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Associations between Daily Asthma Hospital Admissions and Ambient Air Pollutants in Montreal, 1992 to 1999

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A Thesis

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ABSTRACT

Associations between Daily Asthma Hospital Admissions and Ambient Air Pollutants in Montreal, 1992 to 1999

Kim Marie Deschamps

This study investigates the associations between daily asthma hospital admissions and concentrations of ambient pollutants in Montreal, Quebec from 1992 to 1999. Health data was collected from the hospital discharge database (Med-Echo) provided by the Quebec Ministry of Health and Social Services. Selected stationary monitoring sites in and around Montreal were used to obtain daily mean air pollutant concentrations of particulate matter having aerodynamic diameters of 10 μm or less, coefficient of haze, sulfur dioxide, ozone, nitrogen dioxide, carbon monoxide and hydrogen sulfide. Weather data was procured from Environment Canada's weather station at Dorval International Airport (45 28'N, 73 45'W). A time series analysis in the format of a general additive model (GAM) was used to model the logarithm of daily asthma admissions as a function of the daily averaged air pollutants. Non-parametric smoothing functions, specifically locally weighted regression smoothers (LOESS), were used to remove confounding factors such as seasonal fluctuations, calendar year, and weather. The data set was analyzed according to the whole year and seasons (cool and warm periods). Asthma admissions were studied by age. Carbon monoxide and coefficient of haze were positively associated with asthma hospital admissions at lags of 0, 1, and 3-day mean.

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CHAPTER I

INTRODUCTION

AIR POLLUTION AND HUMAN HEALTH

There is nothing that seems more immediate and important than personal health, our own and that of our loved ones. It is here that we feel the greatest urgency to solve problems of environmental pollution, and it is here that the consequences of our actions are most dramatically demonstrated. Cunningham (1994).

Air pollution is not a new phenomenon. In fact, poor air quality has been documented throughout the Middle Ages, into the Industrial Revolution of the 18th and 19th centuries and right up to the present day. As countries industrialized, new anthropogenic air pollutants emerged. Since the industrial revolution, the quantity of air pollutants injected into the atmosphere has increased dramatically, although since the 1950s the quality of the air has been improving. Canadians have been responsible for the release of a wide variety of air pollutants. However, this study area, Montreal, Quebec, has comparatively low levels of air pollutants when compared with other major cities in industrialized nations (Goldberg et al., 2000). In spite of the relatively low levels of air pollutants in Montreal, researchers are finding associations between air pollutants and public health (Goldberg et al., 2000; Rainham, 2000).

1.1 Outdoor Air Pollutants of Interest

Canadians are extremely concerned with the quality of the air they breathe, particularly when it impacts their health. Outdoor air pollution consists of a complex mixture of substances, including sulfur dioxide (SO_2), ozone (O_3), nitrogen oxide (NO), nitrogen dioxide (NO_2), carbon monoxide (CO), particulates, and volatile organic compounds (e.g., benzene). Particulates are a complex mixture of solid and liquid droplets and have wide distributions of size and mass. Coarse particles (particles between 2.5μm and 10μm in aerodynamic diameter) derive from a variety of sources including windblown dust and grinding operations (Hester and Harrison, 1998). Fine particles (particles less than or equal to 2.5μm in aerodynamic diameter) are primarily from the combustion of fossil fuels (Hester and Harrison, 1998). The chemical and physical composition of particles vary widely and may include elemental and organic carbon, sulfates, nitrates, pollen, microbial contaminants, and metals. Chemical reactions of fine particles with SO_2 and NO_x in the atmosphere form strong acids, such as acid aerosols (sulfuric acid, nitric acid) and hydrochloric acid. In addition, urban air contains chemical compounds (e.g., benzene, aldehydes (formaldehyde), ethene, propene, 1,3-butadiene, PAHs) that are carcinogenic. Automobiles and trucks make a major contribution to air pollutants from direct emissions of NO_x, CO, volatile organic compounds, and polycyclic aromatic hydrocarbons, and from atmospheric chemical reactions with these primary pollutants (O_3, acid aerosols, and particles).

The focus of this thesis is in those air pollutants that are likely to cause short-term health effects and tend to be generated by anthropogenic activities. The most commonly measured outdoor air pollutants in Canada include ground-level ozone, particulate matter,

carbon monoxide, sulfur dioxide, and nitrogen oxides. These pollutants "contribute the largest volume of air quality degradation and also are considered the most serious threat of all air pollutants to human health and welfare" (Cunningham, 1994).

1.2 Asthma Admissions and Air Pollution

Asthma can be a debilitating and even life-threatening disease if not managed properly. It causes recurrent episodes of coughing, wheezing, and breathlessness when airways become inflamed, swell up and tighten (Buckman, 2000). Exposure to irritants such as indoor and outdoor air pollution, smoke, dust, pets and many other factors can trigger asthma attacks (Millar, 1998). Various studies have shown significant temporal and geographical increases in the prevalence and incidence of asthma morbidity and mortality (Boezen et al., 1999; Garty et al., 1998; Morgan and Stephen., 1998; Burnett et al., 1994). According to the World Health Organization (WHO) and Statistics Canada over two million Canadians are affected, including 10 to 15 per cent of Canada's children. In Canada, asthma is one of the leading causes of admissions to hospital (Boulet et al., 1999). Each year approximately 500 adults and 20 children die from asthma (Boulet et al., 1999). More than 80 per cent of these deaths could be prevented with proper asthma education and control of their symptoms (Boulet et al., 1999).

In recent years researchers have had difficulty trying to explain the increase in incidence and prevalence of asthma around the world. Some researchers believe that increases in asthma over the last few decades are attributed to a real increase in the severity of the disease while other believe the increases are due to changes in perceptions on the part of the physicians and patients. To some degree, it is accepted that the increase

may be influenced by a lack of awareness on the part of the patient about their illness (Boulet et al., 1999). Other scientists argue that because the rising rates of asthma morbidity and mortality have occurred over such a short period of time (less than a decade) the root of this problem is not due to genetics (Martinez, 2001). They argue that the reasons for the rapid rise in these conditions must be environmental.

Recent epidemiological research has further hinted that the cleanest environments may be the best breeding grounds for allergies and asthma (Martinez, 2001). Several years ago, scientists found that children in large families (particularly the younger siblings) had fewer allergies than children from smaller families (Martinez, 2001). It was believed that exposure to the germs brought home by older siblings protected the younger children from allergies (Martinez, 2001). The hygiene hypothesis has offered one explanation for the increase in asthma morbidity and mortality. Compared to the past, children living in industrialized countries today are exposed to fewer infectious diseases, which are necessary to train their developing immune systems. As a result, their immune systems can overreact to relatively harmless irritants, possibly leading to allergies and asthma.

Whatever cause may explain the increased prevalence and incidence of asthma, that fact remains that various risk factors (triggers) exist for asthma. Of particular interest to this study is the response of asthma hospital admissions to elevated levels of outdoor air pollutants. Reactions to air pollution exposure vary from person to person, depending on the type of pollutant, amount of exposure, and their overall health status. It seems clear that air pollution can cause irritation and inflammation in the lungs (Hester and Harrison, 1998). If asthma is left untreated without proper management, it can lead to a

lower quality of life by making it increasingly difficult to perform simple physical activities, leading to decreased productivity in school or work (including missed days), hospitalization, and even death.

1.3 Motivation of study

The motivation for this study was driven by the fundamental premise that the environment plays a role in human health. Epidemiological and geographical studies have shown that air pollutants can trigger asthma morbidity and mortality. The rising prevalence and incidence of asthma morbidity in Canada is of great concern for all citizens, particularly those afflicted with this disease. Therefore, it is important to understand if hospital admissions for asthma in Montreal are associated with changing levels of air pollutants.

Distinguishing which air pollutants play a role in daily asthma hospital admissions has important public health implications for air pollution and asthma controls (Delfino et al., 1997). The aim of a public health policy should allow the asthma patient to achieve a better quality of life, while providing researchers a better understanding of the disease burdened along with their possible risk factors (Levison, 1991).

1.4 Statement of the Problem

The purpose of this thesis was to (1) estimate the rate of hospital admissions for asthma in Montreal, Quebec (according to different sex and age groups) and to (2) determine whether daily variations in selected air pollutants in Montreal, Quebec, were

associated with daily asthma hospital admissions during the period of April 1st 1992 to March 31st 1999.

Subjects were identified through the provincial hospital discharge database, referred to as Med-Echo. The study population consisted of individuals with a primary diagnosis of asthma living in Montreal and seen at a Montreal Hospital from April 1st 1992 to March 31st 1999. Asthma was coded in the hospitals using the Ninth revision of the International Classification of Diseases (ICD-9). Patients with cancer and cardiovascular diseases were excluded. Statistically, a time-series analysis comparing daily levels of air pollution and daily counts of hospitalization for asthma was used. This investigation thus determined whether increases in air pollution were followed on the same day or future days with increases in the number of hospitalizations for asthma.

1.5 Brief Outline of Thesis

This thesis is presented in five chapters. The second chapter will review English and French epidemiological and geographical literature dealing with the associations between air pollutants and asthma morbidity. The third chapter will describe the material and methodology employed in this study. Chapter four will investigate the results of the time-series analyses while chapter five will discuss the results of this study in light of the findings.

CHAPTER II

LITERATURE REVIEW

The purpose of this review is to present the literature dealing with the associations between ambient air pollution exposure and estimates of prevalence or incidence of asthma. Epidemiologic and geographic studies dealing with asthma published in English and French scientific journals and theses were reviewed. Although associations with risk factors other than outdoor air pollution (e.g., extreme weather and indoor air pollutants) and the prevalence or incidence of asthma have been observed, only studies of outdoor air pollution were included. This chapter will focus on:

i) a brief discussion of the prevalence and incidence of asthma morbidity and how asthma effects the Canadian health care system and afflicted families;

ii) associations between asthma hospital admissions and ambient air pollution.

2.1 Prevalence and incidence of asthma: its burden on the health care system and families

A growing number of studies suggest that the prevalence and incidence of asthma morbidity is increasing (Krahn et al., 1996; Millar and Hill, 1998; Bates and Sizto 1987). In 1986-1987, asthma was the second cause of childhood hospitalization in Canada (next to chronic disease of tonsils and adenoids) (Connors and Millar, 1999). By 1996-1997,

asthma became the leading cause for hospitalization among Canadian children. According to the Canadian Asthma Consensus Guidelines on asthma care, six out of ten Canadians do not have their illness under control. Canadians are needlessly suffering a lower quality of life and are clogging up the health care system due, in part, to poor management. In fact, poor management has led to hospitalization, emergency room visits, and missed days at school or work (Ernst, 2000).

Despite the increased prevalence of childhood asthma, hospitalization rates for children age 1 to 14 fell from 5.7 to 3.6 hospitalization per 1,000 children (1986-1987 and 1996-1997 respectively) (Connors and Millar, 1999), and the average length of stay for asthma hospital admissions decreasing from 3.6 (1986-1987) to 2.3 (1996-1997) days. Asthma symptoms that previously would have warranted admission are now managed in emergency rooms or at home. Therefore, the criteria for hospitalization changed, leaving the beds for the most seriously ill (Connors and Millar, 1999).

The decline in hospitalization rates does not necessarily mean that there are lower health care costs (Connors et al., 1999). Out patient services shift the costs from the hospital and onto the affected individual (e.g. costs related to loss in working days). Incidences of asthma affect the health and wealth of Canadians. A recent study attempted to show the economic stress asthma places on hospital resources as well as "estimating productivity losses caused by illness-related work absence and premature death" (Krahn et al., 1996). The burden asthma places on the health care system, taking into consideration the "direct and indirect costs of asthma in Canada in 1990, was estimated to range between 504 to 648 million dollars" (Krahn et al., 1996).

2.2 Associations between ambient air pollutants and asthma/respiratory morbidity

As stated in the previous chapter, air pollution consists of a complex mixture of substances, including sulfur dioxide (SO_2), ozone (O_3), nitrogen dioxide (NO_2), particulates, and volatile organic compounds. Time series analysis are carried out to examine the following question: *are the increases in asthma morbidity rates on the day (e.g. lag 0) or the next few days (e.g. lag 1, 2 etc) following increases in air pollutant concentrations*. Normally the target population consists of all individuals living in a defined geographic area during a specified time. The estimates used for various studies are the numbers of individuals in the target population who were hospitalized or visited an emergency room or general practitioners office during the study period. Information on subjects is usually obtained from hospital discharge records and air pollution readings are usually acquired from fixed-site monitoring stations. Therefore, it is generally assumed that each subject within the target area is exposed to the same daily average level of air pollution. For some pollutants (PM_{10}, sulfates) this is a reasonable assumption but for others, such as nitrous oxides (NO_X) and sulfur dioxide (SO_2), there may be considerable spatial variation. In most analyses, variables that change over time and may be associated with air pollution, which are referred to as confounding factors, are accounted for in the analyses. For example, weather conditions can confound the associations between air pollutants and asthma morbidity if they are not controlled for in the base model. Many studies have used time-series analysis to assess the associations between elevated levels of air pollutants and increases in asthma morbidity. What positive associations imply is that elevated levels of air pollutants are associated with

increase asthma morbidity. However, what negative associations show is that as elevated levels of air pollutants increase asthma morbidity decreases.

For the past decade, researchers have argued that environmental risk factors such as outdoor air pollutants are to blame for increased asthma morbidity (Millar and Hill, 1998). This section will investigate the literature surrounding the associations of short-term outdoor air pollutants and asthma/respiratory morbidity, and will focus on (1) hospital admissions and (2) emergency room/general practitioner visits. Due to the complex nature of the literature and as a way of avoiding the oversimplification of the results, the data will not be sub-divided according to air pollutant and asthma/respiratory associations. The next two sections will present the literature in a chronological order, based on the year of publication.

2.2.1 Associations between outdoor air pollutants and asthma/respiratory hospital admissions

Data from the Netherlands was used to investigate the effects of short-term air pollution on emergency hospital admissions for respiratory disease for 1977-1989 (Schouten et al., 1996). A time-series regression analysis was implemented controlling for confounding factors (i.e., seasonal variations, weather conditions, and influenza epidemics). Two cities in the Netherlands were under investigation, Amsterdam and Rotterdam. The data was sub-divided into seasons called summer and winter periods. In Amsterdam, exposure to ozone in the summer showed a statistically non-significant positive effect on respiratory admissions among persons ages 65 and older. In Rotterdam, significant positive associations were found with ozone and respiratory admissions. In

Amsterdam a significant negative effect was found with sulfur dioxide and nitrogen dioxide. However, in Rotterdam nitrogen dioxide displayed a non-significant positive effect. Black smoke only showed a small non-significant positive effect in Rotterdam.

Analysis of the relationship between ambient air pollution and non-elderly asthma hospital admissions for the period 1987 to 1994 was performed for Seattle, Washington (Sheppard et al., 1999). A Poisson regression model was used to assess in non-elderly patients associations between daily asthma hospital admissions and elevated levels of particulate matter less than 10 and 2.5 μm in aerodynamic diameter, sulfur dioxide, ozone, and carbon monoxide. Associations were found with particulate matter less than 10 and 2.5 μm in aerodynamic diameter and asthma hospital admissions for a 1-day lag. Also, associations were found with carbon monoxide for single-pollutant models at a 3-day lag and for ozone at a 2-day lag. No associations were found for sulfur dioxide. Multiple pollutant models showed associations between particulate matter less than 10 and 2.5 μm in aerodynamic diameter, carbon monoxide and asthma hospital admissions. The highest increase in risk was estimated during the spring and fall months.

Burnett and others assessed the associations of ozone and sulfates on the frequency of respiratory admissions to Ontario Hospitals 1983 to 1988 (Burnett et al., 1994). Positive associations were found for both pollutants and hospital admissions (lag 0 to 3 days). From May to August, five percent of the admissions were explained by ozone whereas one percent by sulfates. Positive associations were found for ozone-sulfates pollution mix and asthma admissions.

Delfino and others investigated the relationship of urgent hospital admissions for respiratory illnesses to photochemical air pollution levels in Montreal from 1984-1988

(Delfino et al., 1994a). Only the warm periods were investigated for ozone, particulate matter less than 10μm in aerodynamic diameter and sulfate fraction of particulate matter less than 10μm in aerodynamic diameter. They controlled for confounding factors such as seasonal variations, day of the week effects, temperature, and relative humidity. During July and August, there was a statistically significant association for all respiratory admissions. Asthma admissions in May to October were found to increase 2.7% over mean levels for each 12 μg/m3 increase in particulate matter 10μm levels 3 days prior to the admission day.

The associations between respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario, from 1986 to 1988 were assessed (Thurston et al., 1994). Fine aerosol samples (H^+ strong acidity and sulfates) were collected daily at central city sites throughout the study period. The authors only controlled for the effects of temperature, and found that ozone, H^+ and sulfates were positively associated with respiratory and asthma admissions.

Schwartz studied the associations between particulate matter less than 10μm in aerodynamic diameter and ozone with respiratory hospital admissions from 1988 to 1990 (Schwartz, 1996). A time-series analysis was carried out controlling for season, temperature, humidity, and day of the week effect, while controlling for extreme weather. Both particulate matter less than 10μm and ozone were found to be associated with an increased risk of respiratory hospital admissions. Particulate matter less than 10μm associations were insensitive to alternative methods of controls for weather, whereas, ozone was sensitive to various controls for weather.

A large Canadian study was carried out by Burnett summarizing the associations between ozone and hospitalization for respiratory diseases in 16 Canadian cities from April 1, 1981 to December 31, 1991 (Burnett et al., 1997). Burnett was interested in determining if low levels of troposphere ozone contribute to hospitalization for respiratory disease. There were 720,519 admissions for which the primary diagnosis was respiratory disease. This study controlled for various confounding factors including day of the week effect, seasonal and sub-seasonal variations, other air pollutants, and weather. Prior to adjusting for weather and other air pollutants, positive associations were found for a 1-day lag between ozone and respiratory admissions for all 16 cities. However, no associations were found in the winter months. Adjusting for dew point temperature eliminated any associations between ozone and respiratory disease in both Montreal and Vancouver. However, after adjusting for dew point temperature, carbon monoxide and soiling index (coefficient of haze), small associations between ozone and respiratory admissions were found for both Montreal and Vancouver.

Anderson investigated the associations between air pollution, pollen, and daily admissions for asthma in London 1987 to 1992 (Anderson et al., 1998). A Poisson regression model was used to assess the association between ozone, nitrogen dioxide, sulfur dioxide and black smoke and daily emergency admissions for asthma in age group 0-14, 15-64 and the 65 and older. Confounding factors such as pollen, weather, seasonal and sub-seasonal trends were adjusted for. The study period was divided into warm (April to September) and cool (October to March) months. During the warm periods pollen was included in the model only for days when the pollen count was greater then zero. For the all age group over the whole study period, positive associations were found

with nitrogen dioxide, sulfur dioxide and black smoke for various lags. Negative associations with ozone and asthma admissions were found in children (0-14 years) for cool months. While positive associations with ozone and asthma admissions were found for the whole study year and warm months for age 5-64 group, positive associations were also found for nitrogen dioxide and age 0-14 admissions (whole year and warm months) and for age 65 and older (cooler months). In children aged 0-14 years, sulfur dioxide was associated with asthma admissions for the whole year and warm months, while persons aged 65 and over associations were found only in the cool months. Positive associations were found with black smoke and asthma admission for age group 65 and older throughout the whole year. Introducing pollen into the model did not influence the pollutant effects.

Burnett and others investigated the associations in Toronto, Ontario, between ozone and hospitalization for acute respiratory diseases during 1980 to 1994 among children less than 2 years of age (Burnett et al., 2001). A time-series analysis was used to adjust for temporal trends in admissions, weather factors, and exposure to other pollutants. A thirty-five percent increase in the daily respiratory hospital admission was associated with a 5-day moving average of daily 1-hour maximum ozone concentration for May to August. However, no associations were found for hospital admission and ozone for September to April.

Morgan and Corbett investigated the associations of outdoor air pollutants on daily hospital admissions in Sydney, Australia for 1990 to 1994 (Morgan and Corbett, 1998). Utilizing the generalized estimating equation while controlling for weather and seasonal trends, single-pollutant and multiple-pollutant models were assessed. An

increase in the daily maximum 1-hour nitrogen dioxide (lag 0) was associated with a 5.29% increase in asthma hospital admissions for children. A similar increase produced a 3.18% increase in adult asthma admissions. Less significant associations were found with daily levels of nitrogen dioxide. No consistent or significant associations between asthma and ozone or particulate were observed. When the multiple-pollutant models were assessed nitrogen dioxide displayed a slightly larger positive association with childhood asthma admissions.

A study between outdoor air pollutants and hospitalization for respiratory disease in Drammen, Norway was conducted for 1994-1997 (Hagen et al., 2000). A generalized additive model was used with a partial spline while controlling for trends in weather, day of the week effect, seasonal and sub-seasonal variation. Respiratory admission showed the strongest associations with volatile organic compounds (benzene, toluene, and formaldehyde). After studying two and three pollutant models, nitrogen dioxide, sulfur dioxide, and ozone were strongly associated to respiratory hospital admissions then particulate matter less than 10μm.

2.2.2 Association between asthma/respiratory emergency room/general practitioners visits and outdoor air pollutants

Chew and others studied the relationship between ambient air pollution levels (sulfur dioxide, nitrogen dioxide, and total suspended particles, and ozone) on daily asthma admissions and hospital or emergency room visits among children in Singapore for a 5-year period from January 1990 to December 1994 (Chew et al., 1999). A generalized linear model was used while controlling for confounding factors (e.g.

meteorological conditions and day of the week effect). Lags 1 and 2-days were assessed with single air pollutant models. Positive associations between each of these pollutants and daily emergency room visits for asthma was observed in children aged 3-12 years, but not in adolescents and young adults (13-21 years old).

Garty and others assessed the associations between air pollution, weather and airborne allergens with incidences of emergency room visits for children with acute asthma attacks in 1993 (Garty et al., 1998). Nitrogen oxides, sulfur dioxide, ozone, particulate, airborne pollen and spores were assessed. Positive associations were discovered between emergency room visits and concentrations of nitrogen oxides, sulfur dioxide and high barometric pressure, while negative associations were found with ozone and temperature (minimum and maximum). There were no significant correlations between particulate concentrations, humidity, or airborne pollen and spores.

Hajat investigated daily general practitioner (GP) consultations for asthma and other lower respiratory conditions and air pollution in London for 1992 to 1994 (Hajat et al., 1999). They used a time-series analysis controlling for time trends, seasonal fluctuations, day of the week effect, influenza, weather, and pollen levels. Weak positive associations were found across various lags between asthma consultations and nitrogen dioxide, carbon monoxide in children. For adults, positive associations were found for particulate matter of less than 10 μm as well as for adults with lower respiratory admissions and sulfur dioxide. Negative associations were found with ozone and children for both groupings (asthma and respiratory disease). Both of these associations varied greatly by season, particularly for the children. Asthma consultations were greatest in the warm month for children due to nitrogen dioxide, carbon monoxide and sulfur dioxide,

whereas in adults the only positive association found was for particulate matter 10μm and general practitioner consultations.

2.3 Summary of Literature

The literature outlines the possible associations between elevated levels of air pollution and asthma/respiratory morbidity and the role air pollutants play as a risk factor (trigger) for asthma/respiratory hospital admissions emerged (Rainham, 2000). Most of the literature reported air pollutant levels lower then generally expected for urban cities and yet at times showed association between air pollutants and asthma/respiratory morbidity. In summary, the literature review was a brief overview of the published studies covering the temporal and geographical representation of air pollution and asthma/respiratory morbidity. A wide variety of statistical analyses, confounding factors, lags and ways of stratifying data sets were used in these studies.

The following chapters will examine the associations between selected air pollutants and asthma hospital admissions for Montreal, Quebec. The statistical methods described in the studies of this chapter will be useful in defining the relationship (in terms of the implementation of confounding factors and statistical models) between air pollution and asthma morbidity for this study.

CHAPTER III

MATERIALS AND METHODS

Three data sources were used for this analysis: information regarding hospital discharges were abstracted from the Provincial hospital discharge database (Med-Echo); the weather monitoring station at Dorval International Airport in Montreal was used to collect meteorological data; and data from fixed-site monitoring stations were used to calculate the daily average of air pollutants. Each of these data sources will be described further in this chapter, along with a description of the methodology used to determine the study population (asthma patients), weather data and air pollution data, as well a description of the statistical analysis.

3.1 Hospital Discharge Data (Med-Echo)

Med-Echo is a provincially run administrative hospital database that provides hospital discharge records for all patients in the Province of Quebec (Bourdages, 1987; Delfino et al., 1993). This service keeps track of approximately one million discharge records per year for the Province. Upon the discharge or death of a patient, the medical archivists in each hospital are responsible for completing a summary discharge sheet, which lists the health conditions that contributed to the patients' length of stay. The archivists are trained to create this summary form, and it is based on the notes that make up the medical file. The primary diagnosis, coded according to the Ninth Revision of the International Classification of Diseases (ICD9), refers to the health condition that was the main reason for the stay in hospital; a maximum of 15 other conditions that also contributed to the length of stay can be coded. For example, an individual may be

admitted into hospital with a primary diagnosis of asthma and secondary diagnoses of cancer and cardiovascular diseases (according to ICD9 codes).

The form is processed at the date of discharge (or death), and the date of admission is recorded along with other demographic and personal information, including date of birth, age, sex, home address as defined by the Forward Sorting Area (FSA) of the postal code (Delfino et al., 1993). The date of discharge and admission of each patient in our study period was from April 1st 1992 to March 1st 1999.

3.1.1 Data Quality of Med-Echo

Hospital discharge records provide researchers with explicit information concerning the reasons and length of stay for each admission (Table 1). As stated above, upon discharge a file is generated that is sent to the central collection and processing system (Bourdages, 1987). The information that is recorded on the summary sheet is kept with the patient's chart, as completed by the treating physicians. Each acute care hospital is required to report to the system, and is responsible for ensuring the accuracy and submission of its data. Currently, nursing homes and other similar institutions are not required to report. The summary sheet is then analyzed by archivists in each hospital to code (ICD9) the primary (main reason for length of stay) and secondary ($\leq$15 secondary conditions) diagnoses that lead to the individuals length of stay in hospital. The validity of discharge summaries (specifically for respiratory diseases) have been investigated by many researchers (Delfino et al., 1993; Bourdages, 1987; Mayo et al., 1993; Levy et al., 1994) and has been "judged to be sufficiently reliable for use in research relating hospital admission levels to ambient concentrations of air pollution" (Delfino et al., 1993).

Table 1. Hospital Discharge File (Med-Echo)

	Length
1. Encrypted QHIP number	12
2. Type of hospital	2
3. Code of hospital	8
4. Date of accident	6
5. Accident code	5
6. Date of admission	6
7. Diagnosis at admission (ICD9)	5
8. Physician code	6
9. Principal diagnosis	6
10. Secondary diagnoses 1- 15	7
11. Treatments 1- 9	5
12. Encrypted codes of treating physicians 1- 9	6
13. Type of death	1
14. Death ±48 hours	1
15. Date of discharge	6
16. Total amount of time in-hospital	4
17. Discharge destination (type of institution, death, home, etc...)	2
18. Code of discharge destination (type of institution, death, home, etc...)	8
19. Marital status	1
20. Referring institution code	8
21. Referring institution code type	2

As there are numerous weaknesses and strengths of Med-Echo as a primary source for patient information, not all the data will be explored in this work. Some of the limitations of this database are that individual risk factors are difficult to discern when only grouped data is used (Goldberg, 1996). Med-Echo data provides the researcher with only a glimpse of acute health effects (e.g. does not include medical history). It does not consider short-term variations of individual attributes or hospital environments. Some strengths of Med-Echo are that it contains accurate data for location of the hospital, residence of the subject, date of discharge and admission, primary diagnosis, up to 15 secondary diagnostic codes, and up to 9 principal medical procedures performed.

3.1.2 Study Population for this Research

Acute hospital admissions are defined as short-term care for individuals suffering from acute conditions, including serious illnesses or injuries. On the other hand, chronic care is identified as continuous, long-term care for individuals suffering from chronic conditions. Only subjects who were admitted into an acute care hospital were included in this analysis, as the objective was to identify acute health events that may have resulted from changes in air pollution. All hospitals in Montreal were identified by their regional hospital code (otherwise know as Régions Socio-Sanitaires (RSS)) and then all Med-Echo records with an RSS of 06 (Montreal Hospitals) were extracted from the Med-Echo database. Place of residence was then identified using the Forward Sorting Area (FSA; 1st three digits of the postal code) on the record that corresponded to the FSAs in Montreal. In order to identify the location of the study population, the FSA of the patient's residence at time of admission was used. The entire postal code is it not made

available so as to protect the patients' privacy and confidentiality. However, the first three characters of the FSA (postal code) are sufficient to pinpoint a geographic location (e.g. Montreal). The FSA refers to broad geographic areas; for example, eastern Notre Dame de Grace, a part of the City of Montreal, is coded as Hxx. Unfortunately, FSAs were missing for 1990 and 1991 and part of 1992. Therefore, the study period for this research is from April 1st,1992 to March 31st, 1999.

Length of stay is the amount of time, usually measured in days, spent by a patient in hospital. Initially, the data set was generated with a length of stay of 6 months or less. The percentage of patients admitted for this length of time was 98% of the total data set. When a length of stay of 30 days or less was considered, 95% of the data set remained. Therefore, a length of stay of 30 days or less was deemed a sufficient cut off point for hospitalization. Any patient remaining in hospital longer than 30 days was excluded from the analyses. Med-Echo data were only available until March 31st, 1999. Since the data set considers a length of stay of 30 days or less, the data available for a patient that was admitted from March 2nd to March 31st could be incomplete. In order to eliminate possible inconsistencies due to length of stay, March 1st 1999 was set as the cutoff date for admission.

As stated above, on each discharge form there can be as many as 16 diagnoses recorded using the Ninth Revision of the International Classification of Diseases (ICD-9th) codes, one primary and up to fifteen secondary diagnoses. Each of these diagnoses represents a medical reason for stay in hospital. For example, some diagnoses are asthma, injuries or poisoning, pneumonia, respiratory disease and chronic obstructive pulmonary disease. The task at hand was to categorize all individuals according to primary diagnosis

using the ICD-9 codes. All subjects who were admitted into hospital with an ICD-9 code for asthma (493.0, 493.1 and 493.9) listed as a primary diagnosis were extracted from the database. The last step was to ensure that any patient identified as having asthma as a primary diagnosis did not have cancer or cardiovascular diseases as a secondary diagnosis. Cancer and cardiovascular disease have been linked to air pollution (Goldberg et al., 2000). Therefore, in an attempt to generate as pure a data set as possible, for this study, we stipulated that any patient with a primary diagnosis of asthma must be free of cancer and cardiovascular diseases. Thus, if any individuals' Med-Echo form listed either cancer or cardiovascular disease as one of their first four secondary diagnoses, their records were removed from the data set.

Source of origin is a code that identifies where the individual was prior to their admission. This does not indicate a specific address (as with the RSS); rather this refers to the type of physical location (i.e. home, hospital, and daycare). Only the first admission to a hospital was considered for subjects who were readmitted to more than one hospital.

3.2 Weather Monitoring Station

Weather variables were obtained from observations at the suburban Dorval International Airport (45 28'N, 73 45'W) on the Island of Montreal. Weather variables of interest are, daily (maximum, minimum, mean) temperature, daily (maximum, minimum, mean) temperature change, daily mean relative humidity, and change in pressure over the 24 hours ending at 08:00.

The weather data sets were complete, with very few missing values. The only data that was missing was change in pressure for 24 hours ending at 08:00; a total of seven days of readings were missing (01/01/94, 02/12/94, 03/12/94, 20/11/98, 08/01/99, and 09/01/99). The missing days were filled in by calculating the average using 2 days before and after each missing day.

3.3 Air Pollution Monitoring Stations

In 1970, the Provincial of Quebec established the Montreal Urban Community (MUC). It is a regional government that covers the island of Montreal and includes 28 municipalities. Its activities are those of a municipal government but with delegated responsibilities from the provincial government for monitoring the quality of air and water. The MUC ended in 2002 with the merging of the municipalities of Montreal Island into a single entity. Starting January 1st, 2002, some services were transferred to the new City of Montreal and some others to the Montreal Metropolitan Community (MMC) that covers the entire Island of Montreal.

Figure 1 is a map displaying the location of selected air pollution monitoring stations for carbon monoxide (CO), coefficient of haze (COH), hydrogen sulfide (H_2S), nitrogen dioxide (NO_2), ozone (O_3), and sulfur dioxide (SO_2) and some particulate matter stations (PM_{10}). Information regarding these stations was gathered from the MUC prior to the amalgamation of the new Island of Montreal. Station names can be found in Appendix A.1.

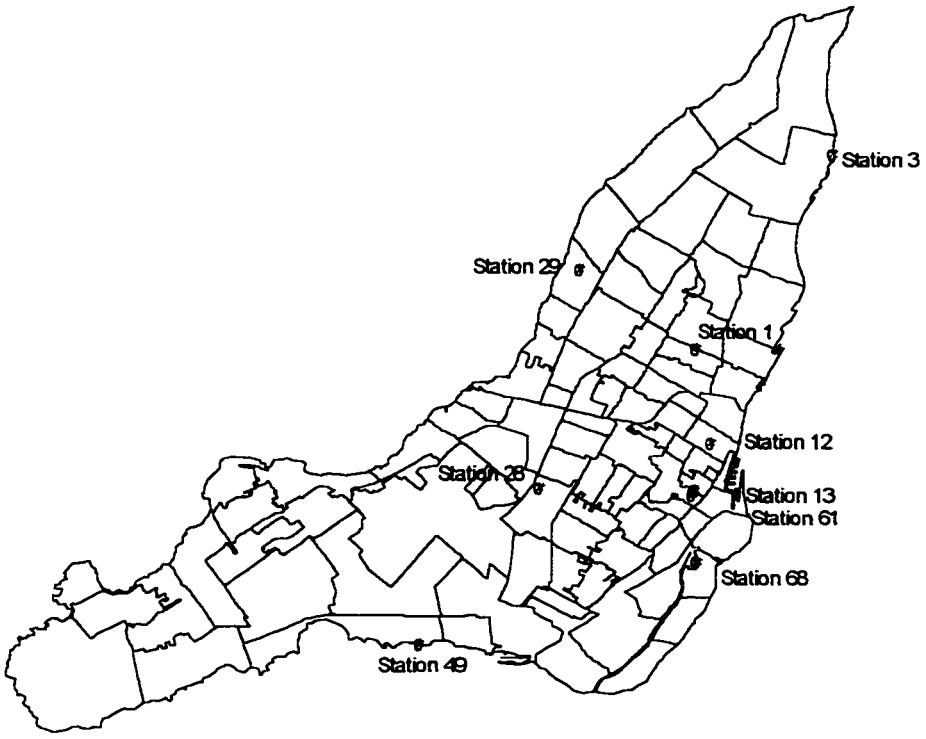

Figure 1. Selected Monitoring Stations for Air Pollutants, Montreal, 1992 to 1999.

The pollutants were measured and analyzed using various techniques. Carbon monoxide (CO) was measured every two hours using infrared absorption (e.g., the measuring device was the Thermo Electron 48). Coefficient of haze (COH), which is a measure of elemental carbon in the ambient air (Goldberg et al., 2000), was measured using a continuous white filter paper. Every two hours, the filter paper moved forward creating iridescent spots whose opacity was assessed with a photometer and expressed in COH units per $0.8m^3$ of air (Gagnon, 1998; and Delfino, 1993). H_2S was read using

Monitor Lab 8780 (Gagnon, 1998). A Thermo Electron 14B measured NO_2 using chemiluminescence. (Goldberg et al., 2000; Gagnon, 1998; Delfino et al., 1993). Chemiluminescence was also used to analyze O_3 using a Bendix 8002 sampler (Gagnon, 1998; and Delfino et al., 1993). The technique used to measure SO_2 had each air sample pass through a bromide solution. This induced a chemical reaction resulting in a change in electrical conductivity of the electrolyte in the same amount as the concentration of SO_2. This analysis employed a Philips 9700 and a Monitor Lab 8850 (Goldberg et al., 2000; Gagnon, 1998; Delfino et al., 1993). PM_{10} was analyzed using the Sierre-Anderson dicotomous sampler. This sampler only operated every sixth day and ran at a flow rate of 16.7 L/minute (Gagnon, 1998; Delfino et al., 1993). Gaseous pollutants were usually assessed hourly. However, non-continuous data on PM_{10} was collected every 6 days for a 24-hour period (midnight to midnight), so the result is an average for that particular day.

Some of the air pollution monitoring stations experienced missing days or hours of readings. Any missing data was left as missing and therefore no imputations were carried out. In addition, zero values were left unchanged since a zero reading indicated that the reading was under the level of detection. Daily averages were taken from every monitoring station for each pollutant (Appendix B.1. to B.2.). Since station data for PM_{10} is provide every six days, daily averages were calculated over the stations only every sixth day (all other air pollutants were measured daily). Then the daily averages for each monitoring station were averaged to attain an overall daily mean, which was the variable used in the analysis.

3.4 Statistical Methodology

The main objective of this thesis was to determine if the number of daily admissions to hospital for asthma increased with increasing levels of ambient air pollutants. This question implies that the interest is in the short-term (day-to-day) effects of air pollution, thereby neglecting the long-term effects. There are many types of study designs that can answer that question. The fundamental design is the longitudinal (or panel) study. The longitudinal (panel) study follows a group of individuals over time and daily health events (asthma hospital admissions) and predictors of these health events (e.g., air pollution) are recorded daily. In this type of study, one can correlate changes in asthma hospital admissions with the air pollutants, but taking into account that an individual may have more than one such event. Longitudinal (panel) studies are extremely expensive and difficult to carry out. Therefore, economists, epidemiologists, and statisticians invented the time series analysis, which is much simpler to carry out and can provide valid information regarding short-term (day-to-day) effects. This type of study is designed so that instead of following a specific cohort of individuals through time, events (e.g., hospitalizations) are examined from an implicit cohort (the general population) and then the number of events per day are correlated with changes in air pollution. In this design, the dependent variable is the number of counts per day and this is distributed approximately as a Poisson variable. (All count data has this feature, as they are generated from a large number of binomial events (namely, being hospitalized or not) that have a low probability of occurrence.) Independent variables are used to predict the counts, and in this study they are the weather and air pollution variables. The

analyses of such data can be done using simple correlation coefficients, but these do not provide estimates of effect (i.e., the number of excess hospitalization per unit increase in air pollution). In addition, a simple correlation analysis will not allow adjustments for potential confounding factors, such as changes in weather conditions that may also influence the rate of hospitalization. Thus, a regression analysis is needed. Simple linear regression, assuming a Gaussian error structure, cannot be used because the dependent variable is distributed as a Poisson variable. Therefore, it is best to use Poisson regression models that are designed to handle count data.

The simplest model has the form:

$$E(\log(Y_i))=\alpha+\beta_1*X1_i+\beta_2X2_i+.... \quad (1)$$

where Y_i is the number of hospitalizations for asthma on day i, X1, X2 are the covariates on day i (i.e., air pollution, weather), α is a coefficient to be estimated from the model and represents the mean number of hospitalizations per day in the absence of the covariates, and the β_j represent the increase in the number of hospitalizations per day per unit change in the covariates.

The Poisson distribution assumes that the mean and variance are equal, but this is not always the case in practice. If the variance is greater than the mean (as with this study), then this implies that the data are over-dispersed. This indicates that there is greater variation than what is expected under the Poisson model. In theory, one can also have less variation than expected, although this very rarely happens. To correct for non-

Poisson variation, a regression technique called "quasi-likelihood" was implemented. This methodology allows for non-Poisson variation and provides an estimate of the degree to which the data does not follow the Poisson distribution (known as the dispersion parameter). All variances are automatically adjusted for this dispersion by multiplying the usual variance by the dispersion parameter. Thus, over-dispersed Poisson models lead to larger variances.

While analyzing these types of data, it has become evident that other structures in the data needed to be considered (Hastie and Tibshirani, 1990). The first is the seasonal nature of the hospitalization data (seasonality in weather and air pollution is a different issue and is related to confounding).

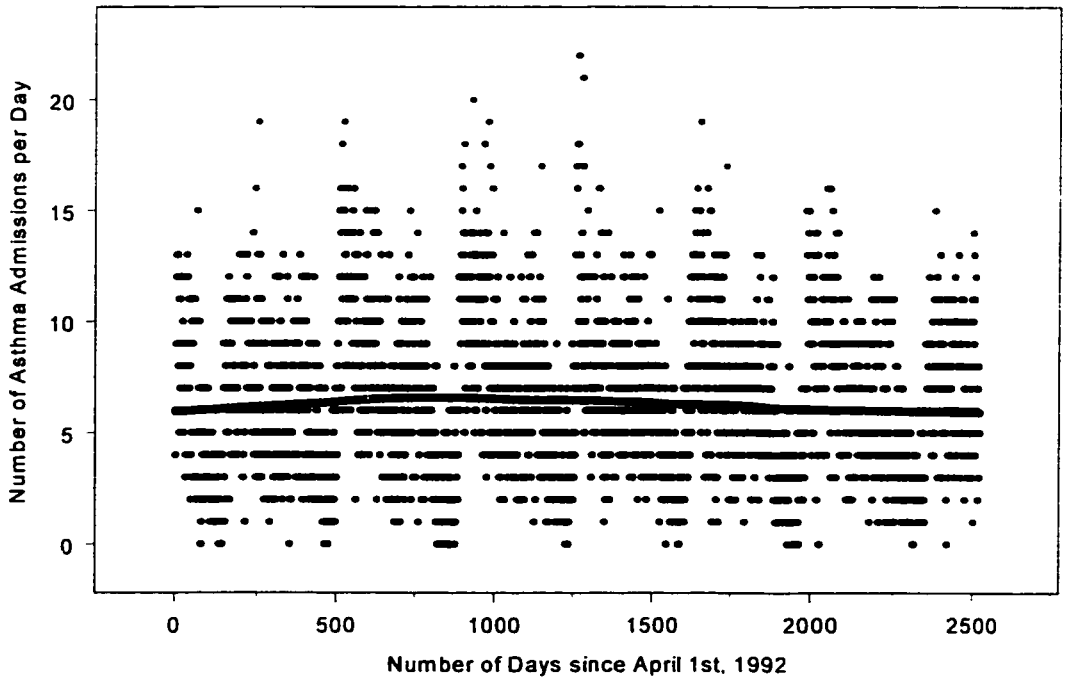

Figure 2. A Scatter-plot of the Daily Asthma Hospital Admissions Span 50%, Montreal, 1992 to 1999

Figure 2. shows the time series for asthma admissions, as the number of asthma hospitalizations increase in the winter and decreases in the summer (Appendix C. 1. Scatter-plot of age 0-9). This type of seasonal affect needs to be accounted for clearly in the statistical model because it induces autocorrelation. What this means is that knowing the number of counts on one day can be used to predict the number of counts on a future day. Such autocorrelation undermines a fundamental assumption of regression, namely that each unit of observation (in this case, the day) is statistically independent of each other. Therefore, additional methods are needed to take this into account. The method that has been used frequently has been to "filter out" the seasonal and sub-seasonal fluctuations using nonparametric functions (Hastie and Tibshirani, 1990).

This figure also shows how these non-parametric functions can be used to fit the data. The generalized additive model (GAM) is the regression analysis that is currently used by most air pollution researchers to handle autocorrelation (GAM; Hastie and Tibshirani, 1990). These statistical regression models allow non-parametric smooth functions to be entered into the regression model in the format of locally weighted regression smoothers (LOESS). When used with temporal variables (i.e., day), the filtering meets the two objectives: (1) it removes the autocorrelation in the data, thereby meeting the independence assumption of regression, and (2) it removes unmeasured confounding factors that influences seasonal patterns in the hospitalization data. Other temporal factors also influence the pattern of hospitalizations, namely day of the week (DOW) and secular trends (i.e., calendar year). The former occurs because hospitals

reduce admissions on the weekend and there are longer trends in hospitalization that may not be picked up by the temporal filter.

The regression model that was considered for this study has the form:

$$E(\log(Y_i)) = \alpha + \text{LOESS}(i,\text{span}=x) + \beta_1 * \text{YEAR}_i + \beta_2 * \text{DOW} + \beta_3 * \text{WEATHER} + \beta_4 * \text{POLLUTANT} \quad (2)$$

Where LOESS(i,span=x) is the temporal filter, YEAR represents calendar year (either as a continuous or categorical variable), DOW represents day of the week effect, WEATHER is some variable or variables representing weather conditions on each day, and POLLUTANT is the value of a pollutant on each day. Hence, in this model it is assumed that weather and air pollution affect hospitalization counts in a linear fashion (on a natural logarithmic scale) whereas seasonal and sub-seasonal variations can have an arbitrary functional form.

There is an additional parameter in the LOESS term, referred to as the span. It is fixed and not estimated by the regression model. LOESS allows us to investigate "local estimates of specific group of data points" within a window/span (Rainham, 2000). Almost all smoothers are local, using adjacent data points (neighourhoods) to evaluate the "predicted" values for each data point. The resulting function that is produced has less variability than the input variable. The larger the window/span, the smoother the curve (Rainham, 2000; Goldberg et al., 2000). There is a delicate balance between over and under filtering (span) which requires us to look at various functions when choosing the best span (Goldberg et al., 2000).

Two main advantages for using LOESS are that end points are managed nicely and it allows researchers the opportunity to adjust the smoothness of the curve (Goldberg et al., 2000). For each asthma admission a temporal filter to rid the data set of seasonal fluctuations was determined. Spans were required to produce the least amount of residual serial autocorrelation and create a filtered time series consistent with white noise process (using Bartlett's Statistic) (Goldberg et al., 2000).

In comparison with other statistical models, GAM provided the most flexibility for this data set. For example, an ordinary linear regression forces the researcher to look only at the family of linear models when determining the best fit for the data. Although data can be transformed into quadratic or log functions ultimately the best fit for this new data set is still from the family of linear models. However, GAM affords us the opportunity to investigate any form of function, known as non-parametric models. Non-parametric models do not force any kind of relationship to the program. It simply finds the best relationship in an infinite family of relationships. In this case, the relationship cannot be expressed with a finite number of coefficients (ßs). The only thing that is forced on the program is the span.

The Akaike's Information Criterion (AIC) is computed from the following formula:

$$AIC = D + 2*p*dispersion,$$

where D is the Residual deviance, p is the number of degrees of freedom (d.f.) used in the model (Null d.f. - residual d.f. + 1) and dispersion is the dispersion parameter. The AIC

deals with the complexities of the model. An advantage of the AIC is that you can compare non-nested models. To use the usual likelihood ratio tests you needed to have nested models (e.g. model with A1 only compared to model with A1 and A2). With the AIC, you can compare models that are not nested (e.g. model with A1 and A2 compared to model with A3 and A4). After accounting for the degrees of freedom, the equation with the lowest AIC values explains the most residual variation (Goldberg, et al., 2000).

As stated before, the daily averaged values for each pollutant across the air pollution stations was calculated. Associations were examined for levels of pollution that occurred on the concurrent day (referred to as lag 0 days), on the previous day (lag 1 day), and averaged over the concurrent and previous two days (3-day mean). Only single-model analysis were performed (e.g. inputting one pollutant at a time) for various lags and by various seasons. Pollutants were regressed against asthma hospital admissions for the whole year and by season according to warm and cool periods. The data was divided by half years into groupings called "warm" (April to September) and "cold" (October to March). Of particular interest were the months from June to August, where hospital admissions rates fell when compared to previous and preceding months (Figure 3). Therefore groupings titled "short" summer (June to August) and "rest" of the year (September to May) will be investigated. The data set was stratified according to 7 classes: all asthma, age 0 to 9, age 0 to 19, age 20 to 64, and age 65 and older. However, for the purpose of this analysis, only all asthma and age 0 to 9 was analyzed.

From this time-series analysis, the percent change in the mean number of daily asthma admissions for an increase in the daily averaged air pollutants equal to its interquartile range was calculated (Goldberg et al., 2000). This is otherwise known as the

mean percent change (MPC) (Goldberg et al., 2000). For each mean percent change (MPC) the upper and lower 95% confidence intervals were also found (Goldberg et al., 2000).

CHAPTER IV

RESULTS OF STATISTICAL ANALYSIS

4.1 The Study Population

In Montreal from April 1st, 1992 to March 31st, 1999 there were 16,472 daily admissions for asthma. This total excludes patients who were not residents of Montreal or Montrealers who were admitted to a hospital outside of the city. In addition, it excluded patients who had cancer and/or cardiovascular disease listed as one of the conditions that contributed to their length of stay (up to and including the 4th secondary diagnosis). As well, those individuals who were transferred between hospitals were included only once (only for the 1st admission).

Table 2 shows the distribution of all asthma admissions by year, gender, and age. It is important to note that 1992 and 1999 are not complete years (1992 data begins April 1st and 1999 data ends March 31st). Table 2 clearly shows that the majority of the admissions for asthma are in children under 9 years old.

Table 2. Distribution of Selected Variables of Asthma for 16,472 Hospitalization Subjects, Montreal, 1992 to 1999

Asthma Classes	Total Number of Asthma Admissions	Percentage
All Asthma Year		
1992	1,743	10.58
1993	2,557	15.52
1994	2,470	15.00
1995	2,429	14.75
1996	2,482	15.07
1997	2,319	14.08
1998	2,070	12.57
1999	402	2.44
Sex		
All Men	8,306	50.42
All Women	8,166	49.58
Age (years)		
0-9	8,840	53.67
0-19	10,731	65.15
20-64	4,115	24.98
≥65	1,626	9.87

4.1.1 Patterns in Hospital Admissions for Asthma

Figure 3, shows the seasonal fluctuations of the distribution of admissions for asthma. Age group 0-19 (or more specifically age 0-9) is the driving force for all of the asthma hospital admissions. The admissions for asthma increase dramatically in September, which corresponds with the beginning of the school year where more time is spent indoors with heating, dust, mould, and infections (Millar and Hill, 1998).

Table 3 displays the distribution of the number of admissions by sex and age. On average, there were 6.5 asthma hospital admissions per day, of which 3.5 of these hospital admissions for asthma were children under nine years of age.

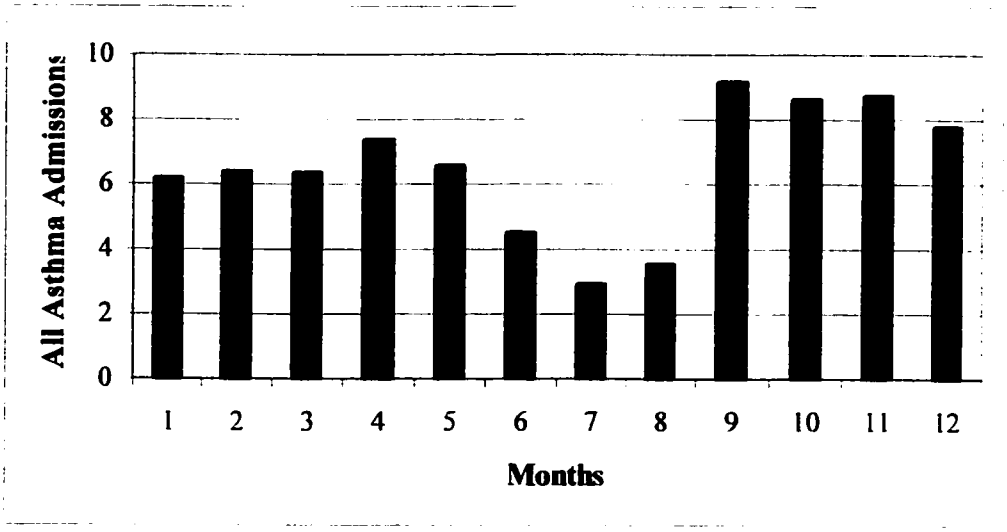

Figure 3. Daily Average of Counts of Hospital Admissions per Month, All Asthma, Montreal, 1992 to 1999

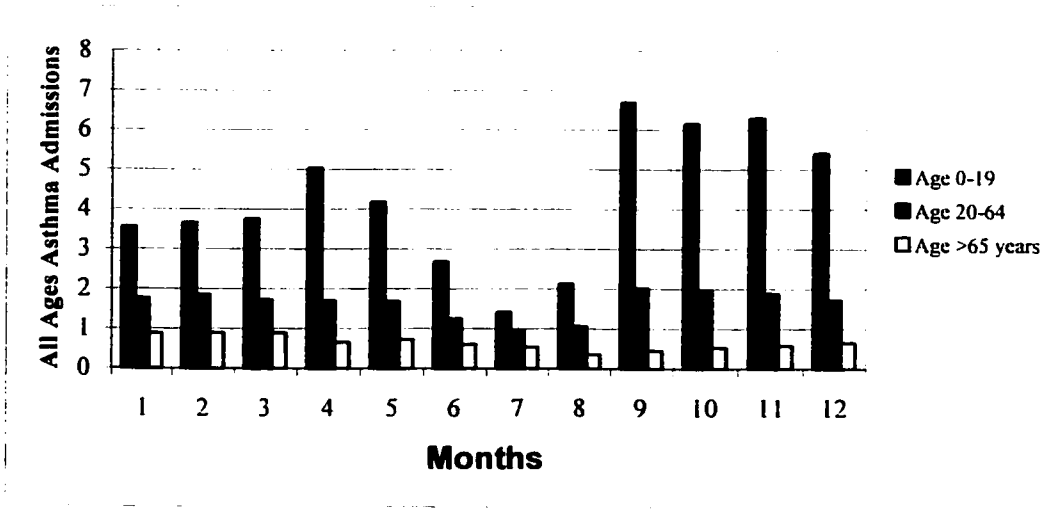

Figure 4. Daily Average of Counts of Hospital Admissions per Month, All Ages, Montreal, 1992 to 1999

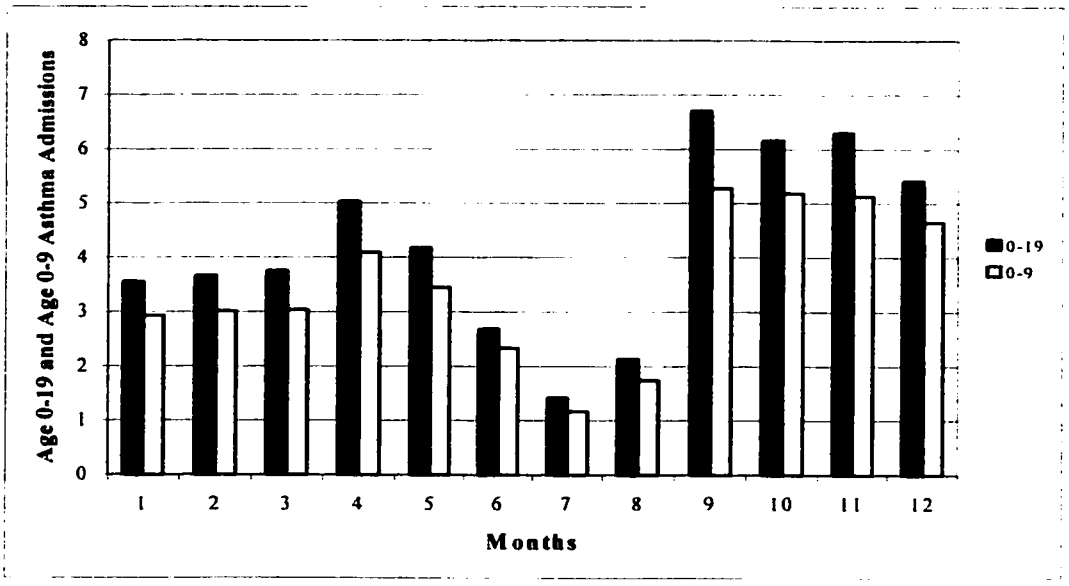

Figure 5. Daily Average of Counts of Hospital Admissions per Month, Age 0-19 and Age 0-9 Asthma, Montreal, 1992 to 1999

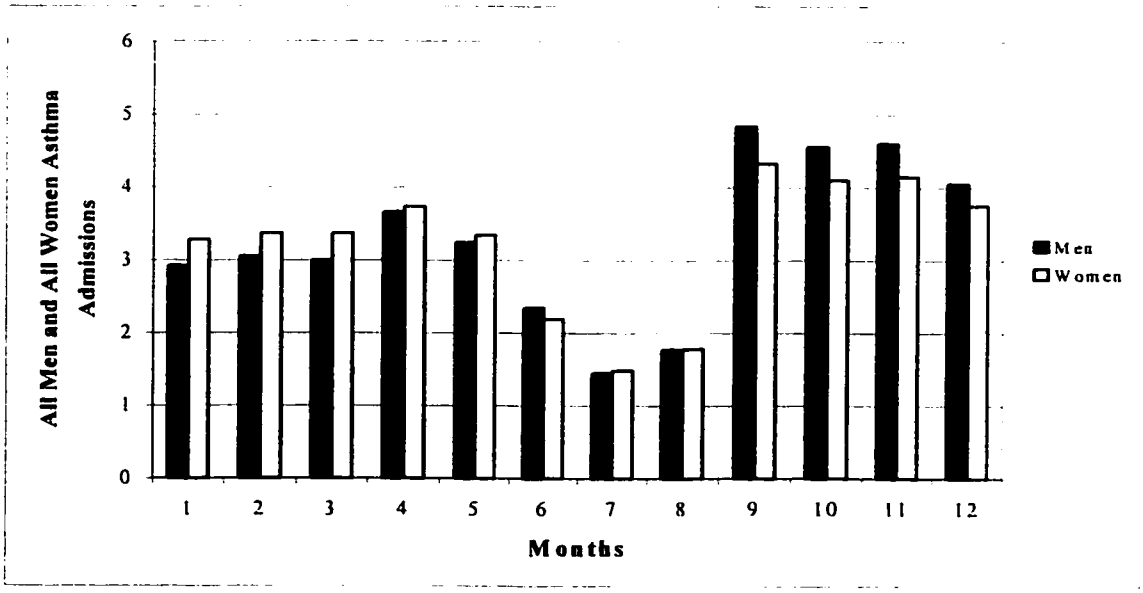

Figure 6. Daily Average of Counts of Hospital Admissions per Month, All Men and All Women Asthma, Montreal, 1992 to 1999

Table 3. Descriptive Statistics of Daily Admissions for Asthma, by Age and Gender, Montreal, 1992-1999

	Total number of admissions	Number of days in which their were no admissions	Mean	Minimum	Percentile				Interquartile Range
					25th	50th	75th	100th	
All Asthma	16,472	33	6.5	0	4	6	9	22	5
All Women	8,166	196	3.2	0	2	3	5	14	3
All Men	8,306	189	3.3	0	2	3	5	14	3
0-9	8,840	224	3.5	0	2	3	5	15	3
0-19	10,731	148	4.2	0	2	4	6	20	4
20-64	4,115	584	1.6	0	1	1	2	9	1
≥ 65 years	1,626	1,363	0.6	0	0	0	1	5	1

4.1.2 Distribution of Length of Stay

Figure 7 shows the length of stay for all asthma admissions in Montreal from April 1st 1992 to March 31st 1999. As the length of stay in days increases for all asthma hospitalizations the number of admissions decrease.

Table 4 shows the median and cumulative percentage for asthma hospital admissions. As expected, length of stay increases with age. We were interested in determining how many asthma patients stayed in hospital for 5 days or less. Approximately 96% of the children age 0-9 stay in hospital for asthma 5 days and less, whereas 53.69% of the elderly population had a length of stay longer than 5 days.

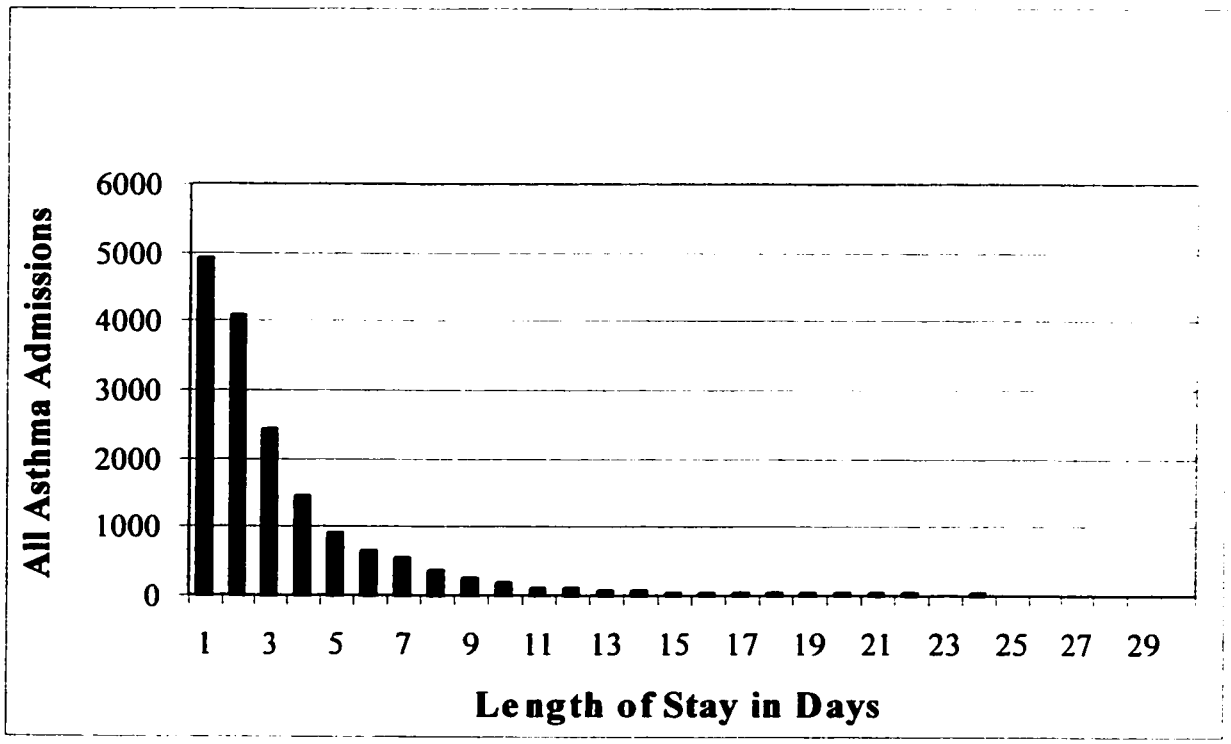

Figure 7. Length of Stay for All Asthma from April 1st 1992 to March 1st 1999, Montreal, Quebec

Table 4. Descriptive Statistics for the Length of Stay of Asthma Hospital Admissions, Montreal, 1992 to 1999

	All Asthma	All Men	All Women	Age 0-19	Age 0-9	Age 20-64	Age ≥ 65
Median	2	2	3	2	2	3	6
Cumulative Percent of 5 days	83.79	89.69	77.79	95.16	96.01	68.94	46.31

4.2 Variations in Daily Weather Patterns

Figure 8 is a time series of the daily mean temperature measured from April 1st, 1992 to March 31st, 1999 (Appendix C. 2. to C.11. scatter-plots of weather variables). Weather variables are extremely complex with considerable temporal variation, much of which is attributed to seasonal and sub-seasonal trends. As stated in the previous chapter, seasonality needs to be accounted for clearly in the statistical model because it induces autocorrelation, although temporal trends were also important. Table 5 shows the distribution of various weather variables under investigation.

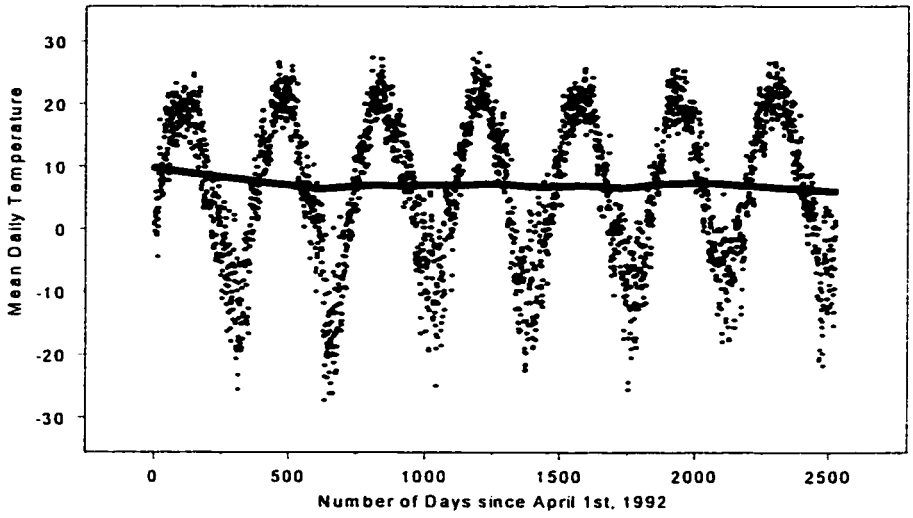

Figure 8. Scatter-plot of Daily Mean Temperature (Span 50%), Montreal, 1992-1999

Table 5. Distribution of Selected Weather Variables, Montreal, 1992 to 1999

	Number of Days Measured	Mean	Minimum	Percentile				Interquartile Range
				25th	50th	75th	100th	
Mean temperature	2526	6.6	-27.3	-2.1	7.6	17.1	28.2	19.2
Maximum temperature	2526	11.4	-23.9	1.9	12.5	22.3	33.9	20.4
Minimum temperature	2526	1.8	-31.8	-6.1	2.6	11.7	24.0	17.8
Mean temperature change	2526	0.0	-15.1	-2.2	0.2	2.2	20.0	4.4
Maximum temperature change	2526	0.0	-23.7	-2.4	0.4	2.7	17.6	5.1
Minimum temperature change	2526	0.0	-18.4	-2.3	-0.3	2.4	23.8	4.7
Mean dew point temperature	2526	1.6	-34.6	-6.3	2.1	11.4	23.0	17.7
Mean dew point temperature change	2526	0.0	-25.5	-2.5	0.4	2.8	28.6	5.3
Mean relative humidity	2526	70.4	31.0	62.0	70.0	79.0	100.0	17.0
Change in pressure ending at 08:00	2097	0.0	-4.2	0.5	0.0	0.5	4.4	0.0

4.3 Fluctuations in Levels of Ambient Air Pollution

As with weather, the time series in Appendix C for air pollutants show seasonal, subseasonal and temporal cycles. Similar to other studies, the air pollutants CO, COH, NO_2, SO_2, PM_{10} had peaks in the winter whereas O_3 peaked in the summer (Appendix C. 12. to C. 16. scatter-plots of air pollutants; Goldberg et al., 2000).

Table 6 shows the level of outdoor air pollutants and the mean values were obtained for each station (Appendix B. 1 to B. 2). Throughout the 7-year study period, ozone ranged from 14.96 to 43.15$\mu g/m^3$. The highest emissions for ozone were found at monitoring stations near industrialized areas and in the heart of the city.

Table 6. Distribution of Daily Concentrations of Regularly Measured Air Pollutants, Averaged Across the Various Monitoring Stations in Montreal, 1992 to 1999

	Units	Number of Monitoring Stations	Number of Days Measured	Mean	Minimum	25th	50th	75th	100th	Interquartile Range
Carbon monoxide	0.1mg/m^3	5	2526	7.0	1.1	4.8	6.4	8.3	34.9	3.5
Coefficient of haze	0.1COH/1000 linear feet	5	2101	2.1	0.0	1.1	1.9	2.9	9.4	1.7
Hydrogen sulfide	0.1ug/m^3	1	2386	9.3	0.0	2.2	6.5	13.1	126.9	10.9
Nitrogen dioxide	ug/m^3	7	2526	39.3	10.3	29.2	37.2	46.8	165.7	17.5
Ozone	ug/m^3	8	2526	29.8	1.2	17.9	27.6	39.3	124.0	21.4
Sulfur dioxide	ug/m^3	5	2526	14.2	0.9	8.1	12.4	17.9	72.7	9.8
Particulate matter less than 10μm	ug/m^3	10	1141	27.8	4.6	16.9	25	34.4	149.4	17.5

4.4 Background Model: Temporal Filters and Weather Conditions.

For the purpose of this thesis, asthma admissions into hospital were investigated for all asthma admissions and asthma age 0-9. These two groupings had the highest incident rates of admissions, and this study set out to determine if these admissions could be attributed to outdoor air pollution concentrations. A time-series analysis was conducted for single air pollution models. As stated before, associations were examined for levels of pollution that occurred on the concurrent day (referred to as lag 0 days), on the previous day (lag 1 day), and averaged over the concurrent and previous two days (3-day mean). The study period investigated both asthma groupings according to all year (April 1st 1992 to March 31st 1999) and seasons; "warm" (April to September), "cold" (October to March), "short" summer (June to August) and "rest" of the year (September to May).

The dependent variable in the regression analysis was the counts per day of admissions for asthma, with separate analyses conducted for all asthma and asthma age 0-9 groups. The baseline model for the regression analyses included those measured variables that could potentially confound the association between daily admissions and levels of air pollution. The following terms were thus included : a temporal filter for time (in days); calendar year; day-of-the-week; and selected weather variables.

The "best" temporal filter for each grouping (all asthma, age 0-9) was chosen so that the time series of residuals would be "relatively compatible with what would be expected from a pure white noise process" (Goldberg et al., 2000). This temporal filter often had the lowest value of the AIC and tended to minimize the serial autocorrelation coefficients. Tables 7 and 8 show the temporal filters that were chosen for the two asthma

groupings. A temporal filter of 2.81 (71/2,526) was chosen for all asthma and age 0-9 as it had the highest p-value for the Bartlett's test of white noise and a reasonable residual autocorrelation coefficients.

Table 7. Selection of the Optimal LOESS Temporal Filter for All Asthma Admissions, Montreal, 1992-1999

	Temporal Filter (span in %)*	Dispersion	AIC	Bartlett Statistics	P-Values from Bartlett Statistics	Serial Autocorrelation Coefficient					
						Lag 1	Lag 2	Lag 3	Lag 4	Lag 5	Lag 6
All Asthma	2.02	1.08	2961.80	1.49	0.02	-0.06	0.00	-0.04	0.00	-0.04	0.03
	2.41	1.09	2978.50	1.17	0.13	-0.04	0.01	-0.02	0.01	-0.02	0.04
	2.81	1.10	3005.10	0.87	0.43	-0.02	0.03	0.00	0.03	-0.01	0.06
	3.21	1.12	3042.50	1.25	0.09	0.00	0.05	0.02	0.05	0.01	0.08
	3.60	1.14	3087.50	1.88	0.00	0.02	0.07	0.04	0.07	0.03	0.09

* Spans in percentage was determined by taking the span divided by the number of days (2,526)

Table 8. Selection of the Optimal LOESS Temporal Filter for Asthma Age 0-9 Admissions, Montreal, 1992-1999

	Temporal Filter (span in %)*	Dispersion	AIC	Bartlett Statistics	P-Values from Bartlett Statistics	Serial Autocorrelation Coefficient					
						Lag 1	Lag 2	Lag 3	Lag 4	Lag 5	Lag 6
Age 0-9	2.02	1.06	3036.22	1.43	0.03	-0.05	-0.04	-0.02	-0.01	-0.05	0.03
	2.41	1.07	3044.38	1.15	0.14	-0.03	-0.02	-0.01	0.00	-0.03	0.04
	2.81	1.08	3061.85	0.88	0.42	-0.01	-0.01	0.01	0.02	-0.02	0.06
	3.21	1.09	3088.58	0.91	0.39	0.00	0.01	0.03	0.03	-0.01	0.07
	3.60	1.10	3122.32	1.44	0.03	0.02	0.03	0.04	0.05	0.01	0.08

* Spans in percentage was determined by taking the span divided by the number of days (2,526)

Weather could confound the association between air pollutants and asthma hospital admissions. Weather variables were graphed with various scatter plots (a matrix 5 by 5 or 6 by 6), plotting weather variables against weather variables along with the Pearson correlation coefficients (r-value) (Appendix D. 1 to D. 2). With the use of the matrix, paired weather variables with low r-values (e.g. so as to avoid co-linearity among the pairs) were considered for the background model. Next, this study set out to determine which weather variable would produce the lowest AIC. No assumptions were made as to whether the effects of weather were concurrent with or proceeded each day of admission. The final weather variables were determined by plugging the weather variables into the regression model. Only those pairs weather variables that explained the most residual variation in the data (minimum AIC). Each of the asthma groupings produced slightly different AIC with the weather variables for lag 0 (2,526) days and lag 1 (2,525) days. The chosen weather variables were daily mean temperature, daily mean temperature change, mean relative humidity, and change in pressure in 24hr ending at 8:00.

The confounding effects of weather were controlled for with the use of temporal filters. The LOESS weather variables were then analyzed for each day of admissions for a lag 0, 1, and average lags zero to two days (3-day mean). As stated above, a time series analysis was developed with temporal filters for calendar year (CALYEAR), day of the week (DOW). Each weather variable was put into the equation using additive (lo(A)+lo(X)) and interactive (lo(A, X)) effects for lag 0 and lag 1 (Table 9). The residual time series analysis for the weather variables with the lowest AIC was used. For the next time series analysis the addition of mean temperature change (TMEANCH) and change

in pressure at 08:00 (PRES08CH) at lag 1 were used. Once these weather variables were chosen it was time to input pollutants into the model.

Table 9. Selected Weather Variables for All Asthma Admissions Time Series Analysis, Montreal, 1992-1999

Weather Variables	Lags	Dispersion	AIC
PRES08CH, TMEAN	1 + 1	1.10	3014.14
PRES08CH, TMEANCH	1 + 1	1.10	3009.40
PRES08CH, RHMEAN	1 + 1	1.10	3012.28
PRES08CH, TMEAN, TMEANCH	1 + 1+ 1	1.10	3016.98
PRES08CH, TMEANCH, RHMEAN	1 + 1+ 0	1.10	3014.24
PRES08CH, TMEAN, RHMEAN	1 + 1+ 0	1.10	3020.09
PRES08CH, TMEAN, TMEANCH, RHMEAN	1 + 1 + 1 + 0	1.10	3022.19
PRES08CH, TMEANCH	1 , 1	1.10	3018.77

1 + 1, only main effects, both with lag 1
1 , 1 means main effects and interaction between the 2 variables, both with lag 1
PRES08CH = change in pressure for 24 hours ending at 08:00
TMEAN = mean temperature
RHMEAN = mean relative humidity
TMEANCH = change in mean temperature

4.5 Statistical Analysis of the Association Between Daily Hospital Admissions for Asthma and Levels of Air Pollution: Results for the Mean Percent Change and 95% Confidence Interval

Analysis was conducted for both asthma admissions (all asthma, age 0-9) for all air pollutants. Each pollutant was analyzed on the day of admission (lag 0), the preceding day (lag1), and the mean of exposures across the concurrent day and the two proceeding days (3-day mean). Tables 10 to 16 shows the mean percent change (MPC) for each asthma grouping and selected air pollutant at various lags across each pollutant's interquartile range (IQR). Each statistical model controlled for day of the week effect, calendar year, seasonal variations and selected weather conditions (e.g. mean temperature change and change in pressure ending at 08:00am). The mean percent change is an estimate of the percent change in daily hospital admissions per increase in air pollutant. Therefore, the mean percent change and its 95% confidence interval are measuring an increase (or decrease) in mean number of hospitalization for an increase in each pollutant equal to the interquartile range. The model was then used to determine the associations between selected air pollutants and asthma hospital admissions.

As stated in Chapter 3 (section 3.4) the study period was divided into warm and cool periods (summer, winter, short summer, and rest of the year). Dividing the year in half provides a better understanding on how concentrations of pollutants affects asthma hospitalization throughout the cool and warm months. The stratification of the data into seasonal groupings titled short summer and rest of the year evolved from the findings in Figure 3, which showed low admission level in June, July and August. The following tables will investigate these seasonal variations in an attempt to determine if elevated

levels of selected air pollutants are associated with increase asthma admission rates for all asthma and age 0-9 at various lags.

Table 10. Mean Percent Change in Daily Asthma Admissions for a Change in the Daily Levels of Air Pollutants Equal to their Interquartile Range, for All Year at Lag 0 Days, Montreal, 1992 to 1999

	CO		COH		H_2S		NO_2		O_3		SO_2		PM_{10}	
	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI
All Asthma	0.46	-1.35, 2.31	2.38	-0.17, 5.00	-0.51	-2.23, 1.25	-0.67	-2.84, 1.54	-4.86	-7.17, -2.50	-1.61	-3.43, 0.24	-0.71	-5.23, 4.03
Age 0-9	0.36	-2.10, 2.88	1.59	-1.93, 5.24	-1.11	-3.41, 1.25	-2.29	-5.23, 0.74	-6.16	-9.30, -2.91	-3.13	-5.64, -0.56	0.57	-5.53, 7.06

All Asthma and Age 0-9 Span 71/2,526

MPC = mean percent change; 95% CI = 95% confidence interval

The statistical model was $E(\log(y_i)) = \alpha + \text{loess}(i, \text{span} = x)) + \beta 1 * YEAR_i + \beta 2 * DOW + \beta 3 * WEATHER + \beta 4 * POLLUTANT$;
where i is an indicator for day and x is the selected span (percent).

Mean percent change calculated for an increase of exposure equal to the interquartile value (Table?)

Table 11. Mean Percent Change in Daily Asthma Admissions for a Change in the Daily Levels of Air Pollutants Equal to their Interquartile Range, for All Year at Lag 1 Days, Montreal, 1992 to 1999

	CO		COH		H_2S		NO_2		O_3		SO_2		PM_{10}	
	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI
All Asthma	0.12	-1.74, 2.01	1.12	-0.17, 5.00	0.70	-1.03, 2.45	0.17	-2.11, 2.50	-1.16	-3.52, 1.26	-1.03	-2.89, 0.86	2.47	-2.53, 7.73
Age 0-9	0.72	-1.80, 3.31	1.85	-1.81, 5.65	0.73	-1.58, 3.09	-0.21	-3.32, 3.00	-3.42	-6.61, -0.13	-1.16	-3.73, 1.49	4.63	-1.58, 11.23

All Asthma and Age 0-9 Span 71/2,526

MPC = mean percent change; 95% CI = 95% confidence interval

The statistical model was $E(\log(y_i)) = \alpha + \text{loess}(i, \text{span} = x)) + \beta 1 * YEAR_i + \beta 2 * DOW + \beta 3 * WEATHER + \beta 4 * POLLUTANT$;
where i is an indicator for day and x is the selected span (percent).

Mean percent change calculated for an increase of exposure equal to the interquartile value (Table?)

Table 12. Mean Percent Change in Daily Asthma Admissions for a Change in the Daily Levels of Air Pollutants Equal to their Interquartile Range, for All Year at Lag 3-Day Mean, Montreal, 1992 to 1999

	CO		COH		H_2S		NO_2		O_3		SO_2	
	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI
All Asthma	-0.60	-2.85, 1.70	1.94	-1.45, 5.45	-0.21	-2.31, 1.93	-1.25	-3.97, 1.54	-3.68	-6.40, -0.88	-2.42	-4.68, -0.10
Age 0-9	1.19	-1.92, 4.40	3.84	-0.93, 8.83	-1.22	-3.99, 1.63	-1.50	-5.21, 2.35	-6.53	-10.18, -2.73	-3.27	-6.41, -0.03

All Asthma and Age 0-9 Span 71/2,526

MPC = mean percent change; 95% CI = 95% confidence interval

The statistical model was $E(\log(y_i)) = \alpha + \text{loess}(i, \text{span} = x)) + \beta 1 * YEAR_i + \beta 2 * DOW + \beta 3 * WEATHER + \beta 4 * POLLUTANT$;
where i is an indicator for day and x is the selected span (percent).

Mean percent change calculated for an increase of exposure equal to the interquartile value (Table?)

Table 13. Mean Percent Change in Daily Asthma Admissions for a Change in the Daily Levels of Air Pollutants Equal to their Interquartile Range, for Summer (April to September) at Lags 0,1, and 3-Day Mean, Montreal, 1992 to 1999

		CO		COH		H_2S		NO_2		O_3		SO_2	
		MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI
All Asthma													
	0	1.41	-2.73, 5.73	5.61	1.16, 10.25	-1.69	-4.27, 0.97	-0.44	-4.33, 3.61	-4.51	-7.89, -1.00	-2.55	-6.48, 1.55
	1	-1.36	-5.54, 3.01	2.46	-2.10, 7.23	0.08	-2.47, 2.70	1.48	-2.64, 5.78	1.46	-2.03, 5.07	1.74	-2.38, 6.04
	3-Day Mean	-3.17	-8.23, 2.16	4.21	-1.62, 10.39	-0.56	-3.72, 2.70	-1.99	-6.97, 3.26	-0.56	-4.83, 3.90	-2.29	-7.73, 3.49
Age 0-9													
	0	-1.11	-6.52, 4.61	3.67	-2.28, 9.99	-0.93	-4.21, 2.47	-0.85	-6.01, 4.59	-3.76	-8.35, 1.06	-3.25	-8.43, 2.22
	1	0.60	-5.03, 6.57	6.15	-0.17, 12.88	0.36	-2.89, 3.71	0.07	-5.34, 5.78	0.31	-4.33, 5.17	0.56	-4.85, 6.27
	3-Day Mean	-2.33	-9.09, 4.94	5.96	-2.06, 14.63	-1.02	-5.01, 3.15	-1.73	-8.36, 5.38	-0.15	-7.15, 4.55	-5.20	-12.19, 2.35

All Asthma and Age 0-9 Span 71/2,526;
MPC = mean percent change and 95% CI = 95% confidence interval;
The statistical model was E(log(yi)) = a + LOESS (i, span =x)) + b1*YEARi + b2*DOW + b3*WEATHER + b4*POLLUTANT; where i is an indicator for day and x is the selected span (percent).

Table 14. Mean Percent Change in Daily Asthma Admissions for a Change in the Daily Levels of Air Pollutants Equal to their Interquartile Range, for Winter (October to March) at Lags 0,1, and 3-Day Mean, Montreal, 1992 to 1999

		CO		COH		H_2S		NO_2		O_3		SO_2	
	Lags	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI
All Asthma													
	0	0.54	-1.59, 2.72	1.06	-2.10, 4.33	0.75	-1.58, 3.14	0.29	-2.40, 3.04	-4.47	-8.84, 0.10	-0.65	-2.93, 1.69
	1	0.99	-1.22, 3.24	1.02	-2.29, 4.43	2.21	-0.15, 4.62	0.89	-1.95, 3.80	-3.72	-8.12, 0.90	-0.70	-3.03, 1.70
	3-Day Mean	0.31	-2.46, 3.16	1.16	-3.14, 5.66	0.70	-2.15, 3.63	1.37	-2.06, 4.92	-4.75	-10.31, 1.16	-1.24	-4.21, 1.83
Age 0-9													
	0	0.76	-2.21, 3.82	0.24	-4.18, 4.87	0.03	-3.24, 3.42	-1.74	-5.49, 2.16	-6.15	-12.10, 0.21	-2.54	-5.78, 0.80
	1	1.28	-1.79, 4.44	0.52	-4.07, 5.34	3.03	-0.29, 6.46	1.68	-2.34, 5.87	-6.35	-12.30, 0.00	-0.45	-3.79, 3.00
	3-Day Mean	2.56	-1.37, 6.64	2.82	-3.28, 9.31	0.58	-3.37, 4.69	2.09	-2.78, 7.20	-8.21	-15.67, -0.10	-1.09	-5.36, 3.37

All Asthma and Age 0-9 Span 71/2,526;
MPC = mean percent change and 95% CI = 95% confidence interval;
The statistical model was E(log(yi)) = a + LOESS (i, span =x)) + b1*YEARi + b2*DOW + b3*WEATHER + b4*POLLUTANT; where i is an indicator for day and x is the selected span (percent).

Table 15. Mean Percent Change in Daily Asthma Admissions for a Change in the Daily Levels of Air Pollutants Equal to their Interquartile Range, for Short Summer (June to August) at Lags 0,1, and 3-Day Mean, Montreal, 1992 to 1999

		CO		COH		H_2S		NO_2		O_3		SO_2	
	Lag	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI
All Asthma													
	0	5.97	-1.86, 14.43	4.24	-2.74, 11.72	-0.36	-4.27, 3.72	1.11	-6.58, 9.44	-5.39	-10.65, 0.17	-0.26	-7.12, 7.11
	1	7.69	-0.61, 16.67	2.64	-4.57, 10.39	-1.73	-5.70, 2.42	6.44	-1.71, 15.27	-0.63	-5.99, 5.04	5.09	-1.91, 12.59
	3-Day Mean	12.07	1.88, 23.27	6.60	-2.35, 16.37	-2.36	-7.49, 3.05	7.85	-2.56, 19.38	-1.01	-7.72, 6.19	8.82	-1.21, 19.86
Age 0-9													
	0	0.63	-9.62, 12.04	2.56	-7.11, 13.25	4.35	-0.64, 9.58	-0.89	-11.13, 10.53	-0.42	-8.00, 7.78	1.84	-7.73, 12.40
	1	13.77	1.84, 27.11	11.31	0.56, 23.22	0.97	-4.20, 6.41	6.97	-4.13, 19.36	-2.24	-9.54, 5.64	6.92	-2.83, 17.65
	3-Day Mean	11.69	-2.26, 27.64	14.50	1.08, 29.69	4.79	-2.28, 12.36	7.89	-6.18, 24.07	-0.88	-10.11, 9.31	13.54	-0.66, 29.77

All Asthma and Age 0-9 Span 71/2,526;
MPC = mean percent change and 95% CI = 95% confidence interval;
The statistical model was E(log(yi)) = a + LOESS (i, span =x)) + b1*YEARi + b2*DOW + b3*WEATHER + b4*POLLUTANT; where i is an indicator for day and x is the selected span (percent).Table 11. Mean Percent Change in Daily Asthma Admissions for a Change in the Daily Levels of Air Pollutants Equal to their Interquartile Range, for Rest Year (September to May) at Lags 0,1, and 3-Day Mean, Montreal, 1992 to 1999

Table 16. Mean Percent Change in Daily Asthma Admissions for a Change in the Daily Levels of Air Pollutants Equal to their Interquartile Range, for Rest of the Year (September to May) at Lags 0,1, and 3-Day Mean, Montreal, 1992 to 1999

		CO		COH		H_2S		NO_2		O_3		SO_2	
	Lags	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI	MPC	95% CI
All Asthma													
	0	0.50	-1.39, 2.43	1.99	-0.75, 4.80	-0.46	-2.37, 1.48	-0.52	-2.79, 1.82	-4.97	-7.76, -2.08	-1.23	-3.17, 0.75
	1	0.08	-1.86, 2.06	0.49	-2.34, 3.40	1.09	-0.82, 3.03	-0.19	-2.59, 2.26	-2.22	-5.06, 0.71	-1.24	-3.22, 0.77
	3-Day Mean	-0.76	-3.13, 1.66	1.30	-2.36, 5.09	-0.17	-2.43, 2.14	-1.40	-4.25, 1.54	-3.39	-6.75, 0.09	-2.21	-4.63, 0.27
Age 0-9													
	0	0.54	-2.06, 3.22	1.04	-2.78, 4.98	-1.70	-4.28, 0.94	-1.84	-4.97, 1.41	-6.40	-10.18, -2.46	-3.04	-5.73, -0.27
	1	0.42	-2.25, 3.15	0.17	-3.74, 4.24	0.86	-1.72, 3.51	-0.35	-3.67, 3.08	-3.71	-7.55, 0.28	-1.57	-4.33, 1.28
	3-Day Mean	1.11	-2.19, 4.53	2.19	-2.93, 7.58	-1.75	-4.76, 1.38	-1.27	-5.22, 2.85	-5.76	-10.27, -1.03	-3.38	-6.76, 0.13

All Asthma and Age 0-9 Span 71/2,526;
MPC = mean percent change and 95% CI = 95% confidence interval;
The statistical model was E(log(yi)) = a + LOESS (i, span =x)) + b1*YEARi + b2*DOW + b3*WEATHER + b4*POLLUTANT; where i is an indicator for day and x is the selected span (percent).

4.6 Summary of Mean Percent Change Results

Tables 10 to 16 are the results for the associations of carbon monoxide (CO), coefficient of haze (COH), hydrogen sulfide (H_2S), nitrogen dioxide (NO_2), ozone (O_3) and sulfur dioxide (SO_2) and asthma admissions, unadjusted for other pollutants. Across all seasons positive associations with the lag 1 and 3-day mean were generally stronger than lag 0. Statistically positive associations were found during short summer and summer with carbon monoxide, coefficient of haze and both asthma admissions. For example, a 3.5mg/m^3 (IQR) increase in carbon monoxide (CO) causes a 12.07% increase in all asthma hospital admission at a 95% CI (1.88-23.27) for the short summer period at lag 3-day mean.

Statistically negative associations were discovered for all year, rest year, summer and winter seasonal sub-divisions for both asthma groupings, ozone and sulfur dioxide. However, no associations were found with increased concentrations of hydrogen sulfide, nitrogen dioxide and particulate matter less then 10μm and asthma hospital admissions (for all asthma and asthma age 0 to 9).

CHAPTER V

DISCUSSION OF RESULTS

The results of this analysis revealed that elevated summer and short summer levels of carbon monoxide and coefficient of haze were positively associated with increased asthma hospital admissions in Montreal for 1992 to 1999. These positive associations were observed for various lags with 1 day and 3-day mean lags showing slightly stronger associations. The strongest positive associations for all asthma hospital admissions was found with carbon monoxide, however the most robust positive association for asthma age 0 to 9 admissions was found with coefficient of haze. Negative associations were found between ozone, sulfur dioxide, and asthma hospital admissions for the whole year (both asthma groupings), rest year (both asthma groupings), summer (all asthma) and winter (asthma age 0 to 9). No associations were found for hydrogen sulfide, particulate matter less than 10μm and nitrogen dioxide with asthma hospital admissions.

Many studies have shown positive associations between air pollutants and increased asthma/respiratory hospital admissions (Schouten, 1996, Schwartz 1996; Bates and Sizto, 1987; Cody et al., 1992; Thurston et al., 1992, Chew et al. 1999, Morgan and Corbett, 1998). In fact, some associations between air pollutant and hospital admissions exist in spite of relatively low air pollutant levels (Pönkä and Virtanen, 1996). To test this notion, one study removed high ozone pollution readings on various days from their analysis and discovered that significantly positive associations remained with low level ozone concentrations and hospital admissions (Thurston et al, 1994).

The literature dealing with ozone and sulfur dioxide at times contradict the results found in this study. As stated above, one of the negative associations found with ozone, sulfur dioxide and both asthma groupings occurred from September to May ("rest year") at lag zero days. However in Toronto, Canada, no associations were found between elevated levels of ozone and acute respiratory admissions for children less than 2 years of age from September to April (Burnett et al., 2001). Other studies in New York, Seattle, Rotterdam and Barcelona found no associations between ozone, sulfur dioxide and morbidity (Kleinman ,1992; Sheppard et al., 1999; Schouten et al., 1996). However, studies in Amsterdam and London support the findings of this analysis whereby, negative associations between ozone and sulfur dioxide and morbidity were found (Hajat et al, 1999 and Anderson et al., 1998). Both authors believe that the negative associations are driven by a lack of statistical power.

Negative associations found between elevated levels of ozone and sulfur dioxide and asthma hospital admissions need to be investigated with care (Burnett, et al., 1994). What a negative association implies is that there is a protective effect occurring with the pollutants and hospital admissions (i.e. when pollutants are high, admissions are low). This protective effect of ozone and sulfur dioxide can be seen for this research in warm months (summer/short summer), cold months (rest year/winter) and for the whole year (all year). Even though ozone is usually highest in the summer, this negative association prevails (Burnett et al., 1994). As Burnett argues "there is no experimental evidence to suggest that ozone (or sulfur dioxide) has a protective effect" (Burnett et al., 1994).

The positive associations found for this study linking coefficient of haze and carbon monoxide with asthma hospital admissions have been found in a number of

studies. (Sheppard et al., 1999; Hajat et al 1999; Burnett et al., 1995; Burnett et al., 1994; Bates and Sitzo, 1987; Thurston et al., 1992; Thurston et al., 1994; Delfino et al., 1994a,1994b; Pönkä and Virtanen, 1996). Some researchers have found associations with particles and respiratory hospital admissions during summer periods (Bates and Sizto 1987; Burnett et al., 1994), while others found associations for the whole year (Pope 1991; Hagen et al., 2000).

The findings of this analysis need to be studied in light of the methodological approach, validity of morbidity data and confounding factors so as to understand the causal nature of air pollutant associations and asthma hospital admissions. A "major argument in favour of causality has been the consistency of results obtained from a wide variety of cities throughout the world" (Anderson et al., 1998) However, the lack of consistency between air pollutant associations and asthma admissions weakens the debate for causality (Anderson et al., 1998). Perhaps asthma admissions are more dependent on the susceptibility of a population and local environment than on air pollutants. However, the results of this analysis indicate that increases of carbon monoxide and coefficient of haze are risk factors for asthma hospital admissions.

5.1 Methodological Framework

The methodology of this analysis was based on a quasi-likelihood regression model of the daily number of asthma hospital admissions against the daily levels of routinely measured air pollutants. Regression models were fitted for different lag structures of the air pollutants as well as accounting for potential confounding by fluctuations in short-term changes in weather, seasonal and subseasonal (e.g., day of the

week) trends. Non-parametric smoothers were used to account for the effect of these confounding factors.

5.1.1 Validity of Morbidity Data

This study assumes that the principal reason for admission into hospital was diagnosed and then coded accurately on the Med-Echo forms and database over the 7-year period. Although asthma is often misdiagnosed with other respiratory diseases (such as bronchitis), if the asthma guidelines are followed correctly by practitioners, these misdiagnoses should be minimized (Levison, 1991). Chapter 3 outlined the weaknesses and strengths of this database. Despite certain weaknesses, Delfino suggests that the data is accurate for identifying asthma hospitalizations in Quebec (Delfino et al, 1993).

5.1.2 Controlling for Confounding Factors

For the purpose of this study, confounding factors were variables that were associated with asthma hospital admissions and air pollution and thus varied over short time scales (Goldberg et al., 2000). Therefore, other essential risk factors for asthma, such as tobacco smoke and moulds in homes which are relatively constant on a population basis, should not confound associations between counts of hospitalizations for asthma and short-term fluctuations in air pollution (Rainham, 2000; Goldberg et al., 2000; Delfino et al., 1993). The temporal time filter should have removed confounding from other unmeasured factors that vary by season, for example influenza epidemics, which occur mostly in the fall and winter, when particle levels are increased (Goldberg et al., 2001). Continuing with this example, since no database could be used to monitor the

effects of influenza epidemics, this factor could not be controlled for directly. Epidemics tend to occur in the winter periods, so consequently the summer period estimates should not have been affected (Goldberg, et al., 2001). Much of this confounding factor was removed by controlling weather variation (Goldberg et al., 2000). However, weather, which does vary over short time scales, is an important confounding variable and was modelled explicitly using non-parametric smoothers. The methodology used to select the functional forms for the temporal and weather variables was based on a purely statistical approach, whereby functional forms and combinations of variables that minimized the AIC (i.e., the residual variance of the model) were selected (Goldberg et al., 2000). Because our understanding of the biological and toxicological processes of air pollution on airway diseases is basic at best and is not useful for selecting appropriate statistical models, this strategy was adopted and is reproducible.

5.1.3 Outdoor Air Pollution Levels

Daily data from selected fixed-site monitoring stations in Montreal that measured daily air pollution levels were used, and averages were taken across each monitoring station for this 7-year study period. Air pollution levels were averaged across each monitoring station in order to "smooth out local scale variations" (Goldberg et al., 2000). Various lag periods were used for this analysis. The lag periods of 0-day lag (concurrent day), 1-day lag, and the 3-day mean (an average of lag 0 to 2 days) were used for two reasons. The first reason is to show the short-term effects of the selected pollutants, and second, to provide insight into the fact that hospital admission does not necessarily occur

immediately after the exposure to risk factors. It may occur on the same day, or possibly within the next few days as seen in many other studies (Goldberg et al., 2000).

Accurate estimates of the risk factors air pollutants pose for human health is important for the implementation of public health policy. The estimates of risk are usually based on concentrations of outdoor air pollutants recorded at fixed monitoring stations. These measurements are subject to biases as most of the population spend the majority of their time indoors (Spengler and Sexton 1983). Also, personal exposure to many pollutants may not be adequately characteristic of the exposure because people spend their time somewhat differently depending on their age, gender, occupation, and socio-economic status (Spengler and Sexton 1983). However, various outdoor (e.g. volatile organic compounds) and indoor air pollutant readings were excluded from this study. The exclusion of these variables was due in part to a lack of available readings for certain outdoor and indoor pollutants making it impossible to account for their potential effects on human health (Goldberg et al., 2000). Most investigators assume that if indoor air pollution concentrations remain relatively constant over short time periods, then their effect will not be sufficient enough to disrupt the relationship between outdoor air pollutants and human health (Goldberg et al., 2000).

5.2 Implication of this Research

The results of this analysis will add to the existing body of knowledge dealing with air pollution associations and human health. It has allowed us to assess whether certain elevated levels of air pollutants cause increases in admission rates for a particular asthma grouping. The positive associations with carbon monoxide, coefficient of haze

and both asthma groupings emphasises the importance of considering susceptibility among sub-populations (Delfino et al., 1997). By identifying susceptible asthma groups we can develop realistic public policies which may improve their quality of life. This study achieved its objectives by: (1) identifying which asthma groupings had the highest incidence of asthma hospital admissions in Montreal (e.g. asthma age 0 to 9 and all asthma) and (2) determining if selected daily air pollutants were risk factors for these identified groupings. In terms of public health it is important to identify "groups at higher than average risk of hospitalization from the short-term effects of air pollution" (Goldberg et al., 2000). This susceptible population can then be targeted for programs (air pollution regulation, education) and for further research to try and determine why these groups have a high susceptibility.

5.3 Limitations of this Study and Further Research

This study provides great insight into the role of air pollution and human health although it does not provide a complete picture of the public health burden incurred by asthma morbidity (Goldberg et al, 2000). Some limitations encountered with this study can be corrected in the further studies, while other limitations were much too complex to avoid. Limitation ranged from the study population to air pollution readings.

This study only investigated asthma morbidity in terms of hospital admissions; leaving out ER admissions, clinic, family physician attendance and mortality. Hospital admissions for asthma likely underestimate of the public health burden due to air pollution in Montreal (Delfino et al., 1997). Therefore the overall public health burden attributed to asthma admissions and air pollution in Montreal could be far greater than

what the findings of this research suggest, if other morbidity and mortality variables were considered (Delfino et al., 1997). This additional analysis would provide a different perspective on the complex issues of health and environment.

This study only investigated the associations of air pollution and asthma admissions for all asthma and age 0 to 9. These two groupings were investigated because they had the highest hospitalization admissions for this study period and we were interested in assessing whether environmental factors were to blame for the raising rates. For future studies, it would be interesting to determine whether air pollutants influence admissions into hospital by other age groupings and sex for the 7-year study period.

Unfortunately, $PM_{2.5}$ readings were unavailable for the study period and only a few monitoring stations in Montreal measured PM_{10} readings. For this analysis selected monitoring stations were chosen, for further research it would be interesting to include more fixed monitoring air pollution stations to get a better sense of the air pollutants distribution. Further research is also required to assess why negative associations exist with ozone, sulfur dioxide and asthma hospital admissions for various lags and seasons.

When assessing whether asthma hospitalizations are associations with air pollutants the number of available beds could have been a confounding factor for admissions. Future analyses should assess this data controlling for the number of available beds by season for this 7-year study period. However, it was difficult to determine if such records are kept for Montreal.

This study controlled for weather as a possible confounding factor. However, the complex role of weather (e.g. extreme weather) and air pollution may change the asthma morbidity/pollution relationship (Rainham, 2000). Additional studies should examine the

relationship between weather and air pollution with respect to their associations with human morbidity.

Researchers believe that pollutants are highly negatively correlated with other pollutants throughout various months (Burnett et al., 1994; Goldstein et al., 1986; Goldberg et al., 2001). However, this analysis did not control for the confounding factors of other air pollutants while investigating the associations of the single air pollutant models. Therefore, further investigation is warranted to assess the joint effects of various pollutants and the risks these pollutants pose on asthma morbidity. Also, the rise of admissions in the spring could be attributed to an increase in various pollen or spores (Anderson et al., 1998). Therefore, further research is also required to assess the possible influence pollen or spores could have on asthma hospital admissions.

5.4 Public Health Policy

Chapter IV Figure 2 shows a decline in the number of asthma hospital admissions. This decline is mostly likely due to better management and prevention of asthma (through the use of inhaled corticosteroids). Despite the fact that asthma incidence was increasing, the use of these drugs only came after the National Institutes of Health consensus conference in the early 1990s concluded that steroids should be used as a first line of treatment. The new drugs prevented major crises, thereby reducing hospitalization. Prior to this all sorts of medications, which often did not work, were used. During that time, the last line of defence was steroids. Since then, new drugs have been put on the market (e.g. advair) which combines long-term steroids with long-term brochodilators, which are intended for daily use by asthma sufferers.

This decline in admissions also corresponds to the introduction of the Canada Health and Social Transfer by the federal government. In 1995 Finance Minister Paul Martin announced that a new federal/provincial cost-sharing program titled Canada Health and Social Transfer, which was created to replace the Canada Assistance Plan and Established Programs Financing. The Canada Health and Social Transfer program was a transfer of cash and tax credits from the federal government to provinces and territories for funding social welfare, health, and education programs. The Canada Health and Social Transfer substantially reduced the flow of cash from the federal government to the provinces and territories by several billion dollars (Health Canada, 2003). Besides the introduction of this new program, the 1990s were a year of intense fiscal pressure for provinces and territories. This lead to rapid cuts to health care, forcing the closure of hospital beds and at times, hospitals (Krahn et al., 1996). According to Statistics Canada there were 6.9 hospital beds per thousand Canadians in 1987/88, however, by 1995/96 the figure had fallen to 4.8 hospital beds (Connors and Millar, 1999). Despite the decrease in the total number of asthma admissions in Montreal, Quebec for 1995, the fact remains that the incidence and prevalence of asthma morbidity and mortality is on the rise worldwide. As stated in the literature review, asthma admissions place a huge burden on the health care system and inflicted families (e.g. loss of workdays). This burden on the health care system can for the most part be avoided if proper prevention (e.g. proper education, minimizing risk factors) and management (e.g. medical treatment) techniques for asthma sufferers are followed.

The impact of air pollution on hospital admissions for asthma from a public health stance is important for policy makers, researchers and public. Asthma policy, according

to the Lung Association, should be driven by both a short-term and a long-term goal (Canada Lung Association, 2003). The short-term goal focuses on the appropriate transfer and application of current knowledge to reduce morbidity and mortality among people with asthma. The long-term goal should aim for the reduction in the prevalence of asthma and perhaps ultimately, eradication.

There are various ways of achieving these goals. We need to continue researching the effects of environment (e.g. air pollution) on health (e.g. asthma morbidity and mortality). By funding research an understanding of the environmental risk factors for asthma can be gained. This would improve the availability of incidence estimates at the federal, provincial or local levels so that health planners can assess the full extent of the disease burden. Data are crucial to defining where programs are needed and what kind of programs they should be, as well as to provide information for educational purposes. There is currently no cure for asthma, but there are preventive measures that can be taken, such as medication and education. As a result, it is important to have a sense of the susceptible population with serious or uncontrolled asthma so that those groups can be targeted. Through education and proper treatment countless individuals have learned to live well with asthma. Asthma education and self-management programs should be made more broadly available to health care providers, patients, and the general population to help asthma sufferers understand and manage their illness. Community structures outside the traditional hospitals and clinics, for example schools, should create asthma care and education programs in order to target the most susceptible asthma grouping (e.g. children age 0 to 9).

This study concludes that there is evidence that various pollutants (carbon monoxide and coefficient of haze) may indeed have an effect on asthma hospital admissions for all asthma and age 0 to 9 groupings. However, it should still be noted that there is a lack of consistency by season, groupings and lags concerning the actual risk ambient air pollutants pose for asthma hospital admissions.

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APPENDIX

APPENDIX A. AIR POLLUTION MONITORING STATIONS

Table A. 1. Names of Air Pollution Monitoring Stations

Monitoring Station	Name of Station
1	Jardin Botanique Montreal
3	St-Jean-Baptisite Montreal
6	Chateauneuf Ville d'Anjou
12	Ontario Est Montreal
13	Drummond Montreal
28	Duncan Montreal
29	Parc Pilon Boul. Pie IX Montreal North
44	Boul. Saint-Michel Montreal
49	Lilas Dorval
61	Boul. De Maisonneuve Montreal
62	Clement Dorval Airport
66	Herve Saint-Martin Dorval Airport
68	Joseph Verdun
99	Sainte-Marie, Sainte-Anne-de-Bellevue

APPENDIX B. Daily Average of Air Pollution Concentrations

Table B. 1. Average of Daily Means Air Pollutants by Station, Montreal, 1992 to 1999

	Monitoring Stations	Number of Days Measured	Number of Missing Days	Average of Daily Mean
CO	3	2446	80	3.66
	12	1449	1077	6.73
	28	2436	90	9.78
	29	2501	25	5.64
	61	2504	22	11.08
COH	3	2006	520	1.50
	28	1949	577	4.13
	29	1919	607	1.46
	61	2057	469	2.73
	68	1948	578	1.29
H_2S	3	2386	140	8.90
NO_2	1	2380	146	35.81
	3	2203	323	29.46
	12	2359	167	37.09
	28	2371	155	51.24
	29	2093	433	34.29
	61	2486	40	54.48
	68	2428	98	32.47
O_3	1	2225	301	31.53
	3	2209	317	34.71
	12	2199	327	27.95
	28	2393	133	20.80
	29	2437	89	30.74
	49	2369	157	43.15
	61	2313	213	14.96
	68	2421	105	30.66
SO_2	1	2288	238	14.05
	3	2442	84	23.74
	28	2460	66	13.73
	61	2494	32	12.29
	68	2420	106	12.07

Table B.1. (continued) Average of Daily Means Air Pollutants by Station, Montreal, 1992 to 1999

	Monitoring Stations	Number of Days Measured	Number of Missing Days	Average of Daily Mean
PM_{10}	3	151	2375	27.01
	6	255	2271	33.94
	12	315	2211	26.10
	13	134	2392	26.13
	44	1	2525	12.85
	49	133	2393	19.98
	62	22	2504	18.71
	66	56	2470	26.60
	68	43	2483	22.63
	99	39	2487	16.27

Table B. 2. Average of Daily Means Air Pollutants by Station and Season, Montreal, 1992 to 1999

	Rest Year		Short Summer		Summer		Winter	
	Monitoring Stations	Average of Daily Means	Monitoring Stations	Average of Daily Means	Monitoring Stations	Average of Daily Means	Monitoring Stations	Average of Daily Means
CO	3	4.01	3	2.71	3	2.93	3	4.45
	12	7.27	12	4.94	12	5.13	12	8.19
	28	10.07	28	8.89	28	8.80	28	10.76
	29	6.17	29	4.04	29	4.36	29	6.89
	61	11.79	61	8.84	61	9.52	61	12.59
COH	3	1.56	3	1.32	3	1.13	3	1.70
	28	3.90	28	4.78	28	4.24	28	4.01
	29	1.55	29	1.19	29	1.16	29	1.78
	61	2.76	61	2.65	61	2.56	61	2.89
	68	1.35	68	1.13	68	1.13	68	1.47
H_2S	3	8.91	3	8.86	3	8.25	3	9.54
NO_2	1	37.61	1	30.22	1	32.04	1	39.76
	3	31.08	3	24.93	3	25.91	3	33.12
	12	38.80	12	31.84	12	33.08	12	40.95
	28	50.43	28	53.69	28	52.90	28	49.57
	29	35.87	29	29.00	29	30.54	29	37.52
	61	54.64	61	54.01	61	54.65	61	54.32
	68	35.52	68	22.80	68	26.50	68	38.49
O_3	1	27.95	1	41.69	1	39.58	1	22.92
	3	31.87	3	41.74	3	40.46	3	28.39
	12	23.53	12	40.64	12	37.07	12	19.40
	28	19.35	28	25.26	28	25.26	28	16.41
	29	27.50	29	41.20	29	39.16	29	22.77
	49	39.10	49	54.73	49	52.06	49	33.83
	61	12.49	61	22.28	61	20.17	61	9.19
	68	26.31	68	44.37	68	40.18	68	21.49

Table B. 2. (continued) Average of Daily Means Air Pollutants by Station and Season, Montreal, 1992 to 1999

	Rest Year		Short Summer		Summer		Winter	
	Monitoring Stations	Average of Daily Means	Monitoring Stations	Average of Daily Means	Monitoring Stations	Average of Daily Means	Monitoring Stations	Average of Daily Means
SO_2	1	15.82	1	7.73	1	8.97	1	18.64
	3	24.46	3	21.70	3	20.89	3	26.77
	28	15.74	28	7.10	28	9.11	28	18.05
	61	13.91	61	7.36	61	8.66	61	15.85
	68	13.44	68	7.57	68	8.00	68	15.86
PM_{10}	3	26.81	3	27.53	3	26.69	3	27.34
	6	34.56	6	32.04	6	31.74	6	36.12
	12	26.40	12	25.19	12	24.40	12	27.88
	13	26.46	13	24.90	13	25.95	13	26.27
	44	26.40	44	NA	44	NA	44	12.85
	49	12.85	49	20.12	49	18.96	49	21.01
	62	19.92	62	NA	62	NA	62	18.71
	66	18.71	66	23.01	66	24.67	66	28.40
	68	22.81	68	22.28	68	21.83	68	24.30
	99	13.22	99	23.14	99	20.85	99	13.08

Appendix C. Time Series Plots for Asthma Morbidity, Weather, and Pollutants

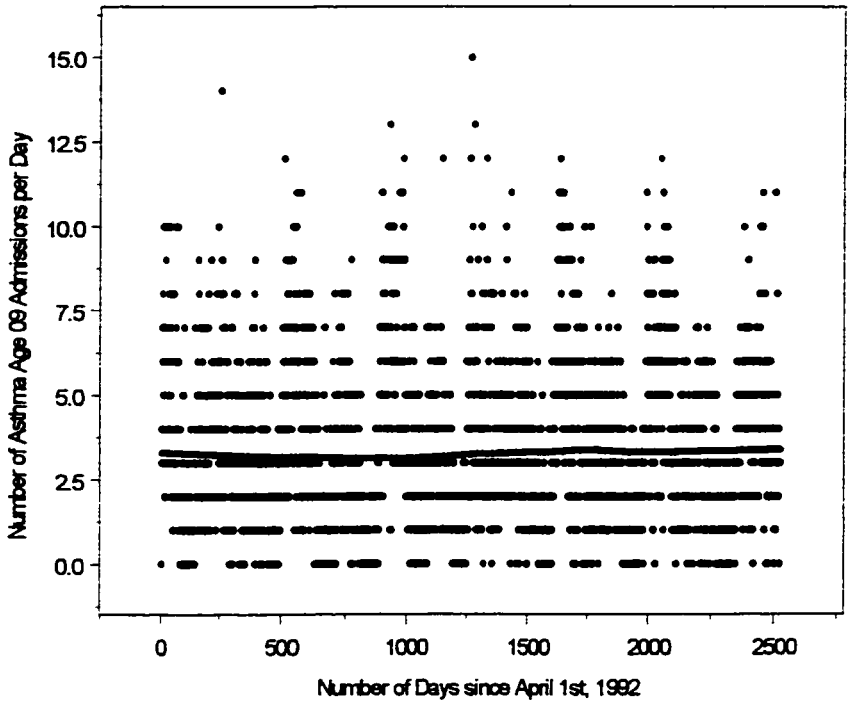

Figure C.1. Daily Hospitalizations from asthma for age 0-9. The solid line is the LOESS smooth representing the long-term trends in the data (span 50%).

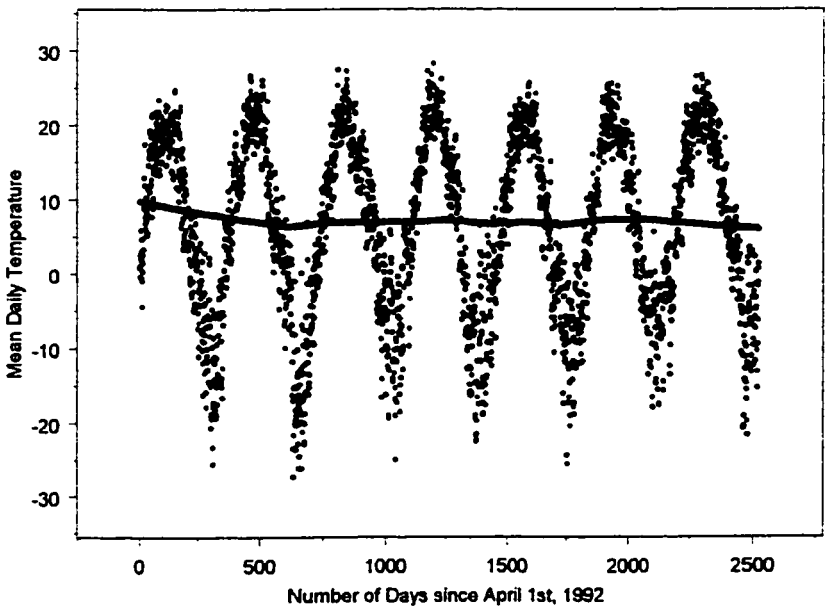

Figure C.2 Mean Daily Temperature. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

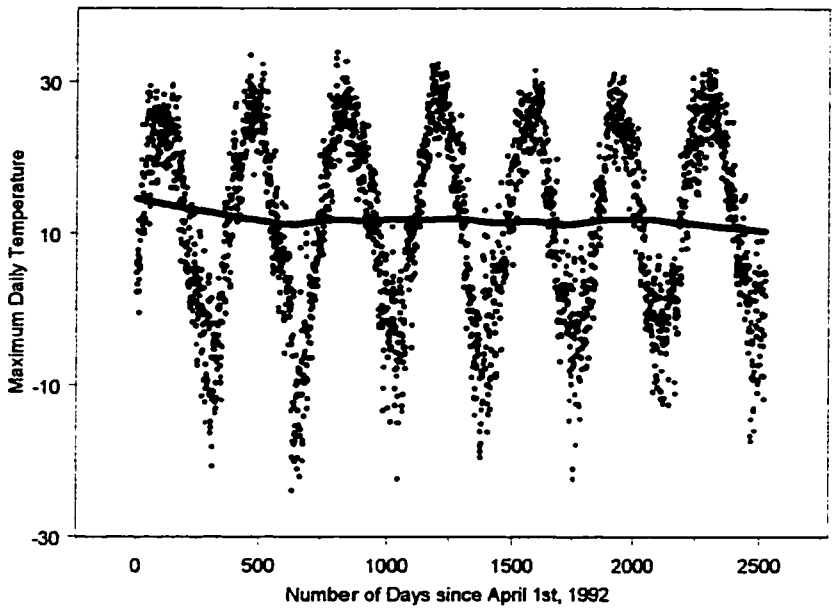

Figure C.3 Maximum Daily Temperature. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

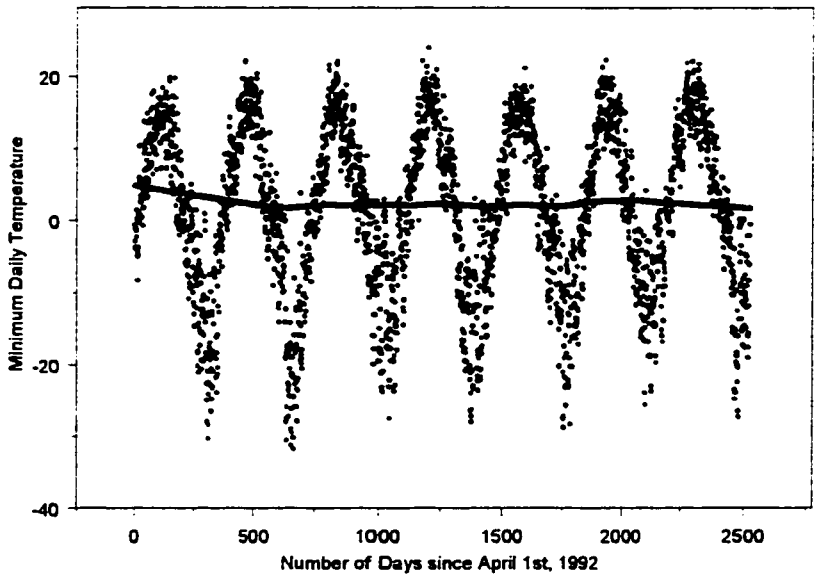

Figure C.4 Minimum Daily Temperature. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

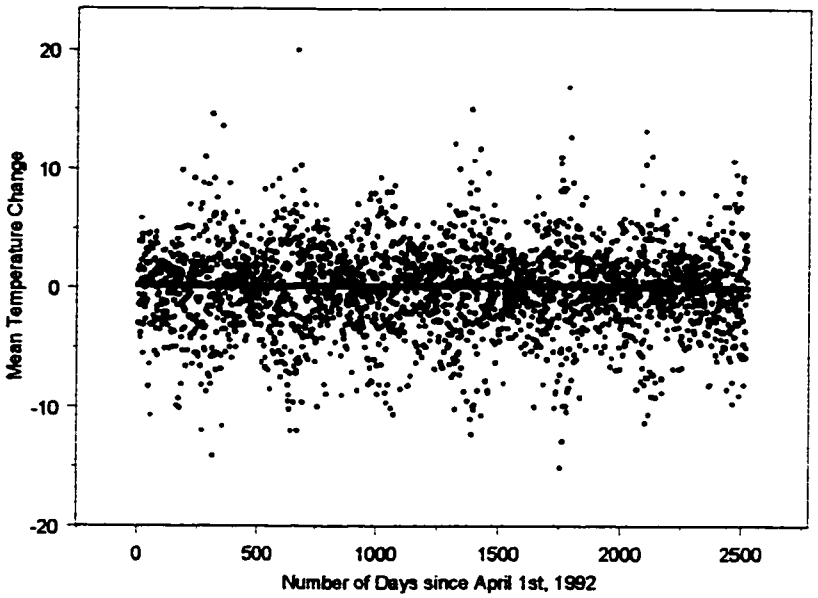

Figure C.5 Mean Temperature Change. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

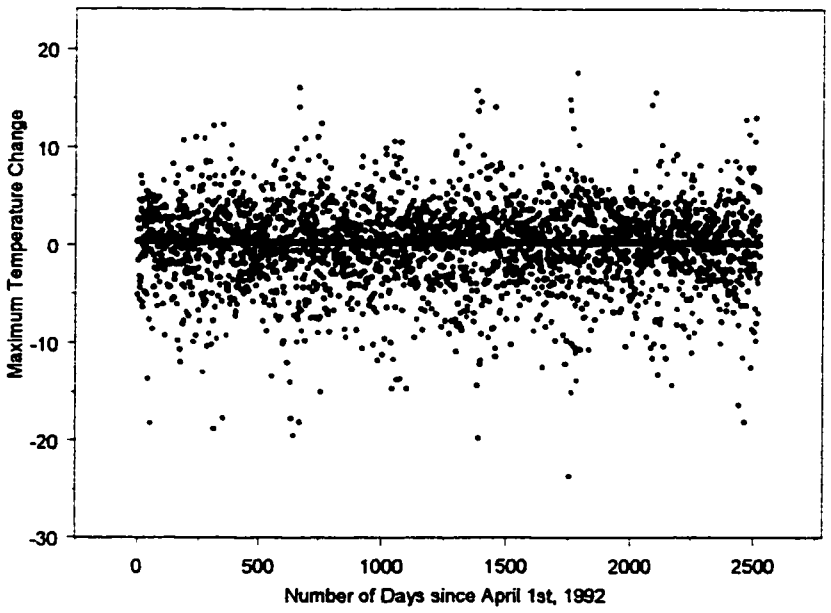

Figure C.6 Maximum Temperature Change. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

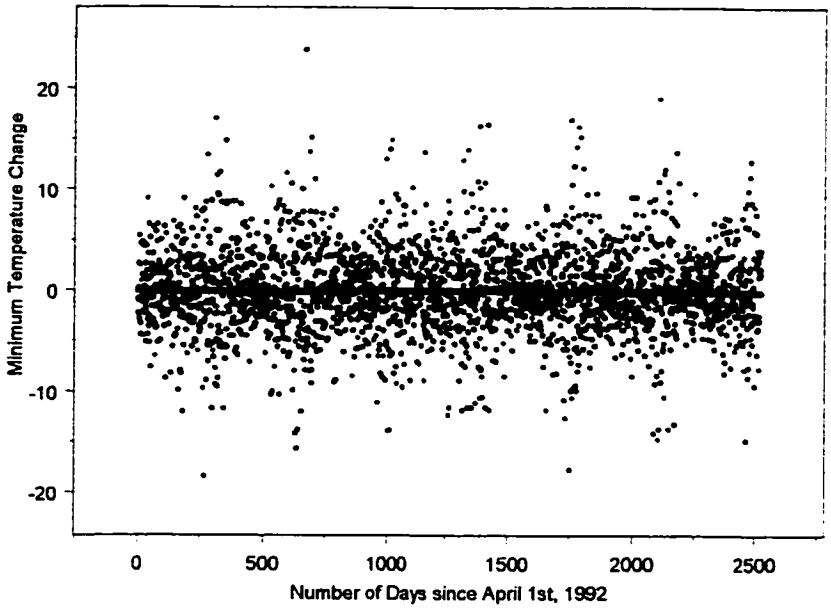

Figure C.7 Minimum Temperature Change. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

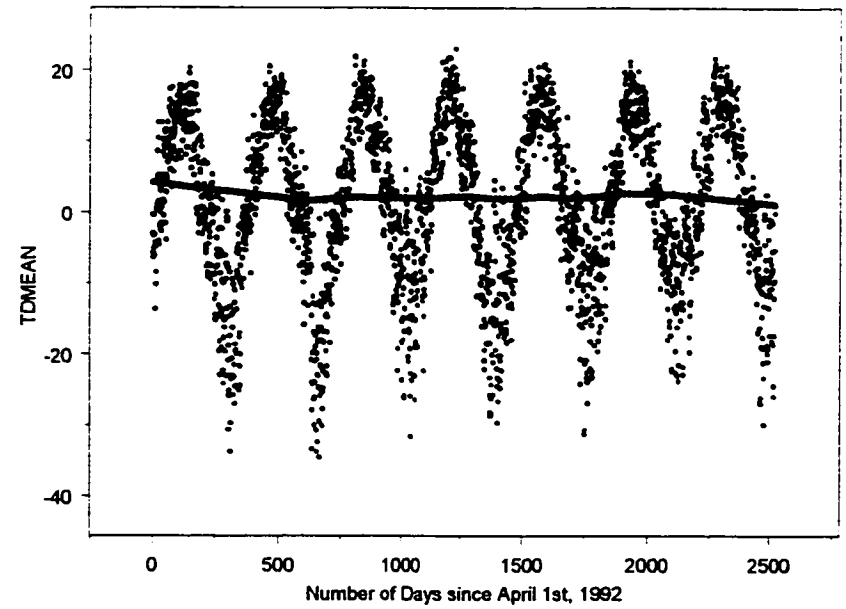

Figure C.8 Mean Dew Point Temperature. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

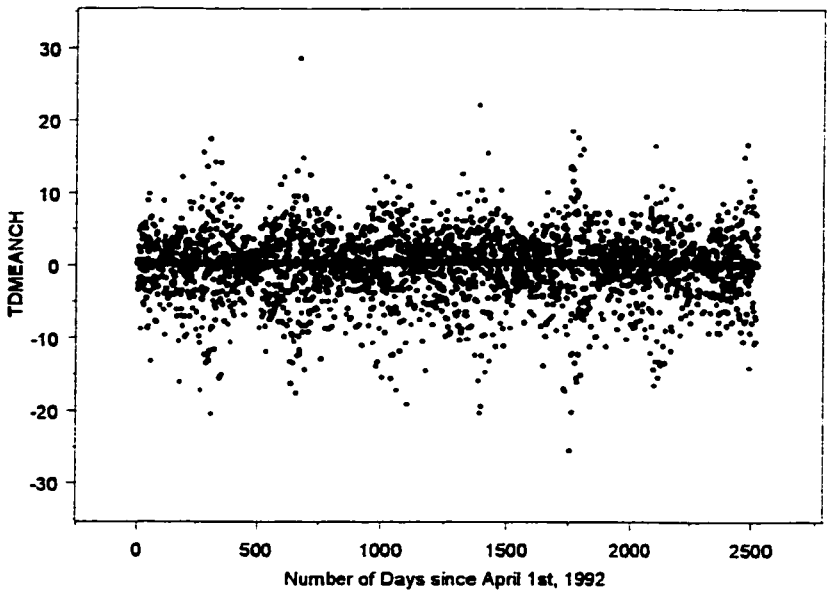

Figure C.9 Mean Dew Point Temperature Change. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

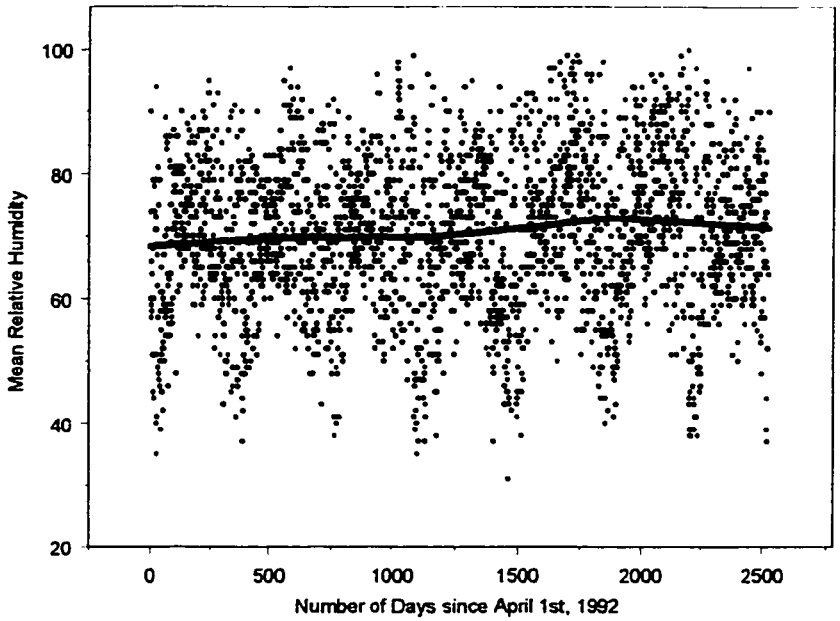

Figure C.10 Mean Relative Humidity. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

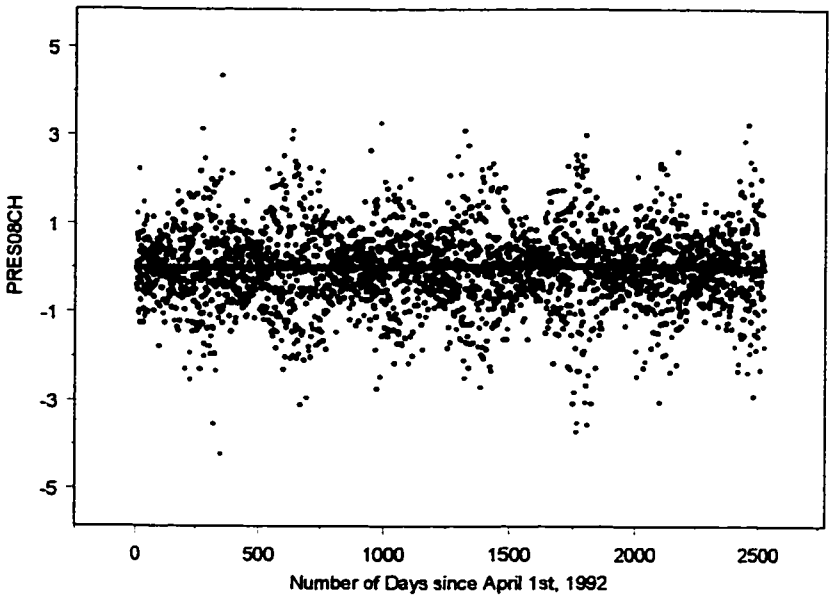

Figure C.11 Change in Pressure. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

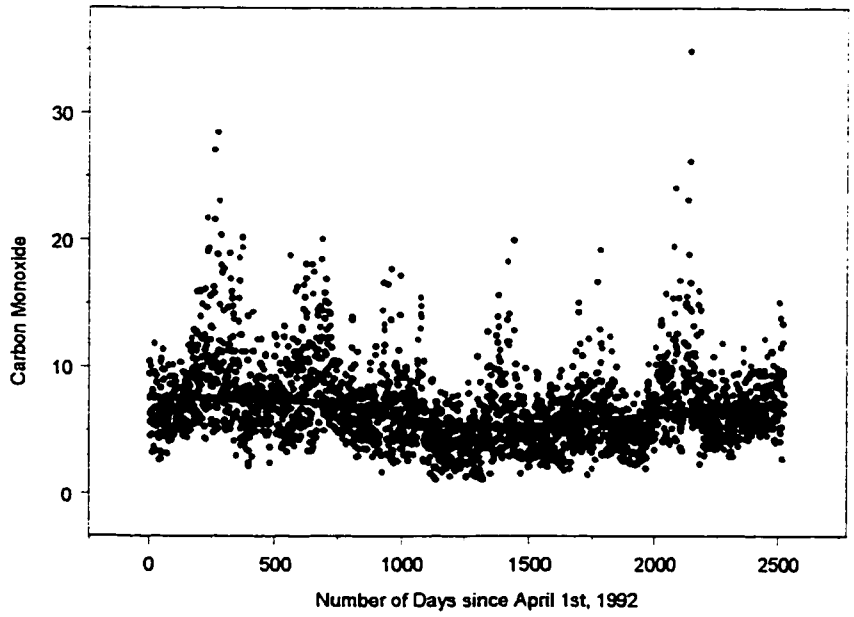

Figure C.12 Mean daily CO. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

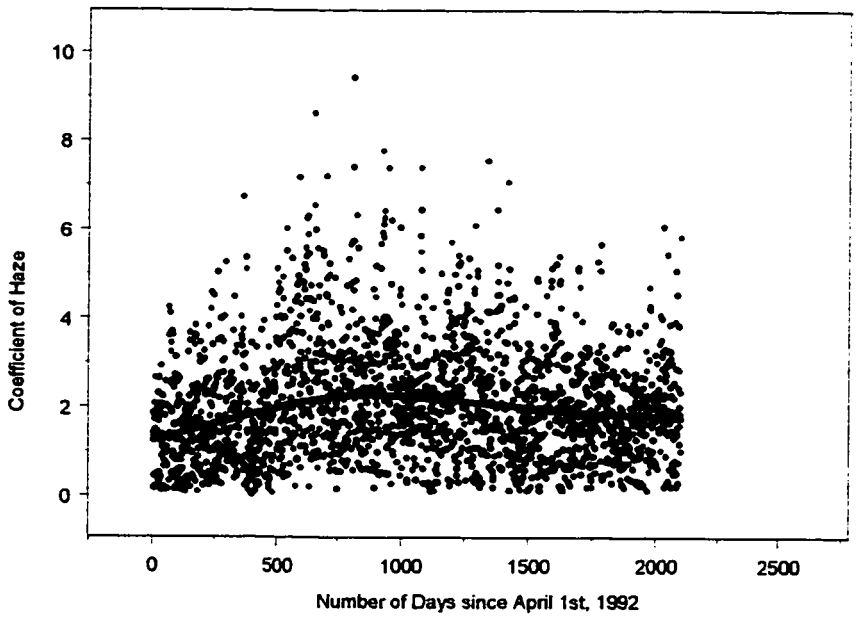

Figure C.13 Mean daily COH. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

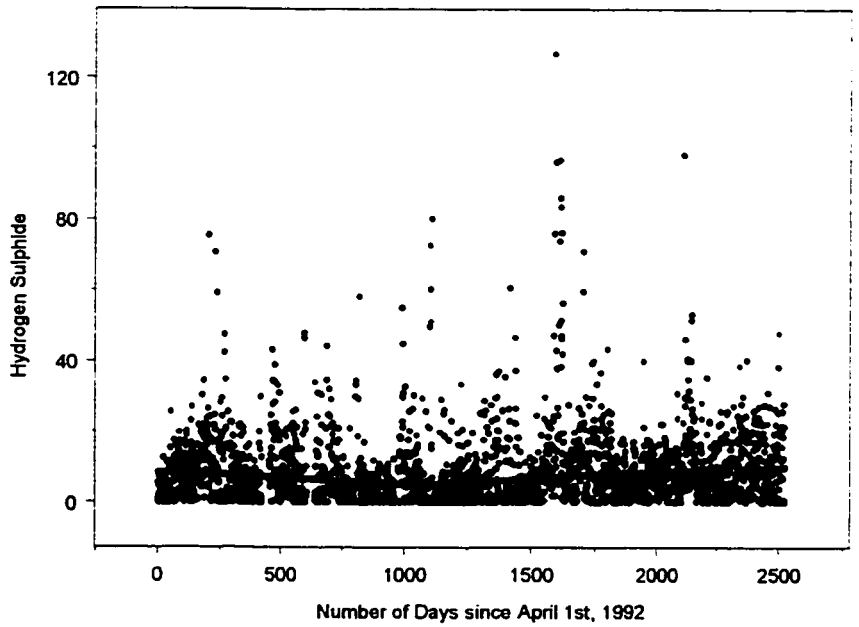

Figure C.14 Mean daily H_2S. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

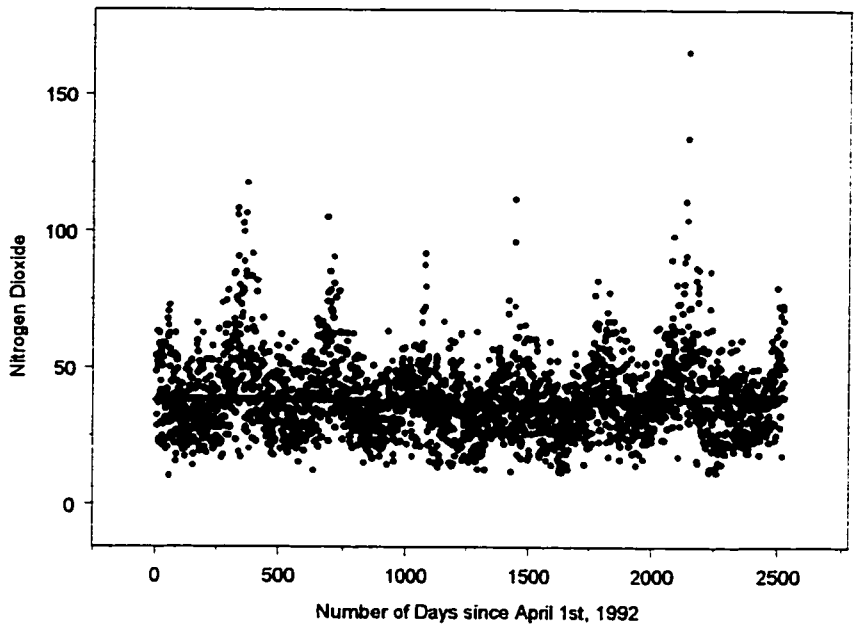

Figure C.15 Mean daily NO_2. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

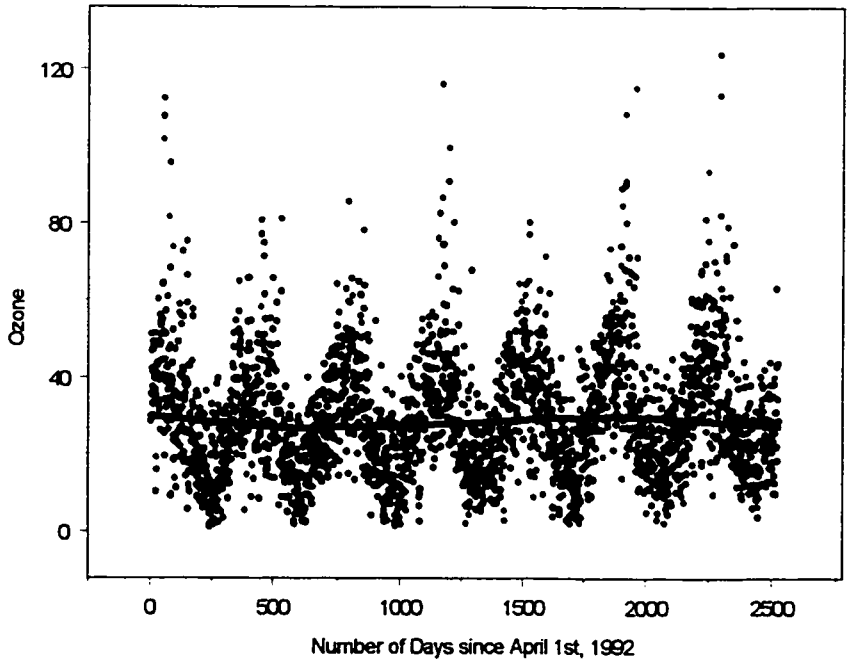

Figure C.16 Mean daily O_3. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

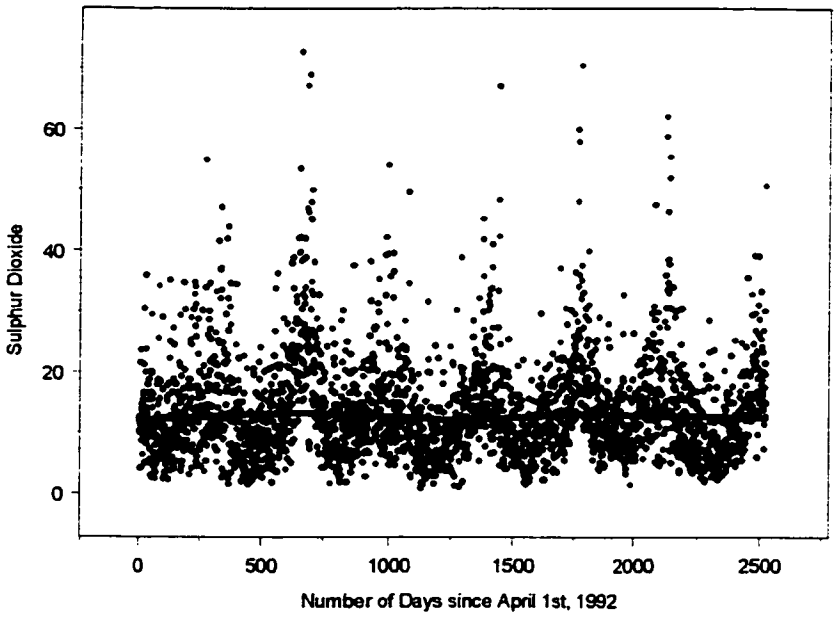

Figure C.17 Mean daily SO_2. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

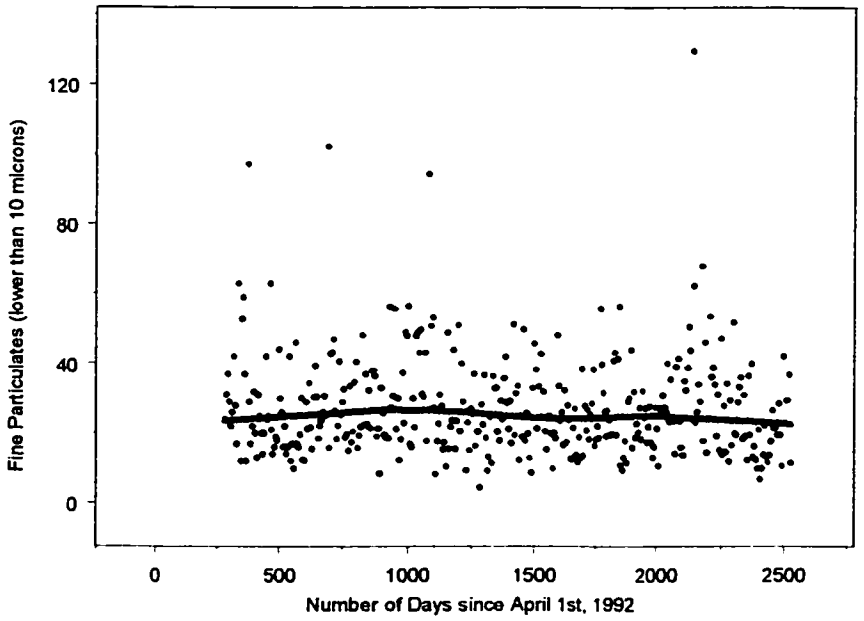

Figure C.18 Mean daily PM_{10}. The solid line is the LOESS smooth representing the long-term trend in the data (span 50%)

APPENDIX D. WEATHER VARIABLES

Table D. 1. Pearson Correlation Coefficient of Weather Variables, Montreal 1992 to 1999

Weather Variables	Pearson Correlation Coefficient
TMEAN	
TMAX	0.99
TMIN	0.99
TMEANCH	0.16
TMAXCH	0.12
TMINCH	0.15
RHMEAN	0.08
PRES08CH	-0.15
TDMEAN	0.97
TDMEANCH	0.15
TMAX	
TMIN	0.95
TMEANCH	0.17
TMAXCH	0.18
TMINCH	0.12
RHMEAN	0.01
PRES08CH	-0.14
TDMEAN	0.95
TDMEANCH	0.15
TMIN	
TMEANCH	0.14
TMAXCH	0.06
TMINCH	0.18
RHMEAN	0.15
PRES08CH	-0.16
TDMEAN	0.97
TDMEANCH	0.14
TMEANCH	
TMAXCH	0.87
TMINCH	0.85
RHMEAN	0.13
PRES08CH	-0.43
TDMEAN	0.16
TDMEANCH	0.85

Table D.1. (continued). Pearson Correlation Coefficient of Weather Variables, Montreal 1992 to 1999

Weather Variables	Pearson Correlation Coefficient
TMAXCH	
TMINCH	0.48
RHMEAN	0.06
PRES08CH	-0.33
TDMEAN	0.12
TDMEANCH	0.69
TMINCH	
RHMEAN	0.17
PRES08CH	-0.42
TDMEAN	0.97
TDMEANCH	0.14
RHMEAN	
PRES08CH	-0.37
TDMEAN	0.27
TDMEANCH	0.33

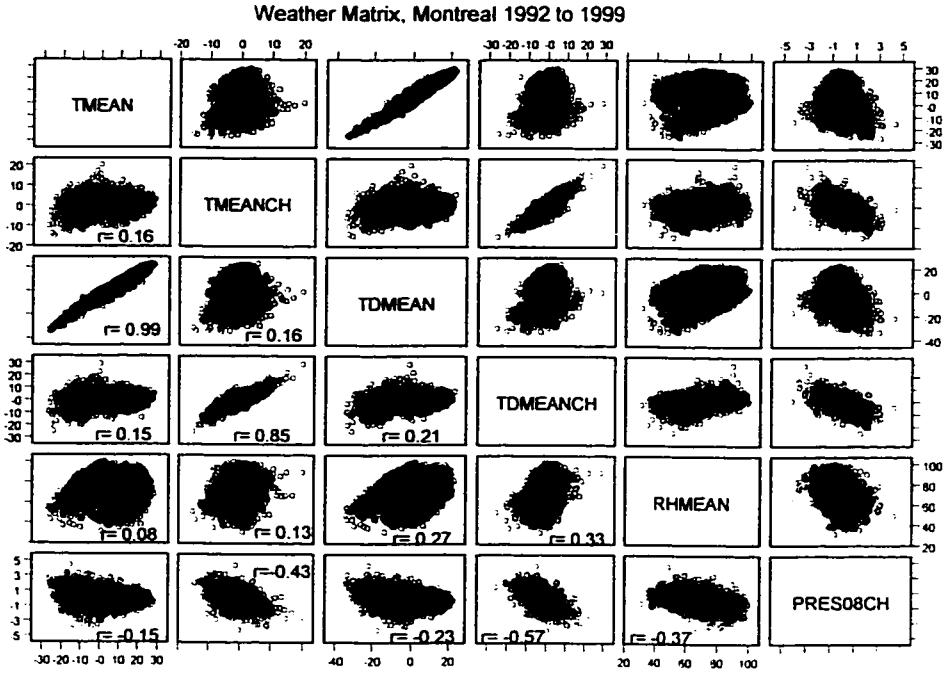

Figure D.2. Scatter-plot Matrix of Selected Weather Variables