studies have examined the relationship between temperature and mortality in Canada.

Metropolitan Toronto is Canada's largest city, and is situated on the north shore of Lake Ontario (43°40'N, 79°24'W). Mainly a commercial rather than heavy industrial urban centre, Toronto has maintained a relatively high population density and a large number of motor vehicles. Due to southern Ontario's variable, temperate climate, the city is susceptible to summer temperature extremes, similar to those experienced in many cities located along the Atlantic seaboard of the United States.[11] Levels of air pollution are relatively low, owing to government regulated emission controls. Recent concern has arisen from the hot and humid summers of 1995, 1998, and 1999, which have motivated the development of a public health heat wave policy for the city.

2.3 Methods

We examined daily variations in human mortality in relation to humidex, a heat stress index that incorporates air temperature and humidity to provide an approximate equivalent of dry air temperature. The study period spanned a 17-year period from 1980 through 1996.

2.3.1 Mortality, Climate, and Air Pollution Data

Statistics Canada provided the mortality database of daily counts of deaths by census division for residents of the Toronto metropolitan area from January 1980 to December 1996. The daily counts were aggregated across six census sub-divisions to comprise the geographical area of the Metropolitan Toronto. Only the daily number of non-accidental deaths

(*International Classification of Diseases, Ninth Revision* [*ICD-9*] codes < 800 and 992) were included. The data were grouped by age (<65 years, 65-74 years, 75-84 years, and >84 years), gender, and selected cause of death (cardiac – ICD9 codes: 410-414, 428; ischemic heart disease – ICD9 codes 410-414; and, non-cardiac mortality – ICD9 codes: all others under 800 and including 992). The final mortality data set consisted of 1-May through 30-September only to represent the period during which heat stress events were likely to occur. Days before May 1 or after September 30 were assigned missing values for modelling purposes.

The Meteorological Service of Canada provided hourly values of dry-bulb and dewpoint temperature from a climate station at the Toronto Pearson International Airport, located approximately 15 kilometres from the urban core. Daily maximum dry-bulb temperature was identified along with the corresponding dewpoint temperature (i.e., the dewpoint temperature that occurred at the same hour as the maximum air temperature, which was not necessarily the maximum dewpoint temperature). Daily values of humidex were calculated according to Masterton and Richardson's equation given below: [12]

$$Humidex(°C) = T + h \qquad (1)$$

where,

T = dry-bulb temperature (°C) and $h = \frac{5}{9}(e - 10)$.

The term 'h' is a correction factor for vapour pressure. The term 'e ' is the vapour pressure calculated using a modified form of the Clausius-Clapeyron equation that relates temperature to pressure as follows:

$$e = 6.11\left\{\exp\left(\frac{M_w L}{R}\right)\left(\frac{1}{273.16} - \frac{1}{T_d}\right)\right\} \qquad (2)$$

where,

6.11	=	saturation vapour pressure (millibars) at a standard temperature of 273.16 °K;
M_w	=	molecular weight of water (constant);
L	=	latent heat of vapourization (constant);
R	=	universal gas constant;
273.16	=	melting point of ice (°K); and,
T_d	=	dewpoint temperature (°K).

The Ontario Ministry of Environment and Energy (OMEE) maintains several ambient air pollution monitoring stations and provided daily data on levels of ozone (O_3), sulfur dioxide (SO_2), and carbon monoxide (CO) from January 1980 to December 1994. Respirable particle data (PM_{10} and $PM_{2.5}$) were only available every six days and not included in this study since short-term effects would be difficult to capture. CO, which has a predominately seasonal cycle that peaks during the winter, was included based on previous studies of Toronto that have indicated strong seasonally independent associations with mortality.[13, 14]

OMEE has located monitoring sites to be representative of the pollution levels experienced by the population as a whole. The deliberate placement of ambient monitors attempts to avoid significant point or local source influences.[15] Daily exposure measures for all gaseous pollutants were calculated from averaging hourly data across four ambient sites. We then calculated daily averages based on the hourly data. Pearson correlations between the monitoring sites for each pollutant were calculated, and matrices are available on request. Average correlations are as follows: r = 0.76-0.89 for O_3; r = 0.38-0.69 for SO_2; and r = 0.23-0.46 for CO. The lower correlations among carbon monoxide sites are most likely due to the nature of the sources. It was estimated that approximately 86%

of air pollution emissions were attributable to transportation related activities in the area.[16]

2.3.2 Statistical Approach

Daily counts of mortality were modeled relating the logarithm of the expected number of deaths to temporally varying mortality predictors. The time series of daily death counts for the entire 17-year study period were pre-filtered to remove long-term trends, seasonal variability, effects of epidemics (sub-seasonal cycles) and weekly effects using a nonparametric smoothed function (Figure 2.1). The smoother is a generalization of a weighted moving average that uses robust locally-weighted regression and smoothing scatterplots (LOESS).[17] Non-parametric smoothed functions were fit to remove day of study, day of the week (mortality rates tend to be highest on Monday), and longer-term fluctuations. The final filtered series shows only small negative autocorrelation for lags of a few days, and thus it was deemed that further modeling of the serial correlation structure was not required.

Untransformed and smoothed twenty-four hour, average lagged values for the four air pollutants were considered for the day of death and one, two, and three days prior to death (0, 1, 2, and 3 day lags), as well as cumulative lags (2 and 3 day moving averages). A final air pollution model was constructed by including the four predictors into a backward stepwise regression procedure using Akaike's Information Criteria (AIC)[18] as the model selection criterion. A similar approach was used to select the lag structure of the humidex variable that best predicted daily mortality after adjustment for a nonparametric smoothed function of day of the study, day of the week, and smoothed functions of air pollution. The relative risk of death attributable to humidex was determined using the generalized additive model function fit in S-Plus.[19]

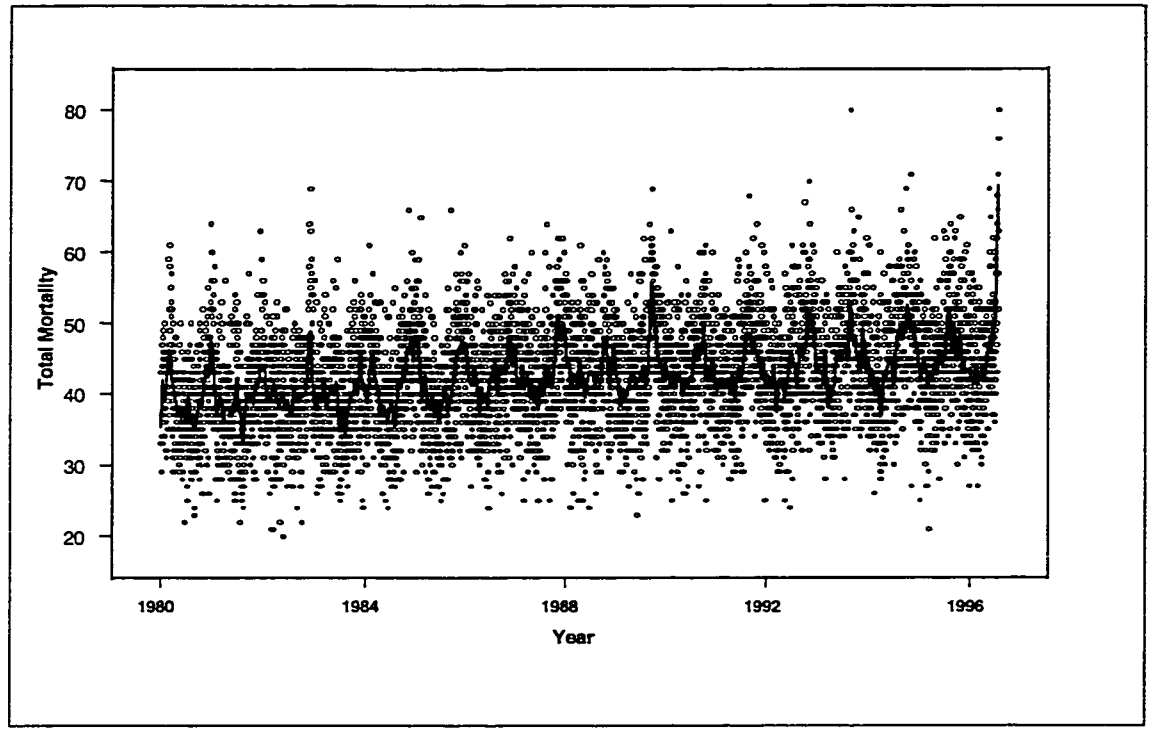

Figure 2.1. Nonparametric smoothed plot of time series for total mortality in Metropolitan Toronto using a 31-day bandwidth from 1980 to 1996.

2.4 Results

There were a total of 105,127 summer deaths from non-accidental causes between 1980 and 1996 in Metropolitan Toronto, with a mean of 40.4 deaths per day (Table 2.1). The correlation matrix for the air pollution and meteorological indicators, shows that nitrogen dioxide is correlated with both carbon monoxide and sulfur dioxide, and ozone is correlated with all measures of temperature (Table 2.2). Deviance residual plots confirm that the nonparametric smoothing for day of study and day of the week removed the seasonal patterns of mortality and controlled for the effects of air pollution on mortality.

2.4.1 Control for Pollutant Effects and Forms of Response

A smoothed one-day lagged function of ozone and sulfur dioxide and a two-day lagged function of carbon monoxide had a lower AIC than

Table 2.1.—Summary Statistics for Daily Values of Mortality, Air Pollution and Climatic Indicators (May 1 to September 30, 1980 to 1996)

Variable	Mean (CV[a])	Minimum	Percentile 5th	25th	50th	75th	95th	Maximum
Mortality (daily)								
Total	40.4 (17)	20	30	36	40	45	52	63
Gender								
Male	20.8 (23)	8	13	18	21	24	29	39
Female	19.6 (23)	6	12	16	19	23	28	34
Age								
< 65	10.2 (32)	1	5	8	10	12	16	22
>= 65	30.3 (20)	11	21	26	30	34	40	52
65 - 74	9.7 (33)	0	5	8	10	12	15	21
75 - 84	11.9 (30)	1	6	9	12	14	18	26
>84	8.8 (37)	1	4	6	9	11	14	22
Cause								
Cardiac	9.7 (34)	1	5	7	10	12	15	26
Ischemic heart disease	4.6 (49)	0	1	3	4	6	9	14
Non-cardiac	35.2 (18)	17	25	31	35	40	46	60
Air Pollutants								
CO (ppm)	1.1 (39)	0	0.5	0.8	1.0	1.3	1.8	4.0
O_3 (ppb)	28.7 (49)	0	10.0	18.7	26.0	36.0	57.0	90.0
NO_2 (ppb)	23.8 (38)	3	12.0	17.0	22.0	29.0	41.0	68.0
SO_2 (ppb)	4.3 (103)	0	0	1	3	6	13	36.0
Weather								
Dry-bulb temp. (°C)	22.6 (23)	5.5	13.0	19.0	23.1	26.5	30.5	36.8
Dewpoint temp. (°C)	12.1 (45)	0	2.3	8.1	12.6	16.3	20.3	26.1
Humidex (°C)	25.4 (30)	3.4	12.4	20.2	25.7	30.9	37.3	50.3

Note. ppm = parts per million, ppb = parts per billion, CO = carbon monoxide, O_3 = ozone, NO_2 = nitrogen dioxide, SO_2 = sulfur dioxide.
[a] CV (%) = coefficient of variation (standard deviation/mean).

other lags and cumulative days considered, with increasing values as the length of the lag grew. The final air pollution model included only non-parametric functions of both ozone and sulfur dioxide based on the backward elimination stepwise regression process. A linear association

Table 2.2.—Correlation Matrix for Daily Values of Air Pollution and Climatic Indicators (May 1 to September 30, 1980 to 1996)

	O_3 (ppb)	NO_2 (ppb)	SO_2 (ppb)	Dewpoint Temp. (°C)	Dry-bulb Temp. (°C)	Humidex (°C)
CO (ppm)	0.058	0.561	0.303	0.211	0.149	0.192
O_3 (ppb)		0.128	0.326	0.313	0.638	0.573
NO_2 (ppb)			0.565	0.145	0.158	0.169
SO_2 (ppb)				0.128	0.223	0.208
Dewpoint Temp. (°C)					0.667	0.851
Dry-bulb Temp. (°C)						0.955

Note. $p < 0.01$ for all correlations, ppm = parts per million, ppb = parts per billion, CO = carbon monoxide, O_3 = ozone, NO_2 = nitrogen dioxide, SO_2 = sulfur dioxide.

with total non-accidental mortality was apparent for nonparametric smoothed functions of ozone, but not for sulfur dioxide (Figure 2.2). A

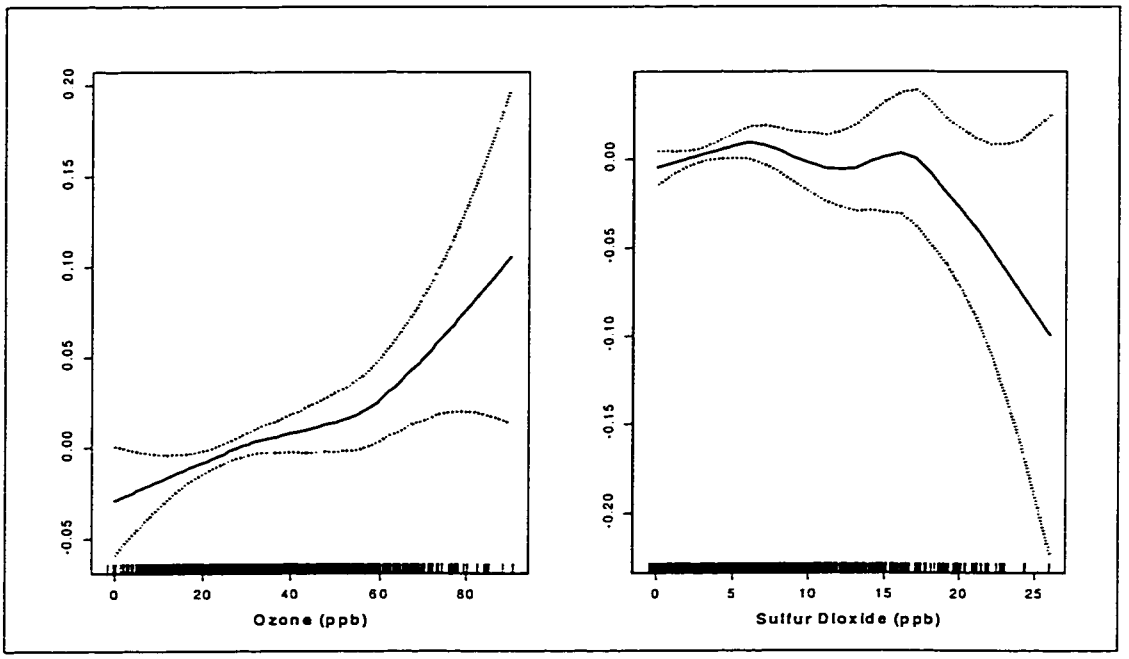

Figure 2.2. Association between daily summer mortality and smoothed function of ozone (one day lag) and sulfur dioxide (one day lag) using a 50% span with 95% confidence bands.

logarithmic association was observed for sulfur dioxide that exhibited some signal decay for outlier observations. These extreme values were removed before proceeding with the analysis. The association between

total non-accidental mortality and humidex (lag 0) is displayed in Figure 2.3 after adjusting for day of the week, day of study, and nonparametric smoothed functions of ozone and sulfur dioxide. A definite "bathtub" shape is apparent for humidex, with monotonic increases of mortality for both cooler and warmer humidex values.

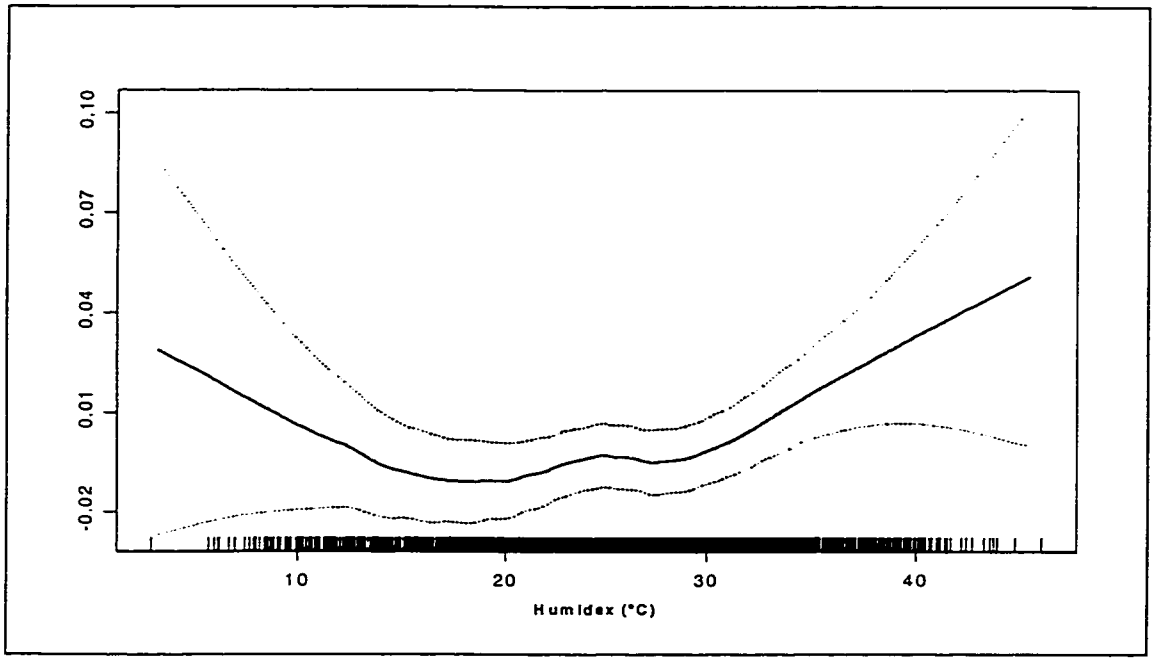

Figure 2.3. A plot of the association between daily summer mortality and a smoothed function of humidex (lag 0) using a 50% span with 95% confidence bands.

2.4.2 Effects of Humidex on Mortality

The results of the generalized additive models for same-day humidex values are summarized in Table 2.3. The percentage increase in mortality by category is given for humidex evaluated at the difference between the 95th and 5th percentiles of its respective distribution, after adjustments for temporal influences and air pollution. Original values were used in all regressions for values of humidex. Humidex (lag 0) was associated with an increase of 2.22% (p = 0.09) in total non-accidental mortality.

Table 2.3.—Percentage Increase in Daily Mortality Associated with an Increase in Humidex from the 5th to 95th Percentile (May 1 to September 30, 1980 to 1996)

Variable	Coefficient	Std. Error	*t* - Statistic	% Increase (95% CI)
Mortality (daily)				
Total	0.00088	0.00053	1.65	2.22 (-0.40, 4.90)
Gender				
Male	0.00088	0.00077	1.15	2.22 (-1.53, 6.10)
Female	0.00139	0.00079	1.77	3.53 (-0.36, 7.58)
Age				
< 65	0.00026	0.00108	0.24	0.65 (-4.52, 6.10)
>= 65	0.00137	0.00063	2.18	3.46 (0.35, 6.67)*
65-74	0.00051	0.00111	0.46	1.29 (-4.06, 6.92)
75-84	0.00007	0.00098	0.07	0.18 (-4.48, 5.07)
> 84	0.00594	0.00113	5.26	15.94 (9.72, 22.51)**
Cause				
Cardiac	0.00493	0.00112	4.40	13.06 (7.05, 19.41)**
IHD	0.00481	0.00161	2.99	12.72 (4.21, 21.94)**
Non-cardiac	0.00039	0.00058	0.67	0.96 (-1.84, 3.84)

Note: IHD = ischemic heart disease,
* $p < .05$; ** $p < .01$

Humidex was significantly associated with elderly non-accidental mortality, with the largest effects occurring among those over 84 years of age. An increase in the daily maximum humidex (lag 0) from the 5th to the 95th percentile was associated with a significant increase (3.46%) in elderly (>64 years) non-accidental mortality. However, the majority of the increase can be accounted for by persons greater than age 84 where the daily humidex (lag 0) was associated with a 15.94% (p <0.01) increase in non-accidental mortality. No consistent or significant associations between daily mortality and humidex were observed for elderly persons under age 85 or for persons under the age of 65.

An increase in humidex (lag 0) from the 5th to the 95th percentile was associated with an insignificant increase of 2.22% (p = 0.25) in male non-

accidental mortality and an increase of 3.53% (p = 0.07) for female non-accidental mortality.

Non-accidental mortality from heart disease was associated with humidex. Increases in humidex (lag 0) from the 5th to the 95th percentile were associated with a highly significant increase of 13.06% (p = 0.007) in total cardiac non-accidental mortality. More specifically, humidex values (lag 0) from the 5th to the 95th percentile were associated with an increase of 12.72% (p = 0.003) in deaths from ischemic heart disease (IHD). No significant associations between non-cardiac mortality and humidex were observed.

2.5 Discussion

The statistical analysis used in this study adequately adjusted for the characteristic autocorrelation and overdispersion found in time-series analyses. The modeling approach also managed to remove the major sources of confounding, namely influences of seasonality, day of the week, and air pollution on human non-accidental mortality. The use of broad categories of mortality, including total non-accidental mortality, reduced the potential problems arising from misclassification of cause of death. However, it is plausible that exposure error was introduced due to the physical location of the weather monitor, which was located away from most of the study population. Humidex effects may actually be more severe than described here due to known phenomena such as urban heat island effects.[21]

This study focused on the association between non-accidental mortality and the full range of ambient summer humidex values. Subsequently, it would be difficult to compare the results of this study, which are based on moderate temperature exposures, with the results of other studies that are mainly concerned with exposures to episodes of excessive heat.[22-24] Our findings indicate that the association between non-

accidental mortality and ambient exposures to humidex is small and not statistically significant. However, larger statistically significant associations were especially apparent for vulnerable populations such as those with pre-existing cardiac conditions and the elderly.

2.5.1 Elderly Mortality

Associations observed between heat stress and elderly mortality have been reported elsewhere.[25,26] A study of heat-related deaths in Texas, which suggested that persistently high ambient temperatures, rather than temperature peaks, were related to death, found a dramatic increase in age-adjusted rates for those over the age of 70 years.[27] The results of this study re-affirm those of two previous case-control studies of heat-related mortality from the 1995 Chicago heat wave. In both studies, elderly persons comprised the majority of deaths.[10, 28] A relatively recent study also found increasing levels of relative mortality risk among the elderly for hot temperature episodes in nursing homes.[29] Nevertheless, all of the studies previously described focused on temperature extremes rather than on daily exposures to humidex fluctuations.

Examination of Figures 2.3 and 2.4 reveals a much stronger relationship between humidex and non-accidental elderly mortality than among the total population. It is unlikely that this could be due to elderly exposures to higher outdoor ambient temperatures; therefore, a likely explanation is the failure of elderly subjects to adapt physiologically to changes in temperature. Variations in mortality/humidex patterns with increasing age may be due to poor heat tolerance. This is a fairly well known phenomenon and has been documented in previous research.[30, 31] Although elderly physiology is an important factor, effects from humidex may also be enhanced by circumstance and behaviour more prevalent among the elderly than younger populations. These include factors related to reduced mobility, isolation, pre-existing health conditions, low

fixed incomes, limited access to air conditioning, and tight household budgets.

2.5.2 Cardiac Mortality

A V-shaped relationship was observed for the relationship between humidex and cardiac mortality (Figure 2.4). This is in contrast to the almost bathtub or U-shaped relationship found between humidex and total non-accidental mortality (Figure 2.3). The low risk range corresponding to the lowest number of deaths from cardiac mortality is similar to the relationship between all-cause mortality and temperature observed in the Netherlands;[2] however, the range is much lower than for countries with similar variable climates.[3,32]

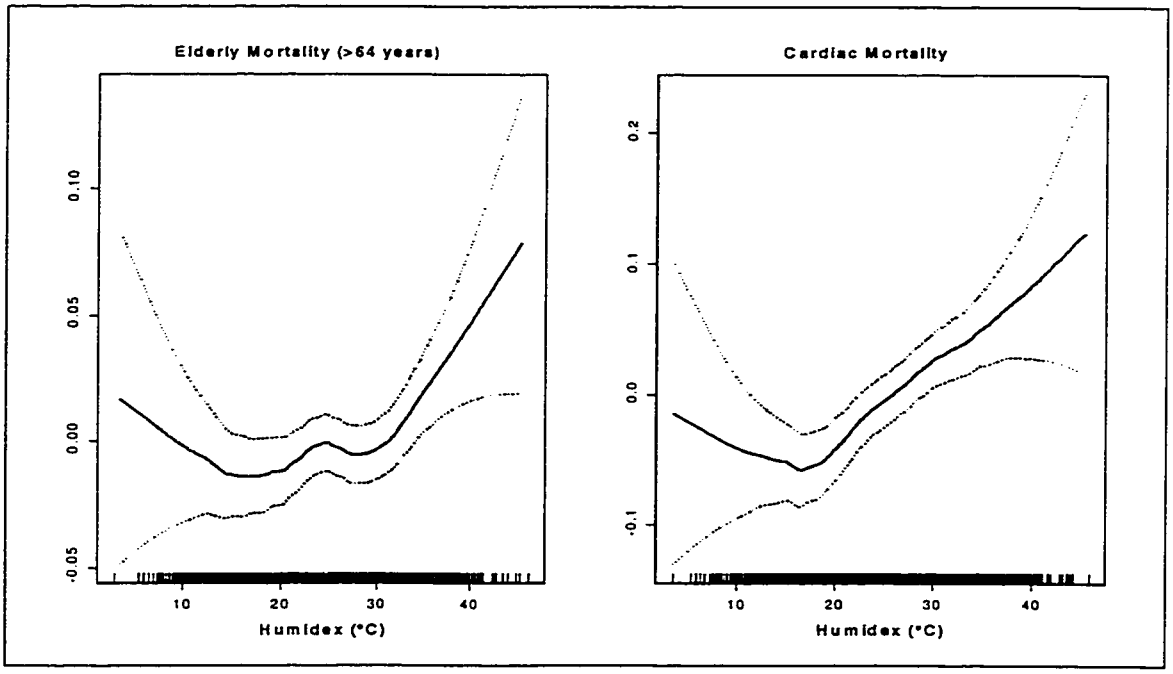

Figure 2.4. Plot of the association between daily elderly and cardiac mortality and a smoothed function of humidex (lag 0) using a 50% span with 95% confidence bands.

Differences in temperature ranges for mortality may be partially attributed to the degree of physiological and behavioural acclimatization,

which previously have been shown to play important roles in the ability of Toronto's population to adjust to summer weather variations.[11]Variations in temperatures, and also humidex, are known to affect people with cardiac problems through well-understood pathophysiological mechanisms. Exposures to increased temperatures have been found to increase blood viscosity and plasma cholesterol.[33]

Patterns of seasonal variability have also been correlated to changes in platelet serotonin concentrations and uptake sites.[34] The significant associations found between humidex and cardiac mortality in this study indicate that variations in temperature, either upward or downward, may help to hasten such events.

2.6 Conclusions

Our study indicates that humidex is associated with increased non-accidental mortality in Metropolitan Toronto. The strongest and most significant associations were between humidex and elderly non-accidental mortality, with the main effects occurring for those greater than 84 years of age. A strong relationship was also evident between humidex and non-accidental cardiac mortality, particularly mortality associated with ischemic heart disease. However, non-significant results were evident for the relationship between humidex and total non-accidental mortality. Thus, any public health policies within Metropolitan Toronto aimed at amelioration of mortality from temperature variability should aim to focus on susceptible populations rather than the city as a whole. These results are consistent with international literature on the short-term health effects of variations in temperature and are coherent with our companion study of daily mortality associated with heat stress days across much of southern Ontario.[35]

The replicability and consistency of the associations between variations of humidex and human non-accidental mortality suggest a

causal relationship. Although the risks observed in the total population are relatively low, the implications for public health are important because of a changing population structure and possible increased temperature variability as a result of climate change. The results from this study would implicate the elderly and health-compromised populations as particularly susceptible.

We recognize that it is difficult to control for the effects of air pollution and other atmospheric risk factors of human mortality. However, it is logical to assume that constant urban expansion, population growth and the resulting increase of privately-owned vehicles, and potential increased climatic variability that may accompany climate change, may well lead to deteriorating atmospheric quality in Metropolitan Toronto. Recent efforts by the Toronto Department of Public Health have sparked research into the development of a weather-watch warning system for the city. A companion work by the authors is underway to examine the potential application of two heat stress indexes for the development of threshold temperature and humidex visualization techniques that will aid public health planning for heat stress events. Further studies should attempt to investigate the relationship between human health and these factors in a more holistic manner so that preventative and adaptive strategies for public health can take into account the diverse factors that influence summer weather/health relationships.

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CHAPTER 3

Effects of Air Pollution on Mortality: An Examination of Seasonality and Weather Interactions

3.1 Abstract

Studies have demonstrated repeatedly that air pollution in Toronto is associated with a small but significant increase in mortality. Past evidence has indicated that high temperatures and other stressful weather conditions can also cause severe excess mortality. This study examined the relationships between daily mortality, air pollution and weather conditions, using time series and synoptic climatological approaches, in Metropolitan Toronto, Canada. A spatial synoptic classification (SSC) procedure was used to classify days with similar weather conditions. Regression models were used to identify risk of mortality from air pollution among seasons and among synoptic weather conditions. Smoothing techniques were utilized to control for temporal influences, autocorrelation, and weather in the seasonal analysis. Mortality risk estimates from air pollution are presented by season and by synoptic weather category. Seasonal patterns were evident for pollutant/mortality associations. Moreover, mortality risk estimates from air pollution were at least twice as high when hot, humid weather categories were present. The results indicate, corroborating previous research findings, that under specific circumstances, weather and air pollution have synergistic effects on human health risk.

3.2 Introduction

Over the past decade, numerous epidemiologic studies have recognized an association between fluctuations of ambient gaseous and particulate air pollution and daily variations in nonaccidental mortality rates.[1-27] At ambient concentrations found to be much lower than in the United States and many industrialized countries, Burnett et al. found statistically significant increases in mortality from air pollution in eleven of Canada's largest cities,[28] with carbon monoxide as the largest contributor to mortality risk.[29] However, a thorough investigation of seasonal pollutant/health relationships and weather/pollutant interactions has been neglected. It has been argued that the potential for effect modification from weather and evidence of seasonal effects can complicate claims of a simple causal association between air pollution and human mortality.[30, 31] Reservations about this argument remain, however, on the basis of insufficient methodological control for influences that contribute to mortality fluctuations.[32]

Associations among meteorological variables, season, and human mortality are well-known. Statistically significant relationships have been identified between cause-specific mortality and seasonal change,[33] and ambient temperatures due to respiratory,[34] coronary and ischemic heart,[35, 36] and cerebrovascular diseases.[37] Mortality rates are generally higher in winter;[38-41] however, the impacts from stressful weather events (such as heat waves for example) are also well documented.[42-44] Furthermore, much of the research described here did not "control" for the influence of air pollution in estimating the independent effects of weather and season on mortality.

To date, little research has attempted to discern the complex independent and/or interacting relationships among air pollution, weather, and human health effects. Lebowitz et al. examined the reactions of susceptible individuals to air pollution and weather and described a

relationship between acute respiratory episodes and days with high air pollution, low temperatures, and high barometric pressure in New York City.[45] Katsouyanni et al. reported a synergistic effect of air pollution and high air temperatures on human mortality in Athens,[46] and Choi and colleagues found a combined effect of NO_2 and high temperatures on lung cancer mortality in Japan.[47] Evidence of interaction between total suspended particulates (TSP) and temperature has also been reported, with the association between mortality and TSP increasing in strength above 29°C.[48]

An alternative method to the traditional descriptive and empiric approaches for examining weather, called synoptic climatology, groups weather patterns according to similar meteorological elements and frequently occurring meteorological complexes.[49] Employing this method, Kalkstein et al. found that effects on mortality from air pollution varied according to selected synoptic (weather) categories.[50] Subsequent research has used the synoptic approach to confirm particle/mortality associations,[51] estimate the impact of climate change on human mortality,[52] predict and forecast air pollution episodes,[53, 54] and explore pollutant effects on human mortality during extreme weather conditions.[55]

In this study, I used air pollution, a spatial synoptic classification system, and mortality data from Metropolitan Toronto for the years 1980 to 1994 to (a) examine the seasonality of the association between air pollution and cause-specific mortality, and (b) investigate how the relationship between exposure to ambient air pollution and death varies with weather using spatial synoptic categories. I also considered the components of the atmospheric mix that best predict cause-specific mortality by season.

Metropolitan Toronto, consisting of six boroughs with an average population of 2.23 million between 1980 to 1994, is situated on the north shore of Lake Ontario. The city maintains a relatively high population

density and a growing population of motor vehicles. The urban centre is oriented towards commercial rather than industrial enterprises powered by a combination of both nuclear and coal electricity production facilities. Due to its temperate climate, the city is susceptible to summer temperature extremes, similar to those experienced in many cities along the U.S. Atlantic seaboard.[56] Air pollutant concentrations are relatively low owing to emission controls regulated at both provincial and federal government levels. Nevertheless, concern for public health has arisen from hot, humid summers of 1995, 1998, and 1999, and an annual increase in the number of summer smog warnings.

3.3 Method

3.3.1 Data.

The 5, 479 days of mortality and air pollution data used in this analysis were supplied by Dr. R. Burnett, of the Environmental Health Directorate of Health Canada, who obtained the mortality data from the Ontario Ministry of Consumer and Commercial Relations (MCCR) and the pollution data from the Ontario Ministry of Energy and the Environment (OMEE). These data have been extensively described elsewhere.[29] The air pollution data included daily average concentrations of carbon monoxide (CO), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), and particulate matter with both 10 and 2.5 micron diameter source fractions (PM_{10} and $PM_{2.5}$). The mortality data only include deaths from non-accidental causes and were subsequently grouped according to age (>64 years), and by cause of death (cardiac – ICD9 codes: 410-414, 428; and respiratory – ICD9 codes: 490-496). The analyses reported in this paper are primarily concerned with data of Toronto's total non-accidental mortality. Only seasonal associations were reported for specific causes of death.

Environment Canada supplied the meteorological data, from Pearson International Airport station, located approximately 15 km north-

west of the downtown core. These data contain hourly measurements of temperature, dew-point, wind speed and direction, sea-level pressure, and cloud cover. Raw meteorological data were employed for climatic modeling with the exception of wind speed and direction, which were converted to north/south and east/west scalars. The data were very complete with only eleven missing values for the fifteen-year period.

3.3.2 Procedure.

Two approaches were employed to achieve the objectives of this study. The first approach (*seasonal associations*) was a re-analysis of the Burnett et al.[29] dataset for Metropolitan Toronto using seasonally based models, with particular attention to proper control for the confounding effects of weather by cause of mortality. In the second approach (*synoptic-based risk modeling*), mean values of filtered mortality, including cause-specific mortality, and air pollution by synoptic weather category by season were described. Synoptic categories with statistically significantly higher or lower than expected values of mortality and air pollution were also identified, and log-linear (Poisson) regressions between total mortality and individual components of the air pollution mix were performed.

3.3.2.1 Seasonal Associations

In the first approach, new seasonal variables for total, elderly, and cause-specific mortality, were created with missing values imputed for other seasons. For example, the new variable for total winter mortality (December–February) would have missing observations assigned for spring (March–May), summer (June–August) and fall (September–November). A Poisson regression within a generalized additive model (GAM)[57] was used to determine the association between daily variations in

environmental factors and fluctuations in human mortality in which an entirely separate model was fitted to each season's data. The GAM allowed for potential over(under) dispersion relative to known Poisson variation, and the inclusion of nonparametric smooth functions to model potential nonlinear dependencies of the mortality data (see Appendix A).

The mortality data showed evidence of long-term trends (from an increasing population), seasonal variability, and subseasonal variations most likely due to epidemics, holidays, and/or stressful weather events. A LOESS[58] smooth function, which is a generalization of a weighted moving average that uses robust locally weighted regression within a moving window or span, was used to prefilter the mortality data prior to the examination of the effects of air pollution. A 31-day span for the smoother was chosen based on examination of the spectral density function and autocorrelation plots of the residuals. Effects from differences in mortality rates across the days of the week were also removed since the highest mortality was found to be on Tuesdays and the lowest on Sundays.

The effects of confounding from weather were also removed from the mortality time-series. Raw and smoothed functions for all weather variables were considered for the day of death and one, two, three, and four days prior to death (i.e., 0-4 day lags), as well as cumulative lags (i.e., 2-4 day moving averages). Weather models were constructed by season and by both age and cause of death since variations in temperature, pressure, and other weather elements, have been shown to affect the elderly and health-compromised populations differently.[59, 60]

The final weather models, determined specifically for each cause of mortality, were derived by including the weather predictors in a backward stepwise regression elimination procedure using Akaike's Information Criterion (AIC),[61] a function of the residual deviance and the model degrees of freedom, as the model selection criterion. A similar approach was used to select the lagged and multiple-day averages of air

pollution that best predicted daily mortality. The lag or averaging period with the largest ratio of the log-relative risk to standard error (*t* value) was chosen for further analysis. All regression models were fit using S-PLUS[62] and compared to previously published risks based on full year associations between ambient air pollution and mortality risk.[29]

3.3.2.2 Mortality Risk Modeling Using Spatial Synoptic Classification (SSC)

The second approach consisted of (a) a description of the average filtered mortality (by cause) and air pollution by synoptic air mass category within each season, (b) identification of seasonally based synoptic categories with significantly higher or lower values of filtered mortality and air pollution, and (c) regressions of air pollution on total mortality stratified by air mass category and by season. Weather for Metropolitan Toronto was classified according to a revamped hybrid synoptic categorization system called spatial synoptic classification (SSC). The procedure described here is the first application of the revamped synoptic classification system that groups days with similar weather characteristics into specific seasonally based air mass categories.

The resultant seven category names as defined by Kalkstein et al.[63] are: Dry Polar (DP), Dry Moderate (DM), Dry Tropical (DT), Moist Polar (MP), Moist Moderate (MM), Moist Tropical (MT), and Transitional (TR). Unlike in previous research using the SSC which could only investigate the association in winter and summer, [32, 50] the latest SSC categorization process allows for the investigation of the air pollution and mortality relationship all year round. By concerning itself only with local, meteorological conditions, conditions for Metropolitan Toronto, the SSC is especially useful for the study of biological responses to the ambient atmosphere as a whole, rather than individual weather elements. The

procedure was run for the full fifteen year study period starting with measurements of temperature, dew point, cloud cover, sea-level pressure, wind speed, and wind direction taken four times daily at 0400, 1000, 1600, and 2200 hours Eastern Standard Time. (Refer to Kalkstein et al.[63] for a detailed explanation of SSC development and operation).

Each air mass can be described by its average meteorological characteristics and origin as follows:[65]

1) Dry Polar (DP) is characterized by cool or cold dry air, little or no cloud cover, and vigorous northerly winds. Its origin is northern Canada and Alaska and it is advected in the rest of North America by a cold-core anticyclone;

2) Dry Moderate (DM) has no traditional source region and is characterized by mild, dry air. Typically this type of air mass comes from the Pacific, is adiabatically warmed and dries out as it traverses over the Rocky Mountains and western plains;

3) Dry Tropical (DT) is associated with clear skies and hot, dry conditions. Its arrival in Metropolitan Toronto would be due to advection from a source region in the south-west portions of the U.S. and northwestern Mexico;

4) Moist Polar (MP) conditions are cool, cloudy, and humid, often with light precipitation;

5) Moist Moderate (MM) air is warmer, cloudy, and more humid than MP air;

6) Moist Tropical (MT) arrives in southern Canada, picking up moisture from the Gulf of Mexico or the southern Atlantic along the U.S. seaboard. The air is very warm and humid, with mostly cloudy conditions in winter and partly cloudy conditions in summer. Precipitation is quite common in summer; and,

7) Transitional (TR) represents days where one air mass yields to another. Conditions are variable, normally with strong winds, possible precipitation, and average cloud cover.

Next, categories were created separately for every season, with seven categories per season, respectively. The resulting air mass calendar provided daily synoptic air mass categories chosen to represent realistic groupings of meteorological variables as they actually occur in Metropolitan Toronto. Short descriptions of the weather characteristics of each category were prepared. Mean synoptically stratified values for each air pollution variable and smoothed values of mortality were calculated by season and compared. These mean values were then evaluated among SSC categories using two-tailed t-tests to examine whether certain categories were associated with significantly higher or significantly lower ($p \bullet 0.05$) air pollution and/or mortality. One and two day lags of mortality were also investigated.

The final step in the SSC approach involved similar smoothing techniques (LOESS) of the *Day of Study* variable to control for known chronological variations in the mortality signals (as in the first approach). Regression models were run within every synoptic category, and for each season, to explore the change in the association between air pollution and smoothed daily mortality as stratified by days with similar weather characteristics. Relative risks were reported using the maximum concentration of the pollutant minus its mean concentration to provide an

indication of the effect of an episode day versus the average day. The fit of the mortality predictions across all synoptic categories was not tested. The purpose of the approach was to explore the changes in the air pollutant relationship with mortality across distinct weather categories, which are assumed to have relatively similar weather characteristics within them.

3.4 Results

Yearly distributions of environmental and mortality data are given in Table 3.1. Daily concentrations for SO_2, PM_{10}, and $PM_{2.5}$ were relatively low. Only on nine occasions did $PM_{2.5}$ exceeded the United States Environmental Protection Agency's (U.S. EPA's) National Ambient Air Quality Standard of 65 $\mu g/m^3$ during the study period. However, Canada's Interim Particulate Guidelines value for PM_{10} of 50 $\mu g/m^3$ was exceeded 441 times (i.e., 8% of all days), and the $PM_{2.5}$ standard of 25 $\mu g/m^3$ was exceeded 857 times (i.e., 15.6% of all days) during the fifteen year study period. The distributions of mortality rates for total, elderly, and cause-specific groupings are also given in Table 3.1. There were an average of 40 daily deaths, for a total of 220 038 non-accidental deaths available for analysis. Of those, 76.6% were greater than 64 years of age, 26.5% were cardiac related, and 6.9% were respiratory related. The variance of each of the mortality categories is greater than the mean, suggesting over dispersion due to deterministic or non-random factors. Adjustments of the time-series using the LOESS smoother removed much of the extra dispersion in the mortality data.

3.4.1 Results from the Seasonal Analysis

The summary statistics of cause-specific mortality and environmental variables used in the seasonal approach are described in Table 3.2.

Table 3.1.—Yearly Distributions of Daily Mean Air Pollution Concentrations, Weather Patterns, and Mortality Rates in Toronto, 1980 to 1994

			Percentiles					
	Mean	CV	5th	25th	50th	75th	95th	100th
Air pollutant concentrations (units)								
CO (ppm)	1.18	42	0.5	0.9	1.1	1.4	2.1	6.1
NO_2 (ppb)	25.2	36	13	19	24	30	42	82
SO_2 (ppb)	5.4	110	0	1	4	8	17	57
O_3 (ppb)	19.6	72	0.3	9	17	27	48	90
PM_{10} ($\mu g/m^3$)	30.2	45	14	21	27	36	56	116
$PM_{2.5}$ ($\mu g/m^3$)	18.0	47	8	12	16	22	34	90
Daily mean weather patterns								
Temperature (°C)	7.8	134	-9.8	0.1	7.8	16.9	23.1	29.4
Dew-point (°C)	3.0	325	-13.9	-3.6	3.0	10.9	17.7	22.4
SLP (hPa)	1016.5	1	1004.3	1011.7	1016.4	1021.2	1029.3	1046.0
Cloud cover (tenths)	6.5	41	2	5	7	9	10	10
Rel. humidity (%)	73.5	15	54	66	74	81	91	99
Wind speed (m/s)	3.4	59	0.6	1.8	3.1	4.6	7.0	14.5
Daily mortality counts								
Total	40	17	29	35	40	45	52	75
Elderly	30	21	20	26	30	34	41	57
Cardiac	11	34	5	8	10	13	17	27
Respiratory	3	65	0	1	3	4	6	13

Notes: CV = coefficient of variation (%), CO = carbon monoxide, NO_2 = nitrogen dioxide, SO_2 = sulfur dioxide, O_3 = ozone, PM_{10} = particulate matter less than 10 microns in aerodiameter, $PM_{2.5}$ = particulate matter less than 2.5 microns in aerodiameter, SLP = sea-level pressure, hPa = hectopascals (standard atmospheric pressure is approx. 1013.25 hPa), m/s = metres per second.

Daily deaths varied by season with higher deaths in winter (n = 43) than in summer (n = 38), and this pattern was consistent for all causes. Ambient concentrations of air pollution also followed seasonal patterns. Gaseous pollution (CO, NO_2, and SO_2) had the highest concentrations in winter and lowest in summer. Ozone showed an opposite pattern with highest concentrations in summer as a result of its relationship and formation with higher temperatures and exposure to sunlight. The particulate measures also showed subtle seasonal variations with highest concentrations in summer and spring for PM_{10}, and summer and fall for

Table 3.2.—Seasonal Statistics of Daily Mean Air Pollution Concentrations, Weather Patterns, and Mortality Rates in Toronto, 1980 to 1994

		Season, Mean (and Standard Deviation)			
	Full Year	Winter (Dec-Feb)	Spring (Mar-May)	Summer (Jun-Aug)	Fall (Sept-Nov)
CO (ppm)	1.18 (0.49)	1.31 (0.55)	1.13 (0.45)	1.07 (0.39)	1.21 (0.54)
NO_2 (ppb)	25.2 (9.0)	26.6 (7.4)	26.3 (9.8)	23.0 (9.0)	25.1 (9.3)
SO_2 (ppb)	5.4 (5.9)	7.5 (7.6)	4.8 (4.8)	4.1 (4.5)	5.1 (5.7)
O_3 (ppb)	19.6 (14.0)	9.4 (5.6)	22.7 (10.3)	32.7 (15.1)	13.2 (9.7)
PM_{10} ($\mu g/m^3$)	30.2 (13.6)	27.4 (8.6)	29.6 (11.3)	34.9 (17.0)	28.6 (14.7)
$PM_{2.5}$ ($\mu g/m^3$)	18.0 (8.5)	18.5 (6.2)	15.8 (6.0)	18.9 (10.2)	18.8 (10.2)
Temperature (°C)	7.8 (10.5)	-4.4 (5.9)	6.4 (2.8)	19.7 (3.6)	9.1 (6.4)
Dew-point (°C)	3.0 (9.6)	-7.5 (6.5)	0.8 (6.9)	13.3 (4.2)	5.0 (6.4)
SLP (hPa)	1016.5 (7.5)	1018.0 (9.1)	1015.3 (7.7)	1015.1 (4.8)	1017.6 (7.5)
Cloud over (tenths)	6.5 (2.7)	7.4 (2.5)	6.5 (2.8)	5.7 (2.5)	6.6 (2.6)
Rel. humidity (%)	73.5 (11.2)	78.9 (7.8)	69.5 (12.8)	68.8 (10.7)	76.8 (8.9)
Wind speed (m/s)	3.4 (2.0)	3.9 (2.1)	3.6 (2.1)	2.6 (1.5)	3.2 (1.9)
Total	40 (7.0)	43 (7.2)	41 (6.8)	38 (6.3)	40 (6.6)
Elderly	30 (6.3)	32 (6.5)	30 (6.2)	28 (5.7)	29 (5.9)
Cardiac	11 (3.6)	12 (3.8)	11 (3.5)	10 (3.4)	10 (3.4)
Respiratory	3 (1.8)	4 (2.0)	3 (1.8)	2 (1.6)	3 (1.7)

Notes: CO = carbon monoxide, NO_2 = nitrogen dioxide, SO_2 = sulfur dioxide, O_3 = ozone, PM_{10} = particulate matter less than 10 microns in aerodiameter, $PM_{2.5}$ = particulate matter less than 2.5 microns in aerodiameter, SLP = sea-level pressure, hPa = hectopascals (standard atmospheric pressure is approx. 1013.25 hPa), m/s = metres per second.

$PM_{2.5}$. Weather variables showed obvious seasonal variations associated with a temperate, four season climate.

The seasonal regression models included indicator variables for day of the week and holidays, a 31-day LOESS function of the day of study for seasonal cycles and chronological effects, stepwise-selected weather specifications selected by season and by cause of mortality (Table 3.3), and each pollutant entered into the regression model individually. In many cases individual weather variable/mortality relationships are non-linear. Appendix B contains plots of the relationship between mortality and weather by season.

Table 3.3.—Final Seasonal Weather Models Based on the Backward Elimination Stepwise Regression Model for Mortality.

Mortality Type	*Weather Model*
Winter	
Total	Relative humidity* (lag 2), maximum temperature, SLP* (lag 2)
Elderly	Relative humidity* (lag 2), dew-point temperature* (lag 2)
Cardiac	Maximum temperature,* wind speed
Respiratory	Dew-point temperature*
Spring	
Total	Mean temperature* (lag 2), dew-point temperature*
Elderly	Relative humidity (lag 1), cloud cover* (lag 1), wind speed (lag 2), SLP*
Cardiac	Maximum temperature* (lag 2)
Respiratory	Minimum temperature, SLP* (lag 2)
Summer	
Total	2100h Dew-point temperature,* wind speed
Elderly	2100h Dew-point temperature, cloud cover* (lag 2)
Cardiac	Dew-point temperature* (lag 2)
Respiratory	Minimum temperature, wind speed* (lag 2)
Fall	
Total	Dew-point temperature*
Elderly	Dew-point temperature*
Cardiac	Minimum temperature,* SLP (lag 2)
Respiratory	2100h Dew-point temperature,* minimum temperature (lag 2)

* nonparametric smoothed function (LOESS) using a 50% span
Notes: lag n = n-day lagged function, SLP = sea-level pressure

The models were then evaluated to assess the association between air pollution and daily mortality while controlling for all previously mentioned confounders. Relative risk values for each pollutant were evaluated using the most significant lag (as indicated by the *t*-statistic) and at the difference between the 95th minus the 5th percentiles of their respective distributions. Single pollutant relative risk values, based on the 95th – 5th percentile distribution, are shown in Figure 3.1 for each mortality category. Tables 3.4 to 3.7 give the pollutant 95th-5th percentile distribution, the pollutant coefficient, standard error, *t*-statistic, and RR for each mortality outcome. For comparison, Table 3.4 also provides the results from Burnett et al.'s study[29] of the relationship between air pollution and total nonaccidental mortality.

3.4.1.1 Total Mortality

For winter, positive associations were observed for all mortality-pollution combinations, except for ozone and sulfur dioxide (Table 3.4). Air pollution was only weakly associated with total mortality (i.e., *t* statistic less than 2). Pollutant-mortality relationships were strongest in spring. The pollutants most strongly associated with total nonaccidental mortality were PM_{10} ($t = 6.27$), NO_2 ($t = 4.12$), and CO ($t = 4.07$). Similarly, $PM_{2.5}$ ($t = 3.55$), SO_2 ($t = 2.53$), and O_3 ($t = 2.22$) were also strongly associated with total mortality. Statistically significant associations were also found for the summer season, although less so than for spring and fall. The strongest associations in summer were for CO ($t = 3.23$), NO_2 ($t = 2.80$), and O_3 ($t = 2.34$). No significant associations were found for SO_2 or either of the particulate measures. In fall, significant pollutant-mortality associations were found for all pollutants, with the strongest associations for CO ($t = 5.41$) and the other gases. Lesser, but still significant associations were found for O_3 and both respirable particulate measures. All pollutant-mortality associations showed seasonal variability. For the gases (CO, NO_2, and SO_2), the pollutant-mortality association showed a bimodal distribution with peak associations in spring and fall. For ozone, the association was lowest in winter and increased in strength until the fall season. For PM_{10} and $PM_{2.5}$, pollutant-mortality associations were smallest in summer and strongest in spring. Overall, the largest total nonaccidental mortality risk estimates were found in spring and fall.

3.4.1.2 Elderly Mortality

All pollutants examined, except O_3, PM_{10}, and $PM_{2.5}$, yielded significant associations with elderly winter mortality (Table 3.5). The pollutants most strongly associated with elderly mortality were the gases of CO ($t = 3.39$), NO_2 ($t = 2.66$), and SO_2 ($t = 2.44$). In spring, only two of

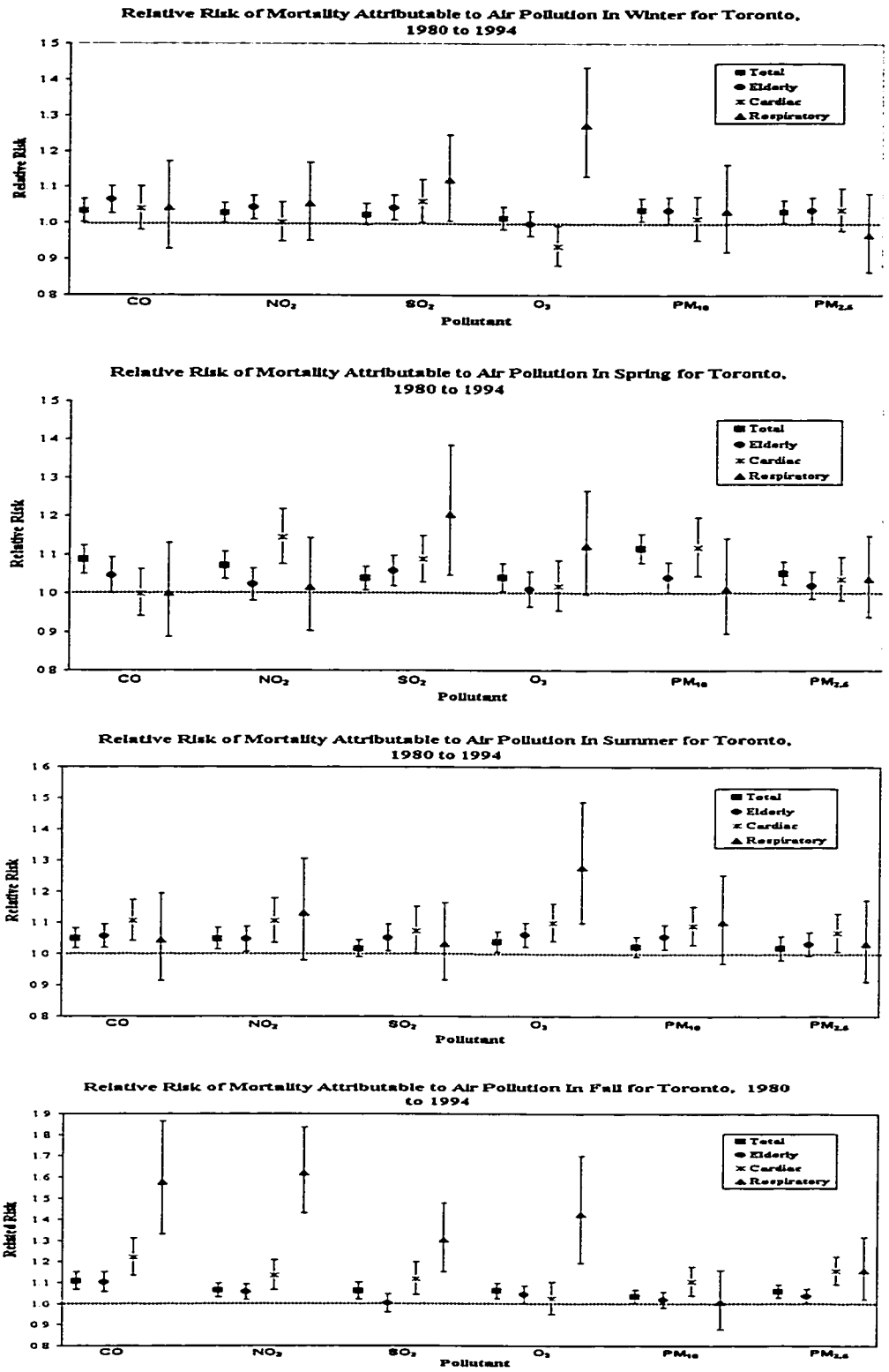

Figure 3.1. The relative risk of mortality attributable to air pollution for winter, spring, summer, and fall, in Toronto from 1980 to 1994.

Table 3.4.—Total Non-accidental Mortality Attributable to the Difference between the 95th and 5th Percentiles of the Air Pollution Distribution and Based on a Single Pollutant Model for Toronto, 1980 to 1994.

	Full Year (Results from Burnett et al. [29])				
	95th-5th	Coefficient	SE	*t*-Statistic	RR (95% CI)
CO (2d MA)	1.4	0.0478	0.0055	8.57	1.070 (1.053, 1.087)
NO_2	29.0	0.0008	0.0002	3.26	1.024 (1.010, 1.039)
SO_2	17.0	0.0057	0.0022	2.52	1.017 (1.004, 1.030)
O_3 (lag 1)	45.0	0.0001	0.0001	0.73	1.006 (0.990, 1.023)
PM_{10} (2d MA)	42.0	0.0006	0.0001	3.95	1.029 (1.015, 1.044)
$PM_{2.5}$ (2d MA)	22.0	0.0018	0.0002	6.28	1.042 (1.029, 1.056)
			Winter Season		
CO (lag 1)	1.7	0.0194	0.0094	2.06	1.034 (1.002, 1.067)
NO_2 (lag 1)	23.0	0.0012	0.0006	1.96	1.028 (1.000, 1.057)
SO_2 (lag 1)	23.0	0.0073	0.0045	1.60	1.024 (0.995, 1.053)
O_3	18.0	0.0007	0.0008	0.85	1.013 (0.983, 1.044)
PM_{10} (lag 1)	27.7	0.0013	0.0005	2.32	1.037 (1.006, 1.069)
$PM_{2.5}$ (lag 1)	19.6	0.0015	0.0007	1.99	1.031 (1.000, 1.063)
			Spring Season		
CO (2d MA)	1.4	0.0611	0.0130	4.07	1.086 (1.049, 1.124)
NO_2 (2d MA)	26.8	0.0025	0.0006	4.12	1.071 (1.037, 1.107)
SO_2	15.0	0.0136	0.0054	2.53	1.039 (1.009, 1.070)
O_3 (3d MA)	25.3	0.0015	0.0006	2.22	1.040 (1.005, 1.076)
PM_{10} (3d MA)	28.3	0.0038	0.0006	6.27	1.116 (1.078, 1.154)
$PM_{2.5}$ (3d MA)	14.3	0.0035	0.0009	3.55	1.052 (1.023, 1.082)
			Summer Season		
CO (2d MA)	1.1	0.0466	0.0144	3.23	1.050 (1.019, 1.082)
NO_2 (2d MA)	24.0	0.0019	0.0007	2.80	1.049 (1.014, 1.084)
SO_2 (lag 1)	13.0	0.0066	0.0051	1.29	1.018 (0.991, 1.045)
O_3 (lag 1)	50.0	0.0007	0.0003	2.34	1.038 (1.006, 1.071)
PM_{10} (2d MA)	46.3	0.0004	0.0003	1.38	1.023 (0.991, 1.055)
$PM_{2.5}$ (2d MA)	32.7	0.0005	0.0005	0.95	1.018 (0.981, 1.057)
			Fall Season		
CO (2d MA)	1.5	0.0688	0.0127	5.41	1.109 (1.068, 1.151)
NO_2 (2d MA)	14.0	0.0025	0.0006	3.97	1.064 (1.032, 1.098)
SO_2 (3d MA)	12.0	0.0231	0.0075	3.07	1.061 (1.022, 1.102)
O_3	31.0	0.0018	0.0005	3.26	1.060 (1.001, 1.098)
PM_{10} (2d MA)	37.3	0.0008	0.0004	2.09	1.034 (1.002, 1.067)
$PM_{2.5}$ (2d MA)	25.6	0.0022	0.0005	2.84	1.058 (1.028, 1.090)

Notes: 2d MA = two day moving average, 3d MA = 3d moving average, CO = carbon monoxide, NO_2 = nitrogen dioxide, SO_2 = logarithm of sulfur dioxide plus one unit, O_3 = ozone, PM_{10} = particulate matter less than 10 microns in aerodiameter, $PM_{2.5}$ = particulate matter less than 2.5 microns in aerodiameter, SE = std. error, RR = relative risk.

the gases, CO (t = 1.96) and SO_2 (t = 3.00) remained significantly associated with elderly mortality. PM_{10} (t = 2.07) was also significantly associated with elderly mortality; however, concentrations of $PM_{2.5}$, NO_2, and O_3 did not approach statistical significance. In contrast, all pollutants

Table 3.5.—Elderly Mortality Attributable to the Difference between the 95th and 5th Percentiles of the Air Pollution Distribution and Based on a Single Pollutant Model for Toronto, 1980 to 1994.

	95th-5th	Coefficient	SE	*t*-Statistic	RR (95% CI)
			Winter Season		
CO (lag 1)	1.7	0.0365	0.0107	3.39	1.064 (1.027, 1.103)
NO_2 (lag 1)	23.0	0.0018	0.0006	2.66	1.043 (1.011, 1.076)
SO_2 (lag 1)	23.0	0.0128	0.0052	2.44	1.042 (1.008, 1.076)
O_3	18	-0.0008	0.0009	-0.90	0.998 (0.992, 1.003)
PM_{10} (lag 1)	27.7	0.0012	0.0006	1.90	1.035 (0.999, 1.072)
$PM_{2.5}$ (lag 1)	19.6	0.0017	0.0009	1.94	1.035 (1.000, 1.072)
			Spring Season		
CO (2d MA)	1.4	0.0332	0.0169	1.96	1.046 (1.000, 1.094)
NO_2 (3d MA)	23.4	0.0009	0.0008	1.06	1.022 (0.981, 1.065)
SO_2	15.0	0.0205	0.0068	3.00	1.059 (1.020, 1.099)
O_3 (lag 2)	32.0	0.0003	0.0007	0.43	1.010 (0.966, 1.056)
PM_{10} (lag 2)	35.9	0.0011	0.0005	2.07	1.041 (1.002, 1.081)
$PM_{2.5}$ (lag 2)	18.0	0.0010	0.0009	1.13	1.020 (0.985, 1.056)
			Summer Season		
CO (2d MA)	1.1	0.0525	0.0172	3.05	1.057 (1.020, 1.095)
NO_2 (3d MA)	19.2	0.0024	0.0010	2.40	1.048 (1.008, 1.089)
SO_2 (3d MA)	9.5	0.0211	0.0088	2.38	1.051 (1.009, 1.095)
O_3 (3d MA)	32.4	0.0017	0.0005	3.13	1.060 (1.022, 1.099)
PM_{10} (3d MA)	42.4	0.0012	0.0004	2.77	1.054 (1.016, 1.094)
$PM_{2.5}$ (3d MA)	26.6	0.0012	0.0007	1.64	1.031 (0.994, 1.070)
			Fall Season		
CO (2d MA)	1.5	0.0655	0.0144	4.53	1.103 (1.057, 1.151)
NO_2 (2d MA)	24.0	0.0022	0.0007	3.05	1.056 (1.020, 1.094)
SO_2 (3d MA)	12.0	0.0118	0.0085	1.39	1.003 (0.961, 1.047)
O_3	27.0	0.0015	0.0007	2.01	1.043 (1.001, 1.086)
PM_{10}	44.0	0.0004	0.0004	0.97	1.018 (0.982, 1.056)
$PM_{2.5}$ (2d MA)	25.6	0.0014	0.0006	2.17	1.037 (1.004, 1.072)

Notes: 2d MA = two day moving average, 3d MA = 3d moving average, CO = carbon monoxide, NO_2 = nitrogen dioxide, SO_2 = logarithm of sulfur dioxide plus one unit, O_3 = ozone, PM_{10} = particulate matter less than 10 microns in aerodiameter, $PM_{2.5}$ = particulate matter less than 2.5 microns in aerodiameter, SE = std. error, RR = relative risk.

were significantly associated with elderly mortality in summer, except for concentrations of $PM_{2.5}$ (t = 1.64). The strongest association was for O_3 (t = 3.13), followed by CO (t = 3.05), PM_{10} (t = 2.77), NO_2 (t = 2.40), and SO_2 (t = 2.38). In fall, the strongest individual association between elderly mortality and air pollution was for CO (t = 4.53). Similarly, NO_2 (t = 3.05) was also strongly associated with elderly mortality, while $PM_{2.5}$ (t = 2.17) and O_3 (t = 2.01) were less so. Associations of elderly mortality with SO_2 and PM_{10} were not statistically significant. In summary, pollutant-mortality associations varied with season, and CO, NO_2, and $PM_{2.5}$ relationships were strongest in fall and weakest in spring. For SO_2, the strongest relationship with elderly mortality occurred in spring and diminished towards fall. For both O_3 and PM_{10} the strongest pollutant-mortality associations were in summer, whereas the weakest for O_3 were in winter, and the weakest for PM_{10} in fall.

3.4.1.3 Cardiac Mortality

Estimates of the association between air pollution and cardiac mortality were characterized by very high or very low risk estimates and large confidence intervals (Table 3.6). For the relationship between cardiac mortality and air pollution in winter, only SO_2 yielded a significant association (t =2.03). None of the other pollutants considered in this season were significantly associated. For the spring season, only NO_2 (t = 4.24), PM_{10} (t = 3.24), and SO_2 (t = 2.96) were strongly associated with cardiac mortality. In contrast, strong associations were observed for all pollutants and cardiac mortality in summer. The strongest individual association was observed for O_3 (t = 3.37). Similar associations were also observed for CO (t = 3.32), NO_2 (t = 3.07), and PM_{10} (t = 2.98), with slightly weaker relationships noted for $PM_{2.5}$ (t = 2.20) and SO_2 (t = 2.01). The pollutants that demonstrated the strongest associations with cardiac

Table 3.6.—Cardiac Mortality Attributable to the Difference between the 95th and 5th Percentiles of the Air Pollution Distribution and Based on a Single Pollutant Model for Toronto, 1980 to 1994.

	95th-5th	Coefficient	SE	*t*-Statistic	RR (95% CI)
			Winter Season		
CO (lag 1)	1.7	0.0230	0.0175	1.32	1.040 (0.981, 1.103)
NO_2 (lag 1)	23.0	0.0001	0.0012	0.09	1.002 (0.950, 1.058)
SO_2 (lag 2)	23.0	0.0182	0.0089	2.03	1.060 (1.002, 1.121)
O_3	18.0	-0.0037	0.0016	-2.24	0.935 (0.881, 0.991)
PM_{10} (lag 1)	27.7	0.0004	0.0011	0.38	1.012 (0.953, 1.075)
$PM_{2.5}$ (lag 1)	19.6	0.0018	0.0014	1.24	1.037 (0.979, 1.098)
			Spring Season		
CO (lag 1)	1.4	-0.0004	0.0222	-0.02	0.999 (0.940, 1.062)
NO_2 (3d MA)	23.4	0.0057	0.0013	4.24	1.144 (1.075, 1.218)
SO_2	15.0	0.0303	0.0102	2.96	1.037 (1.012, 1.063)
O_3	33.0	0.0005	0.0009	0.55	1.088 (1.029, 1.150)
PM_{10} (3d MA)	28.3	0.0039	0.0012	3.24	1.119 (1.045, 1.198)
$PM_{2.5}$ (3d MA)	14.3	0.0025	0.0019	1.34	1.037 (0.983, 1.094)
			Summer Season		
CO (3d MA)	1.0	0.1034	0.0311	3.32	1.106 (1.042, 1.173)
NO_2 (3d MA)	21.0	0.0047	0.0015	3.07	1.106 (1.037, 1.178)
SO_2 (3d MA)	9.5	0.0303	0.0150	2.01	1.074 (1.002, 1.151)
O_3 (3d MA)	34.8	0.0027	0.0008	3.37	1.099 (1.040, 1.160)
PM_{10}	52.4	0.0016	0.0005	2.98	1.089 (1.030, 1.153)
$PM_{2.5}$	32.6	0.0019	0.0009	2.20	1.067 (1.007, 1.131)
			Fall Season		
CO (2d MA)	1.5	0.1331	0.0245	5.42	1.221 (1.136, 1.312)
NO_2 (2d MA)	24.0	0.0052	0.0013	3.96	1.135 (1.066, 1.208)
SO_2 (2d MA)	14.0	0.0411	0.0129	3.18	1.118 (1.044, 1.198)
O_3 (lag 2)	32.0	0.0007	0.0011	0.62	1.024 (0.951, 1.103)
PM_{10} (2d MA)	37.3	0.0026	0.0085	3.12	1.104 (1.038, 1.176)
$PM_{2.5}$ (2d MA)	25.6	0.0056	0.0011	5.00	1.157 (1.092, 1.225)

Notes: 2d MA = two day moving average, 3d MA = 3d moving average, CO = carbon monoxide, NO_2 = nitrogen dioxide, SO_2 = logarithm of sulfur dioxide plus one unit, O_3 = ozone, PM_{10} = particulate matter less than 10 microns in aerodiameter, $PM_{2.5}$ = particulate matter less than 2.5 microns in aerodiameter, SE = std. error, RR = relative risk.

mortality in fall were CO (t = 5.42), $PM_{2.5}$ (t = 5.00), and NO_2 (t =3.96). Slightly weaker but still strongly significant associations were found for SO_2 (t = 3.18) and PM_{10} (t = 3.12). Ozone was not significantly associated with cardiac mortality in fall. The distribution of pollutant-cardiac mortality associations by season was different for every pollutant. For CO the strongest association was found in fall and the weakest in spring. NO_2

exhibited a bimodal relationship with associations peaking in spring and fall. Mortality associations with SO_2 remained fairly consistent throughout the year with the most significant relationship in fall. O_3 was only significantly associated with cardiac mortality in summer. For PM_{10} the relationship with mortality was fairly constant from spring to fall and weakest in winter. Lastly, $PM_{2.5}$ yielded the strongest associations with cardiac mortality in fall; however, no statistically significant relationships were found in winter and spring.

3.4.1.4 Respiratory Mortality

Surprisingly, the association between O_3 and respiratory mortality was very strong in winter (t = 3.98). SO_2 was the only other pollutant significantly associated with respiratory mortality (t = 2.07) in winter. The pollutant most strongly associated with respiratory mortality in spring was SO_2 (t =2.60), although the O_3 coefficient did approach statistical significance (t = 1.92). Ozone was the only pollutant to show significant associations with respiratory mortality in summer (t = 3.17). However in fall, by far the strongest pollutant-mortality relationship was found for NO_2 (t = 7.55). Very strong associations were also uncovered for CO (t = 5.30), SO_2 (4.19), and O_3 (t = 3.94). $PM_{2.5}$ concentrations were also significantly associated with respiratory mortality (t = 2.30); however, concentrations of PM_{10} were not.

The distribution of the pollutant-mortality associations was similar for CO, NO_2, and $PM_{2.5}$ in that they were only significant in the fall season. The association between SO_2 and respiratory mortality was bimodal, with the strongest associations in spring and fall. Ozone showed consistently strong associations with respiratory mortality and was only marginally insignificant in spring. No significant associations were present for PM_{10} for any season.

Table 3.7.—Respiratory Mortality Attributable to the Difference between the 95th and 5th Percentiles of the Air Pollution Distribution and Based on a Single Pollutant Model for Toronto, 1980 to 1994.

	95th-5th	Coefficient	SE	*t*-Statistic	RR (95% CI)
			Winter Season		
CO (lag 3)	1.7	0.0249	0.0348	0.72	1.043 (0.929, 1.172)
NO_2 (lag 3)	23.0	0.0022	0.0022	1.00	1.054 (0.951, 1.167)
SO_2 (lag 2)	23.0	0.0356	0.0171	2.07	1.120 (1.006, 1.246)
O_3 (2d MA)	15.5	0.0156	0.0039	3.98	1.274 (1.131, 1.435)
PM_{10} (lag 3)	27.7	0.0011	0.0021	0.55	1.034 (0.919, 1.163)
$PM_{2.5}$ (lag 3)	19.9	-0.0017	0.0029	-0.59	0.967 (0.863, 1.083)
			Spring Season		
CO	1.4	0.0004	0.0442	0.01	1.001 (0.886, 1.130)
NO_2	32.0	0.0004	0.0018	0.26	1.016 (0.903, 1.143)
SO_2 (3d MA)	11.0	0.0748	0.0287	2.60	1.204 (1.047, 1.385)
O_3 (lag 2)	32.0	0.0036	0.0019	1.92	1.124 (0.997, 1.266)
PM_{10}	36.2	0.0003	0.0017	0.19	1.012 (0.895, 1.143)
$PM_{2.5}$	18.2	0.0020	0.0028	0.74	1.039 (0.939, 1.149)
			Summer Season		
CO (2d MA)	1.1	0.0427	0.0646	0.65	1.045 (0.915, 1.194)
NO_2	28.0	0.0044	0.0026	1.70	1.131 (0.981, 1.305)
SO_2 (lag 2)	13.0	0.0123	0.0231	0.53	1.033 (0.916, 1.165)
O_3 (3d MA)	34.8	0.0070	0.0022	3.17	1.278 (1.098, 1.486)
PM_{10} (lag2)	52.5	0.0018	0.0012	1.49	1.102 (0.970, 1.252)
$PM_{2.5}$ (lag3)	32.2	0.0010	0.0019	0.52	1.034 (0.912, 1.172)
			Fall Season		
CO (2d MA)	1.5	0.3036	0.0572	5.30	1.577 (1.333, 1.866)
NO_2 (lag 2)	28.0	0.0172	0.0022	7.55	1.620 (1.429, 1.836)
SO_2 (lag 2)	16.0	0.0936	0.0223	4.19	1.304 (1.151, 1.476)
O_3 (3d MA)	26.0	0.0136	0.0034	3.94	1.425 (1.195, 1.700)
PM_{10} (lag 2)	44.6	0.0002	0.0015	0.14	1.010 (1.159, 0.879)
$PM_{2.5}$ (2d MA)	25.6	0.0057	0.0025	2.30	1.160 (1.022, 1.316)

Notes: 2d MA = two day moving average, 3d MA = 3d moving average, CO = carbon monoxide, NO_2 = nitrogen dioxide, SO_2 = logarithm of sulfur dioxide plus one unit, O_3 = ozone, PM_{10} = particulate matter less than 10 microns in aerodiameter, $PM_{2.5}$ = particulate matter less than 2.5 microns in aerodiameter, SE = std. error, RR = relative risk.

3.4.2 Air Mass / Pollution / Mortality Relationships

Air mass categories were determined for Metropolitan Toronto, for a fifteen year period from 1980 to 1994. Summary statistics for meteorological variables were computed for each category and each season (Tables 3.8 to 3.11). The air mass classification system identified seven air masses for each season with one air mass assigned to each day.

The majority of days in winter can be classified into the dry polar (36.6%), moist polar (24.4%), and moist moderate (16.5%) air masses. The appearance of relatively dry and warm air masses was limited to approximately 10.6% of winter days. Moist tropical air masses accounted

Table 3.8.—Meteorological Composition of Winter Air Mass Types for Toronto, 1980 to 1994.

Air Mass Category Description	Days (%)	Time (LST)	T_a (°C)	T_d (°C)	SLP (hPa)	Cloud (10ths)	Winds
Winter Dry Moderate	133	03.00	-2.2	-4.8	1019.7	5.5	Moderate, W
Mild and dry. Modified	(9.8%)	09.00	0.2	-3.6	1020.3	5.6	Moderate, SW
dry polar air mass,		15.00	3.2	-2.2	1017.9	5.6	Moderate, SW
zonal flow aloft.		21.00	0.4	-2.9	1018.1	5.9	Moderate, SW
		24-hr	**0.2**	**-3.5**	**1019.1**	**5.9**	**Moderate, SW**
Winter Dry Polar	495	03.00	-11.5	-14.6	1021.9	5.2	Moderate, N
Cold, dry air.	(36.6%)	09.00	-10.4	-14.2	1024.2	5.4	Moderate, N
Little cloud, anti-		15.00	-7.3	-12.3	1023.2	6.0	Moderate, W
cyclonic polar source		21.00	-9.8	-13.1	1023.7	5.7	Moderate, W
		24-hr	**-9.8**	**-13.5**	**1023.0**	**5.7**	**Moderate, W**
Winter Dry Tropical	1	03.00	3.7	-0.3	1008.9	0.0	Light, SW
Warm, dry, and clear	(0.0%)	09.00	7.2	0.7	1008.6	0.0	Light, SW
skies. Source is		15.00	14.8	2.5	1004.1	9.0	Moderate, SW
southwestern U.S.		21.00	6.0	0.0	1003.7	9.0	Light, S
		24-hr	**7.5**	**0.8**	**1006.8**	**3.0**	**Light, SW**
Winter Moist Moderate	224	03.00	1.3	-0.5	1013.8	9.5	Moderate, SE
Warmer and more	(16.5%)	09.00	2.2	0.3	1013.7	9.5	Moderate, SW
humid than moist polar		15.00	3.1	0.5	1012.3	9.6	Moderate, W
air with cloud.		21.00	1.6	-0.2	1013.1	9.2	Moderate, W
		24-hr	**2.0**	**0.0**	**1013.4**	**9.4**	**Moderate, SW**
Winter Moist Polar	330	03.00	-3.4	-5.8	1014.7	9.2	Moderate, N
Cool, cloudy, with	(24.4%)	09.00	-2.8	-5.5	1015.4	9.1	Moderate, N
precipitation.		15.00	-1.9	-5.1	1014.3	9.1	Moderate, W
		21.00	-3.3	-5.8	1015.1	8.8	Moderate, N
		24-hr	**-2.8**	**-5.5**	**1014.8**	**8.9**	**Moderate, N, W**
Winter Moist Tropical	12	03.00	4.7	3.7	1016.9	10.0	Light, Calm
Air is warm and	(0.9%)	09.00	7.2	5.7	1015.2	9.8	Moderate, Var.
humid, cloudy.		15.00	10.6	8.4	1011.4	9.7	Moderate, SW
		21.00	8.9	7.3	1010.4	10.0	Moderate, SW
		24-hr	**7.7**	**6.1**	**1014.0**	**9.7**	**Light, SW**
Winter Transitional	158	03.00	-5.6	-8.0	1015.1	7.3	Moderate, N
Transition between two	(11.7%)	09.00	-4.6	-7.4	1015.2	7.9	Strong, N, NW
air mass types. Strong		15.00	-3.3	-6.8	1014.0	7.7	Strong, SW
winds.		21.00	-5.2	-8.2	1015.2	6.9	Strong, W
		24-hr	**-4.6**	**-7.6**	**1014.9**	**7.4**	**Strong, W**

Notes: Values in bold represent daily averages, Time = LST (local standard time), T_a = dry-bulb temperature, T_d = dew-point temperature, SLP = sea-level pressure, Winds = general description of average wind conditions. Speed is defined as: light: 0-2m/s, moderate: 3-5m/s, strong: >5m/s. Direction is approximated to nearest 45°.

for only 0.9% of all winter days. Toronto's coldest winter air mass was the dry polar with a mean air temperature of –9.8°C.

In Spring, 414 out of 1380 days were characterized by a dry polar air mass. This air mass was also the coldest with a mean temperature of –4.4°C and strong afternoon winds. The dry tropical and moist tropical air

Table 3.9.—Meteorological Composition of Spring Air Mass Types for Toronto, 1980 to 1994.

Air Mass Category Description	Days (%)	Time (LST)	T_a (°C)	T_d (°C)	SLP (hPa)	Cloud (10ths)	Winds
Spring Dry Moderate	235	03.00	4.2	0.7	1016.9	3.9	Light, N
Mild and dry. Modified	(17.0%)	09.00	11.4	2.3	1017.8	5.2	Moderate, N
DP air mass with added		15.00	14.5	2.5	1015.7	5.6	Strong, SE
moisture.		21.00	8.7	2.3	1016.2	5.2	Moderate, N
		24-hr	**9.5**	**1.9**	**1016.8**	**5.2**	**Moderate, N**
Spring Dry Polar	414	03.00	-2.2	-5.6	1019.5	3.5	Moderate, N
Cool, dry air and	(30.0%)	09.00	3.1	-5.0	1021.6	4.3	Moderate, N
northerly winds. Little		15.00	6.2	-4.4	1019.9	5.3	Strong, N
cloud.		21.00	1.4	-4.8	1020.5	3.9	Moderate, N
		24-hr	**1.9**	**-4.9**	**1020.3**	**4.6**	**Moderate, N**
Spring Dry Tropical	48	03.00	11.7	7.5	1014.3	2.7	Light, N
Hot and dry with clear	(3.5%)	09.00	21.5	10.2	1014.7	3.2	Moderate, W
skies.		15.00	25.0	10.4	1012.1	4.2	Strong, W
		21.00	16.9	9.7	1012.5	2.6	Moderate, W
		24-hr	**18.4**	**9.2**	**1013.6**	**3.8**	**Moderate, W**
Spring Moist Moderate	173	03.00	7.6	5.6	1011.6	9.0	Moderate, N
Cloudy, modified moist	(12.5%)	09.00	9.8	6.7	1011.8	9.4	Moderate, N
polar air mass.		15.00	11.4	7.1	1010.3	9.2	Moderate, N
Generally warm.		21.00	8.2	5.9	1011.1	8.5	Moderate, N
		24-hr	**9.3**	**6.2**	**1011.4**	**8.9**	**Moderate, N**
Spring Moist Polar	267	03.00	2.7	0.2	1011.5	8.9	Moderate, N
Cool, cloudy, humid,	(19.3%)	09.00	4.3	0.7	1012.2	9.4	Strong, N
often with light		15.00	5.4	1.1	1011.5	9.4	Strong, N
precipitation.		21.00	3.1	0.3	1012.9	7.8	Moderate, NW
		24-hr	**3.9**	**0.6**	**1012.0**	**8.8**	**Moderate, N**
Spring Moist Tropical	70	03.00	13.7	10.6	1012.2	7.6	Light, Calm
Very warm & humid.	(5.0%)	09.00	18.8	13.3	1012.5	8.2	Moderate, N
Partly cloudy,		15.00	20.9	13.6	1010.6	7.6	Strong, Variable
convective precipitation		21.00	15.6	12.1	1011.1	6.9	Light, SW, Calm
		24-hr	**17.3**	**12.2**	**1011.8**	**7.7**	**Light, SW**
Spring Transitional	173	03.00	4.0	0.6	1011.8	6.9	Moderate, N
Transition between two	(12.5%)	09.00	7.6	1.7	1011.9	7.5	Strong, SW
air mass types. Strong		15.00	9.4	1.9	1011.2	7.3	Strong, NW
winds.		21.00	4.8	0.3	1013.5	5.8	Strong, NW
		24-hr	**6.4**	**1.1**	**1012.2**	**7.1**	**Strong, NW**

Notes: Values in bold represent daily averages, Time = LST (local standard time), T_a = dry-bulb temperature, T_d = dew-point temperature, SLP = sea-level pressure, Winds = general description of average wind conditions. Speed is defined as: light: 0-2m/s, moderate: 3-5m/s, strong: >5m/s. Direction is approximated to nearest 45°.

masses were represented across the least number of days, 48 and 70 respectively, and were characterized by higher than seasonal temperatures and light to moderate winds.

In summer, the moist tropical air mass, normally associated with hot, humid and oppressive weather conditions, accounted for 249 days

Table 3.10.—Meteorological Composition of Summer Air Mass Types for Toronto, 1980 to 1994.

Air Mass Category Description	Days (%)	Time (LST)	T_a (°C)	T_d (°C)	SLP (hPa)	Cloud (10ths)	Winds
Summer Dry Moderate	392	03.00	14.0	11.3	1016.8	2.8	Light, N
Mild and dry. Modified	(28.4%)	09.00	22.8	12.7	1017.8	4.1	Mod., Variable
dry polar air mass with		15.00	25.4	12.1	1016.2	5.3	Moderate, SE
added moisture.		21.00	19.1	12.7	1016.5	3.9	Light, Variable
		24-hr	**20.1**	**12.2**	**1016.8**	**4.4**	**Light, Variable**
Summer Dry Polar	234	03.00	10.1	7.3	1017.4	2.4	Light, N
Dry, cooler air &	(17%)	09.00	18.2	7.8	1019.1	4.1	Moderate, N
northerly winds.		15.00	20.7	7.5	1017.9	4.7	Moderate, SE
Generally little cloud.		21.00	14.6	7.8	1018.6	3.2	Light, N
		24-hr	**15.8**	**7.7**	**1018.1**	**3.9**	**Light, N**
Summer Dry Tropical	33	03.00	18.1	13.3	1016.7	2.8	Light, NW
Hot and dry conditions,	(2.4%)	09.00	29.0	15.7	1016.7	3.3	Moderate, SW
clear skies.		15.00	32.1	15.5	1014.4	4.7	Strong, W
		21.00	24.3	16.0	1014.7	3.8	Light, W
		24-hr	**25.5**	**14.9**	**1015.8**	**4.2**	**Light, W**
Summer Moist Moderate	272	03.00	17.2	15.2	1012.8	8.2	Light, Calm
Cloudy, warm with little	(19.7%)	09.00	20.5	16.2	1013.3	8.8	Moderate, N
humidity, high cloud.		15.00	22.0	16.3	1012.2	8.4	Mod., Variable
		21.00	18.3	15.7	1013.1	6.6	Light, N
		24-hr	**19.6**	**15.8**	**1012.9**	**7.9**	**Light, N**
Summer Moist Polar	85	03.00	13.4	10.6	1012.9	7.9	Light, N
Cool, cloudy, humid, and	(6.2%)	09.00	16.3	11.0	1013.9	8.9	Moderate, N
light precipitation.		15.00	17.7	11.0	1013.5	8.5	Moderate, N
		21.00	14.0	10.5	1014.4	4.1	Light, N
		24-hr	**15.5**	**10.8**	**1013.6**	**7.8**	**Light, N**
Summer Moist Tropical	249	03.00	16.2	13.2	1010.8	6.6	Light, Calm
Warm, very humid,	(18.0%)	09.00	20.6	14.0	1011.2	7.5	Moderate, W
partly cloudy. Possible		15.00	21.4	12.8	1010.9	6.9	Moderate, W
convective precipitation.		21.00	15.8	11.1	1012.8	5.1	Light, SE
		24-hr	**23.9**	**18.3**	**1014.2**	**6.0**	**Light, W**
Summer Transitional	114	03.00	18.0	16.5	1021.0	0.0	Light, Calm, W
Transition between two	(8.3%)	09.00	24.5	18.7	1021.9	2.0	Strong, NW
air mass types. Strong		15.00	27.2	17.2	1019.5	7.0	Strong, N
winds.		21.00	NA	NA	NA	NA	Moderate, NW
		24-hr	**18.7**	**12.9**	**1011.4**	**6.6**	**Moderate, NW**

Notes: Values in bold represent daily averages, Time = LST (local standard time), T_a = dry-bulb temperature, T_d = dew-point temperature, SLP = sea-level pressure, Winds = general description of average wind conditions. Speed is defined as: light: 0-2m/s, moderate: 3-5m/s, strong: >5m/s. Direction is approximated to nearest 45°.

(18%). Average daily temperatures were approximately 24°C, cloud cover was moderate, and winds were light. The dry moderate air mass category had the greatest representation consisting of 28.4% of all summer days. Daily temperatures averaged 20°C, and barometric pressures were relatively high and stable.

Table 3.11.—Meteorological Composition of Fall Air Mass Types for Toronto, 1980 to 1994.

Air Mass Category Description	Days (%)	Time (LST)	T_a (°C)	T_d (°C)	SLP (hPa)	Cloud (10ths)	Winds
Fall Dry Moderate	314	03.00	6.8	4.6	1019.2	3.9	Light, Calm
Mild and dry. Modified	(23.0%)	09.00	12.9	7.1	1020.1	5.0	Mod., Variable
dry polar air mass with		15.00	15.6	6.5	1018.2	5.4	Mod., Variable
added moisture.		21.00	10.3	6.8	1018.8	4.6	Light, W
		24-hr	**11.1**	**6.0**	**1019.1**	**5.0**	**Light, SW**
Fall Dry Polar	308	03.00	1.6	-0.7	1023.1	3.6	Light, N, NW
Cool, dry air, northerly	(22.6%)	09.00	6.5	0.2	1025.3	4.7	Moderate, N
winds.		15.00	8.6	0.1	1023.8	5.1	Moderate, N
		21.00	3.9	0.4	1024.3	3.5	Light, N
		24-hr	**4.9**	**0.0**	**1024.0**	**4.5**	**Light, N**
Fall Dry Tropical	4	03.00	11.3	9.9	1018.9	1.0	Light, Calm
Warm, dry conditions	(0.3%)	09.00	21.3	13.3	1019.5	4.0	Moderate, SW
and clear skies.		15.00	26.9	15.0	1016.5	3.3	Strong, SW
		21.00	19.7	14.7	1017.5	5.3	Light, SW
		24-hr	**19.3**	**13.0**	**1018.4**	**3.0**	**Light, SW**
Fall Moist Moderate	207	03.00	11.2	9.6	1015.3	8.9	Light, N
Warm but cooler than	(15.2%)	09.00	13.1	10.6	1015.2	9.5	Moderate, N
MT. High cloud,		15.00	14.1	10.7	1013.6	9.1	Moderate, N
seasonal temperatures.		21.00	11.7	9.9	1013.9	8.2	Light, Variable
		24-hr	**12.5**	**10.1**	**1014.6**	**8.9**	**Light, N**
Fall Moist Polar	299	03.00	4.4	1.9	1014.4	8.3	Moderate, N
Cool, cloudy and	(21.9%)	09.00	5.9	2.0	1015.6	8.7	Strong, SW
humid. Light		15.00	6.6	1.8	1015.0	8.9	Strong, W
precipitation.		21.00	4.3	1.5	1016.3	7.5	Moderate, W
		24-hr	**5.3**	**1.8**	**1015.2**	**8.3**	**Moderate, W**
Fall Moist Tropical	83	03.00	16.7	14.9	1015.2	7.4	Light, Calm
Very warm and humid.	(6.1%)	09.00	21.2	16.7	1015.1	7.3	Moderate, SW
Convective		15.00	23.4	16.8	1013.0	7.6	Moderate, SW
precipitation		21.00	19.0	15.9	1012.9	7.5	Moderate, S
		24-hr	**19.9**	**15.8**	**1014.3**	**7.6**	**Moderate, SW**
Fall Transitional	150	03.00	9.4	7.1	1012.6	7.2	Mod., Variable
Transition between two	(11.0%)	09.00	11.9	7.9	1011.9	8.3	Strong, W
air mass types. Strong		15.00	12.5	6.4	1010.8	7.4	Strong, W
winds.		21.00	8.6	5.3	1012.8	6.3	Strong, W
		24-hr	**10.6**	**6.7**	**1015.2**	**7.3**	**Moderate, W**

Notes: Values in bold represent daily averages, Time = LST (local standard time), T_a = dry-bulb temperature, T_d = dew-point temperature, SLP = sea-level pressure, Winds = general description of average wind conditions. Speed is defined as: light: 0-2m/s, moderate: 3-5m/s, strong: >5m/s. Direction is approximated to nearest 45°.

In fall, the dry moderate (23%), dry polar (22.6%), and moist polar (21.9%) air masses dominated. These air masses are relatively cool, with average daily temperatures ranging from 4.9° to 11.1°C. Winds were light for the DM and DP categories, and moderate in the MP category. The warmest category was the moist tropical, with average temperatures of 19.9°C and cloudy conditions.

3.4.2.1 Air Pollution and Mortality Trends by Air Mass Type

A description of the mean air pollution and daily mean mortality for each air mass, and each season including the results of the two-tailed *t*-tests, are given in Table 3.12. Bolded values indicate that mean air pollution or mortality values are significantly higher or lower in the category when compared to values from all other categories.

Winter air pollution was significantly higher in the DM and MM air mass categories with the exception of significantly lower O_3 concentrations in the MM air mass. However, significantly elevated O_3 concentrations occurred on days characterized as DP and TR air mass types. DP air masses are characteristically cool, sunny and calm, all excellent conditions for the formation of O_3. The higher O_3 concentrations during winter TR days is peculiar due to the variable, windy weather conditions, typical of frontal passages. In contrast, significantly lower ambient concentrations of air pollution were evident on days of the MP or TR air mass types. Both categories are typically cool, cloudy, windy, and wet; these are not favourable conditions for the accumulation of high air pollution concentrations.

Significantly higher total and respiratory mortality rates were found on TR air mass days. Conversely, significantly higher cardiac mortality was found for days characterized by the cold and dry DP air masses. This result coincides well with previous studies that have

Table 3.12.—T-test Results for Air Pollution and Mortality Between Synoptic Air Mass Categories by Season for Toronto, 1980 to 1994.

	Air Masses Derived from Spatial Synoptic Classification (SSC)						
	Dry Moderate	Dry Polar	Dry Tropical	Moist Moderate	Moist Polar	Moist Tropical	Transition
			Winter Season				
Days (%)	133 (9.8)	495 (36.6)	1 (0.0)	224 (16.5)	330 (24.4)	12 (0.9)	158 (11.7)
	Mean Air Pollution (95% C.I.)						
CO (ppm)	1.38 ± 1.10	1.28 ± 0.05	NC	**1.47 ± 0.08^**	**1.26 ± 0.05˘**	1.87 ± 0.70	**1.14 ± 0.08˘**
NO_2 (ppb)	**28.8 ± 1.6^**	26.9 ± 0.6	NC	26.9 ± 0.9	26.2 ± 0.6	29.3 ± 6.4	**23.7 ± 1.2˘**
SO_2 (ppb)	**10.1 ± 1.5^**	7.4 ± 0.7	NC	7.9 ± 0.7	**6.2 ± 0.7˘**	**10.8 ± 2.2^**	6.9 ± 1.1
O_3 (ppb)	9.2 ± 0.9	**11.6 ± 0.5^**	NC	**5.4 ± 0.6˘**	**8.2 ± 0.6˘**	6.6 ± 4.8	**10.9 ± 0.8^**
PM_{10} ($\mu g/m^3$)	**29.9 ± 1.7^**	27.7 ± 0.7	NC	**28.6 ± 1.1^**	**25.7 ± 0.9˘**	27.8 ± 6.2	**26.2 ± 1.2˘**
$PM_{2.5}$ ($\mu g/m^3$)	**19.8 ± 1.3^**	**17.6 ± 0.5˘**	NC	**21.0 ± 0.8^**	**17.7 ± 0.6˘**	20.4 ± 4.8	17.9 ± 0.8
	Mean Mortality and Lags* (95% C.I.)						
Total	42.5 ± 0.6	42.9 ± 0.3	NC	**42.3 ± 0.5˘**	42.7 ± 0.3	**40.3 ± 2.9˘**	**43.4 ± 0.5^**
LAG1	42.5 ± 0.6	42.9 ± 0.3	NC	**42.3 ± 0.5˘**	42.7 ± 0.3	**40.4 ± 2.9˘**	**43.4 ± 0.5^**
LAG2	42.5 ± 0.5	42.9 ± 0.3	NC	**42.3 ± 0.5˘**	42.7 ± 0.3	**40.3 ± 2.9˘**	**43.4 ± 0.5^**
Elderly	32.2 ± 0.5	32.3 ± 0.3	NC	**31.9 ± 0.4˘**	32.1 ± 0.3	30.4 ± 2.6	**32.7 ± 0.5^**
LAG1	32.2 ± 0.5	32.3 ± 0.3	NC	**31.8 ± 0.5˘**	32.1 ± 0.3	30.5 ± 2.5	**32.7 ± 0.5^**
LAG2	32.3 ± 0.5	32.3 ± 0.3	NC	**31.8 ± 0.5˘**	32.1 ± 0.3	30.4 ± 2.5	**32.7 ± 0.5^**
Cardiac	**11.0 ± 0.3˘**	**11.8 ± 0.1^**	NC	11.5 ± 0.3	11.5 ± 0.2	11.9 ± 0.6	11.8 ± 0.3
LAG1	**11.0 ± 0.3˘**	**11.8 ± 0.1^**	NC	11.4 ± 0.2	11.5 ± 0.2	11.9 ± 0.7	11.8 ± 0.3
LAG2	**11.0 ± 0.3˘**	**11.8 ± 0.1^**	NC	11.4 ± 0.2	11.5 ± 0.2	11.9 ± 0.7	11.8 ± 0.3
Respiratory	3.4 ± 0.2	3.4 ± 0.1	NC	3.3 ± 0.1	**3.3 ± 0.1˘**	**2.8 ± 0.4˘**	**3.4 ± 0.1^**
LAG1	3.4 ± 0.2	3.4 ± 0.1	NC	3.3 ± 0.1	3.3 ± 0.1	**2.8 ± 0.4˘**	3.4 ± 0.1
LAG2	3.4 ± 0.1	3.4 ± 0.1	NC	3.3 ± 0.1	3.3 ± 0.1	**2.9 ± 0.4˘**	3.4 ± 0.1
			Spring Season				
Days (%)	235 (17.0)	414 (30.0)	48 (3.5)	173 (12.5)	267 (19.3)	70 (5.0)	173 (12.5)
	Mean Air Pollution (95% C.I.)						
CO (ppm)	1.16 ± 0.06	**1.04 ± 0.04˘**	**1.29 ± 0.14^**	**1.28 ± 0.08^**	1.13 ± 0.05	**1.28 ± 0.10^**	**1.07 ± 0.07˘**
NO_2 (ppb)	**30.3 ± 1.5^**	**23.4 ± 0.7˘**	**34.9 ± 3.5^**	**28.8 ± 1.4^**	**24.4 ± 0.1•**	**31.3 ± 2.4^**	**23.5 ± 1.4˘**
SO_2 (ppb)	**6.7 ± 0.8^**	**4.1 ± 0.4˘**	**7.2 ± 1.5^**	4.5 ± 0.6	**3.4 ± 0.5•**	**6.8 ± 1.1^**	4.9 ± 0.7
O_3 (ppb)	**26.7 ± 1.3^**	22.8 ± 0.6	**40.3 ± 3.9^**	**16.9 ± 1.4˘**	**16.8 ± 0.8•**	**30.9 ± 3.7^**	23.4 ± 1.5
PM_{10} ($\mu g/m^3$)	**34.0 ± 1.5^**	**25.6 ± 0.8˘**	**47.6 ± 4.2^**	**31.4 ± 1.3^**	**24.9 ± 1.1•**	**41.9 ± 2.9^**	28.9 ± 1.7
$PM_{2.5}$ ($\mu g/m^3$)	**16.8 ± 0.8^**	**13.3 ± 0.4˘**	**19.9 ± 1.6^**	**18.8 ± 0.9^**	**15.1 ± 0.8•**	**21.4 ± 1.4^**	15.2 ± 0.9
	Mean Mortality and Lags* (95% C.I.)						
Total	40.6 ± 0.4	**40.9 ± 0.3^**	**39.4 ± 0.7˘**	**40.3 ± 0.5˘**	**41.0 ± 0.4^**	**39.6 ± 0.5˘**	40.7 ± 0.5
LAG1	40.5 ± 0.4	**40.9 ± 0.3^**	**39.4 ± 0.7˘**	**40.2 ± 0.5˘**	**41.0 ± 0.6^**	**39.8 ± 0.6˘**	40.7 ± 0.5
LAG2	40.5 ± 0.4	**40.9 ± 0.3^**	**39.5 ± 0.7˘**	**40.2 ± 0.5˘**	**41.0 ± 0.4^**	**39.9 ± 0.7˘**	40.7 ± 0.5
Elderly	30.5 ± 0.4	**30.7 ± 0.3^**	**29.2 ± 0.7˘**	30.2 ± 0.4	30.7 ± 0.4	**29.4 ± 0.5˘**	30.6 ± 0.5
LAG1	30.5 ± 0.4	30.6 ± 0.3	**29.2 ± 0.8˘**	30.1 ± 0.5	30.7 ± 0.4	**29.7 ± 0.6˘**	30.6 ± 0.5
LAG2	30.4 ± 0.4	30.6 ± 0.3	**29.2 ± 0.8˘**	30.1 ± 0.4	30.7 ± 0.4	**29.9 ± 0.7˘**	30.6 ± 0.5
Cardiac	10.8 ± 0.2	10.9 ± 0.1	10.6 ± 0.3	10.8 ± 0.2	11.0 ± 0.2	**10.2 ± 0.3˘**	10.9 ± 0.2
LAG1	10.9 ± 0.2	10.9 ± 0.1	10.7 ± 0.3	10.8 ± 0.2	11.0 ± 0.2	**10.3 ± 0.3˘**	10.9 ± 0.2
LAG2	10.9 ± 0.2	10.9 ± 0.1	10.6 ± 0.3	10.8 ± 0.2	11.0 ± 0.2	**10.3 ± 0.4˘**	10.9 ± 0.2
Respiratory	3.0 ± 0.1	3.0 ± 0.1	**2.7 ± 0.1˘**	2.9 ± 0.1	**3.0 ± 0.1^**	**2.8 ± 0.1˘**	3.0 ± 0.1
LAG1	3.0 ± 0.1	3.0 ± 0.1	**2.7 ± 0.1˘**	2.9 ± 0.1	**3.0 ± 0.1^**	**2.8 ± 0.1˘**	3.0 ± 0.1
LAG2	3.0 ± 0.1	3.0 ± 0.1	**2.7 ± 0.1˘**	2.9 ± 0.1	**3.0 ± 0.1^**	2.9 ± 0.1	3.0 ± 0.1

Notes: * = predicted mortality using nonparametric LOESS smooth with 31 day span, **bolded** values indicate value for synoptic category is significantly different ($p \leq 0.05$) than the others according to a two-tailed *t*-test, ^ = mean is greater than comparison categories, ˘ = mean is less than comparison categories, NC = no comparison due to only one observation.

revealed a particular vulnerability of those with pre-existing cardiac conditions to fairly minor upward or downward temperature variations (refer to Chapter Two, Section 2.5.2). Evidence of this phenomenon is also apparent for the warm and mild DM days which had significantly lower cardiac mortality when compared to other air mass types. Significantly reduced total and elderly mortality were found during MM air masses. The warmer MT air mass was associated with significantly lower total and respiratory mortality.

A greater consistency in air pollution concentrations by air mass was found in spring. The DM, DT, MM, and MT air mass types had significantly higher pollutant concentrations. Significantly lower ambient concentrations of air pollution were found for the DP and MP air mass categories. In addition, TR air mass days had significantly lower concentrations of CO and NO_2.

Significantly higher rates of mortality in spring were found for the DP and MP air mass types. Higher rates of total mortality were evident for both categories, whereas higher rates of respiratory mortality were limited to the MP air mass days. Significantly little mortality was found during days characterized by the warmer and mild DT and MT air mass categories. Days characterized by a DT air mass had lower rates of total , elderly, and respiratory mortality; while MT air mass days retained lower rates of total, elderly, cardiac, and respiratory mortality. Considering that there is no evidence of significantly increased mortality during air masses regarded as possessing significantly higher concentrations of air pollution suggests that mortality in spring is associated more closely with variations in weather conditions than with polluted air.

Summer concentrations of air pollution were significantly higher for the DT and MT air mass categories. In fact, average values for most pollutants, in either air mass, were higher than for any other category at any other time of the year. For example, average particulate

Table 3.12.—T-test Results for Air Pollution and Mortality Between Synoptic Air Mass Categories by Season for Toronto, 1980 to 1994 (continued).

	Air Masses Derived from Spatial Synoptic Classification (SSC)						
	Dry Moderate	Dry Polar	Dry Tropical	Moist Moderate	Moist Polar	Moist Tropical	Transition
				Summer Season			
Days (%)	392 (28.4)	234 (17.0)	33 (2.4)	272 (19.7)	85 (6.2)	249 (18.0)	114 (8.3)
	Mean Air Pollution (95% C.I.)						
CO (ppm)	**1.02 ± 0.03˘**	**0.88 ± 0.04˘**	**1.52 ± 0.29^**	**1.11 ± 0.05^**	1.02 ± 0.07	**1.24 ± 0.06^**	1.05 ± 0.08
NO_2 (ppb)	22.8 ± 0.9	**18.9 ± 0.9˘**	**31.0 ± 3.2^**	**24.8 ± 1.1^**	**19.5 ± 1.6˘**	**26.2 ± 1.1^**	**21.4 ± 1.5˘**
SO_2 (ppb)	4.4 ± 0.5	**2.6 ± 0.4˘**	**8.5 ± 2.1^**	**3.6 ± 0.4˘**	**2.3 ± 0.6˘**	**5.6 ± 0.6^**	3.7 ± 0.7
O_3 (ppb)	**35.6 ± 1.3^**	**24.5 ± 1.1˘**	**62.2 ± 5.2^**	**27.8 ± 1.5˘**	**19.4 ± 1.8˘**	**44.3 ± 1.7^**	**27.3 ± 2.5˘**
PM_{10} ($\mu g/m^3$)	**33.1 ± 1.4˘**	**23.6 ± 1.1˘**	**58.5 ± 8.4^**	34.3 ± 1.6	**25.7 ± 2.5˘**	**50.3 ± 2.5^**	**32.1 ± 0.7˘**
$PM_{2.5}$ ($\mu g/m^3$)	**17.3 ± 0.8˘**	**12.0 ± 0.7˘**	**28.9 ± 4.7^**	**20.0 ± 1.0^**	**14.5 ± 1.5˘**	**27.9 ± 1.5^**	**17.2 ± 1.5˘**
	Mean Mortality and Lags* (95% C.I.)						
Total	37.5 ± 0.2	37.5 ± 0.3	**39.3 ± 0.6^**	**37.1 ± 0.3˘**	37.3 ± 0.4	**37.7 ± 0.3^**	37.7 ± 0.4
LAG1	37.5 ± 0.2	37.5 ± 0.3	**39.2 ± 0.6^**	**37.1 ± 0.3˘**	37.2 ± 0.4	**37.7 ± 0.3^**	37.7 ± 0.4
LAG2	37.4 ± 0.2	37.5 ± 0.3	**39.1 ± 0.6^**	**37.2 ± 0.3˘**	37.3 ± 0.4	**37.7 ± 0.3^**	37.7 ± 0.4
Elderly	27.7 ± 0.2	27.6 ± 0.3	**29.4 ± 0.5^**	**27.3 ± 0.3˘**	**27.3 ± 0.4˘**	**27.9 ± 0.3^**	27.7 ± 0.4
LAG1	27.7 ± 0.2	27.6 ± 0.3	**29.3 ± 0.5^**	**27.3 ± 0.3˘**	**27.3 ± 0.4˘**	**27.9 ± 0.2^**	27.8 ± 0.4
LAG2	27.6 ± 0.2	27.6 ± 0.3	**29.3 ± 0.5^**	**27.3 ± 0.3˘**	**27.3 ± 0.4˘**	**27.9 ± 0.3^**	27.8 ± 0.4
Cardiac	9.7 ± 0.1	9.9 ± 0.2	**10.5 ± 0.5^**	9.8 ± 0.2	**10.1 ± 0.3^**	**9.7 ± 0.2˘**	9.9 ± 0.3
LAG1	9.7 ± 0.2	9.9 ± 0.2	**10.5 ± 0.5^**	9.8 ± 0.2	**10.1 ± 0.3^**	**9.7 ± 0.2˘**	9.9 ± 0.3
LAG2	**9.7 ± 0.1˘**	9.9 ± 0.2	**10.5 ± 0.5^**	9.8 ± 0.2	**10.1 ± 0.3^**	**9.7 ± 0.2˘**	9.8 ± 0.3
Respiratory	2.4 ± 0.0	2.3 ± 0.1	2.5 ± 0.2	**2.3 ± 0.1˘**	2.3 ± 0.1	2.4 ± 0.1	2.3 ± 0.1
LAG1	2.4 ± 0.0	2.3 ± 0.1	2.4 ± 0.2	**2.3 ± 0.1˘**	**2.2 ± 0.1˘**	2.4 ± 0.1	2.3 ± 0.1
LAG2	2.4 ± 0.1	2.3 ± 0.1	2.5 ± 0.2	**2.3 ± 0.1˘**	**2.2 ± 0.1˘**	2.4 ± 0.1	2.3 ± 0.1
				Fall Season			
Days (%)	314 (23.0)	308 (22.6)	4 (0.3)	207 (15.2)	299 (21.9)	83 (6.1)	150 (11.0)
	Mean Air Pollution (95% C.I.)						
CO (ppm)	**1.31 ± 0.06^**	**1.09 ± 0.06˘**	1.38 ± 0.40	**1.34 ± 0.08^**	1.21 ± 0.06	**1.38 ± 0.10^**	**1.02 ± 0.08˘**
NO_2 (ppb)	**30.2 ± 0.3^**	**22.7 ± 0.9˘**	32.3 ± 18.3	25.4 ± 1.2	**22.7 ± 0.7˘**	**29.2 ± 0.9^**	**21.6 ± 1.2˘**
SO_2 (ppb)	**8.2 ± 0.8^**	**3.8 ± 0.6˘**	12.5 ± 13.8	**4.6 ± 0.7˘**	**3.3 ± 0.2˘**	**8.1 ± 1.1^**	**4.1 ± 0.7˘**
O_3 (ppb)	**16.0 ± 1.2^**	13.2 ± 0.6	**41.5 ± 34.9^**	**10.7 ± 1.3˘**	**7.8 ± 0.5˘**	**26.3 ± 4.6^**	13.3 ± 1.2
PM_{10} ($\mu g/m^3$)	**35.2 ± 2.0^**	**22.5 ± 1.1˘**	**53.9 ± 15.8^**	29.0 ± 2.5	**22.9 ± 1.1˘**	**48.6 ± 4.2^**	**26.6 ± 1.9˘**
$PM_{2.5}$ ($\mu g/m^3$)	**22.4 ± 1.4^**	**14.7 ± 0.8˘**	**29.9 ± 5.9^**	**20.0 ± 1.1^**	**15.7 ± 0.8˘**	**30.7 ± 2.7^**	**17.0 ± 1.3˘**
	Mean Mortality and Lags* (95% C.I.)						
Total	39.8 ± 0.3	39.8 ± 0.3	37.4 ± 4.5	**39.2 ± 0.4˘**	**40.1 ± 0.3^**	39.3 ± 0.7	**40.2 ± 0.4^**
LAG1	39.8 ± 0.3	39.9 ± 0.3	37.0 ± 4.8	**39.2 ± 0.4˘**	**40.1 ± 0.3^**	39.3 ± 0.7	**40.2 ± 0.5^**
LAG2	39.8 ± 0.3	39.9 ± 0.3	37.2 ± 5.6	**39.3 ± 0.4˘**	40.0 ± 0.3	39.3 ± 0.7	40.1 ± 0.4
Elderly	29.7 ± 0.3	29.6 ± 0.3	28.1 ± 5.6	**29.0 ± 0.4˘**	29.8 ± 0.3	29.2 ± 0.7	29.9 ± 0.4
LAG1	29.7 ± 0.3	29.6 ± 0.3	27.8 ± 5.6	**29.0 ± 0.4˘**	29.7 ± 0.3	29.2 ± 0.7	**29.9 ± 0.4^**
LAG2	29.7 ± 0.3	29.7 ± 0.3	28.0 ± 6.0	**29.1 ± 0.4˘**	29.6 ± 0.3	29.2 ± 0.7	29.9 ± 0.4
Cardiac	10.3 ± 0.2	10.4 ± 0.2	9.5 ± 1.3	10.5 ± 0.2	**10.5 ± 0.1^**	**9.9 ± 0.2˘**	10.2 ± 0.2
LAG1	10.3 ± 0.2	10.4 ± 0.2	9.5 ± 1.5	10.5 ± 0.2	**10.5 ± 0.1^**	**9.9 ± 0.2˘**	10.3 ± 0.2
LAG2	10.3 ± 0.1	10.4 ± 0.2	9.6 ± 1.6	10.5 ± 0.3	**10.5 ± 0.2^**	**9.9 ± 0.2˘**	10.3 ± 0.2
Respiratory	2.5 ± 0.1	2.5 ± 0.1	2.5 ± 0.1	2.5 ± 0.1	**2.6 ± 0.1^**	2.5 ± 0.1	2.5 ± 0.1
LAG1	2.5 ± 0.1	2.5 ± 0.1	2.4 ± 0.6	2.5 ± 0.1	**2.6 ± 0.1^**	2.5 ± 0.1	2.5 ± 0.1
LAG2	2.5 ± 0.1	2.5 ± 0.1	2.5 ± 0.7	2.5 ± 0.1	2.6 ± 0.1	2.5 ± 0.1	2.5 ± 0.1

Notes: * = predicted mortality using nonparametric LOESS smooth with 31 day span, **bolded** values indicate value for synoptic category is significantly different ($p \leq 0.05$) than the others according to a two-tailed *t*-test, ^ = mean is greater than comparison categories, ˘ = mean is less than comparison categories, NC = no comparison due to only one observation.

concentrations for both categories would violate the newly proposed Canada-Wide Standards (CWS) for PM_{10} and $PM_{2.5}$ of 60 and 30$\mu g/m^3$ respectively. Significantly lower air pollution concentrations were found for all other air mass categories.

Mean summer mortality estimates were significantly higher for DT and MT air mass types. Significantly lower rates of total and elderly mortality were apparent for the MM air mass days, and lower rates of cardiac mortality were evident for MT air masses. It is noteworthy that the results from spring and summer are quite dissimilar. Air pollution concentrations in spring and summer were significantly higher for the DT and MT air masses. However, mean mortality rates for the spring DT and MT air masses were significantly lower, whereas in summer they were significantly higher. One hypothesis is that, for spring, the warm, mild conditions somehow reduce the risk of death from air pollution, or that they lower mortality risk compared to colder conditions. Conversely, the combination of hot, humid temperatures of the air mass and the significantly higher concentrations (in summer) of air pollution suggest a potential synergistic effect resulting in significantly elevated mortality.

The result of significantly lower cardiac mortality rates during MT days is somewhat counterintuitive. As previously mentioned, previous research found that those with pre-existing cardiac-related conditions particularly susceptible to temperature extremes. The MT air mass is characteristically warm and humid, and in Metropolitan Toronto, typically associated with high concentrations of air pollution. Research from the air pollution literature has also found differential effects of ambient pollution concentrations for those with cardiac conditions. Mortality risks are generally higher for those with pre-existing chronic medical conditions. Thus it is unclear why there is significantly lower cardiac mortality for MT air masses.

Ambient air pollution concentrations in fall are significantly higher for the DM and MT air mass categories (Table 3.12). Interestingly, the DT air mass had significantly higher concentrations of O_3, PM_{10}, and $PM_{2.5}$; concentrations of the gaseous pollutant measures were not significant but still high. This is most likely due to the small number of observations (n = 4).

Total, cardiac, and respiratory mortality rates were significantly higher during MP air mass days. Significantly lower rates for total and elderly mortality were allied with MM air mass types; lower cardiac mortality was evident during MT air mass days.

In summary, mean mortality rates lower in summer. Rates of winter mortality are elevated during conditions with significantly lower air pollution concentrations and days with unstable (windy), transitional-type weather. Furthermore, significantly lower death counts occur during days with warmer temperatures. However, only cardiac mortality seems to be related to days with much colder temperatures. In spring the relationship between weather and mortality is even more apparent. Deaths decrease significantly on days represented by warm, moist air masses. In contrast, average daily deaths are significantly greater when colder temperatures dominate weather patterns. In fall, these patterns are much less evident, although total and elderly mortality is lowest when moist, moderate air masses are present.

Air mass/pollutant/mortality relationships are less nebulous in summer. Significantly elevated mortality, with cardiac mortality as the exception, occurs during air masses that also have the highest air pollutant concentrations and the greatest temperatures (i.e., dry tropical and moist tropical). Other characteristics of these air masses include relatively light winds, and moderate to little cloud cover. These results imply the possibility of a weather/pollution interaction during the summer months when either a DT or MT air mass is present. In contrast, significantly

lower mortality occurs during air masses with cooler temperatures and lower pressures. When the relationships between air pollution and mortality are stratified by air mass category, variation in mortality for each of the four seasons seems to coincide more closely with changes in weather, rather than with fluctuations of air pollution concentrations.

3.4.2.2 Relative Risk of Mortality from Air Pollution by Season and Air Mass Type

The total mortality relative risks for each pollutant evaluated by season at the difference between the maximum and the average values of their respective distributions are given in Tables 3.13 to 3.16, after adjusting for day of the week and nonparametric smoothed function of day of study.

For the winter season, significant positive associations were observed only for air masses characterized by cold, stable air, and light wind conditions. The highest relative risk was observed for $PM_{2.5}$ (1.056, t = 2.45), and SO_2 (1.044, t = 2.19) during dry polar (DP) air mass categories. Only two other associations were found to be significant during winter. The relationship between O_3 and mortality was significant for days characterized by a MP air mass (1.039, t = 2.09), and was only just significant between SO_2 and mortality for days characterized by a TR air mass (1.057, t = 1.97).

The MM and MP air mass categories exhibited the most consistent air pollution/mortality associations in spring. For the MM category, positive associations were observed for each of the variables except for ozone and $PM_{2.5}$. The largest relative risk was found for NO_2 (1.118, t = 2.37), followed by PM_{10}, CO, and SO_2. The largest relative risk in the MP category was also found for NO_2 (1.143, t = 3.80), followed by $PM_{2.5}$, PM_{10}, and SO_2. Two pollutant/mortality associations were significant for the DT air mass category. The relative risk for $PM_{2.5}$ (1.211, t = 2.14) was highest

Table 3.13.— Estimated Mortality Risk Attributable to Air Pollution by Air Mass Category for Winter, in Toronto, 1980 to 1994.

	Mean	Coefficient	SE	*t*-Statistic	RR (95% CI)
Dry Moderate (DM) n = 133					
CO	1.38	0.0137	0.0233	0.59	1.019 (0.957, 1.085)
NO_2	28.8	0.0003	0.0017	0.16	1.008 (0.917, 1.108)
SO_2 (lag 1)	10.1	0.0093	0.0156	0.60	1.023 (0.950, 1.101)
O_3 (lag 2)	9.2	0.0035	0.0028	1.26	1.033 (0.982, 1.085)
PM_{10} (lag 1)	29.9	0.0007	0.0019	0.35	1.020 (0.913, 1.140)
$PM_{2.5}$ (lag 1)	19.8	0.0012	0.0024	0.50	1.024 (0.932, 1.125)
Dry Polar (DP) n = 495					
CO (lag 1)	1.28	0.0107	0.0159	0.67	1.014 (0.974, 1.055)
NO_2 (lag 1)	26.9	0.0018	0.0010	1.81	1.050 (0.996, 1.108)
SO_2 (2d MA)	7.4	0.0204	0.0093	2.19	1.044 (1.005, 1.086)
O_3	11.6	0.0002	0.0015	0.16	1.003 (0.970, 1.037)
PM_{10} (lag 1)	27.7	0.0014	0.0010	1.46	1.040 (0.987, 1.095)
$PM_{2.5}$ (lag 1)	17.5	0.0031	0.0013	2.45	1.056 (1.011, 1.102)
Dry Tropical (DT) n = 1					
One observation only.					
Moist Moderate (MM) n = 224					
CO (lag 1)	1.47	0.0213	0.0204	1.04	1.032 (0.973, 1.094)
NO_2 (lag 1)	26.9	-0.0003	0.0013	-0.21	0.992 (0.925, 1.065)
SO_2 (lag 2)	7.8	-0.0014	0.0114	-0.12	0.997 (0.950, 1.047)
O_3 (2d MA)	5.4	0.0026	0.0028	0.95	1.014 (0.985, 1.044)
PM_{10} (lag 1)	28.6	-0.0011	0.0012	-0.90	0.969 (0.906, 1.037)
$PM_{2.5}$ (lag 1)	21.0	-0.0008	0.0016	-0.48	0.984 (0.921, 1.051)
Moist Polar (MP) n = 330					
CO (lag 1)	1.26	0.0260	0.0203	1.28	1.033 (0.983, 1.086)
NO_2 (lag 1)	26.2	0.0009	0.0014	0.63	1.023 (0.953, 1.098)
SO_2 (lag 1)	6.2	0.0161	0.0090	1.78	1.032 (0.997, 1.069)
O_3 (3d MA)	8.2	0.0047	0.0022	2.09	1.039 (1.002, 1.077)
PM_{10}	25.7	0.0008	0.0012	0.66	1.021 (0.960, 1.087)
$PM_{2.5}$	17.6	0.0015	0.0017	0.84	1.026 (0.966, 1.090)
Moist Tropical (MT) n = 12					
CO	1.86	0.0506	0.0608	0.83	1.099 (0.880, 1.371)
NO_2	29.3	0.0039	0.0063	0.62	1.121 (0.780, 1.612)
SO_2 (lag 2)	10.8	0.0443	0.0586	0.76	1.116 (0.840, 1.481)
O_3 (lag 1)	6.6	0.0126	0.0069	1.84	1.087 (0.995, 1.187)
PM_{10} (lag 2)	27.8	0.0048	0.0056	0.87	1.143 (0.844, 1.548)
$PM_{2.5}$ (lag 2)	20.3	0.0079	0.0082	0.96	1.173 (0.845, 1.627)
Transition (TR) n = 158					
CO (lag 1)	1.14	0.0358	0.0290	1.23	1.042 (0.976, 1.112)
NO_2 (lag 2)	23.7	0.0033	0.0019	1.77	1.081 (0.992, 1.178)
SO_2 (lag 1)	6.9	0.0267	0.0136	1.97	1.057 (1.000, 1.116)
O_3 (lag 2)	10.9	-0.0024	0.0024	-0.99	0.975 (0.926, 1.025)
PM_{10}	26.2	0.0016	0.0019	0.86	1.044 (0.946, 1.152)
$PM_{2.5}$	17.9	0.0028	0.0027	1.03	1.051 (0.956, 1.156)

Notes: 2d MA = two day moving average, 3d MA = three day moving average, CO = carbon monoxide, NO_2 = nitrogen dioxide, SO_2 = logarithm of sulfur dioxide plus one unit, O_3 = ozone, PM_{10} = particulate matter less than 10 microns in aerodiameter, $PM_{2.5}$ = particulate matter less than 2.5 microns in aerodiameter, SE = std. error, RR = relative risk.

followed by SO_2 (1.166, t = 2.32). In the DM category, the association between mortality and CO was also significant (1.086, t = 2.62).

Table 3.14.— Estimated Mortality Risk Attributable to Air Pollution by Air Mass Category for Spring, in Toronto, 1980 to 1994.

	Mean	Coefficient	SE	*t*-Statistic	RR (95% CI)
Dry Moderate (DM) n = 235					
CO (2d MA)	1.16	0.0713	0.0272	2.62	1.086 (1.021, 1.156)
NO_2 (lag 1)	30.3	0.0005	0.0010	0.52	1.016 (0.956, 1.081)
SO_2 (lag 2)	6.7	0.0202	0.0117	1.73	1.042 (0.994, 1.092)
O_3 (lag 2)	26.7	0.0018	0.0012	1.55	1.049 (0.988, 1.114)
PM_{10} (2d MA)	34.0	0.0011	0.0011	1.01	1.038 (0.965, 1.117)
$PM_{2.5}$ (2d MA)	16.8	0.0019	0.0020	0.95	1.033 (0.966, 1.104)
Dry Polar (DP) n = 414					
CO (lag 1)	1.04	0.0390	0.0230	1.69	1.041 (0.994, 1.091)
NO_2	23.4	0.0019	0.0012	1.59	1.045 (0.990, 1.103)
SO_2	4.1	0.0096	0.0095	1.01	1.016 (0.985, 1.047)
O_3 (lag 1)	22.8	0.0001	0.0014	0.09	1.003 (0.944, 1.066)
PM_{10}	25.6	0.0010	0.0012	0.86	1.026 (0.968, 1.088)
$PM_{2.5}$ (lag 1)	13.3	0.0016	0.0019	0.88	1.022 (0.973, 1.073)
Dry Tropical (DT) n = 48					
CO	1.29	0.0690	0.0595	1.16	1.093 (0.940, 1.271)
NO_2 (3d MA)	34.9	0.0068	0.0035	1.94	1.268 (0.998, 1.612)
SO_2	7.2	0.0731	0.0315	2.32	1.166 (1.024, 1.328)
O_3 (lag 1)	40.3	-0.0002	0.0018	-0.09	0.993 (0.860, 1.147)
PM_{10} (3d MA)	47.6	0.0036	0.0028	1.30	1.187 (0.917, 1.538)
$PM_{2.5}$	19.9	0.0096	0.0045	2.14	1.211 (1.016, 1.444)
Moist Moderate (MM) n =173					
CO (2d MA)	1.28	0.0698	0.0307	2.28	1.093 (1.013, 1.181)
NO_2 (2d MA)	28.8	0.0039	0.0016	2.37	1.118 (1.019, 1.227)
SO_2	4.5	0.0372	0.0159	2.34	1.065 (1.010, 1.123)
O_3 (lag 2)	16.9	0.0013	0.0012	1.10	1.023 (0.983, 1.064)
PM_{10}	31.4	0.0032	0.0014	2.20	1.104 (1.011, 1.206)
$PM_{2.5}$	18.8	0.0036	0.0021	1.76	1.071 (0.993, 1.155)
Moist Polar (MP) n = 267					
CO (lag 1)	1.13	0.0259	0.0253	1.03	1.030 (0.974, 1.089)
NO_2 (2d MA)	24.4	0.0055	0.0014	3.80	1.143 (1.067, 1.224)
SO_2 (2d MA)	3.4	0.0305	0.0137	2.22	1.046 (1.005, 1.089)
O_3 (lag 1)	16.8	0.0005	0.0011	0.46	1.009 (0.973, 1.046)
PM_{10}	24.9	0.0022	0.0011	1.94	1.056 (1.000, 1.116)
$PM_{2.5}$	15.1	0.0037	0.0014	2.68	1.058 (1.015, 1.102)
Moist Tropical (MT) n = 70					
CO	1.27	0.0586	0.0452	1.30	1.077 (0.963, 1.206)
NO_2	31.2	0.0027	0.0023	1.21	1.088 (0.948, 1.249)
SO_2 (lag 2)	6.8	0.0302	0.0235	1.29	1.064 (0.968, 1.170)
O_3 (lag 1)	30.9	-0.0002	0.0011	-0.16	0.995 (0.929, 1.064)
PM_{10} (lag 2)	41.9	0.0013	0.0014	0.93	1.058 (0.940, 1.191)
$PM_{2.5}$ (lag 2)	21.4	0.0048	0.0030	1.58	1.107 (0.975, 1.257)
Transition (TR) n = 173					
CO (lag 1)	1.07	0.0283	0.0329	0.86	1.031 (0.962, 1.104)
NO_2 (lag 2)	23.5	-0.0004	0.0016	-0.26	0.990 (0.921, 1.065)
SO_2 (lag 2)	4.9	0.0050	0.0140	0.36	1.009 (0.961, 1.059)
O_3 (lag 2)	23.4	-0.0015	0.0015	-1.03	0.965 (0.900, 1.033)
PM_{10} (lag 2)	28.9	-0.0009	0.0014	-0.65	0.979 (0.920, 1.043)
$PM_{2.5}$ (lag 2)	15.2	0.0012	0.0024	0.49	1.034 (0.904, 1.183)

Notes: 2d MA = two day moving average, 3d MA = three day moving average, CO = carbon monoxide, NO_2 = nitrogen dioxide, SO_2 = logarithm of sulfur dioxide plus one unit, O_3 = ozone, PM_{10} = particulate matter less than 10 microns in aerodiameter, $PM_{2.5}$ = particulate matter less than 2.5 microns in aerodiameter, SE = std. error, RR = relative risk.

In summer, the most consistent and significant air pollution/mortality associations were found during days characterized by a DT air mass. The largest relative risk for the DT category was observed for ozone (1.415, t = 2.53), followed by NO_2, $PM_{2.5}$, PM_{10}, and CO. The relationship between SO_2 and mortality was not significant. Other significant air pollutant/mortality relationships were found for the DM, DP, MM, and TR air mass categories. In the DM category, the largest relative risk was observed for CO (1.078, t = 2.39), followed by O_3, and SO_2. Only the association between SO_2 and mortality was significant in the DP category (1.052, t = 2.96). In the MM category, only NO_2 was significantly associated with mortality (1.114, t = 2.92). For the TR category, the association between NO_2 and mortality was marginally significant (1.086, t = 1.99).

Unlike in summer, the relationships between air pollution and mortality in fall were spread across five air mass categories. The MM category had the most consistent relationships and the largest relative risk values observed for CO (1.138, t = 3.14), NO_2, PM_{10}, and SO_2. Positive associations were also observed for CO (1.083, t = 2.27) and O_3 during days characterized by DM category conditions. A positive association was also found between CO and mortality (1.057, t = 2.23) for DP category days. In the DT category, positive associations were found for NO_2 (1.265, t = 2.02) and SO_2. The associations between mortality and NO_2 (1.121, t = 3.51) and $PM_{2.5}$ were also significant during days characterized by MP air mass conditions. No significant positive associations were observed for the MT and TR air mass categories.

In summary, the association between air pollution and total mortality generally was most apparent in air mass categories characterized by moderate temperatures, average barometric pressures,

Table 3.15.— Estimated Mortality Risk Attributable to Air Pollution by Air Mass Category for Summer, in Toronto, 1980 to 1994.

	Mean	Coefficient	SE	*t*-Statistic	RR (95% CI)
Dry Moderate (DM) n = 392					
CO (2d MA)	1.02	0.0739	0.0309	2.39	1.078 (1.014, 1.147)
NO_2 (2d MA)	22.8	0.0025	0.0013	1.90	1.057 (0.998, 1.120)
SO_2 (lag 1)	4.4	0.0219	0.0092	2.38	1.038 (1.007, 1.070)
O_3 (lag 1)	35.6	0.0013	0.0007	2.02	1.049 (1.001, 1.098)
PM_{10} (3d MA)	33.1	0.0012	0.0008	1.45	1.041 (0.986, 1.099)
$PM_{2.5}$ (3d MA)	17.3	0.0012	0.0014	0.88	1.021 (0.975, 1.069)
Dry Polar (DP) n = 234					
CO (lag 1)	0.87	0.0493	0.0368	1.34	1.044 (0.980, 1.111)
NO_2 (lag 1)	18.9	0.0021	0.0016	1.32	1.040 (0.981, 1.104)
SO_2 (lag 1)	2.6	0.0392	0.0133	2.96	1.052 (1.017, 1.087)
O_3 (lag 2)	24.5	0.0009	0.0008	1.11	1.021 (0.984, 1.060)
PM_{10} (lag 1)	23.6	0.0010	0.0009	1.20	1.025 (0.985, 1.066)
$PM_{2.5}$ (lag 1)	11.9	0.0012	0.0015	0.79	1.014 (0.979, 1.051)
Dry Tropical (DT) n = 33					
CO	1.52	0.1203	0.0371	3.24	1.201 (1.075, 1.341)
NO_2 (lag 1)	31.0	0.0089	0.0036	2.45	1.316 (1.057, 1.638)
SO_2	8.5	0.0644	0.0558	1.15	1.156 (0.904, 1.479)
O_3 (2d MA)	62.2	0.0056	0.0022	2.53	1.415 (1.081, 1.852)
PM_{10}	58.5	0.0036	0.0015	2.41	1.233 (1.041, 1.461)
$PM_{2.5}$ (2d MA)	28.8	0.0076	0.0028	2.68	1.244 (1.061, 1.460)
Moist Moderate (MM) n = 272					
CO	1.11	0.0509	0.0320	1.59	1.058 (0.987, 1.134)
NO_2 (2d MA)	24.8	0.0044	0.0015	2.92	1.114 (1.036, 1.198)
SO_2 (lag 2)	3.5	0.0018	0.0105	0.17	1.003 (0.972, 1.034)
O_3 (lag 1)	27.8	0.0011	0.0007	1.67	1.031 (0.995, 1.068)
PM_{10}	34.3	0.0014	0.0008	1.82	1.049 (0.996, 1.104)
$PM_{2.5}$ (2d MA)	19.9	0.0018	0.0013	1.40	1.036 (0.986, 1.089)
Moist Polar (MP) n = 85					
CO	1.02	0.0906	0.0738	1.23	1.097 (0.946, 1.271)
NO_2	19.5	0.0055	0.0029	1.89	1.113 (0.996, 1.245)
SO_2 (lag 1)	2.3	0.0482	0.0250	1.93	1.059 (0.999, 1.123)
O_3	19.4	0.0037	0.0025	1.48	1.073 (0.978, 1.179)
PM_{10}	25.7	0.0023	0.0019	1.24	1.062 (0.966, 1.167)
$PM_{2.5}$	14.5	0.0028	0.0030	0.94	1.041 (0.957, 1.133)
Moist Tropical (MT) n = 249					
CO (lag 1)	1.23	0.0399	0.0276	1.23	1.050 (0.983, 1.123)
NO_2	26.2	-0.0013	0.0015	-0.89	0.966 (0.894, 1.043)
SO_2	5.6	-0.0029	0.0128	-0.23	0.994 (0.949, 1.043)
O_3 (lag 1)	44.3	0.0002	0.0007	0.23	1.008 (0.946, 1.073)
PM_{10} (lag 1)	50.3	0.0073	0.0059	1.25	1.446 (0.812, 2.574)
$PM_{2.5}$ (lag 1)	27.9	0.0007	0.0009	0.77	1.020 (0.970, 1.072)
Transition (TR) n = 114					
CO	1.04	0.0789	0.0406	1.94	1.085 (0.999, 1.179)
NO_2	21.4	0.0039	0.0020	1.99	1.086 (1.001, 1.179)
SO_2	3.7	0.0226	0.0151	1.50	1.036 (0.989, 1.084)
O_3	27.3	0.0017	0.0011	1.59	1.048 (0.989, 1.110)
PM_{10}	32.1	0.0017	0.0011	1.60	1.055 (0.988, 1.127)
$PM_{2.5}$	17.2	0.0041	0.0018	2.24	1.072 (1.009, 1.140)

Notes: 2d MA = two day moving average, 3d MA = three day moving average, CO = carbon monoxide, NO_2 = nitrogen dioxide, SO_2 = logarithm of sulfur dioxide plus one unit, O_3 = ozone, PM_{10} = particulate matter less than 10 microns in aerodiameter, $PM_{2.5}$ = particulate matter less than 2.5 microns in aerodiameter, SE = std. error, RR = relative risk.

Table 3.16.— Estimated Mortality Risk Attributable to Air Pollution by Air Mass Category for Fall, in Toronto, 1980 to 1994.

	Mean	Coefficient	SE	*t*-Statistic	RR (95% CI)
Dry Moderate (DM) n = 314					
CO (2d MA)	1.31	0.0611	0.0269	2.27	1.083 (1.011, 1.161)
NO_2 (lag 1)	30.2	0.0000	0.0010	0.02	1.001 (0.942, 1.064)
SO_2 (lag 2)	8.2	0.0180	0.0115	1.56	1.041 (0.990, 1.094)
O_3 (lag 2)	16.0	0.0031	0.0011	2.74	1.051 (1.014, 1.089)
PM_{10}	35.2	0.0003	0.0009	0.36	1.012 (0.950, 1.077)
$PM_{2.5}$	22.4	-0.0004	0.0016	-0.29	0.990 (0.924, 1.060)
Dry Polar (DP) n = 308					
CO (lag 1)	1.08	0.0514	0.0230	2.23	1.057 (1.007, 1.110)
NO_2	22.7	0.0020	0.0012	1.72	1.047 (0.994, 1.104)
SO_2	3.8	0.0101	0.0095	1.06	1.016 (0.987, 1.046)
O_3 (lag 1)	13.1	-0.0005	0.0014	-0.39	0.993 (0.959, 1.028)
PM_{10}	22.5	0.0007	0.0012	0.63	1.017 (0.966, 1.071)
$PM_{2.5}$ (lag 1)	14.7	0.0029	0.0019	1.53	1.043 (0.988, 1.100)
Dry Tropical (DT) n = 4					
CO	1.37	0.0744	0.0614	1.21	1.107 (0.939, 1.306)
NO_2 (3d MA)	32.3	0.0073	0.0036	2.02	1.265 (1.007, 1.590)
SO_2	15.5	0.0695	0.0333	2.09	1.215 (1.012, 1.459)
O_3 (lag 1)	41.5	-0.0006	0.0019	-0.32	0.975 (0.838. 1.136)
PM_{10}	53.9	0.0018	0.0022	0.82	1.100 (0.875, 1.384)
$PM_{2.5}$	29.9	0.0091	0.0047	1.94	1.313 (0.997, 1.728)
Moist Moderate (MM) n = 207					
CO (2d MA)	1.34	0.0968	0.0308	3.14	1.138 (1.050, 1.235)
NO_2 (2d MA)	25.4	0.0037	0.0017	2.26	1.099 (1.013, 1.193)
SO_2	4.6	0.0364	0.0160	2.28	1.065 (1.009, 1.124)
O_3 (lag 2)	10.7	0.0012	0.0012	0.99	1.013 (0.988, 1.039)
PM_{10}	29.0	0.0031	0.0014	2.14	1.094 (1.008, 1.187)
$PM_{2.5}$	20.0	0.0038	0.0021	1.82	1.078 (0.994, 1.169)
Moist Polar (MP) n = 299					
CO (lag 1)	1.21	0.0213	0.0251	0.85	1.026 (0.967, 1.089)
NO_2 (2d MA)	22.7	0.0051	0.0014	3.51	1.121 (1.052, 1.196)
SO_2 (2d MA)	3.3	0.0262	0.0137	1.92	1.039 (0.999, 1.080)
O_3 (lag 1)	7.8	0.0006	0.0011	0.54	1.005 (0.988, 1.022)
PM_{10}	22.9	0.0021	0.0011	1.85	1.049 (0.997, 1.103)
$PM_{2.5}$	15.7	0.0034	0.0014	2.41	1.054 (1.010, 1.100)
Moist Tropical (MT) n = 83					
CO	1.38	0.0615	0.0455	1.35	1.089 (0.962. 1.231)
NO_2	29.2	0.0028	0.0023	1.23	1.085 (0.953, 1.235)
SO_2	8.1	0.0170	0.0254	0.67	1.038 (0.930, 1.159)
O_3 (lag 1)	26.3	-0.0002	0.0011	-0.18	0.995 (0.939, 1.054)
PM_{10} (lag 2)	48.6	0.0013	0.0014	0.92	1.067 (0.930, 1.224)
$PM_{2.5}$ (lag 2)	30.7	0.0050	0.0030	1.66	1.166 (0.972, 1.398)
Transition (TR) n = 150					
CO (lag 1)	1.02	0.0290	0.0327	0.89	1.030 (0.965, 1.100)
NO_2 (lag 2)	21.6	-0.0002	0.0016	-0.12	0.996 (0.932, 1.064)
SO_2 (lag 2)	4.1	0.0038	0.0139	0.27	1.006 (0.963, 1.052)
O_3 (lag 2)	13.3	-0.0015	0.0015	-1.00	0.980 (0.943, 1.019)
PM_{10} (lag 2)	26.6	-0.0009	0.0014	-0.63	0.977 (0.910, 1.050)
$PM_{2.5}$ (lag 2)	17.0	0.0013	0.0024	0.56	1.023 (0.945, 1.106)

Notes: 2d MA = two day moving average, 3d MA = three day moving average, CO = carbon monoxide, NO_2 = nitrogen dioxide, SO_2 = logarithm of sulfur dioxide plus one unit, O_3 = ozone, PM_{10} = particulate matter less than 10 microns in aerodiameter, $PM_{2.5}$ = particulate matter less than 2.5 microns in aerodiameter, SE = std. error, RR = relative risk.

and stable conditions. The number of significant associations was higher for spring, summer, and fall than for winter. In summer, the most significant air pollution/mortality associations were observed for DT air masses, which are hot and humid, but drier than MT air masses. This result is similar to that from the *t*-test analysis and reinforces the theory of pollutant/weather interactions during the summer season.

3.5 Discussion

The existence of a comprehensive data set for Metropolitan Toronto, Canada, has allowed for a thorough investigation of several aspects of mortality, weather, and air pollution in a single location. First, this paper has extended Burnett et al.'s analysis by assessing the sensitivity of previous findings to seasonal influences, and by including the specification of weather in models of daily mortality. The effects of several pollutants—CO, NO_2, SO_2, O_3 and measures of respirable particulates—were estimated simultaneously for distinct mortality outcomes. Second, synoptic climatological procedures were employed to identify weather patterns with statistically significant variations of air pollution and mortality, and to control for the confounding effects of weather in the air pollution/ mortality association.

3.5.1 Seasonal Analysis

Results from the seasonal analysis demonstrate positive and statistically significant associations between ambient concentrations of several air pollutants and the various mortality outcomes considered. Separate regression models for each pollutant and mortality outcome were fit for each season and adjusted for several potential confounders including day of the week, day of the study, and weather. Special attention was given to using a nonparametric smoother to reduce the confounding that could occur due to temporal patterns and weather.

The relationship between weather and daily mortality has been examined by several investigators,[36, 37, 40, 66] sometimes as part of an investigation of air pollution.[32, 45, 51] Most researchers reported a U or V-shaped relationship.[35-37] In this study, I found that the relationship varied by season, age and cause of death. For total mortality, deaths were mainly influenced by measures of temperature; however, weather models for winter included variables for humidity and atmospheric pressure, and summer models included values of wind speed and mid-evening temperatures. Previous research has alluded to the cumulative effects of temperature on mortality from insufficient night-time cooling.[42] Although also highly influenced by changes in temperature, elderly mortality was also related to cloud cover, wind speed, and barometric pressure in the spring and summer seasons. Cardiac deaths were particularly sensitive to colder temperatures, especially in winter and summer, whereas respiratory deaths were more influenced by the effects of cold weather throughout the year. These findings were fairly consistent across seasons and were used as a basis for the calculation of mortality risks from air pollution. Disaggregation of the relationships by age and cause of death also provided more comprehensive effect estimates, and has been recognized as an imperative procedure for estimating potential impacts from climate change.[67]

Ambient air pollutants, with major source contributions of vehicular origin, showed strong and statistically significant associations with total mortality which varied for elderly and cause-specific mortality outcomes. Consistent with findings from annual regression models, average CO concentrations (lagged one day in winter, and averaged over two days for other seasons), were a strong and significant predictor of total and elderly mortality. The pollution/mortality relationships were robust; they persisted despite how weather was modeled, i.e., fitting individual weather elements and/or the SSC method. The findings are

consistent and are within a range of risks reported for CO concentrations in other locations, such as Mexico City,[15] Philadelphia,[16] Sao Paulo,[68] and Buffalo.[26] Previous research has described pathophysiological mechanisms by which CO affects people with pre-existing cardiac conditions.[69] An association with cardiac deaths was found for this study, although only during summer and fall. For respiratory mortality, a significant effect was only detectable for fall. These results for both cardiac and respiratory mortality point toward the potential for weather to influence risk estimates, even after careful control. Furthermore, the lack of statistical power for both cardiac and respiratory values, due to the low frequency of deaths per day, may introduce instability into the relative risk estimates, producing large fluctuations and confidence intervals.

Positive and significant associations with total mortality were observed for NO_2 over all seasons studied, although effects were clearly stronger in summer and fall. Some studies have indicated short term effects of NO_2 on hospital admissions and visits to emergency departments.[69, 70] Positive associations of NO_2 on daily mortality have been reported in a European meta-analysis,[71] and in a study from Los Angeles.[2] However, from the studies available, the independent role of NO_2 on mortality is difficult to separate from that of particles, as they both often originate from similar sources.[14, 22] For elderly deaths, the association varied with season and was statistically and positively associated with NO_2 for all seasons except spring. In contrast, the association between NO_2 and cardiac mortality was very strong for spring, summer, and fall, but not significant in winter. For respiratory mortality, strong positive associations with NO_2 were found only in fall. These results are comparable with those from Milan which found an overall 7.6% increase in mortality from NO_2.[23] The results from this study indicate average mortality increases of between 5.3%, for total mortality, and approximately 60% for fall respiratory mortality. Again, caution should be

applied to the interpretation the respiratory death estimates due to the small number of observations available for analysis.

Seasonal influences were apparent in the association between SO_2 and all of the mortality outcomes under study, except for cardiac mortality, which showed significant positive associations all year round. For total mortality, associations with SO_2 were significant in all seasons except winter. SO_2 associations with elderly and respiratory mortality were also seasonally dependent, with no significant association in fall for elderly, and no significant summer association for respiratory mortality. The strength of the associations from this analysis are similar to those found in Athens,[72] New York, [73] and Shenyang, China,[27] but are observed at much lower concentrations. Previous research into the association between SO_2 and human mortality has been controversial, with some studies of cities in the eastern U.S. reporting no association,[74] and other studies describing the dependence of the effects on levels of particulates.[3] The results from this study only suggest an association between each pollutant singly, and mortality. In fact, mortality risk estimates are likely inflated due to multi-collinearity among the air pollutants. In essence, it is possible to assign too much risk to individual pollutants. A study design with an examination of multi-pollutant effects would be required to sort out the effects of highly collinear pollutants such as SO_2 and particulates.

The associations between ozone and total mortality, elderly mortality, and cardiac mortality exhibited seasonal dependencies, whereas positive and statistically significant associations with respiratory mortality were observed with O_3 all year round. In winter, for example, a significant 27% increase in respiratory mortality was observed from ambient concentrations of ozone. In contrast, ambient winter concentrations of O_3 only resulted in an insignificant 1.3% increase in total mortality. Associations between mortality and ozone have been reported in many previous studies,[2, 10, 16, 17, 25, 26] but not in all studies.[11, 15, 74] No study has

previously reported the magnitude of the effects for respiratory mortality from ozone found here for Metropolitan Toronto. The results of the present study also add to the base of information that suggests that increased O_3 concentrations are associated with increases in mortality. In this study, no indication of a threshold in the association was found. However, the stability of the relative risk estimates was subject to influences from weather, especially in winter. Concentrations of O_3 were higher on winter days with slightly warmer temperatures and lots of sunshine. For example, O_3 may statistically have a protective effect; however, it is likely the warmer winter temperatures that are proving beneficial to human health. Thus overall risk estimates may be considerably lower owing to low ozone concentrations, when contrasted with other seasons, and unseasonably warm weather.

Recent interest has focused on the particulate matter (PM_{10} and $PM_{2.5}$) association with daily mortality as evidenced by several earlier studies.[4, 8, 20-22, 25, 26] The results here indicate a strong and statistically significant association between total mortality and particulates, but only for winter, spring, and fall. For elderly mortality, the relationship with particulates was more tenuous. Positive significant associations were found with PM_{10} for spring and summer, and only for fall with $PM_{2.5}$. Similar patterns (spring and summer only associations) were found between cardiac mortality and PM_{10}. However, the relationship between cardiac mortality and $PM_{2.5}$ was significant only in summer and fall. Furthermore, respiratory mortality was only associated with $PM_{2.5}$ during the fall season. These seasonal influences on the relationship between particulates and mortality were not investigated by Burnett et al.,[29] who reported a 1.9 and 0.7% increase in total mortality for every 10 $\mu g/m^3$ increase in $PM_{2.5}$ and PM_{10}. The results from this research indicate a range (i.e., the smallest and largest percentage increase from any season) of increased mortality for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ of 0.5 to 3.5% for total

mortality, 1.0 to 1.7% for elderly mortality, 1.8 to 5.6% for cardiac mortality, and 1.0 to 5.7% for respiratory mortality. These results are consistent with six previous time series studies of total and cause-specific mortality from particulate air pollution.[4, 8, 10, 11, 13, 14] The results are also similar to another study that examined mortality rates and air pollution in Toronto from 1970 to 1990.[75] The authors, however, used a different time period, air pollution monitoring stations, and statistical procedures in their investigation than the one presented here, indicating a robustness of the particulate/mortality association.

The seasonal analysis was able to detect seasonal variability among the mortality risk estimates from air pollution. Overall, mortality/pollutant associations were stronger in spring and fall, even after inclusion of seasonally based and mortality-specific weather relationships.

3.5.2 Air Mass /Air Pollution / Mortality Relationships

Unlike previous analyses, which have utilized synoptic weather modeling to control for the confounding effects of weather,[32, 51] the purpose of this study was to explore the distribution of both air pollution and total mortality when stratified by synoptic weather categories. Very little research has examined the distribution of mortality and air pollution according to air mass categories, and understanding their association may lead to a better understanding of weather/air pollution/mortality relationships. For example, previous research has been limited to summer and winter due to limitations of the categorization procedure. The results from this study covered all four seasons.

The exploration of weather/pollution/mortality relationships using synoptic categorization has revealed some fascinating results. One major finding is that significantly elevated levels of winter mortality appear to be related to transition situations (TR) for total, elderly, and

respiratory mortality. Transition air masses are usually characterized by a frontal passage, with changing atmospheric pressures and dew point temperatures. TR air masses are present as the weather situation changes from one air mass to another. The TR air mass is also characterized by significantly lower levels of air pollution. This is reasonable due to the high winds and constant mixing associated with TR air mass types. Weather/mortality relationships are acute and maintained for at least a two-day lag. Increased risk of cardiac mortality was related to the presence of a dry polar (DP) air, also for lags extending to two days. This result is consistent with previous findings of increasing mortality during DP air masses in winter, although excess values tend to be much lower than for deaths associated with summer categories.[52] Counter-intuitively, some winter air masses emerged as significantly protective. For example, the moist moderate (MM) and moist tropical (MT) air masses had significantly higher concentrations of CO, SO_2, and particulates, yet daily values of total and elderly mortality were significantly lower in the MM category, and values of total and respiratory mortality were significantly lower in the MT category.

Unlike in other studies that have utilized synoptic categorization in winter,[76] no winter air mass was characterized by elevated values of both air pollution and daily mortality. The protective effect from milder conditions seems to counter-act any potential harmful air pollution impacts. Moreover, a previous investigation of Canadian heat-related mortality under present and $2xCO_2$ conditions, resulting from climatic change, indicated that any increases in heat-related mortality will not be offset by decreases in winter mortality.[56] In contrast, the results from this study suggest significantly lower mortality during the presence of the warm, mild DT and MT air masses. There is a clear indication that an increase in the frequency of these air masses would actually offset potentially elevated summer mortality rates from heat stress.

A similar pattern of counterintuitive results with regard to air pollution was found for spring air pollution/mortality relationships. The dry tropical (DT) and MT air masses were associated with significantly higher ambient concentrations of air pollution across all pollutant types. In the seasonal analysis previous described, it was noted that associations between air pollution and mortality were particularly strong and statistically significant for both spring and fall. Examination of the daily mortality for the DT and MT air mass types in spring and fall reveals significantly lower mortality, for all mortality outcomes under study. This finding remained consistent using smoothed values of mortality, after controlling for day of the week effects and other temporal influences, after fitting weather models for each season and for each mortality outcome, and for up to two days after the appearance of each air mass. Significant increases in total mortality were detected only for the DP and moist polar (MP) air mass types. This result is indicative of a mortality response associated more with cooler temperatures than with air pollution. Furthermore, the DP and MP air mass types had, on the whole, significantly lower concentrations of air pollution when compared to other categories.

High risk air mass categories were uncovered for Metropolitan Toronto in summer. The two hottest air masses, MT and DT, exhibited the highest mean air pollution concentrations and the highest mean total and elderly mortality. The DT air mass, which is very hot and dry, possessed the highest air pollution concentrations. This finding differs from previous research of Birmingham, Alabama, and Philadelphia, where the hottest air mass did not possess the highest O_3 and TSP (particulate) concentrations, although levels were above the period mean.[55] However, due to Toronto's more northerly location, the DT and MT air masses are likely to have picked up air pollution from the northern border states, in particular from the Ohio Valley region. An experiment using air pollution sampling

equipment along the southern edge of the Ontario landmass may assist to test and validate this hypothesis. Values for O_3 and particulates (PM_{10} and $PM_{2.5}$) were on average higher than the proposed Canada-Wide Standard 24-hour averages of 60 and 30 $\mu g/m^3$ respectively.

The presence of the MT air mass accounted for 18% of all summer days in the study period, and was the third most prevalent air mass type. Daily average mortality was higher for DT days than MT days. Physiological studies have revealed differential impacts between DT and MT air mass types on human health. The relatively high humidity associated with the MT air can lessen the body's ability to regulate evaporative heat loss by perspiration and vasodilation.[77] During DT days, vapour pressure gradients are sufficiently large to enable perspiration, but opportunities for evaporation can increase to such a level that perspiration production can be insufficient, leading to dehydration. The resulting hyperthermal conditions may lead to death.[78] The combination of high temperatures (DT days) and humidities, especially for MT days, combined with excessively high concentrations of air pollutants supports the findings from previous research of the potential for interaction between these variables and the resulting increased risk to human mortality.[46-48, 55]

Fall patterns of mortality among air mass types were similar to those found in spring. Statistically higher total, cardiac, and respiratory mortality was found during cool, damp conditions associated with the MP air mass and increased total mortality occurred during transition (TR) situations. Air pollution concentrations were on average significantly lower for these categories, implying a stronger relationship between mortality and weather characteristics than with air pollution concentrations. These results are somewhat dissimilar from the seasonal analysis which found strong pollutant/mortality associations in fall for all mortality outcomes. The reason is likely due to the difference in approach. The seasonal analysis employed techniques to reduce the effects of

confounding from weather in the determination of mortality risk estimates. In the air mass analysis, however, the effects of weather on mortality rates have not been removed since the analysis is performed in the context of synoptic weather conditions.

The discussion so far supports past research on winter mortality, which indicates that threshold meteorological conditions leading to higher mortality are either difficult to find or non-existent altogether.[56] In addition, the coldest air mass is associated only with slightly increasing cardiac mortality. It is, however, clear that summer offensive air masses, such as the DT and MT types from this study, have the potential to severely amplify human mortality, especially in areas such as Metropolitan Toronto where severe heat waves and air pollution episodes are infrequent, and the population is not well acclimatized to more extreme weather conditions .

Vigilance and care should be applied to the interpretation of the results from the within category regression analysis, where the logarithm of daily deaths was regressed against concentrations of air pollution after controlling for day of the week and non-random temporal influences. The purpose of the approach was to explore the potential for effect modification by estimating separate coefficients for each relationship between concentrations of air pollution and daily mortality via stratification by air mass type. The main intent was to develop relative risk values for each pollutant for each air mass category, and thus examine the change in the pollutant/mortality relationship between air mass types. One particular consequence of the approach was that little variation between days remained, a characteristic necessary for the time-dependent modelling approach utilized in this study. Similar methods in previous studies identified the potential for loss of precision, and for the potential to overinterpret variation among the coefficients, most likely due to statistical fluctuations rather than effect modification.[32]

Pollutant/mortality associations from the winter season were sporadic and no patterns were detected (Table 3.13). Thus no discussion of the results from the winter within-category analysis will be provided.

Within-category regressions of the association between air pollution and daily mortality in spring were consistent and strong for the cooler, more humid MM and MP air mass types and for the hot, dry DT air mass category (Table 3.14). For both MM and MP air mass categories, positive and statistically significant associations with mortality were observed for ambient air pollutants that have major contributions from vehicular sources. Percentage increases in mortality, according to a 95^{th} minus 5^{th} percentile distribution, were between 2.9 and 9.4% for CO, between 11.8 and 14.3% for NO_2, and between 7.0 and 9.8% for SO_2, for the MM and MP air mass types respectively. In comparison, and keeping in mind that the analysis was for all days rather than stratified by air mass type, percentage increases in mortality for these gases from the seasonal analysis were 8.6% for CO, 7.1% NO_2, and 5.2% for SO_2 (Table 3.4). Particle/mortality relationships for PM_{10} were less strong for MM and MP days. The percentage increase in mortality from PM_{10} for the MM air mass was 10.4%, and for the MP air mass was 5.6%. In comparison, the percentage increase in mortality from the seasonal analysis was 11.6%. For $PM_{2.5}$, however, the percentage increase in mortality was 5.8% for the MP air mass. The seasonal analysis results for spring showed a 5.2% increase in total mortality from ambient concentrations of $PM_{2.5}$ (Table 3.4). In addition, significant and very strong pollutant/mortality associations were found for DT air mass days for SO_2 and $PM_{2.5}$. The percentage increase in mortality for SO_2 was 19.1% greater for DT days when compared to all spring days. Similarly, the percentage increase in mortality from $PM_{2.5}$ for days with a DT air mass type was 16% greater when compared to all remaining spring days.

For summer, within-category regressions revealed statistically significant associations between ambient air pollution concentrations and mortality in the DM, DP, DT, MM, and TR air mass categories (Table 3.15). However, only the results from the DT air mass will be discussed because of the dramatic differences between mortality increases from the seasonal analysis (Table 3.4) versus those found in the DT air mass. Significant associations between CO, NO_2, O_3, and both particulate measures and total mortality were found for DT air mass days. The percentage increase in mortality from CO was 15.1% greater for DT days versus all remaining summer days combined. Similar marked differences in the percentage increase in mortality were also found in summer for NO_2 (26.7%), O_3 (37.7%), PM_{10} (21.0%), and $PM_{2.5}$ (22.6%). Further, it is interesting to note that the lower confidence interval value for DT summer air mass mortality is still usually higher than the relative risk value from the summer seasonal analysis (Table 3.4).

In fall, statistically significant within-category regressions were observed between air pollution and daily mortality in the DM, DP, DT, MM, and MP air mass categories (Table 3.16). The largest increases in mortality, when compared to seasonal regressions, were found for the DT and MM air mass categories. Positive and statistically significant associations were found between concentrations of NO_2 and SO_2, and daily mortality during DT air mass days. Percentage differences in increased mortality between days during a DT air mass and all days, as reported in the seasonal analysis, are 25.2% for NO_2 and 14.0% for SO_2. For days categorized as an MM air mass day, positive and statistically significant associations were found between concentrations of CO, NO_2, SO_2, and PM_{10}, and daily mortality from the within-category regressions. Percentage difference increases in mortality, noted as the difference between MM days and all days from the seasonal analysis, are 3.0% for CO, 3.5% for NO_2, 1.2% for SO_2, and 6.0% for PM_{10}.

In conclusion, the present study found significant seasonal associations between air pollution and daily mortality in Metropolitan Toronto. Risk of mortality varied with the air pollutant under investigation, with age, and by cause of mortality. The effect of collinearity among the pollutants using regression models with multiple pollutants was not performed and remains a limitation of this study.

Several time series studies have reported stronger effects of air pollution on daily mortality in warmer months than in cooler months.[12, 14, 19] Other studies have reported similar findings using synoptic climatological classification procedures.[32, 51, 52, 55] The results suggest that in spring, summer, and fall, especially on exceptionally warm days, both weather and air pollution strongly affect short-term mortality variations, most likely through some interaction between weather and air pollution. There is weak evidence from previous epidemiological studies that high temperatures interact with relatively high pollution levels,[46-48] and the same has been reported for acutely low temperatures.[79] This is the first study to quantify effect modification by weather on the relationship between ambient concentrations of air pollution and daily mortality using a synoptic weather classification system.

There are three possible, non-mutually exclusive explanations of the findings. First, in the warmer seasons and days characterized by warm and dry or moist air masses, people tend to spend more time outdoors or open windows to promote cooling. Therefore, exposure to air pollutants is higher and closer to what is being measured by the fixed ambient monitors. This phenomenon also plays a role in the reduction of bias from misspecification of exposure since outdoor concentrations would approach indoor concentrations on warmer days. Time-activity pattern data would be useful to clarify whether the results found in this study are the result of pollution/weather interactions, or higher individual exposures based on modified activity patterns. Second, other

determinants of daily mortality, such as acute respiratory infections, are less influential during warmer weather conditions. Third, it is well known that temperature and possibly other elements of weather affect mortality through various biological and physiological mechanisms, and it is plausible that a biological interaction between air pollution and temperature may exist, thereby increasing the effect of pollution on human health.

The most notable outcome of the present study concerns the way in which studies of air pollution/health linkages should be tackled. This study has shown clearly the potential for synoptic-based climatological approaches to elucidate variations of the severity of the air pollution/mortality relationship among a number of weather and air quality conditions. The utility of this approach and the findings presented should send a signal to public health and environmental policy makers that methods exist, possibly in the form of weather/pollution watch-warning systems rather than just weather (only) watch-warning systems, for preventing deaths from atmospheric risk factors.

3.6 References

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CHAPTER 4

Summary and Directions for Future Research

4.1 Research Summary

Using methodologies from environmental epidemiology, this thesis achieved its stated goals to (1) unite methodological frameworks from investigations of human health outcomes from atmospheric risk factors, and (2) provide comprehensive information on the epidemiology of human mortality from air pollution in an urban setting. The preceding chapters reported the results of two ecologic epidemiological studies that examined the daily association of atmospheric risk factors, such as weather and ambient concentrations of air pollution, and nonaccidental, short-term human mortality among the general population in Toronto, Ontario, from 1980 to 1996 (to 1994 in Chapter Three).

Chapter One identified the possible causal mechanisms of human mortality from atmospheric risk factors and reviewed previous research from the areas of biometeorology, environmental epidemiology, medical climatology, and statistics. Specific attention was devoted to investigations of environmental relationships with human mortality, urban populations, and modern statistical approaches from time series analyses. Although the focus was on literature from the past decade, except for a few older seminal papers, and included numerous study designs and data sources, several uncertainties, methodological issues, and pertinent findings were identified.

Arguably, the additive effects from weather and air pollution account for approximately 10 to 30% of the daily variation in human mortality. Many studies have identified both long-term seasonal, and acute effects from variations in weather.[1-6] Studies with similar approaches and methodologies have identified a consistent association between low ambient concentrations of air pollution and human mortality.[7-10] Increased risk of mortality, from either weather and/or air pollution, has been identified among susceptible populations, particularly the elderly,[11-13] and those with pre-existing or chronic health conditions.[14, 15] The literature review also identified significant statistical issues. These issues included the requirement for proper control of potential confounding factors,[16, 17] accurate estimations of exposure,[18] and evaluation of potential interactions between atmospheric risk factors.[19, 20] The essential challenge in many epidemiological investigations of atmospheric risk factors on human mortality is that of dependably distinguishing the differential impacts from fluctuations in weather patterns and small contributions of air pollution.

Chapter Two examined and quantified the relationship between summer weather, using a weather index, and daily fluctuations of human mortality. This was an ecologic time-series analysis that employed nonparametric approaches (smoothing or filtering) to control for the confounding effects of seasonality, temporal trends, changes in population size over time, and air pollution. The review from Chapter One revealed that many investigations of impacts on human mortality from weather failed to control for the confounding effects of air pollution and thus it was important to account for the relationship when estimating mortality risk. Smoothing the data also dealt with serial correlation evident from tests of autocorrelation of the mortality data. The findings indicated that the association between total nonaccidental mortality and summer weather, as measured by humidex, is small yet significant.

The relationship between humidex and daily mortality was reported by gender, age, and cause of death. There was no significant (p • 0.05) association between humidex and total, male, or female mortality evaluated at the difference between the 95th and 5th percentiles of the humidex distribution; however, the association with female mortality was stronger than for males and did approach significance (p = 0.07). In addition, the relationship between humidex and mortality varied with age. Positive and statistically significant associations between humidex and mortality were found for the elderly subpopulation with the largest risk found for those over the age of eighty-four. Incremental increases in risk above the age of sixty-five, determined by ten year increments, were not significant, most likely due to the small number of observations in these groups.

Total mortality was also subdivided into cardiac and non-cardiac subpopulations, with the cardiac group further divided into those who died from ischemic heart disease (IHD). Strong and statistically significant associations between humidex and mortality were found for cardiac mortality and mortality from ischemic heart disease, with percentage increases of 13.1% and 12.7% evaluated at the difference between the 95th and 5th percentile distribution of humidex respectively. An interesting result from the study was the shape of the response curve between elderly and cardiac mortality. The shape of the response for the elderly was more of a bathtub, or U shape, while the shape of the curve for cardiac mortality could be defined as a V shape, with a much steeper slope. The steeper curve for cardiac mortality indicates that this cause of death is particularly sensitive to both cold and hot temperature extremes.

The magnitude of the relationships and the shape of the response for elderly and cause-specific mortality in Metropolitan Toronto were similar to that found in the Netherlands.[21] Variations in temperature, as represented by the humidex, are known to affect humans through well-

understood pathophysiological mechanisms.[22,23] A notable outcome of the study was the consistency of the association over a broad range of humidex values. Many previous studies tended to investigate mortality risks from extreme weather only. The research described in Chapter Two established that significant increases in mortality risk can result from small variations in what are generally considered to be comfortable atmospheric conditions. Further research in other large cities with temperate climates may help to solidify the consistency of the results described here.

Chapter Three explored the relationship between atmospheric risk factors and human mortality from a slightly different perspective. Instead of estimating increased risk from the effects of weather, the third chapter investigated the association between ambient levels of air pollution and nonaccidental mortality in Metropolitan Toronto from 1990 to 1994. However, several techniques were utilized to examine how this association varied with other atmospheric conditions, such as weather. This alternative approach focused less on the traditional empirical methods used to control for the confounding effects of weather; rather, the objective of the investigation was to explore the potential interactions or synergism between weather and air pollution, and their combined effects on human mortality. To achieve this objective, Chapter Three used data from a previously published study, of air pollution and human mortality in Metropolitan Toronto, which found positive and statistically significant associations.[24]

The first tactic examined the seasonal association between ambient concentrations of air pollution and mortality by fitting separate regression models for each season and each pollutant, across four separate mortality outcomes (total, elderly, cardiac, and respiratory mortality). Similar control for temporal influences using smoothing was applied and risk estimates compared to the previously published estimates. The second

and less traditional procedure involved the development of synoptic air mass categories derived from a set of weather elements. The distribution of air pollution and daily mortality was described according to each resulting category. In addition, regressions were performed within each category to investigate the variability of estimated risk of death from air pollution between days with significantly different weather patterns.

There were two major outcomes from the second study in Chapter Three. First, it was found that the association between mortality and air pollution changes in strength from season to season. Stronger relationships were evident in spring and fall, and less so in summer and winter. Furthermore, no systematic pattern of variation could be identified between pollutants with similar sources. In some cases, relative risk estimates were below the value of one. Rather than interpreting these results to indicate that air pollution is beneficial to health, instead it is more plausible that weather during these days was a protective factor that compensated for any potentially acute health effects from increased air pollution.

Second, the division of the weather data into synoptic categories allowed for the identification of certain weather conditions associated with significantly higher and/or lower air pollution and mortality. Regression model estimates from analyses within each category revealed some striking and some counter-intuitive results. In winter, spring, and fall, increased mortality was limited to weather conditions associated with lower mean concentrations of air pollution, suggesting the larger role of weather as a risk factor for mortality. However, in summer, risk estimates were markedly higher for categories previously identified as having significantly elevated concentrations of air pollution and characteristics of weather previously shown to be harmful, i.e., high temperatures, and very dry or very humid conditions. For many pollutants the resultant

percentage increase in mortality was between 5 and 25% higher than those results found previously from seasonal regression estimates.

Few previous studies have identified or quantified mortality risk estimates, across multiple mortality outcomes, from either the interactive,[25] or differential,[26] impacts of both weather and air pollution using weather categories. Two studies of data from Philadelphia reported contrasting results.[27, 28] Further research into the relationship between air pollution and mortality using synoptic weather modeling is required. This thesis is best viewed as exploratory and an initial stride in research examining atmospheric risk factors of human mortality.

At present, it is not clear whether the relationships here can be generalized to other locales with similar climatic, industrial, and demographic conditions. The ecologic design, large sample size, and reasonably accurate estimates of exposure have provided the backdrop for an analysis of the impact from both weather and air pollution on the population of Metropolitan Toronto, Ontario. In my view, the results of this study have made a small but important contribution to the body of knowledge in environmental epidemiologic research.

4.2 Future Directions

Recent time series studies of daily mortality and hospitalization have found statistically significant relationships with air pollution concentrations at present ambient levels, with little or no indication of threshold relationships. In essence, there is little proof or direct information on changes that might occur from the imposition of further air pollution controls. Further examination of data from any given location and time period can help in this regard. The synoptic-based approach in the second study of this thesis found what can be considered a counterintuitive result; the mortality risk from ozone was highest during low ambient temperatures and low ozone concentrations for respiratory

mortality, as opposed to during hot weather and high ozone concentrations. This finding was reinforced by mortality risk estimates for respiratory mortality from ozone in the seasonal analysis. Further research should attempt to help clarify such findings by strengthening our understanding of physiological responses, or through the development of study designs and statistical models capable of better understanding weather/air pollution interactions.

It was also clear, from both studies in this thesis, that there exists a strong positive association between weather and/or air pollution and mortality. Unfortunately, it is not clear what percentage of deaths were due to the phenomenon known as harvesting, or mortality displacement. Further research is required to find out, under the scenario of mortality displacement, the number of those people who would have died shortly after their actual date of death in the absence of atmospheric risk factors.

Further emphasis should also be placed on alternative study designs, preferably ones that can estimate risks at an individual level of analysis. There is no doubt that the ecologic design used in this thesis provides the necessary information to guide further research. However, because of statistical difficulties due to uncertainties in actual exposures, the details of the relationship between human mortality and exposures to weather and air pollution remain essentially unobservable. For example, it has become very clear that much more time is spent indoors than previously estimated. This type of exposure pattern introduces misclassification bias into the study, and has a profound effect on the ability of an ecologic study to fully capture actual exposures to weather extremes and air pollution. Explorations of viable study designs to address these issues would also strengthen existing arguments of a causal relationship between atmospheric risk factors and human health impacts.

4.3 Addressing Causality

The study of environmental epidemiology, akin to many other applied sciences, rarely warrants conclusions about causation. According to Susser, causation must usually be inferred and never absolutely.[29]

> *"Only judgement can prevent the hypercritical rejection of useful results."*[i]

This statement should keep epidemiologists and health researchers mindful that many interpretations of findings in epidemiology are based ultimately on the judgement of "experts", rather than on the statistical conclusions from one study. An inherent dilemma for air pollution and human health research, for example, is the existence of multiple causes and results. Even systematic and careful estimation of relationships and functional forms does not grant permission to the investigator to conclude causal association based on statistical inference. Thus, it is pertinent to briefly summarize how the results from this thesis can be evaluated against the criteria for causation discussed in Chapter One. If air pollution and/or weather are responsible for causing mortality, then exposure to them should occur prior to the response.

To investigate the temporal relationship hypothesis, and the temporal direction of the associations, this thesis examined the correlations of the environmental exposures (air pollution and weather) on the day of and days prior to the date of mortality. The temporal direction of the associations can be determined by connecting the rate of mortality on any day to the amount of exposure on the following day. Although this phenomena has never been reported in the literature and was not evident

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[i] Susser M. Causal thinking in the health sciences: Concepts and strategies of epidemiology. New York: Oxford University Press, 1973, p. 141.

in this thesis, temporal associations that are forward in time could occur due to positive serial correlations in time of the exposure variable. This type of correlation was reduced in this thesis by utilizing the LOESS nonparametric smoother.

The strength of the association between mortality and both air pollution and weather exposures, in this thesis, was measured using the relative risk (RR). A relative risk of 1.0 indicates a null result or that exposure to air pollution does not increase risk of mortality. Alternatively, a relative risk below 1.0 or above 1.0 could be interpreted as being either protective or harmful respectively. In general, the results from this thesis reported relative risks above 1.0; however, a few results were below most likely due to the influence of above average temperatures in winter, and cooler temperatures in summer.

A bathtub shape is characteristic of the does-response plots (curves) in Chapter Two, illustrating the relationship between nonaccidental mortality and values of humidex. The result suggests a dose-response relationship between much cooler or warmer humidex values and significant increases in daily mortality. Plots of the seasonal concentration response relationships between air pollution and nonaccidental mortality were generally linear. The associations between air pollution and nonaccidental mortality stratified by air mass types were not plotted.

Both studies in this thesis reported relative risks that were in the range of risks reported by many other investigations from the literature and support the hypothesis that air pollution and weather, either independently or synergistically, affect rates of nonaccidental mortality. Thus this thesis contributes to the literature and supports similar research in terms of consistency between studies. Moreover, care was taken to control for confounding factors that could lead to unstable effect estimates. Bivariate relationships between known confounders and nonaccidental mortality were examined, and significant predictors were kept to adjust

for the influence of potential risk factors. In addition, only the effects from individual pollutants were identified thereby reducing the potential for confounding from covariation with other pollutants over time. One drawback of this approach is the identification of a "true" risk estimate based on actual exposures to multiple pollutants.

It would be presumptuous of this author to conclude a causal relationship between the atmospheric risk factors examined in this thesis, from humidex and air pollution, and nonaccidental mortality. The two remaining criteria for causation, those dealing with biological plausibility and specificity of the association, were not dealt with in this thesis. Adjustment to the methods described in this thesis could account for the specificity of the association with relative ease. This would entail the additional examination of a 'control' series of events not *a priori* thought to be related to variations in humidex and to ambient air pollution. The hypothesis would be that the association between the control events and the exposures would be less strong than for those outcomes examined in this thesis. Unfortunately, the study design employed here cannot directly assess the biological mechanisms of the observed relationships. The literature revealed several experimental studies designed to investigate the effects of air pollution on animal subjects; however, definitive conclusions are few and the biological mechanisms vague. Additional research in this area may support or refute claims of causal effects.

Many of the criteria proposed by Hill[30] have been met in this thesis, and to a greater extent, in the area of environmental epidemiology. It is the opinion of this author that the research described here supports the notion of a true causal effect rather than a random statistical artefact or correlation. Thus, there is no real "safe" level of air pollution, and human health is affected by the tail ends of climate variability. Nevertheless, one question still remains: *What is the risk we are willing to accept knowing that exposures to both atmospheric risk factors are largely preventable?*

It has long been recognized that the relationship between the quality of the environment and human health is inextricable. Recent research has reinforced notions that waste residuals from society (such as air pollution) are but one of the stresses that interrupt ecosystem function, resulting in a potentially disastrous array of negative consequences.[31] In these situations it is often difficult to tease apart the contributions of any single stress to any single effect. Public health officials, politicians, researchers, and society itself, must begin to accept the fact that research, policies, and actions must reflect the complexities of ecosystem function within which we live. Air pollution can be eliminated and health effects from weather reduced. Further efforts should focus on the potential to prevent not only negative human health outcomes, but also degradation of the environment on which we depend.

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APPENDICES

APPENDIX A

Generalized additive models (GAMs) are an additive extension of the family of generalized linear models. Generalized linear models are themselves a generalization of linear regression models. The predictor effects are assumed to be linear in the parameters, but the distribution of the responses, as well as the *link* between the predictors and this distribution, can be quite general.

Generalized additive models extend generalized linear models in the same manner that the additive model extends a linear regression model, that is, by replacing the linear form $\alpha + \Sigma_i X_i \beta_i$ with the additive form $\alpha + \Sigma_i f_i (X_i)$.[1] The additive predictor replaces the linear predictor (possibly by a smoothed term). GAMs also represent a method of fitting a smooth relationship between two or more variables through a scatterplot of data points.[2] They are useful when the relationship between the variables is expected to be of a complex form, not easily fitted by standard linear or non-linear models. No *a priori* reason for using a particular model is required. GAMs do not involve strict assumptions about the relationship that are implicit in standard parametric regression where such assumptions may force the fitted relationship away from its natural path at critical points. They generally suggest a curved relationship which may more accurately reflect the progress of the condition. GAMs can also deal with categorical variables, often easier than continuous ones.

Smoothing parameter selection, and even assessment of model fit, can take place either using cross-validation techniques or by using Akaike's Information Criterion (AIC):

$$AIC = -2[\log(L) - q],$$

where log(L) is the log-likelihood evaluated at the maximum likelihood estimates of the model parameters, and q is the number of parameters in the model. The first term is a measure of how well the model fits the data,

and the second term is a penalty for the addition of parameters (and hence model complexity).[3] The model giving the smallest value of AIC is selected by the criterion.

There is always the risk of overfitting the data and interpreting spurious features of the fitted curves. Width of standard error bands, approximate deviance tests and residual plots give support to important features of the fit, and give warnings about features that might be spurious.

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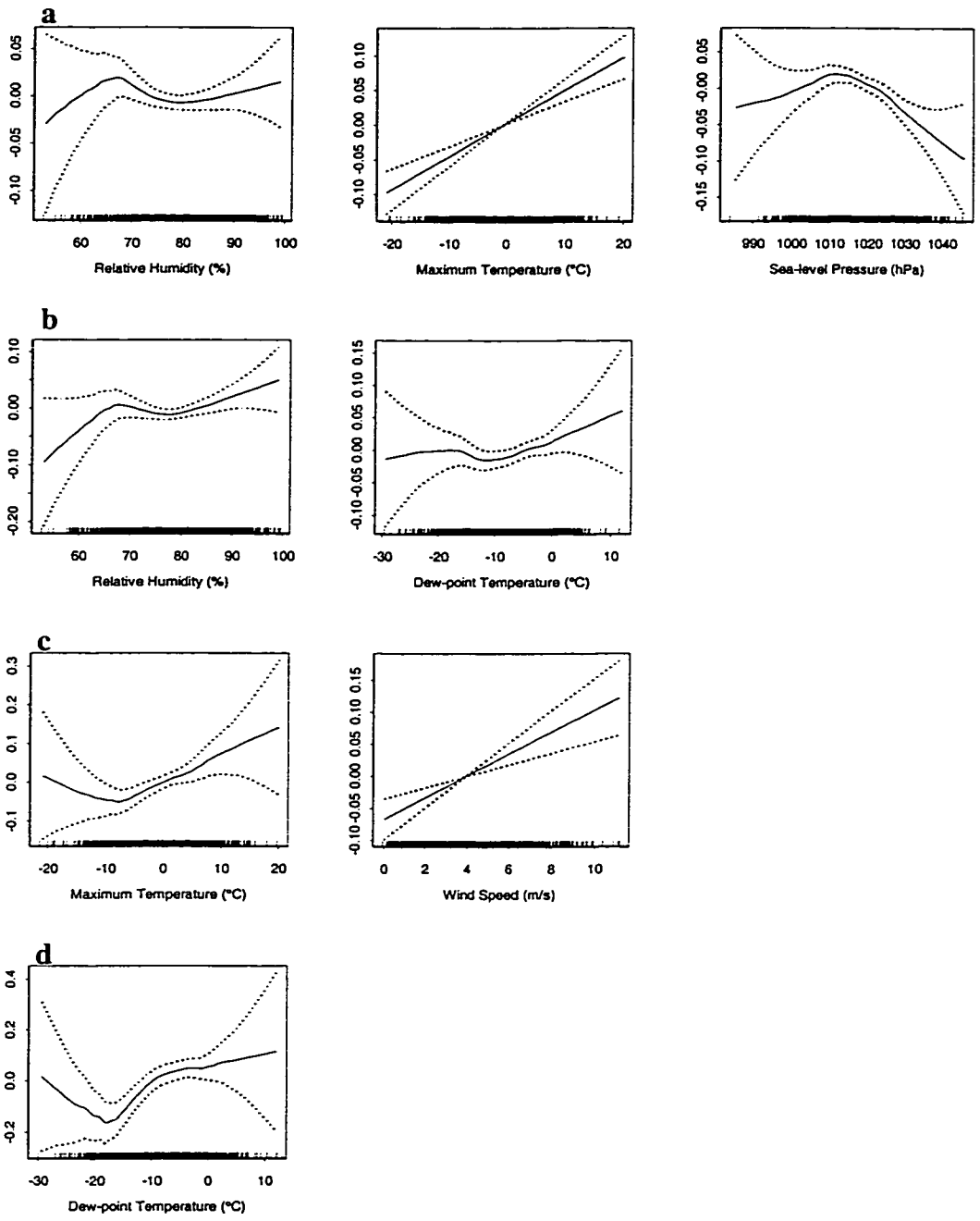
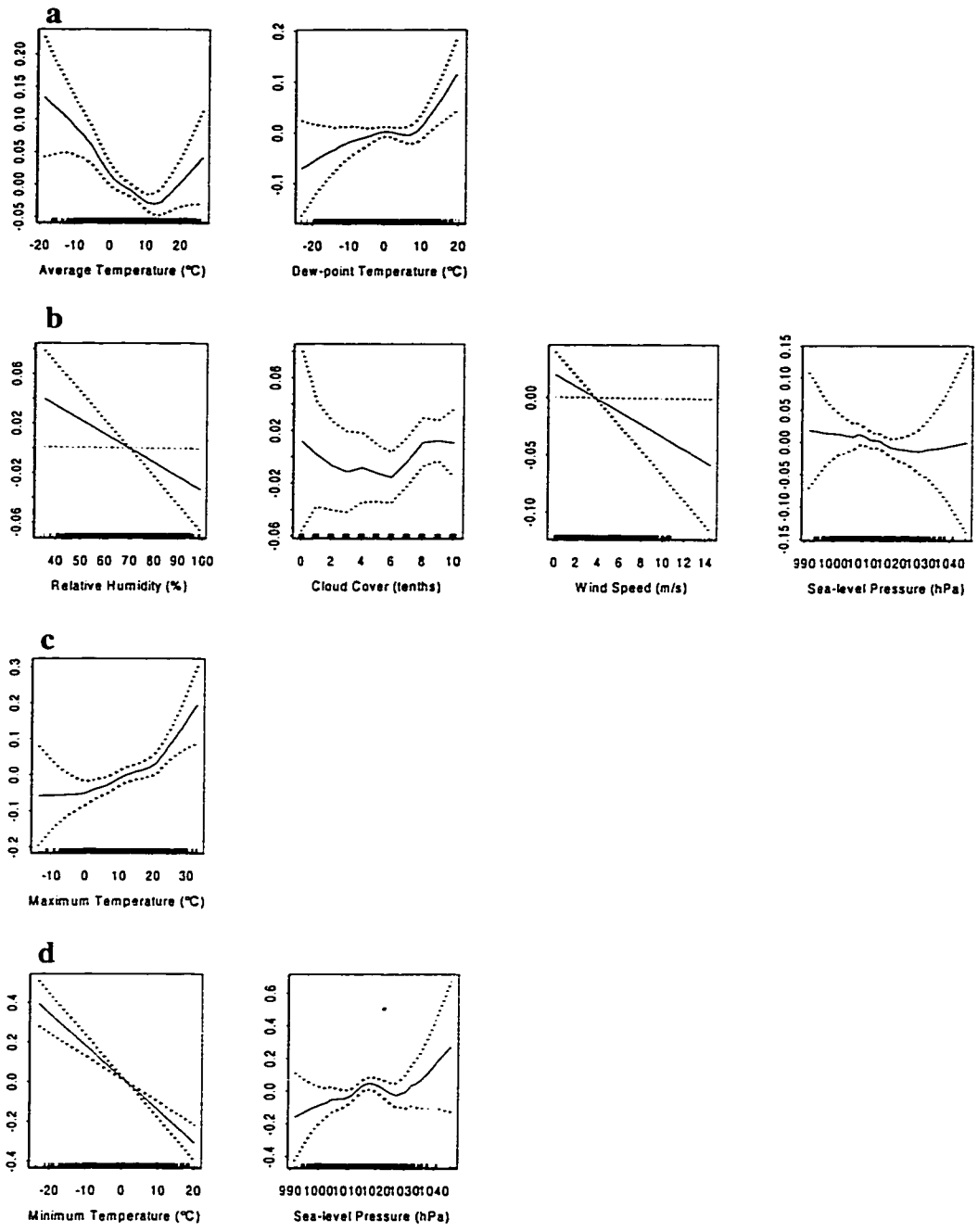
APPENDIX B



Winter. Association between (a) total mortality and smoothed two-day lagged relative humidity, same day maximum temperature and smoothed two-day lagged sea-level pressure, (b) elderly mortality and smoothed two-day lagged relative humidity and smoothed two-day lagged dew-point temperature, (c) cardiac mortality and smoothed same day maximum temperature and same day wind speed, and (d) respiratory mortality and smoothed same day dewpoint temperature. Fits achieved using LOESS with a 50% span (95% confidence intervals given by dashed lines and data represented by tick marks).



Spring. Association between (a) total mortality and smoothed two-day lagged average temperature and smoothed same-day dew-point temperature, (b) elderly mortality and smoothed three-day lagged relative humidity, smoothed three-day lagged cloud cover, two-day lagged wind speed and smoothed same-day sea-level pressure, (c) cardiac mortality and smoothed two-day lagged maximum temperature, and (d) respiratory mortality and same-day minimum temperature and smoothed three-day lagged sea-level pressure. Fits achieved using LOESS with a 50% span (95% confidence intervals given by dashed lines and data represented by tick marks).