Hamilton-Wentworth
Air Quality Initiative

H A Q I

Human Health Risk Assessment for Priority Air Pollutants

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Hamilton-Wentworth Air Quality Initiative
Human Health Work Group

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Executive Summary

In consultation with the coordinating body of the Hamilton-Wentworth Air Quality Initiative and the residents of the Hamilton-Wentworth (H-W), the Human Health Workgroup was assigned three tasks:

(i) in association with The Lung Association of Hamilton-Wentworth and Environment Canada, undertake a "user-friendly" survey of the scientific literature regarding the health effects of air pollution,

(ii) develop a priority "target" list of air pollutants that are found in the Hamilton-Wentworth airshed, and if possible

(iii) undertake the appropriate human health risk assessments on these identified air pollutants.

(i) For the *first* task, a data base of over 4000 recently published scientific papers examining the health effects of air pollution was created and according to a set of specific criteria, a set of over 200 papers most relevant to the H-W situation were obtained. From these papers, six fact sheets in a series called "The Air Pollution Picture" were written to acquaint high school students and members of the public with the basic issues of air pollution and its effects. The first two fact sheets describe the types and sources of the air pollutants of greatest concern; the next three describe the health effects of ozone, sulphur dioxide and sulphates, and PM_{10} (particulate) air pollution; and the sixth provides the scientific literature background details on which the first five were based. The fact sheets are available from the Lung Association, Hamilton-Wentworth.

(ii) For the *second* task, and after a review of many pollutant lists such as those from the United States Environmental Protection Agency, the Canada-Ontario Agreement, Ontario Regulation 346, the National Pollutant Release Inventory and the Smog Plan of Ontario, a priority "target" list together with appropriate health endpoints and groupings was prepared. For each selected pollutant, data had to be applicable and measurable (real-world data), a specialist in the field of risk assessment had to be available to undertake the assignment, and the assessment had to be done within six months. The objective for this second task was to determine which air pollutants were the greatest public concern and which had the greatest potential to cause adverse impacts on human health in H-W (Table I).

a) The first three groups ("The Criteria Pollutants") listed in the priority "target" list contain pollutants for which there are measurable acute or immediate health effects in the population attributable to distinct air quality variables. Measurable health effects can be addressed by applying projections from the epidemiological literature (real data on real people) to the local population. These projections are based on real measurable effects such as hospital admissions and premature death associated with ambient air levels of these pollutants in numerous cities in North and South America and Europe.

b) The fourth group ("The Air Toxics") contains carcinogens and substances with other chronic or long-term health effects and whose impacts can be estimated by risk assessment procedures. These risk assessment procedures provide an estimate of the health risk to the local population according to the concentrations found in ambient air and are based on animal testing or human epidemiology studies.

c) The fifth group ("Odour and Aesthetics") contain two contaminants which are very difficult to quantify with respect to human health effects. This is due to the facts that the odour detection of total reduced sulphur compounds occurs at concentrations at least 100 times lower than that which leads to adverse health effects due to toxicological mechanisms, and for the black particulate fallout of northeast Hamilton, the exact composition and source(s) remain unknown.

Table I - The Priority "Target" List

Contaminant	Comments	Group Identification
PM_{10} and Sulphates ($SO_4^=$)	The Smog Plan elements. Mortality and hospital admissions endpoints.	Group #1
Ground Level Ozone (O_3)		
Sulphur Dioxide (SO_2)	Acute COPD and other respiratory effects.	Group #2
Acid Aerosols		
Carbon Monoxide (CO)	Special interest - cardiovascular effects	Group #3 Groups #1,#2 and #3 are often referred to as the "The Criteria Pollutants"
Nitric Oxide (NO)	Special interest - possible mortalities and hospital admissions endpoints	
Nitrogen Dioxide (NO_2)		
Cadmium	Chronic health effects and cancer endpoints	Group #4 "The Air Toxics"
Hexavalent Chromium		
Manganese		
Lead		
Benzene		
1.3-Butadiene		
Benzo[a]pyrene		
Black Particulate Fallout	Health effects very difficult to quantify	Group #5 "Odour and Aesthetics"
Total Reduced Sulphurs		

iii) For the *third* task, experts from MOEE and McMaster University have taken the local air monitoring data and using the results of the best studies carried out in other areas, have estimated the health impacts for these pollutants to be expected in H-W. The pollutants have been prioritized (Table II) according to their health importance in H-W together with the estimates of their risk including hospitalization and mortality in the community.

General Health Impacts:

For the "Criteria Pollutants"

Inhalable particulate (PM_{10}) has been linked to increased mortality. This association is found in metropolitan areas in North America including Toronto, Detroit and Los Angeles. Studies show that an increase of 10 $\mu g/m^3$ could increase the total mortality rate by 1 percent. Certain groups, such as children and the elderly, seem to be more sensitive to this problem.

There is considerable discussion currently on the issue of what are the important inhalable particulate species and what is their biological mechanism of action. Some animal and clinical exposure studies have pointed towards sulphates ($SO_4^=$) as the most important species on the basis of its association mainly with the smaller respirable fraction and its chemical reactivity

Admissions to hospitals for respiratory conditions such as asthma and bronchitis tend to increase during periods of elevated sulphur dioxide (SO_2) and/or sulphate concentrations. Sulphur dioxide was identified as one of the pollutants responsible for inducing premature mortality in susceptible persons in the London, England smog episodes in 1952. Sulphur dioxide can react in the atmosphere to form sulphate particles. More recent studies have shown increased mortality and hospitalization, especially in people with cardio-respiratory diseases, with increases in sulphate level.

Ozone (O_3) is particularly harmful to people with respiratory conditions such as asthma or chronic bronchitis. Studies have linked increases in ozone levels with increases in cardio-respiratory diseases. An increase of 10 ppb in ozone concentration could increase the respiratory hospital admission rate by 0.9 percent. Ozone also reduces lung function, even at concentration levels equal to the one-hour Ontario ambient air quality criterion of 80 ppb.

Carbon monoxide (CO) has long been recognized as a pollutant with adverse health effects, and in high concentrations, is lethal. Recent papers have also related exposure to low concentrations of CO with hospitalization for congestive heart failure in patients over 65 years of age.

There have been many studies on the effects of nitrogen oxides (NO and NO_2) on health; particularly on respiratory symptoms or pulmonary function in children. However, there have been few studies on hospital admissions or mortality, except for an European study of 15 European cities in 10 countries.

For the "Air Toxics"

Cadmium, hexavalent chromium, benzene, benzo[a]pyrene and 1,3-butadiene are considered to be carcinogens. Cadmium can also induce damage to the kidney. Lead and manganese have adverse effects on the nervous system. The emphasis of the toxics health assessment is primarily on cancer outcomes. Only the risk from inhalation was estimated as part of the HAQI, and therefore, the assessment may underestimate the risk from all routes of exposure for these contaminants.

For the "Odour and Aesthetics"

Odour detection is considered to be the critical effect of total reduced sulphur compounds whereas a health effect assessment for the black particulate fallout material is very difficult to determine due to its inconsistent composition, spatial and temporal distributions, and its unknown source(s).

Specific Results:

The health effect results, as summarized in Table II, demonstrate a substantial burden of illness and premature deaths associated with air pollution in Hamilton-Wentworth. The estimated health effects for the air pollutants have been expressed as the number of premature mortalities, hospital admissions and excess cancer cases. As noted earlier, air pollutants such as inhalable/respirable particulates and sulphates, ozone, sulphur dioxide, carbon monoxide and nitrogen oxides are associated with hospital admissions and premature death. Air toxics are associated with chronic or long-term effects such as cancer.

Premature mortalities are estimates of how many people die per year in Hamilton-Wentworth due to air pollution. The Human Health Workgroup recognized that there are varying levels of scientific consensus and therefore confidence in the estimates. The confidence levels indicated in Table II reflect the strength of association between the health effects and the ambient concentrations for each pollutant.

The estimated number of premature mortalities range from 90 to as high as 321 (the latter value not shown in Table II). Estimates with high and medium confidence are shown in the 'premature mortality' columns of this table. There is a high level of confidence for the estimate of 90 premature mortalities whereas there is a medium level of confidence for the estimate of 214 premature mortalities and a low level of confidence for the estimate of 321 premature mortalities. The strongest evidence for health effects is found in the associations between particulate pollution (specifically inhalable particulates, and sulphates which are mainly found in the respirable particle fraction) and premature mortality. In comparison, Ontario's Smog Plan (IP/RP Workgroup, 1996) has estimated a total of approximately 1,800 premature mortalities and 1,400 hospital admissions per year in all of Ontario are primarily due to the effects of inhalable particulates alone.

The hospital admissions estimates show how many people per year in Hamilton-Wentworth are admitted to hospital for cardiac and respiratory disease associated with air pollution. This is shown

in the 'hospital admissions' columns of Table II. The high confidence level estimate is approximately 300 hospital admissions per year due to air pollution.

Table II - Human Health Impacts in Hamilton-Wentworth of the Priority Air Pollutants

Pollutant	Estimated Premature Mortalities (incidences/year)		Estimated Hospital Admissions (incidences/year)		Estimated Excess Cancer Cases (Incidences/year)
	Confidence Level		Confidence Level		Average Case
	High	Med	High	Med	
Inhalable Particulate Matter (PM_{10}) and Sulphates	85 (51)*	92 (163)*	145 (193)*		na na
Ozone	5	11	40		na
Sulphur Dioxide		40	28		na
Nitrogen Oxides			25		na
Carbon Monoxide			15		na
"Air Toxics"	na	na	na	na	<1
TRS and Black Particulate Fallout	na	na	na	na	na
Total	90[a]	214[b]	301		<1

* sulphate is part of particulate matter
a using the higher of the PM_{10} and sulphate estimate (i.e., 85)
b using the higher of the PM_{10} and sulphate estimate (i.e., 163)
na not applicable/available

As noted in Table II, inhalable particulates and especially their sulphate component have the greatest impact on air pollution related premature mortalities and hospital admissions in Hamilton-Wentworth.

Air pollution related premature mortality, based on the medium confidence estimates, is attributed to the following pollutants:

- 76% to sulphates (or 64% when considered as inhalable particulate); and 24% to sulphur dioxide and ozone.

Air pollution related hospital admissions are attributed to the following pollutants:

- 64% to sulphates (or 57% when considered as inhalable particulate); and 23% to sulphur dioxide and ozone.

It should be recognized that these serious health outcomes of hospital admissions and death are just the tip of the iceberg and that these pollutants lead to a number of other morbidity effects (e.g., adult chronic bronchitis, hospital emergency room visits, asthma symptom days, restricted activity days, acute respiratory symptom days, bronchitis in children), which were not quantified in this analysis but which can affect a much larger number of people.

The health effects of "Air Toxics" can be addressed in two ways. Most of these substances are carcinogens and therefore the estimated impact has been expressed as the number of 'excess cancer cases' expected per year in Hamilton-Wentworth. Inhalation of these air pollutants at average levels is estimated to result in less than one additional cancer case per year as indicated in the 'excess cancer cases' column of Table II.

Overall, inhalation exposures to the selected cancer-causing compounds (cadmium, hexavalent chromium, benzene, benzo[a]pyrene and 1,3-butadiene) are estimated to result in less than one percent of the expected annual incidence of all lung cancers based on 1994 Ontario cancer statistics. For the 1994 Hamilton-Wentworth population (467,900 in 1991), inhalation of these air pollutants could mean an average of 0.7 additional cancer cases (90^{th} percentile - 2 cases) per year or approximately 0.05% of the cancer incidences.

When considering the noncarcinogenic health effects of the "air toxics" (cadmium, lead and manganese), it is felt that the current airborne concentration levels of manganese suggests further study. Although manganese ambient air levels do not exceed the current Ontario Ministry of Environment and Energy (MOEE) limits, more stringent air quality criteria for this compound in other jurisdictions suggest that this MOEE limit should be re-examined.

The incidence of cancer due to "Air Toxics" is quite low in comparison to the incidence of premature mortality of the "Criteria Pollutants".

Air pollution is estimated to be responsible for close to 1% of all non-trauma hospitalizations in Hamilton-Wentworth and up to 9% of all non-trauma mortalities. Fine particulate pollution (and especially its sulphate component) represents the greatest air pollution problem in Hamilton-Wentworth being responsible for nearly half of the air pollution related hospitalizations and premature mortalities.

The major concern associated with total reduced sulphur compounds in air is considered to be odour detection which occurs in the range of 1-5 ppb. This range is at least 100 times lower than concentrations that lead to adverse health effects due to toxicological mechanisms.

The inhalable particulate (i.e. the PM_{10} fraction) and the benzo[a]pyrene (B[a]P) components of the black fallout in northeast Hamilton are probably the most important elements with respect to human health. Although no statistical difference has been determined between the airborne concentrations of PM_{10} measured in northeast Hamilton and that measured in downtown Hamilton, it is realized that since B[a]P is adsorbed onto particulates, the reduction of any particulate emissions from the many combustion sources in the industrial sector of Hamilton should also help reduce airborne B[a]P concentrations. In fact, this is the current abatement strategy being followed by the Ministry. The health risk associated with the black particulate fallout remains very difficult to assess since an agreement as to what exactly constitutes this material has yet to be established.

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PART ONE: A "User-Friendly Literature Review" of the health effects of air pollution

1.1 Objective

The objective of the first task of the Human Health Working Group (HHWG) of HAQI was to produce a "user-friendly" survey of the scientific literature regarding the health effects of air pollution. Two summer research assistants were engaged to carry out this work: a "Scientific Literature Assistant" (SLA; J.P. Knox) and a "Lay Literature Assistant" (LLA; Jenny Cooper). Both were supervised by the Chair of the HAQI HHWG. The task of the SLA was to assemble a database of recent literature with respect to the effects of air pollution on health, to develop a short list of the most relevant recent literature and from this, to assemble the basic facts from which the final product would be written. The task of the LLA was to obtain as much lay information on the same topic as was reasonably accessible, to review it, and to make judgements about its communications effectiveness. The results of this survey would then be available to establish design principles for the development of the HAQI "Fact Sheets".

After discussion between the HHWG Chair and the two research assistants, it was decided that the user-friendly review should consist primarily of the series of brochures or "Fact Sheets" and if time permitted, a more detailed monograph. The Fact Sheets were designed to complement a static display recently completed by The Lung Association Hamilton-Wentworth through funding from Health Canada. The static display was designed to be accessible to high school students or community groups, and to stimulate interest in air pollution issues. Those attracted to the display would then be able to obtain the brochures, or "Fact Sheets" which offer quick, basic information on specific topics. More detailed information could then be provided by the agencies participating in the Hamilton-Wentworth Air Quality Initiative.

1.2 Scientific Literature Review

The task for the SLA was subdivided into five subgoals:

1. The development of a basic understanding of the principles of air pollution science and the related health effects.
2. The compilation of a database of bibliographies and abstracts.
3. The generation of a list of articles to be read, using specified criteria and the database.
4. Summarization of the articles, creating a list of facts to be used in the finished product.
5. Developing, designing and creating the finished set of 6 "Fact Sheets" in collaboration with the research assistant who had appraised the lay literature.

In order to develop understanding of the current state of air pollution knowledge, the SLA consulted several reference books and read review articles of the scientific literature. The most useful information was derived from an extensive reading of the review literature.

The data base was obtained from larger data bases such as "Medline" using the key words "air pollution" and "health", and downloaded into the "Reference Manager" system: it contains 4472 references, covering the period 1928-1996. There were 1595 references from 1977-1986, and 2536 references from 1987-1996. From this data base, 205 papers were selected on the basis of title and/or abstract for retrieval of the whole article from serials in the McMaster and University of Toronto libraries, or by interlibrary loan. These were subsequently reviewed and further selections were made by formal criteria including study design, author, and suitability of outcome measures to support the expression of a dose-response relationship.

Articles were selected which were thought to be particnlarly useful. In doing so, two strategies were followed: First, several authors were selected whose names appeared frequently in citations of air pollution research. Copies of their articles were ohtained from library sources and compiled into books; one for each author. Secondly, the database was analysed year by year for recent years, starting with the present, and articles were selected using keywords which in part focussed on general themes, such as "suspended particles" or "epidemiological studies", and in part examined issues which were more specific, such as "children's health". These selections were gathered into books, one for each year from 1996 to 1993.

The books were then summarized into a brief table, (Table 1) noting the variables, manipulations and results. References from which this table was prepared are given at the end of Part 1. This table represents the main source for the facts used to generate the end product. It should be noted that the second set of books, which are selections from each year, is not necessarily representative of all of the literature produced in that year. Since these selections were chosen because they added eclectic pieces of information, papers on more obscure topics are over-represented.

The books of articles served as the main source of scientific information for the *user-friendly review*. Additional information was provided by the LLA, who obtained information on such topics as sources and regulations regarding air pollution.

The set of 10 books comprising over 200 articles are available in the Urban Air Environment Group Laboratory, Room 3E27, Health Sciences Centre, McMaster University.

Master List of Studies, Populations, Pollutants and Effects

Table 1a

Study	Pollutant # 1	Pollutant #2	Pollutant #3	Level of Pollution	Population	Location
Dockery 3	ozone			high	Exercising children with respiratory symptoms	Mexico City
Dockery 4	H^+	PM_{10}	ozone	ambient	Children partitioned via questionnaire	Pennsylvania
Dockery 6	TSP	sulfur dioxide		high	hospital outpatient visitors	Beijing
Dockery 10	TSP	sulfur dioxide		high	all included	residential Beijing
Dockery 11	$PM_{2.5}$	sulfates	TSP, H^+, SO_2, NO_2	ambient	adults observed longitudinally over 14-16 yrs.	six US cities
Dockery 13	sulfur dioxide			high	all included	Erfurt, Germany
Dockery 14	ozone			high	children	Mexico City
Dockery 15	PM_{10}	$PM_{2.5}$	sulfates, H^+	ambient	all included	St. Louis & Tenn
Dockery 19	TSP	sulfur dioxide		high	adult never smokers (40 - 69 yrs.)	Beijing
Pope 3	sulfates	$PM_{2.5}$		ambient	adults	US
Pope 4	PM_{10}			ambient	elderly	Sao Paulo
Pope 5	PM_{10}			ambient	smokers with mild to moderate COPD	Salt Lake City
Pope 6	PM_{10}			ambient	all included	Utah Valley
Pope 7	PM_{10}			high	symptomatic & asymptomatic children	Utah Valley
Pope 8	PM_{10}			high	all included	Utah Valley
Pope 9	PM_{10}			high	all included	Utah Valley
Brunekreef 2	ozone	sulfur dioxide	NO_2, PM_{10}	ambient	children	the Netherlands

Table 1a ctd.

Study	Effect #1	Significance	Effect #2	Significance	Effect #3	Significance
Dockery 3	spirometry	no effect				
Dockery 4	H^+ and spirometry	significant in symptomatic children only	PM_{10} & spirometry	significant in symptomatic children only	ozone and spirometry	weak effect
Dockery 6	TSP & # of visits	significant (strongest in summer)	SO_2 & # of visits	significant (strongest in summer)		
Dockery 10	TSP & mortality	weakly positive effect	SO_2 & mortality	strong effect		
Dockery 11	$PM_{2.5}$ & sulfate and mortality	strong effect	TSP & mortality	weak effect	H^+ or SO_2 or NO_2 & mortality	weaker effect
Dockery 13	SO_2 & mortality	weak effect				
Dockery 14	ozone & spirometry	significant effect				
Dockery 15	PM_{10} & mortality	weakly significant	$PM_{2.5}$ & mortality	weak	sulfate & H^+, & mortality	weaker
Dockery 19	TSP or SO_2 & spirometry	weak effect				
Pope 3	sulfate & pulmonary mortality	significant	$PM_{2.5}$ & pulmonary mortality	significant		
Pope 4	PM_{10} & mortality	strongly significant				
Pope 5	PM_{10} & spirometry	weak effect				
Pope 6	PM_{10} & mortality	strong effect				
Pope 7	PM_{10} & spirometry	significant, stronger in symptomatic kids	PM_{10} & incidence	no effect		
Pope 8	PM_{10} & respiratory admissions	significant effect, but no measurement of confounding pollutants				
Pope 9	PM_{10} & respiratory admissions	strong effect, particularly in children	PM_{10} & monthly admissions, 1 month lag	strong effect, implies cumulative effect		
Brunekreef 2	O_3, SO_2, NO_2, PM_{10} & incidence	no effect				

Table 1b

Study	Pollutant # 1	Pollutant #2	Pollutant #3	Level of Pollution	Population	Location
Brunekreef 3	ozone			ambient	adult amateur cyclists, implies high ventilation	the Netherlands
Brunekreef 4	sulfur dioxide	nitrogen dioxide		ambient	children	the Netherlands
Brunekreef 5	ozone			high	children	the Netherlands
Brunekreef 6	ozone	nitrogen dioxide	PM_{10}, H^+,	ambient	children	the Netherlands
Brunekreef 7	sulfur dioxide	nitrogen dioxide	PM_{10}	ambient	children with chronic respiratory symptoms	the Netherlands
Brunekreef 9	TSP	ozone		high	children	the Netherlands
1993-1	TSP	ozone	sulfur dioxide	ambient	adult, non-smoking 7th Day Adventists	California
1993-6	nitrogen dioxide			ambient	children	Italy
1993-13	ozone	sulfates	coefficient of haze	ambient	adults	California
1993-16	sulfur dioxide	black smoke		ambient	all included	Barcelona
1993-18	VOCs				children	West Virginia
Bates 4	ozone	acid aerosols	$SO_4^=$, $PM_{2.5 \& 10}$, TSP	ambient	all included	Ontario
Bates 8	ozone	sulfates		ambient	all included	Ontario
1995-1	PM_{10}			ambient	non-smoking 7th Day Adventists	California
1995-5	PM_{10}	air Fe			adults	the Netherlands
1995-11	air pollution			high	children	Poland
1995-13	air pollution			ambient	adults	Denmark

Table 1b ctd.

Study	Effect	Significance	Effect #2	Significance	Effect #3	Significance
Brunekreef 3	O_3 & spirometry	small but significant effect				
Brunekreef 4	SO_2 & spirometry	no effect	NO_2 & spirometry	small bu significant effect		
Brunekreef 5	O_3 & spirometry	significant effect	O_3 & symptomatic, & spirometry	no effect		
Brunekreef 6	NO_2 & spirometry	no effect	O_3 &PM_{10} & H^+, & spirometry	significant effect	incidence & spirometry	no effect
Brunekreef 7	SO_2 & PM_{10}, &spirometry	small but significant effect	NO_2 & spirometry	no effect	SO_2 & PM_{10}, & wheeze	small effect
Brunekreef 9	TSP & Ozone, & spirometry	response variability did not did not change, implies no sensitive subgroups				
1993-1	symptoms & TSP	significant effect	symptoms & O_3	significant effect	symptoms & SO_2	no effect
1993-6	NO_2 & spirometry	significant effect				
1993-13	O_3 & symptoms	significant	sulfates & symptoms	significant	CoH & symptoms	no effect
1993-16	SO_2 & admissions	significant winter and summer	BS & admissions	significant in winter, weaker in summer		
1993-18	VOCs & incidence	significant	VOCs % asthma	significant		
Bates 4	O_3 & admissions	significant, strongest	H^+ & admissions	significant	$SO_4^=$,$PM_{2.5}$,PM_{10},TSP & admissions	weakly positive
Bates 8	O_3 & summer admissions	significant	sulfates & summer admissions	significant	$SO_4^=$, O_3 & winter admissions	not significant
1995-1	PM_{10} & symptoms	significant				
1995-5	PM_{10} & spirometry	significant	air Fe & spirometry	no effect		
1995-11	air pollution & spirometry	significant				
1995-13	air pollution & prevalence	significant				

Table 1c

Study	POLLUTANT # 1	Pollutant #2	Pollutant #3	Level of Pollution	Population	Location
1994-3	ozone	sulfates		ambient	all included effected infants>adults>elderly	Ontario
1994-5	ozone			ambient	all included	Montreal
1994-7	air pollution			ambient	children	the Netherlands
1994-19	sulfur dioxide	nitrogen dioxide		ambient	all included	Helsinki
1994-20	ozone	sulfates		ambient	children	rural Canada
1996-1	ozone	black smoke	SO_2, NO_2	ambient	all included	London, UK
1996-2	sulfur dioxide	TSP		high	all included	Slovakia
1996-5	ozone	sulfur dioxide		high	children	London, UK
1996-6	ozone	PM_{10}	SO_2	ambient	all included	Paris, France
1996-7	ozone			ambient	all included	London, UK
1996-20	ozone	sulfur dioxide	NO_2, TSP	ambient	all included	Helsinki
1996-23	ozone	sulfur dioxide	NO_2, TSP	ambient	all included	the Netherlands
1996-25	sulfur dioxide	TSP	nitrogen dioxide	high	all included	Cologne
1996-26	black smoke	sulfur dioxide	O_3, NO_2	ambient	all included	Barcelona
1996-27	sulfur dioxide	black smoke	carbon monoxide	ambient	all included	Athens
1996-29	sulfur dioxide	TSP		ambient	all included	Milan
1996-31	nitrogen dioxide	ozone	SO_2, PM_{10}	ambient	all included	Lyon

Table 1c ctd.

Study	Effect #1	Significance	Effect #2	Significance	Effect #3	Significance
1994-3	O_3 & respiratory admissions	significant	$SO_4^=$ & respiratory admissions	significant but weaker		
1994-5	O_3 respiratory admissions	strong, but fades to insignificance when coregressed with temp				
1994-7	air pollution & Bronchial Resp.	significant, seems independent of atopics, asthmatic & spirometric factors				
1994-19	SO_2 & respiratory admissions	significant	NO_2 & respiratory admissions	significant		
1994-20	O_3 & spirometry	significant	$SO_4^=$ & spirometry	significant		
1996-1	O_3 & all admissions	significant	BS & all admissions	significant	SO_2, NO_2 & all admissions	significant, weaker
1996-2	SO_2 & all admissions	not significant	TSP & all admissions	not significant		
1996-5	O_3 & wheeze admissions	significant	SO_2 & wheeze admissions	significant, weaker		
1996-6	O_3 & respiratory admissions	no effect	SO_2 & respiratory admissions	significant	PM_{10} & respiratory admissions	significant
1996-7	O_3 & respiratory admissions	significant				
1996-20	O_3 & asthma admissions	significant in children < 15 years old	SO_2 & asthma admissions	significant in those > 15 years old	TSP & NO_2, & asthma admissions	weakly significant
1996-23	O_3 & respiratory admissions	weakly positive	SO_2 & respiratory admissions	no effect	NO_2 & respiratory admissions	weakly positive
1996-25	SO_2 & mortality	significant	TSP & mortality	weakly significant	NO_2 & mortality	no effect
1996-26	BS & total mortality	significant	SO_2 & total mortality	significant	NO_2 & O_3, & elderly mortality	significant
1996-27	SO_2 & total mortality	significant, winter effect present	BS & mortality	significant	CO & mortality	significant
1996-29	SO_2 & resp. adms. & total deaths	significant	TSP & resp. adms. & total deaths	significant		
1996-31	NO_2 & any cause of death	no effect	O_3 & any cause of death	no effect	SO_2 or PM_{10} & respiratory deaths	weakly positive

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1.3 Lay Literature Review

1.3.1 Data Collection

In most cases, literature was collected by contacting government agencies, environmental groups, health agencies and large industries or industrial associations. Personal visits were made to the Ministry of Environment and Energy office in Hamilton, and to the Hamilton Public Library. Some information was also obtained from the Internet.

1.3.2 Evaluation Criteria

The publications received were evaluated on several measures. In order to receive a "good grade", the publication must be easy to obtain from the distributing organization; be clear and easy to understand; contain a thorough discussion of the topic (the sources of air pollution, effects on health, methods of prevention and /or avoidance, information on data collection and/or monitoring techniques); include a contact for more information; discuss the Air Quality Index (AQI) and/or other government initiatives to improve air quality and educate the public; emphasize the importance of good air quality; and mention the future goals in the field of research and regulations of air pollutants and the associated health effects.

These standards were derived from a set of questionnaires distributed to schools, a Lung Association self-help patient group (Broncho-Busters) and a community group. All collected material was evaluated according to these measures. Information received that was irrelevant to the topic of air pollution was not included.

Although the publications were evaluated on several aspects, the most important one was the extent of their description of the health effects associated with air pollution. In general, those publications graded in the "A" range contain detailed information about the health effects of air pollution and may be considered an excellent resource; those in the "B" range contain brief discussions of the health effects of air pollution and are useful sources of general information; those in the "C" range have minimal information on health effects but may contain other interesting material; and those in the "D" range contain no information on the health effects associated with air pollution and are of very little use for our purposes.

1.3.3. Results of the Evaluation, by Source

In general, the information received from industries rarely mentioned anything about the health effects of their air pollution. The focus for these groups was the amount of money that they have put into environmental controls and the reductions in their emissions as a result. However the Lambton Industrial Society (an association of several industries in the Sarnia area committed to improving environmental quality) had an excellent collection of information when the entire package was viewed together.

The Region of Hamilton-Wentworth had only limited amounts of information. A State of the Environment Report was received, but it had not been possible to obtain any other information.

The MOEE publications were very technical but were absolutely necessary to obtain detailed information on the levels of the measured pollutants in the province. The LLA contacted the MOEE Public Information Line 3 times to request a publications catalogue, and finally received a notice that it was on back order. It has not yet been received.

There was a wide range in the quality of Environment Canada and Health Canada publications, and calls often had to be made more than once to receive information. One publication was requested twice from Health Canada, and was never received. We found that a copy was available in the lab files, and used it instead.

The lay literature at the Hamilton Public Library was very outdated: no useful material was found there. Publications received from environmental agencies such as Greenpeace were very dramatic and interesting but it was hard to tell if the data were accurate (it appeared that their publications were a bit biased in order for them to make their point). They do make for good reading and when conducting a literature search it is useful to have their point of view. The Lung Association has produced some interesting publications, although many of them do not provide enough detailed information. It was very surprising that the 2 asthma organizations contacted did not have any information on air pollution. People with asthma need to know about the potential risk of air pollution exposure.

The Friends of the Earth Foundation did not return calls although messages were left on 3 different occasions. Overall, the LLA was impressed with the amount of literature available if the researcher is willing to take the time and effort to look for it. Most people that were dealt with were very friendly and helpful, often directing the LLA to other sonrces if they did not have any information to give. In most cases publications were received within a week of the request. The literature search was a very helpful in assisting us in the creation of our own material. We have been able to combine the good features from several documents into a (hopefully) very useful series of fact sheets.

Table 1d - Scores of Lay Literature Reviewed by Source

Grade	Total	Industry	Government	*NGOs
A	13	0	5	8
B	25	5	14	6
C	16	6	9	1
D	36	22	11	3

*NGOs - Non-Governmental Organizations

1.4. Development of Fact Sheets

In August of 1996, the SLA and the LLA worked together to produce the first drafts of the fact sheets. There were several guiding principles inherent in their design:

1. The overall framework was the static display developed by The Lung Association, Hamilton-Wentworth in contract with Health Canada: the display was called "The Air Pollution Picture", was in cartoon form, and was designed to appeal to senior high school students. The "Fact Sheets" were intended to complement this display.

2. There was to be a "set" of Fact Sheets, so that each one could cover a topic in some depth. However there had to be a common design theme among the set serving as a linkage between the Fact Sheets.

3. The set was designed to be "open", so that additional topics could be added at a later date.

4. There were to be 3 content themes: Health Effects, Sources and Weather Effects. The Health Effects were focussed on 3 pollutants: ground-level ozone, particles, and sulphur dioxide and sulphates.

5. There would be no detailed reference citations in the theme areas; these would be consolidated in a single "Further Readings" Fact Sheet.

6. The graphic design used the gatefolds of the sheets to deliver the basic message first, and to expand on it as the piece was opened. On the back of each piece there would be indications of current government action, pathways through which the individual can act, and the date and source of the Fact Sheet.

First drafts of the Fact Sheets were completed by the research assistants by the end of Augnst. Some revisions to these drafts were then made, and first presented to the Lung Association Air Pollution Community Action Program Committee (APCAP) in mid-September. These drafts were subsequently provided to a grade 11 high school teacher who is also a Lung Association volunteer. The teacher then used her class to assess the Fact Sheets for readability and communications effectiveness. In general, the students found them to be understandable, but they had a number of useful comments for change which were incorporated in the next draft. One of the most substantive changes was to replace X-Y or "line" charts (e.g.,hospitalization vs ozone) with "bar" charts, which they found much more understandable. There were also some wording changes recommended by the students. Following these changes, the sheets were reviewed and re-drafted on a total of three more occasions.

Consultation on the final drafts of the Fact Sheets was carried out with a joint meeting of the APCAP committee of The Lung Association, and a subcommittee of the Human Health Working Group of

HAQI. Some useful suggestions for change were provided by the subcommittee, and the final product was approved for printing in November, 1996. The set of Fact Sheets has been completed and has had an initial printing of 1000 copies. They are available from The Lung Association, Hamilton-Wentworth.

PART TWO: Development of Priority "Target" List of Pollutants

2.1 Background

Over the years, our environment has been subjected to literally thousands of natural (biogenic) and manmade (anthropogenic) chemicals. Currently there are an estimated 40 to 60,000 chemicals in everyday use and according to a recent United States Environmental Protection Agency statement, 1,000 new chemicals are still being made (or found) each month. The *found* chemicals refer to those chemicals that are now being brought to light by much improved analytical instruments and methodologies, and by mankind's better understanding of the environment and the interaction of its many elements. As examples, consider the advances in medicine and the chemical understanding (modelling) of the "Ozone Hole".

Although the exact number is unknown, these chemicals may be classified or sorted by many various schemes and plans depending on the user. As an example, some may be considered as being toxic and some are not; some are long-lived, some are not; some are bio-accumulative, some are not; some are volatile (a gas), some are not (a particle); some are carcinogenic, some are not; some are mutagenic, some are not; some are beneficial, some are not; some are manmade, some are not; some are ..... To put this in perspective, chemists and other practitioners (environmental, human health, sociol-scientists, urban planners, etc.) have prepared numerous lists for their own use. As an example, a 1968 Encyclopaedia of Chemicals and Drugs (the Eighth Edition of the MERCK Index) includes uearly 10,000 descriptions of individual substances, more than 4,500 structural formulas and about 42,000 names. The World Health Organization (WHO) and the International Association for Research in Cancer (IARC) have identified over 4,700 airborne mainstream environmental tobacco smoke components and at least 43 are known human carcinogens. The Ontario Regulations 346 and 337 contain lists of over 300 contaminants for which point-of-impingement (POI) standards are codified or maximum desirable ambient air quality criteria (AAQC) are identified respectively - with endpoints of human health, enjoyment of property, odour, ecosystem health, particulates, etc..

2.2 Rationale

Of these many compounds, those that appear on the lists from the United States Environmental Protection Agency (such as the Hazardous Air Pollutants - HAPs (189 chemicals)), the Canada-Ontario Agreement (COA), the Ontario Regulation 346, the National Pollutant Release Inventory (NPRI) and the Ontario Smog Plan were deemed most appropriate as the starting point for our investigation into the human health effects from exposure to airborne contaminants in Hamilton-Wentworth. For a health risk assessment to be done for airborne contaminants (pollutants /

chemicals) in Hamilton-Wentworth, the assessment must be done on real data. To this end, the MOEE, the academia and the many environmental groups within Hamilton-Wentworth have participated in numerous air quality studies conducted over the years in this area. As an example in 1994, the MOEE TAGA (a mobile Trace Atmospheric Gas Analyzer unit - a mobile tandem mass spectrometer) conducted a one-week summer study in the vicinity of Columbian Chemicals. This is the Ministry's most sophisticated and most sensitive mobile analytical laboratory. Their findings indicated that eighteen different volatile organic compounds were detected in measurable amounts downwind of Columbian Chemicals, but none at concentrations that were in excess of the MOEE Standards. Furthermore, the airshed characteristics were typical of emissions from a heavy industrial area.

2.3 Criteria for Selection

Since the Human Health Risk Assessment wonld be a major component of the HAQI report and timing would be important, three selection criteria were established for preparing the list of priority pollutants. First, the data have to be applicable and measurable (real world) data; secondly, a specialist in the field of risk assessment must be willing to undertake the assignment; and thirdly, the assessment must be done within 6 months.

2.4 Results of Selection (Definition of Groups #1, #2, #3, #4 and #5)

Using the aforementioned information and listening to the concerns and experiences of the public and industry within Hamilton-Wentworth, the compounds or groups of compounds as shown in Table 2.1 were selected for detailed human health risk assessments. For the particulate matter, sulphates, ozone, sulphur dioxide and acid aerosols, the human health endpoints were mortality, morbidity, hospital admissions, acute chronic obstructive pulmonary disease (COPD) and other respiratory effects. For carbon monoxide, the main endpoint was cardiovascnlar effects. For the oxides of nitrogen (nitric oxide and nitrogen dioxide), the main endpoints were possible mortalities and hospital admissions. For the "air toxics": cadmium, hexavalent chromium, manganese, lead, benzene, 1,3-butadiene and benzo[a]pyrene, the endpoints were chronic health effects (morbidity) and cancer. Although black particulate fallout and total reduced sulphur compounds were to be handled by the aesthetics and odour working group as their endpoints of concern were soiling, property damage and enjoyment of property, these contaminants were also be included in this list as they were a health concern of the public.

Table 2.1. Priority "Target" List of Pollutants

Contaminant	Comments	Group Identification
PM_{10} and Sulphates ($SO_4^=$)	The Smog Plan elements. Mortality and hospital admissions endpoints.	Group #1
Ground Level Ozone (O_3)		
Sulphur Dioxide (SO_2)	Acute COPD and other respiratory effects.	Group #2
Acid Aerosols		
Carbon Monoxide (CO)	- Special interest - cardiovascular effects	Group #3 Groups #1,#2 and #3 are often referred to as the "The Criteria Pollutants"
Nitric Oxide (NO)	- Special interest - possible mortalities and hospital admissions endpoints	
Nitrogen Dioxide (NO_2)		
Cadmium	Chronic health effects and cancer endpoints	Group #4 "The Air Toxics"
Hexavalent Chromium		
Manganese		
Lead		
Benzene		
1.3-Butadiene		
Benzo[a]pyrene		
Black Particulate Fallout	Recommendations from the aesthetics group	Group #5 "Odour and Aesthetics"
Total Reduced Sulphurs		

PART THREE: Estimate of Adverse Health Outcomes in Hamilton-Wentworth Associated with Ambient Air Pollution.

3.1 Background

The Region of Hamilton-Wentworth and, in particular, the City of Hamilton have recognized and examined their air pollution problems for over 40 years. Several studies of both adults and children have identified negative health impacts of air pollution in Hamilton during this period. However, because of the design of these studies, it has been difficult to estimate the population burden of illness and death, and as a consequence, some indication of the economic cost associated with this burden.

Information on the burden of illness might be obtained by direct measurement in Hamilton (a "Health Study"), but there are many drawbacks to this approach. The most important of these are: the potential for lack of statistical power to show a significant and clinically important outcome; the cost of a large enough study to have the required statistical power; the time required to execute such a study; and the potential need for strengthening the resources for pollution monitoring in order to obtain adequate exposure information for a study.

Hamilton-Wentworth is fortunate to have a good historical record (for nearly 20 years) of the concentration levels of many common air pollutants at several sites in the Region and to have this maintained at a high level of quality by the West Central Region of the Ontario Ministry of Environment and Energy (WCR-MOEE). In addition, there is additional information available about other pollutants of more recent concern, such as "air toxics" and fine particles.

Table 3.1 gives the population data of the various municipalities within the Hamilton-Wentworth Region according to the 1991 census. Data are from the "Health Action Task Force Report, March 1996" of the H-W District Health Council.

Table 3.1 Population of Hamilton-Wentworth Region by Municipality*

Municipality	Population
Ancaster	21,630
Dundas	21,600
Flamborough	29,245
Glanbrook	9,465
Hamilton	337,955
Stoney Creek	48,005
Region of Hamilton-Wentworth	467,900

* Health Action Task Force March 1996. Statistics Canada Census Data; 1991

In common with other Public Health Units in Ontario, in Hamilton-Wentworth, we have access to mortality and health care resource utilization (hospital admission) data compiled by the Ontario Ministry of Health and Statistics Canada.

In addition and as a result of the presence of the Faculty of Health Sciences of McMaster University in Hamilton, we have a number of academic departments, institutes, centres and research units whose members have interest and expertise in interpreting health care and environmental data and their interrelationships. This expertise has been mobilized together with experts from MOEE's Standards Development Branch to bring the air pollution and health outcome measurements together with information from the scientific literature to generate estimates of the air pollution-associated health outcomes for the Region.

3.2 Objective

To determine the morbidity and mortality effects associated with air pollution in Hamilton-Wentworth; by pollutant species; and to prioritize the pollutants examined by the quantity of adverse outcomes associated with each pollutant.

3.3 Rationale

Given that the concentrations of a variety of air pollutants in Hamilton-Wentworth are known at several sites for many years, and given that hospital admission and mortality data by disease classification for Hamilton-Wentworth are available for many of the same years, and given that risk ratios relating health outcomes to levels of many pollutant species have been established by several studies reported in the recent scientific literature; it is possible to estimate from this information the number of deaths attributable to exposure to a number of different pollutants in a given year in Hamilton-Wentworth. Similar estimates of hospital admissions and mortality can also be made.

3.4 Methods

3.4.1 Data Sources.

Information of three types was necessary to make the required estimates: air quality, hospital admissions and mortality, and dose-response estimates relative to the pollutants and outcomes. A decision was made to obtain data as recent as possible for a 3-year period. This translated into data for 1992, 1993 and 1994. Owing to the fact that the health data for hospitalization were only available up to 1992, estimates have been made for this year only.

3.4.1.1 *Air Quality*

Air quality information (measured primarily by WCR-MOEE) was integrated centrally and managed by the Environment Working Group. After the HHWG had established the list of pollutants of interest, the nature of the metric (e.g., 24-hr daily average for all days by year) and the format of the computer file, the data were transferred to the HHWG worksite computer(s). The list is given in Table 3.2.

Table 3.2 List of Parameters Needed for HHWG Risk Assessment of the Criteria Pollutants.

Pollutant 24 hr-av daily, by year	carbon monoxide CO*	nitrogen dioxide NO_2	nitric oxide NO	sulphur dioxide SO_2	ozone O_3*	inhalable particulate PM_{10}	sulphate in Total Suspended Particulate $SO_4^=$ in TSP
1992	X	X	X	X	X	X	X
1993	X	X	X	X	X	X	X
1994	X	X	X	X	X	X	X
* CO and O_3 data should also include daily maximum 1hr values							

Exposure

Ozone

For the purposes of risk assessment for Hamilton-Wentworth, we have assumed no threshold for any pollutant with the exception of ozone, for which we have assumed two possible thresholds: 25 and 40 ppb.

Table 3.3 Estimates of Ozone Exposure; Site 29000, 1990
(median concentration for the year was 31 ppb.)

Number of days of O_3 > 25 ppb	235
Average O_3 concentration for days when O_3 > 25 ppb	43.6
Average O_3 concentration for days when O_3 < 25 ppb	18.6
Percent of year O_3 concentrations > 25 ppb	64%
Yearly average concentration over threshold	11.9 ppb

Number of days of O_3 > 40 ppb	112
Average O_3 concentration for days when O_3 > 40 ppb	56.3
Average O_3 concentration for days when O_3 < 40 ppb	16.3
Percent of year O_3 concentrations > 40 ppb	31%
Yearly average concentration over threshold	5.1 ppb

Sulphate ($SO_4^=$)

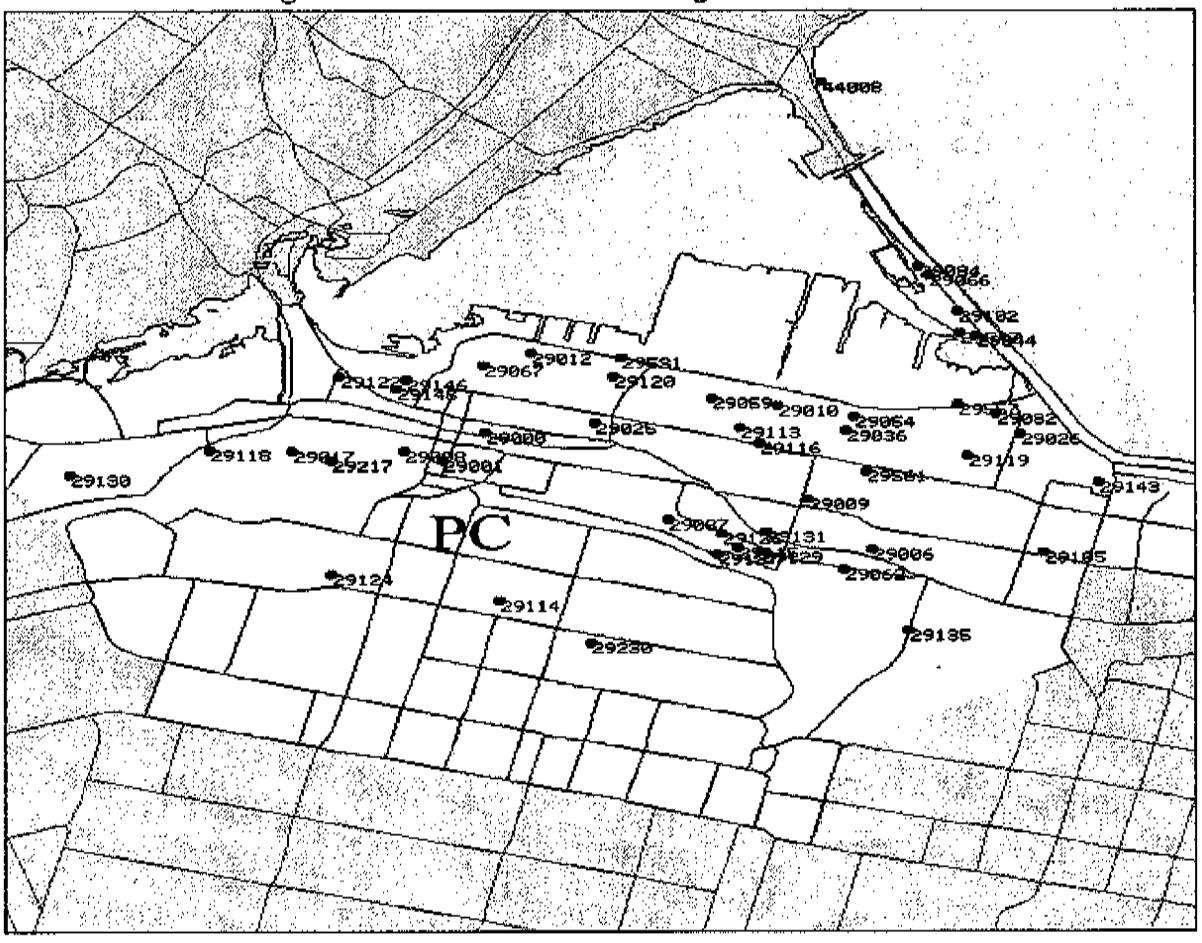
There are potentially two sources for $SO_4^=$ information in H-W. There are the $SO_4^=$ in PM_{10} data and there are the SO_4 in TSP data; the latter being more numerous. However, there is a technological problem with the $SO_4^=$ in TSP data, in that the sample is collected on a glass-fibre filter which has the property that it can convert SO_2 passing through the filter to $SO_4^=$, thus giving a falsely

high $SO_4^=$ reading. A correction for this artifact which is applicable to southern Ontario has been published by Burnett et al. (1994) and is given in the note to Table 3.4. We have used the corrected TSP $SO_4^=$ values in the estimates for H-W.

Site Selection.

Figure 1 is a map of the Hamilton area showing the air monitoring network. At first glance it might appear that there is a dense network of monitoring sites which should be more than sufficient for providing good population-based ambient exposure estimates. Forty-two of the 47 regional sites are shown on this map, but only 6 provide 24 hour monitoring, which is necessary for the assessment of the gaseous pollutants. There are 18 dustfall and 18 total suspended particulate sites; some of which are co-located and in any event, will not be used in this risk assessment, except for those few TSP sites for which sulphate analysis of the sample has been carried out. Another potentially useful site (44088, not shown, at the northeast side of the map) is technically outside the region; being in Burlington. Data acquired at sites 44088, 29102 and 29026 are not good indicators of population exposure. During the prevailing southwesterly winds, these three sites are strongly influenced by industrial emissions which are being blown away from the Hamilton population and during east or northeast winds, are influenced by the lake, and thus would underestimate population exposure.

Figure 1 - Network Air Monitoring Sites in Hamilton



Statistics Canada provides geographic-referenced data on electronic files and with this they provide the "population centroids" of the census areas they have measured. Hamilton-Wentworth is "Census Division 25" and it is made up of the "census subdivisions" of the 6 municipalities, as shown in the Table 3.1. The population centroid represents the geographical population-weighted "centre of gravity" so to speak, of the area: the location which is most representative of the population. The population centroid is shown by the letters "PC", and can be seen to lie almost midway along a line drawn between sites 29000 and 29114, and almost midway between a line drawn between sites 29118 and 29105. Sites 29025 and 29000 are just over 1 km apart from each other.

One approach that could be used in determining ambient exposure would be to weigh the data from a given site according to distance from the population centroid. This might be necessary if there was substantial asymmetry to the site locations. However, it appears that the Hamilton sites are symmetrical to the population centroid, and for the most part we feel that a simple averaging of the results is justified.

A summary of the data used for this risk assessment is given in Table 3.4.

3.4.1.2 *Hospital Admissions and Mortality*

Data were required for both total and disease-specific events for both of these outcomes for the years of interest. The selection of the disease categories for which hospital admission and mortality data was based on the results of the literature survey reported in Part One of this report. An example of this type of information is given in the Table 3.5, in which Dockery and Pope made a summary of disease outcomes with respect to PM_{10} exposure (Dockery and Pope 1994).

Of all these outcomes in Hamilton-Wentworth, data are available only for mortality (total and disease-specific) and for hospital admissions (total and disease-specific). The hospital admissions data are actually in the form of "separations" and include deaths in hospital following admission (Table 3.6).

Data for the years 1990 to 1994 inclusive were found to be available. This information was provided by the McMaster University Health Priorities Analysis Unit. The data were provided in detailed form, disaggregated by ICD-9 (International Classification of Diseases) chapter and major subclassification, as well as by sex and age category, and by year for Hamilton-Wentworth. Data were subsequently aggregated to include both males and females, and all ages together, for major classifications. Details of the data classification are given in Table 3.6 and the resultant numerical data are given in Table 3.7.

Table 3.4 Input Air Quality Data for HAQI Risk Assessment

	Arithmetic Mean					Geometric Mean ($\mu g/m^3$)			
Site & Year	CO (ppm)	NO_2 (ppb)	NO (ppb)	SO_2 (ppb)	O_3 (ppb)	PM_{10}	PM_{10} $SO_4^=$	TSP $SO_4^=$	corr* $SO_4^=$
A ll Available									
92	0.94	18	18	6.8	17	26	4.9	10.4	6.6
93	0.93	20	18	6.2	18	22	3.7	9.2	5.6
94	0.75	20	18	4.9	20	26			
3 Yr Avg	0.87	19	18	5.9	18	25	4.3	9.8	6.1
29000									
92	1.20	19	15	7.0	17	26	4.9		
93	1.11	22	16	5.9	16	22	3.7		
94	0.85	22	18	5.1	17	26			
25025									
92				8.2				11.4	7.5
93				9.4				9.5	5.8
94				6.1					
29105									
92				6.8	17				
93				4.1	19				
94				2.9	20				
29114									
92		17	9	6.3	19			9.3	5.7
93		18	10	6.7	21			8.9	5.3
94		18	9	6.0	23				
29118									
92	0.68	18	29	5.7	17				
93	0.75	19	28	4.7	17				
94	0.65	20	28	4.4	19				

* correction made as in Burnett et. al., 1994: $SO_4^=$ (corr) = -2.61 + 0.89 TSP $SO_4^=$

Table 3.5 Dockery & Pope 1994 - Hospital Admissions and Mortality

Combined effect estimates of daily mean particulate pollution		% Change in health indicator per each 10 $\mu g/m^3$ increase in PM_{10}
Increased daily mortality		
	total	1.0
	respiratory	3.4
	cardiovascular	1.4
Increased hospital use (respiratory)		
	admissions	0.8
	emergency department visits	1.0
Exacerbation of asthma		
	asthmatic attacks	3.0
	bronchodilator use	2.9
	emergency department visits	3.4
	hospital admissions	1.9
Increased respiratory symptom reports		
	lower respiratory	3.0
	upper respiratory	0.7
	cough	1.2
Decrease in lung function		
	forced expiratory flow (FEF)	0.15
	peak expiratory flow (PEF)	0.08

Table 3.6 Disease Classification of Outcome Events

	Non-traumatic Mortality			Non-traumatic Hospital Admissions			
Name	All	Cardio-vascular	Respira-tory	All	Cardio-vascular	Congest. Heart Failure	Respira-tory
ICD-9	001-799	390-459	460-519	001-799	390-459	428	460-519
Total events, both sexes, all ages; data required by year, 1990-1994 inclusive. ICD - International Classification of Diseases; codes							

Table 3.7 Input Data for Health Effects Calculations in Hamilton-Wentworth

Year	Popu-lation*	Annual Non-Trauma Mortalities*			Annual Non-Trauma Hospital Separations*				
		Total	Cardio-vascular	Respir-atory	Total	Cardio-vascular	Congest hrt. fail	Respiratory (incl. asthma)	Asthma only
1990	446,680	3466	1449	301	47285	7283		3842	468
1991	468,129	3624	1584	347	47040	7470		3974	430
1992	470,362	3529	1595	299	45303	7433		3701	534
1993	472,693	3521	1444	295					
1994	475,521	3804	1516	340					
mean 90-94	466,677	3589	1518	316	46543	7395	633	3839	477

Note: * see references below

The sources of the information provided by the Health Priorities Analysis Unit are as follows:

- Hamilton-Wentworth population estimates by age and sex, 1991-1995. *Statistics Canada,* unpublished final intercensal (1991), final postcensal (1992-93), updated postcensal (1993-95).
- Hospital separations rates by disease category by age and sex, Hamilton-Wentworth 1986-1992. *Ontario Ministry of Health*, Morbidity Reports 1986-1992.
- Mortality by disease category by age and sex, Hamilton-Wentworth 1990-1994. *Ontario Ministry of Health,* Mortality Database, 1990-1994.

3.4.1.3 *Cancer Incidence and Mortality*

The risk assessment for "air toxics", which is covered in another Section, is primarily concerned with cancer incidence or mortality as endpoints. The results specifically for Hamilton-Wentworth are presented for the years 1992-93 in Table 3.8.

Table 3.8 - Cancer Incidence and Cancer Mortality

Year	Incidence	Mortality
1992	1966	958
1993	1966	1028

3.4.1.4 *Dose-Response Estimates*

The most recent scientific information available was desired to provide the most comprehensive and technically supportable estimates of outcomes for this project. Two main sources of information were used: the first was the comprehensive literature review and assembly of a data base which was carried out for the HHWG by McMaster University in support of the development of the "user-friendly" literature review and the second, was background information (government documents, manuscripts submitted, etc.) known to various members of the Working Group.

The data base was obtained from larger data bases such as "Medline" using the key words "air pollution" and "health", and downloaded into the "Reference Manager" system: it contains 4472 references, covering the period 1928-1996. There are 1595 references from 1977-1986, and 2536 references from 1987-1996. From this data base, 205 papers were selected on the basis of title and/or abstract for retrieval of the whole article from serials in the McMaster and University of Toronto libraries, or by interlibrary loan. These were subsequently reviewed and further selection was made by formal criteria including study design, author, and suitability of outcome measure to support the expression of a dose-response relationship.

As indicated above, other documents were also used which were known to various members of the working group, such as the "Hagler-Bailly Report" (Hagler-Bailly 1995) and the supporting document for 'Towards a Smog Plan for Ontario" (MOEE Smog 1996). A complete list of references used for this work is included in Section 1.2.2.

3.4.2 Interpretation of Studies in the Literature

As a result of recent scientific work and the corresponding development of rationale and criteria documents for ozone and fine particles, there have been several summaries of the health effects associated with these pollutants and these reviews have focussed on the results of epidemiological studies.

Separating out the relatively small health effect(s) attributable to short-term variations in pollution versus the relatively large impact of other causes is accomplished in several ways in these large epidemiological studies. The first is to use a time-series design, with the day as the unit of time and a large relatively stable population in which changes in important health determinants such as socio-economic status, age and smoking behaviour are highly unlikely to occur on a day to day or even week to week basis. The second is to include filters for short-term subseasonal variations in rates than what might be associated with events such as influenza epidemics or health service work stoppages or the like. The third is to include terms in the modelling equations which reflect weather patterns, long-term changes in the health outcome over time and other such trends that may be important. The resultant statistical models are highly complex but are able to detect very small risk ratios that may be missed using other kinds of epidemiological approaches.

In general, compared to PM_{10}, $PM_{2.5}$ and other PM metrices, there has been much less information available on the gaseous pollutants SO_2, NO_2, NO and CO. However in 1996, the results of a major collaborative epidemiological study of air pollution were reported from Europe (the so-called "APHEA Study: Air Pollution and Health; European Approach") which focussed primarily on SO_2 and particles in 15 European cities in 10 countries. In some cities (notably London, England), SO_2 levels were relatively low and so other pollutants, such as O_3 and NO_2 were also included. The APHEA study was designed with the intention of combining the results into a comprehensive analysis (i.e., a meta-analysis).

In addition, there have been a small number of recent papers examining the association between CO and congestive heart failure in elderly patients, and a study of cerebrovascular disease associated with exposure to NO.

3.4.2.1 *Relative Risk or Risk Ratio*

Epidemiological studies which have not been designed *a priori* to link one with another are often difficult to compare, owing to differences in the way the outcome measure is expressed. In addition, often some "baseline" information is not provided, such as the rate of hospitalization for specific disease entities, or the background level of pollutant in the study area. In the APHEA study and in a number of other recent studies, the outcome is expressed as a "relative risk" or "risk ratio" for a given change in the pollutant of interest. In addition, the 95% confidence intervals around the computed ratio are often given to indicate the statistical strength of the association. If, for example, an association is made (with suitable statistical design rigour, accounting for the effects of other interfering variables) between hospital admission for congestive heart failure in patients over 65 and the concentration level of ambient CO, and is expressed as a relative risk of 1.05 for a change of 2.0 ppm CO; this implies that if the ambient level of CO rises by 2 ppm for a day, then the uext day, (usually) 5% more elderly patients would be admitted for congestive heart failure. If the level were to decrease by 1 ppm, then 2.5% fewer admissions would be made. In the estimates of outcome made for Hamilton, data from the literature have been used which are either in the form of risk ratios or which were easily converted to risk ratios.

As a rule, risk ratios are usually expressed as a decimal, where a value of 1.00 means no difference in risk of one circumstance compared with another. A relative risk of 2.00 means that the circumstance being examined is twice as likely to happen as that to which it is being compared; for example a "normal" circumstance. Compared to other risks found in public health (such as the risk of getting lung cancer from cigarette smoking), risks of death or hospitalization *for the individual* from the effects of air pollution are very small. However, since the whole community is exposed to this risk, the chance of an adverse outcome for someone in the community is much greater. Thus risk ratios used in air pollution epidemiology are often also expressed as a percentage difference. A risk ratio of 1.05 can also be expressed as 105%, or a 5% (105-100) risk change (risk *increase*, in this case). In this document we have chosen to define this as a "percent health outcome change", with the symbol $\Delta H\%$.

In most of the studies used, the outcomes have been estimated on a daily basis, as daily time series data were the source of the information. In most cases, we have integrated the daily results over the period of a year by using the yearly average of daily values to compute the number of outcome events. This procedure carries with it at least two assumptions: first, linearity in the dose/response relationship, and second, that there is no "threshold" of effect (which is actually a special case of the first assumption). That is to say: a change of 1ppm CO from zero to 1ppm will have the same outcome (2.5% increase in CHF (congestive heart failure) 65+ admissions) as a change of 1 ppm from 2 to 3 ppm.

3.4.2.2 *Shape of the Dose-Response Relationship.*

There is some evidence that dose-response relationships for some pollutant species and outcomes are logarithmic; that is to say there is a greater response (the curve is steeper) at lower pollutant levels than at higher levels. This is sometimes given as evidence for a "harvesting" effect: that there is a limited supply of those who respond in the population and the most sensitive respond first; leaving a reduced population from which to draw as those who are less sensitive respond to higher levels.

However in general, so long as the pollutant levels are not far removed from those for which the risk ratios were originally established, the linear assumption is reasonable. If the observed levels are higher, the outcome may be over-estimated: if it is lower, they may be under estimated.

3.4.2.3 *Threshold*

If there is a supportable assumption that no outcomes will occur below a certain level of pollutant, then it would not be appropriate to include "below threshold" levels in the average calculated for a yearly exposure. For example, if the dose-response relationship relating hospital admissions to O_3, were only supported for values greater than 25 ppb high-hour or "maximum" (and increased with a risk ratio of 1.045/50 ppb above this level) and at no time in the year did the high-hour value exceed 25 ppb, then there would be no excess risk (i.e., no air pollution associated increase in admissions). However, if the mean of the yearly high-hour values was 24.9 ppb, the

implication would be that on some days, the high-hour value exceeded the threshold and on those days, there would be excess morbidity. Thus it would be necessary to subtract 25 from the daily maximum, and if the result were positive, do two things: count the number of days for which this was true in the year; and calculate the mean "excesses" for those days. This would result in the mean "excess" over the threshold, and the percent of the year for which this occurred. The product of this (divided by 100) would equal the "yearly excess" concentration from which the outcomes could be derived using the risk ratio as before.

For O_3 and PM_{10}, there has been a great deal of discussion about the evidence for or against the existence of a threshold for mortality or hospital admissions. Hagler-Bailly (*Supplemental Report 2*, pp 4-6) has acknowledged that there is no threshold for either O_3 or PM_{10} for health effects, based on the available epidemiological evidence. The primary assumptions used in their analysis were 40 ppb, corresponding to background levels for O_3 (Hagler-Bailly, *Summary Report*, pp 3-5) and zero $\mu g/m^3$, that is no threshold, for PM_{10} (Hagler-Bailly, *Supplemental Report 2*, p 5). However, they have conducted some sensitivity analyses to determine the impact of the assumption of thresholds at certain levels for both of these pollutants. The thresholds they have selected for PM_{10} (24 hr) are 25 and 50 $\mu g/m^3$, and those selected for O_3 are 25 and 80 ppb high-hour values. The lower thresholds in both cases are based on an analysis of Ontario data by Burnett (for PM_{10} estimated from sulphate) and on data from 16 Canadian cities for O_3.

For the purposes of risk assessment for Hamilton-Wentworth, we have assumed no threshold for any pollutant with the exception of O_3, for which we have assumed two possible thresholds: 25 and 40 ppb.

3.4.3 Dose-Response Relationships by Pollutant

3.4.3.1 *Carbon Monoxide (CO)*

Carbon monoxide has long been recognized as a pollutant with adverse health effects and, in moderate concentrations, is lethal. It can be measured in the exhaled air of heavy (2 pack/day) smokers at concentrations from 50 to 75 ppm. The industrial "threshold limit value" for an 8-hr day is 50 ppm. The many indoor sources for CO include gas cooking stoves, portable non-electric space heaters and cigarette smoking. Since the introduction in the 1970s of catalytic converters on automobiles and light trucks, urban levels of CO have dropped substantially and until recently, it was thought that CO was no longer a pollutant problem. This has changed within the last two years.

There are three important recent papers relating exposure to CO to hospitalization for congestive heart failure in patients over 65 years of age: Morris et. al.(1995), Schwartz and Morris (1995), and Burnett et. al.(1997). The Schwartz and Morris paper (covering 7 cities) expanded on their earlier paper, which examined only Detroit, Michigan. The very recent Burnett paper is of particular interest because the data were obtained from 10 Canadian cities; one of them Hamilton (actually Hamilton-Wentworth). Furthermore, specific risk ratios were estimated for Hamilton, and the author has provided us with the base rate of hospitalization for CHF which was observed in this study. In

general, the burden of illness associated with CO estimated by Burnett et. al. for Hamilton is similar to that found by Morris and Schwartz in seven U.S. cities. (Risk ratio of 1.05 for increment of 2 ppm, compared with 1.05 to 1.39 [mean 1.25] for an increment of 10 ppm.) We have used the Burnett figure (1.05 per 2 ppm) for this analysis.

The data we have currently available for CO are in the form of 24-hr average data. The metric used by Morris and Schwartz is the "daily maximum" or "maximum daily 1-hour reading". Burnett et. al. used the same metric, but they also reported coefficients for the 8-hour running average which were 50% higher than the 1-hr maximum coefficients. In the absence of the 1-hr maximum data (which would be greater than the 24-hr data), we are using the coefficient for 1 hr. We expect that this is a conservative estimate, i.e., the correct data will yield higher numbers of outcomes.

The only current data we have on premature mortality and CO exposure are from the APHEA study in Athens by Touloumi et al. (1996). In this study, a risk ratio for premature mortality of 1.01 was found associated with an increase of 1.0 ppm CO.

3.4.3.2 *Nitrogen Dioxide (NO_2)*

Although there have been many studies of the effect of NO_2 on health, many of these have been cohort studies examining health outcomes; particularly in children measured in terms of respiratory symptoms or pulmonary function change. Both NO_2 and CO are pollutants which have major indoor sources from gas cooking, non-electric portable space heaters and cigarette smoking. As a result, there are few studies in the literature which have shown a statistically significant association with hospital admissions or mortality. The APHEA study in London (Ponce de Leon et. al.1996) determined a risk ratio for respiratory hospital admissions of 1.011 for a 27 ppb change in NO_2. The study from Barcelona (Sunyer et al.1996) determined a risk ratio for mortality of 1.034 for a 29 ppb change in NO_2 .

3.4.3.3 *Sulphur Dioxide (SO_2)*

In a severe and well documented episode in London, England in 1952, SO_2 was identified as one of the air pollutants responsible for inducing premature mortality in susceptible persons and animals. From the '50s to the '60s, there were many studies which explored the role of SO_2 and particles in the air as air pollutants which were associated with adverse health outcomes. Steps to regulate emissions to improve air quality in that period were primarily aimed at reducing the levels of these two pollutants. In many jurisdictions, success was achieved in lowering the levels of both SO_2 and the coarser fraction of suspended particles, and by the mid-60s, attention in North America was turning towards the growing problem of ozone, or "photochemical smog". With the growth of automobile traffic in North America, the ground-level ozone concentration was becoming steadily higher, especially in the summer months, and it was clear that adverse health effects were also attributable to this pollutant. For about 20 years, there was less attention paid to the health effects of SO_2 and there was no change in the SO_2 air quality standards from those based on the '50s and '60s research.

In the late '80s, attention was again focussed on SO_2 and more particularly, $SO_4^=$ as a problem coexisting with O_3 in northeastern North America. In addition, it was recognized at this time that the smaller size particles (less than or equal to 10 micrometers (μm) in diameter; so-called PM_{10} and the even smaller particles, $PM_{2.5}$) had marked association with premature mortality, as well as various morbidity endpoints. In Ontario, about one-third of $PM_{2.5}$ is particulate sulphate, which has been shown in a number of studies to increase mortality and hospitalization in persons with cardiorespiratory disease. The epidemiological studies showed little evidence for a "threshold" for the health effects; that is to say, no evidence of a "safe" level below which no adverse effects were observed.

The APHEA Study

In 1991, a group of European and American investigators designed a large collaborative air pollution epidemiology study funded by the European Union to examine the health effects of air pollution in 15 different cities in 10 countries of eastern and western Europe. The population base was over 25 million people and all aspects of the design were agreed upon at the beginning. Thus the results could appropriately be combined in a meta-analysis at the end of the study.

The basic objective of the overall study was "to provide quantitative estimates of the short-term health effects (using the total and cause-specific number of deaths and emergency hospital admissions) of air pollution, taking into consideration interactions between different pollutants and other environmental factors." (Katsouyanni et. al., 1996) The association between the daily time series of several pollutants and the daily number of events (cause-specific deaths, hospital admissions) were assessed using Poisson regression and adjusting for a number of time-related factors.

In all of the individual studies, the pollutants examined included SO_2 and some measure of particulate as a minimum. In addition, in some but not all of the studies, other pollutants such as ozone, carbon monoxide or nitrogen dioxide were also measured. The outcomes, in most cases, were expressed as "risk ratios" for the events examined, with respect to a "standard" increase in pollutant level (usually the 95th compared to the 5th percentile of data; for SO_2, on the order of 100 $\mu g/m^3$ over 24hr). Because of the common methods and outcome metric, it is possible to compare directly the findings of the 11 individual studies.

An abbreviated summary of the basic results of the APHEA study is given in Table 3.9. In 10 out of the 11 studies, SO_2 at ambient levels was shown to have adverse health effects. The highest median levels of 24-hr SO_2 were observed in Milan, Italy and Cracow, Poland (66, 74 $\mu g/m^3$) and the lowest was observed in Paris, France (23 $\mu g/m^3$). The largest range of values for SO_2 was observed in Italy and the smallest was reported in London, England.

In Table 3.9, a "sig" or numeric entry implies a statistically significant association; "ns" implies the relationship was tested and not found significant; and a "blank" entry signifies that a relationship was not examined. In 7 of the 8 studies where it was examined, significant associations were found

between premature mortality and daily levels of SO_2 and in all 5 studies where it was examined, significant associations were found between hospital admissions and SO_2. Values of the risk ratios for the different pollutants examined have been converted to a constant exposure difference where necessary and are given in Table 3.9. Although significant results were found in studies conducted in Finland and Poland, data were not provided from which risk ratios could be obtained.

From the original papers, estimates of relative risk of total mortality for a difference in 24-hr SO_2 exposure of 95 to 100 $\mu g/m^3$ varied from 1.04 to 1.13 and for cause-specific mortality, from 1.08 to 1.16 (corrected from 50 $\mu g/m^3$). Estimates for respiratory hospital admissions for a difference in 24-hr SO_2 exposure of 100 $\mu g/m^3$ varied from 1.03 to 1.12 (corrected from 31 $\mu g/m^3$).

On the basis of these results, it was possible to estimate an overall risk of daily non-traumatic mortality of approximately 1.09 / 100 $\mu g/m^3$ SO_2 and for cause-specific mortality of 1.10 / 100 $\mu g/m^3$ SO_2. A meta-analysis of these data supports estimates of a risk ratio (for a difference of 24-hr SO_2 exposure of 50 $\mu g/m^3$) of approximately 1.03 to 1.05 for both total and disease specific mortality. Evidence from these studies suggests that these estimates for SO_2 are little influenced by the levels of particulate occurring at the same time and further, that lower estimates may be more appropriate in Eastern European cities.

We may conclude from the APHEA studies that there is good evidence that a change of the 24-hr concentration level of SO_2 from 10 $\mu g/m^3$ to 60 $\mu g/m^3$ would be associated with a 3% increase in total daily mortality, a 4% increase in cardiac and respiratory mortality, and a 2% increase in daily respiratory hospital admissions.

3.4.3.4 *Ozone (O_3)*

Ozone (and more recently fine particles) have been recognized as problem pollutants in developed and developing countries throughout the world. Since the processes of producing, transporting and consumption of fossil fuels have been identified as the main contributors of ground-level ozone, there is great resistance (on economic grounds) to taking steps to control this pollutant in most jurisdictions. The great difficulty with ozone (and to a lesser degree fine airborne particulates) is that it is generated by reactions in the atmosphere from other pollutants. Only by controlling the "ozone precursors" can ozone be controlled. The problem is compounded by the fact that ozone and its precursors can be transported hundreds of kilometres. For reasons which will not be expanded upon here, the United States Environmental Protection Agency (EPA) has been reluctant until very recently to recognize the growing epidemiological evidence of the adverse health effects of O_3 and thus it has taken some time for this aggregate of scientific evidence to emerge.

One of the most useful recent reviews of this literature was carried out under contract to Environment Canada to support an initiative of the Canadian Council of Ministers of the Environment (CCME) called the "Task Force on Cleaner Vehicles and Fuels" (Hagler-Bailly 1995). It is of interest that one of the papers that this report regards as most important is the Canadian study of Burnett et. al.(1994). Having reviewed the literature, Hagler-Bailly arrived at the risk ratio for

respiratory hospital admissions of 1.045 associated with a 50 ppb increase of O_3. It is difficult from the information provided to calculate a risk ratio for mortality.

The common outcome metric (risk coefficient) used in the Hagler-Bailly analysis is the "individual daily risk per unit of pollutant". To estimate the number of events (hospital admissions, deaths) predicted to be associated daily with a given level of pollutant in a community, the risk coefficient is multiplied by the total population in the community, and the concentration of the pollutant in the same units. This is a useful approach for estimating impacts over a wide jurisdiction (say, nationally or provincially), where the population is in the millions or tens of millions. Implicit in this approach is an assumption of mortality or morbidity rates which are uniform over the area of interest. Unfortunately this is not the best method for estimating local impacts (such as those for Hamilton-Wentworth), because it does not allow for the inclusion of local morbidity or mortality rates. In the present analysis we have used "risk ratios", which do allow for the inclusion of local rates, and thus it has been necessary when using the data from Hagler-Bailly to obtain the original risk ratios from which the individual risk coefficients were derived. This has been done either directly, where the risk ratios were quoted in the text, or by calculation from the information provided, where it has been sufficient.

For example, in Section 3 (Premature Mortality, page 18) of the Hagler-Bailly (1995) analysis, the "central" PM_{10} mortality risk is calculated from the original risk ratios. On the basis of results from recent time-series studies, they have selected a $\Delta H\%$ of total non-traumatic mortality of 1% per 10 $\mu g/m^3$ of PM_{10}; that is to say a $\Delta H\%$ of 0.1% per $\mu g/m^3$ PM_{10}. They then state (from WHO data) that the average daily Canadian non-accidental mortality rate is 18.4 per million population, and obtain a coefficient of 1×10^{-3} (0.1%) $\times$ $18.4 \times 10^{-6} = 1.8 \times 10^{-8}$ deaths per person per $\mu g/m^3$ per day. Knowing this relationship, it is possible to calculate the risk ratio for total mortality associated with ozone. The individual daily risk coefficient is 5.0×10^{-9} (page 20) /person/ppb O_3. This translates to a $\Delta H\%$ of $0.5/1.84 \times 10^{-1} = .027$ per cent per ppb. For an exposure of 50 ppb this translates to a $\Delta H\%$ of 1.35%, which has been used in this analysis. One of the APHEA studies (Sunyer et al., 1996) estimated a relative risk of total mortality of 1.048 for 47 ppb ($100 \mu g/m^3$) for ozone, and because it was not available for the review by Hagler-Bailly, it was not used in their analysis. Obviously this would yield a larger number of outcomes if it were used here. Since the Hagler-Bailly estimate was based on Toronto data and is more conservative, and the Sunyer study related to Barcelona, we have used the Toronto estimate here. The Sunyer study was one of the three APHEA studies to examine O_3 mortality and the only one to find a statistically significant relationship.

Table 3.9 - Risk Ratios from APHEA Studies

COUNTRY	CO		NO_2		SO_2		O_3		PM_{10}	
	D =1.0 ppm		D =100 µg/m³		D =100 µg/m³		D =100 µg/m³		Δ =100 µg/m³	
	mortality	resp adm	motality	resp adm	motality	resp adm	motality	resp adm	mortality	resp adm
Slovakia					ns				ns	
Netherlands				1.08(#)		1.05		1.05(*)		1.10 (bs,#)
France(Lyon)			ns		1.12		ns		1.08(r)	
Poland					sig				sig(bs)	
France (Paris)			ns	ns	1.09(^)	1.04	ns	ns	1.17(r)	1.045
Greece	1.01				1.12				1.05(bs)	
Germany			ns		1.04				1.03	
Finland				ns		sig(as)		sig(as)		ns
United Kingdom				1.012		1.129		1.056		ns
Italy					1.12(r)	1.04			1.12(t)	1.05(t)
Spain			1.034		1.127		1.048		1.07(bs)	

Notes: * = summer; # = winter; ^ = 1 hr; r = respiratory; as = asthma; t = TSP; bs = British Smoke.
Specific volumes @ 21 °C: CO = 0.862 l/g NO_2 = 0.293 l/g SO_2 = 0.368 l/g O_3 = 0.503 l/g
Matheson gas data book (1971) e.g., 1 mg/m³ CO = 0.862 ppm; 1 µg/m³ SO_2 = 0.368 ppb.

3.4.3.5 *Particles and Sulphate (TSP, PM_{10} and $SO_4^=$)*

The strongest association found in the Hagler-Bailly report and that to which the greatest economic consequences are ascribed, is between exposure to ambient fine particles and mortality, either total or cause-specific. It is true, however that relatively few studies have actually measured particulate pollution using the PM_{10} measurement system (with the notable exception of the studies of Pope et al. in Utah). Dockery and Pope, in their review of the health effects of particulate pollution, had to make conversions (e.g., from TSP to PM_{10}) in their meta-analysis of different studies and is the same process used in Hagler-Bailly. It is of interest that although Hagler-Bailly used Burnett's (1995) study to estimate hospital admissions, rather than provide the estimate in terms of exposure to sulphate, a conversion was made to PM_{10} (1 $\mu g/m^3$ PM_{10} = 0.18 $\mu g/m^3$ $SO_4^=$) based on Ontario data. We have chosen to use the actual data for $SO_4^=$ obtained for Hamilton-Wentworth and where health outcomes in the literature have been related directly to $SO_4^=$, we have used those risk ratios. Outcomes have been estimated based on both PM_{10} and $SO_4^=$ and we have chosen to use whichever of these two measures shows the largest number of effects; but have not summed them together. This may give rise to an underestimate of outcomes as there may well be components of PM_{10} which have effects other than those attributed to $SO_4^=$.

PM_{10}

For total mortality, we have used the central estimate from Hagler-Bailly and for cause-specific mortality, the estimates of Dockery and Pope (1994).

For hospital admissions, we have used the estimates of Burnett et al. (1994,1995) based on sulphates, deriving, as in Hagler-Bailly, an estimate based on PM_{10} using a Hamilton-specific conversion of sulphates to PM_{10} .Thus the Ontario-wide conversion of 1 $\mu g/m^3$ PM_{10} = 0.18 $\mu g/m^3$ $SO_4^=$ used in Hagler-Bailly was substituted with 1 $\mu g/m^3$ PM_{10} = 0.254 $\mu g/m^3$ $SO_4^=$.

Sulphate ($SO_4^=$)

There is currently a considerable amount of discussion focussed on the issue of: "From the health perspective, what are the important fine particle species and what is their biological mechanism of action?"

Some investigators are convinced that current evidence points towards $SO_4^=$ as the most important species on the basis of its small size (in continental, as opposed to marine environments, of less than 0.5 µm), its chemical reactivity (via its association with the hydrogen ion) and on the basis of animal and clinical exposure studies. Lippmann and Thurston (1996) state, "Sulfate has advantages over $PM_{2.5}$ for retrospective epidemiology, at least in the U.S., because considerably more data were collected in recent decades and there is a broader epidemiologic data base in the literature for comparison to other studies. While $SO_4^=$ *per se*, is an unlikely causal factor for mortality or morbidity, it is often closely correlated with variations in the strong acid component of ambient particulate matter (H^+) and $PM_{2.5}$ concentrations (especially in summer) which are more likely causal factors."

In *Sulphur in Fuels* (1997), an attempt is made to provide low, central and high estimates of the associations between mortality and sulphate exposure. The approach uses both the time-series epidemiological studies of daily mortality counts (i.e., mortality from acute exposures) and cross-sectional studies of annual mortality rates (i.e., mortality from chronic exposures). This has been brought about as a result of the very strong evidence from a cross-sectional study put forward by Pope and colleagues (1995) who concluded a 7.5% health outcome change per 10 $\mu g/m^3$ $SO_4^=$.

Sulphur in Fuels derive a low estimate of premature mortality using the results from a study by Schwartz (1996) which uses time-series epidemiological methods and a high estimate from the cross-sectional study by Pope(1995). The central estimate is based on a weighted mean from the two previous studies, two-thirds to one-third relative weighting of the time-series and cross-sectional studies respectively. Thus the low estimate is based on 1.7%/10 $\mu g/m^3$ $SO_4^=$ (i.e., mean mortality effect size minus one standard deviation) from Schwartz(1996) while the high estimate is based on 8.5 %/10$\mu g/m^3$ $SO_4^=$ (i.e., mean mortality effect size plus one standard deviation) from Pope (1995). Using the weighting above, the central estimate is 3.8%/10 $\mu g/m^3$ $SO_4^=$.

For the purposes of the HAQI analysis, we have assumed that the mean mortality estimates based on acute studies (Schwartz et al, 1996) are a lower bound to the estimate of $SO_4^=$ mortality. The mean mortality estimates based on chronic studies (Pope et al, 1995), similar to the approach in *Sulphur in Fuels* but without the standard deviation adjustments, provide an upper bound.

For cardiovascular and respiratory hospital admissions, the estimates of Burnett et al. (1994,1995) have been used. As indicated above, sulphate levels have been obtained from the TSP data, corrected by the method of Burnett et al.(1994).

3.5 Estimates of Hospital Admissions and Mortality Related to Pollutants in Hamilton-Wentworth

Tables 3.11 and 3.12 integrate the information on air pollution, hospitalization and mortality rates from Hamilton-Wentworth with the risk ratios obtained from the literature. The data in these tables are for the year 1992.

Each table is in four parts:

- The first line of numbers represent the pollution level used for the risk assessment, and in most cases it is made up of the yearly average of daily mean (24-hr) data. In the case of ozone, it represents the daily mean of the one-hour maximum values for those days of the year when the one-hour maximum exceeded 25 ppb, less 25 ppb. The number is further reduced by the ratio of the number of days of exceedance of 25 ppb to total days in the year (365).

- The second part shows the expected per cent change in outcome for Hamilton-Wentworth. This is obtained by taking the percent health outcome change (from the literature) and multiplying it by the ratio of the Hamilton pollutant level to the level used in the literature to define that change. For example, for respiratory hospital admissions and PM_{10}, the $\Delta H\%$ is 0.5%, the literature pollutant change is 10 $\mu g/m^3$; and the average H-W level is 26 $\mu g/m^3$. Therefore the H-W %change in outcome is:

$$0.5\% \times 26/10 = 1.3\%.$$

- The third section gives the "base" or yearly average values of hospital admissions or mortality in each category; and

- The final section shows the result of the calculation (for each category where data were available) of the number of events associated with a specific pollutant by multiplying the H-W %change in outcome by the base number of events. For example, respiratory admissions associated with PM_{10} gives 7433 x 1.3% or 96.6 yearly admissions associated with exposure to PM_{10}.

3.5.1 References to Table 3.10

1. Hagler Bailly Consulting.(1995). *Environmental and Health Benefits of Cleaner Vehicles and Fuels. Supplemental Report 2: Selected Concentration-Response Functions for Human Health Effects.* Canadian Council of Ministers of the Environment Task Force on Cleaner Vehicles and Fuels. Note: For PM_{10} hospital admissions see text, this document in "**PM_{10}**" section.
2. See text, this document in "**SO_2**" section.
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Table 3.10 Percent Health Outcome Change (ΔH%) for a Specified Change in Pollutant

Pollutant		CO (ppm)	NO_2 (ppb)	SO_2 ($\mu g/m^3$)	O_3 (ppb)	PM_{10} ($\mu g/m^3$)	$SO_4^=$ in TSP ($\mu g/m^3$)
Pollutant change		1	29.3	50	50@	10	10
Mortalities	total (acute exp)	1%(8)*	3.4%(9)	3%(2)	1.35%(1)	1% (1)	2.2%(7)
	total (chronic exp)						7.5%(10)
	cardiovascular			4%(2)		1.4%(6)	
	respiratory			4%(2)		3.4%(6)	
Pollutant change		2@	27	50	50@	10	10
	cardiovascular					0.6%(1)	2.5% (1)
Hospital admissions	CHF65+**	5%(4,5)					
	respiratory		1% (3)	2%(2,3)	4.5%(1)	0.7%(1)	2.7% (1)

Notes:

* References are on previous page

** Congestive heart failure in elderly patients (65+)

@ Daily high-hour

Table 3.11 Hospital Admissions Related to Pollutants in Hamilton-Wentworth 1992

Pollutant / Level used for R.A. (yearly avg. for period)		CO (ppm)	NO_2 (ppb)	SO_2 ($\mu g/m^3$)	O_3 (ppb)*	PM_{10} ($\mu g/m^3$)	$SO_4^=$ in TSP ($\mu g/m^3$)	Total
		0.94	18	18.7	11.9	26	6.6	
Risk increase for H-W: (per cent)	CV					1.3	1.7	
	CHF65+	2.35						
	Resp		0.67	0.75	1.07	1.3	1.8	
Relevant admission base	All N.T.							45,303.
	CV							7,433
	CHF65+(#)							633
	Resp							3,701
		CO	NO_2	SO_2	O_3	PM_{10}	$SO_4^=$ in TSP	Total (~)
Additional admissions	CV					116	126.4	126.4
	CHF65+	14.9						14.9
	Resp		24.8	27.8	39.6	67.4	66.6	159.6
Total (~)	All classes	14.9	24.8	27.8	39.6	183.4	193	301

Notes: * Based on 1990 O_3 data. Mean of (level - 25) for days when level > 25. Represents 64% of days in year.
Burnett 1997; and personal communication of Hamilton daily rate (R. Burnett): 1.735
~ Not including PM_{10} data. See text

Table 3.12 Non-Trauma Mortality Related to Pollutants in Hamilton-Wentworth 1992

Pollutant		CO (ppm)	NO_2 (ppb)	SO_2 ($\mu g/m^3$)	O_3 (ppb)*	PM_{10} ($\mu g/m^3$)	$SO_4^=$ in TSP ($\mu g/m^3$)	Total
Level used for R.A. (yearly avg. for period)		0.94	18	18.7	11.9	26	6.6	
Risk increase for H-W: (per cent)	acute exp	0.94	2.09	1.12	0.32	2.6	1.45	
	chron exp						3.17	
	CV			1.5		3.64		
	Resp			1.5		8.84		
Relevant mortality base	All N.T.							3,529
	CV							1,595
	Resp							299
		CO	NO_2	SO_2	O_3	PM_{10}	$SO_4^=$ in TSP	Total (~)
Additional mortality	acute exp	33	74	39.5	11.3	91.8	51.2	250
	chron exp						163	163
	CV			23.9		58.1		113
	Resp			4.5		26.4		
Low est	all cats			40	5	85	51	130
High est	all cats	33	74	40	11	92	163	321

Note: * Based on 1990 O_3 data. Mean over year of (level - 25) for days when level > 25. Occurs 64% of days in year.
~ Including either PM_{10} or $SO_4^=$ data. See text

3.6 Discussion

The results of this analysis demonstrate a substantial burden of illness and premature death associated with air pollution in Hamilton-Wentworth. In 1992 there were 301 hospital admissions for cardiac and respiratory disease and between 90 and 321 premature deaths (see Table 3.13).

Air pollution is not the only factor contributing to ill health and premature death in the region. There were 45,303 admissions for non-traumatic illness in 1992 and air pollution was associated with less than 1 (0.66) % of these admissions. In the same year, there were 3529 non-trauma associated deaths, for which air pollution was estimated to contribute to in the range between 90 and 321; a much larger share at 9%.

Six pollutants were considered in this aspect of the analysis: carbon monoxide (CO), nitrogen dioxide (NO_2), sulphur dioxide (SO_2), ozone (O_3), inhalable particulate (PM_{10}) and sulphate ($SO_4^=$). Data from the literature allowed us to make estimates of hospital admissions for all of these pollutants and information currently available, allowed us to also make estimates of premature mortality. Since there are varying levels of confidence for estimates of mortality depending on pollutant species, we have indicated a low estimate and a high estimate for this health endpoint.

3.6.1 Relative Health Effects of Pollutants

Of the pollutants studied, inhalable particulate matter and its sulphate component has by far the greatest impact in Hamilton-Wentworth in terms both of hospital admissions and premature deaths. When assessed as sulphate, it represents 64% of the air pollution-related hospital admissions and up to 51% of the mortalities. If the particulate pollution is assessed as PM_{10} rather than sulphate, it represents 61% of the hospital admissions and up to 29% of the mortalities.

There has been considerable discussion in the Human Health Working Group regarding the confidence in the estimates of mortality and hospital admissions associated with the various pollutants. The working group has greatest confidence in the associations between particulate pollution (specifically PM_{10} and SO_4 which is mainly found in the fine particle fraction $PM_{2.5}$) and mortality. We have somewhat less confidence in the associations between ozone and mortality, and the associations between CO or NO_2 and mortality are only supported by a very small number of studies.

Of the gaseons pollutants, O_3 has the next highest burden for hospital admissions and SO_2 for mortality (neglecting the one study on NO_2). Both CO and NO_2 have quantifiable but smaller impacts on hospitalization.

Table 3.13 summarizes the premature mortality and hospital admission estimates and the relative confidence, in terms of 'high', 'medium' and 'low', that the working group had in these estimates. Some additional observations and caveats in very recent publications by expert groups and panels are noted below which have partially influenced the confidence assigned to the estimates.

Table 3.13 Summary and Relative Confidence in the Mortality and Hospital Admission Estimates (in Tables 3.11 and 3.12) in Hamilton-Wentworth 1992

Pollutant	**Premature Mortality** (incidences/year)			**Hospital Admissions** (incidences/year)		
	Confidence in Estimates			*Confidence in Estimates*		
	High	*Medium*	*Low*	*High*	*Medium*	*Low*
Particulate Matter (PM_{10}) and Sulphate* ($SO_4^=$)	85 (51)*	92 (163)*		183 (193)*		
Ozone (O_3)	5	11		40		
Sulphur Dioxide (SO_2)		40		28		
Nitrogen Oxides (NO and NO_2)			74	25		
Carbon Monoxide (CO)			33	15		
Total	90[a]	214[b]	321[c]	301[d]		

* Sulphate is part of particulate matter
[a] Using the higher of the PM_{10} and Sulphate estimate (i.e., 85)
[b] Using the higher of the PM_{10} and Sulphate estimate (i.e., 163)
[c] NO_2 and CO associated low confidence estimates added to the other pollutants in the previous (i.e., 'medium') column
[d] Using the higher of the PM_{10} and Sulphate estimate (i.e., 193)

The Sulphur in Fuels Expert Panel noted the following:

- When attempting to deal with associations between mortality and acute sulphate exposures vs chronic sulphate exposures, they have selected to use an acute study (selected from many studies with similar results) as the basis of their low estimates and the single chronic study as a basis of their high estimate. However, in producing their central estimate, the relative weights they have assigned to the acute/chronic studies was 2/3 to 1/3 (*Sulphur in Fuel* , 1997, pp. 4-10) . Thus the panel suggested that a central estimate of mortality associated with sulphates is closer to the results of the acute studies than to the single chronic study.

- In addressing sulphur dioxide, even though this was the pollutant indicated to be most reduced as a result of reducing sulphur in fuels, the panel felt that the health effects relationships in the literature for this pollutant were ill-defined (*Sulphur in Fuel*,1997, pp. 1-1). In their discussion of uncertainties addressing the relationship between sulphate and other pollutants they make the following observations. They note that most U.S. studies of the population-based health effects of SO_2 have been unable to separate SO_2 from the collinear effects of various measures of particulate matter (PM); mainly TSP and black smoke. They later add that although some European studies have found that the SO_2 results

sometimes appear to be more robust than the PM results, the SO_2 results have usually been regarded as less plausible. Thus, the panel's decision was to focus on sulphate as the index pollutant (*Sulphur in Fuel*, 1977, pp. 7-5).

The final summary report by Katsouyanni, Schwartz et al. (1997) on the APHEA project has recently been published and ends with a rather ambivalent note regarding sulphur dioxide:

- Sulphur dioxide, on the other hand, is a highly reactive gas with a short half-life indoors. It is a known respiratory irritant and bronchoconstrictor. Although sensitivity to exposure varies, its effects seem limited to patients with asthma and bronchitis. Exposure to sulphur dioxide may therefore not completely explain the observed increase in mortality; it may rather serve as a surrogate of other substances. Since sulphur dioxide is highly correlated with the levels of fine particles in some American cities, Schwartz et. al. postulated that sulphur dioxide may be a marker of fine particles. However, the fact that we found a consistently significant effect of sulphur dioxide on mortality in all western European cities, whatever the level and composition of particles in each one, may suggest that sulphur dioxide has a direct effect. The role of outdoor peak exposures to sulphur dioxide in the increase of daily mortality should be further investigated."

These latter two observations made by the Sulphur in Fuels Expert Panel and the report by Katsouyanni respectively, appear to suggest that it may be too early to assign a high confidence to mortality estimates associated with sulphur dioxide in this Hamilton-Wentworth study.

Thus, even though Table 3.13 reflects a range of estimates for the different pollutants with varying degrees of confidence associated with them, the Human Health Working Group is in full agreement that, from the human health protection point of view, the most important thing is to identify the appropriate marker(s) of the air pollution mix rather than identify the specific toxic agent in the air pollution mix (if indeed, there is only one toxic component in the mix). We are in agreement that sulphur dioxide emissions (through conversion in the atmosphere to sulphates), nitrogen oxide emissions (through conversion to nitrates), as well as, primary emissions of inhalable particles (i.e., both PM_{10} and $PM_{2.5}$) all contribute to increasing the ambient air burden of inhalable particulate matter and especially its fine fraction component. We are also in agreement that the strength and consistency of the epidemiological evidence for mortality and morbidity effects of current levels of inhalable particulate air pollution is remarkable, robust, consistent and compelling.

Thus from an overall point of view, of pollutants other than the "air toxics" and total reduced sulphur compounds and black particulate fallout (Groups # 4 and #5, Table 2.1), the most important, from a health outcome perspective, are fine particles (and especially the sulphate component), SO_2 (because it is this pollutant that produces atmospheric sulphates) and O_3. Of less, but still significant importance are NO_2 and CO.

3.6.2 Sulphate ($SO_4^=$) Levels in H-W

By its physical nature, since sulphate pollution is almost entirely in the form of very fine particles, it is included in the measurement of PM_{10}. The fraction of PM_{10} that $SO_4^=$ represents, however varies considerably from region to region. It appears that Hamilton-Wentworth has a larger amount of $SO_4^=$ in ambient air than the fraction in PM_{10} would indicate.

Sites 29000 and 29025 are approximately 1 km apart, and allow for a comparison of $SO_4^=$ in TSP and in PM_{10}. In 1992, the geometric mean for TSP at 29025 was 62 $\mu g/m^3$; the sulphate component at that location was 11.4 $\mu g/m^3$. The geometric mean for PM_{10} at site 29000 for that year was 26 $\mu g/m^3$; the sulphate component was 4.9 $\mu g/m^3$. A direct comparison can be made at site 29102 in 1992: TSP was 52 with 11.2 $\mu g/m^3$ $SO_4^=$; and PM_{10} was 25 with 6.2 $\mu g/m^3$ $SO_4^=$.[1] Thus there is some evidence that sulphate levels determined from TSP measurements are approximately twice that found from the PM_{10} data. Part of the explanation is the type of filter medium that is used for the two different kinds of measurement, as Teflon-coated filters are used in the PM_{10} measurement to avoid the artifacts described in an earlier Section. It may also be possible that in Hamilton-Wentworth, a portion of the sulphate burden is emitted directly as a primary pollutant from local industry, and may be of larger particle size, which appears in the TSP measurement but not in PM_{10}.

3.6.3 Health Impacts of Sulphate ($SO_4^=$)

The risk ratios for estimating the burden of mortality from $SO_4^=$ were obtained in part from the very large cross-sectional study of Pope et al. This study has avoided the usual problems of confounding variables by its prospective design, which allowed for the collection of information on individual smoking habits, occupational exposure, alcohol use age, sex, etc. They found that the elevated mortality risks were similar for both smokers and non-smokers in higher pollution locations. This is probably the most robust information available which relates mortality to $SO_4^=$ levels, but it must be borne in mind that this relates to chronic exposures. The relationship between chronic exposure-related mortality from $SO_4^=$ and that related to acute exposure has been previously discussed, and the rationale for the approach used here described.

The risk ratios for estimating hospital admissions were obtained from the studies of Burnett et al., which by virtue of the use of $SO_4^=$ and health data measured in Ontario are most relevant, and are also well accepted as the best available information. Thus we have confidence that the health burden of sulphate has been assessed in a valid manner. Sulphate should be recognized as the environmental pollutant deserving of the highest priority for strong abatement measures within Hamilton-Wentworth, as well as major efforts to minimize inputs to the region from sources upwind.

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[1] Data from "1991-1992 Air Quality data Summary Regional Municipality of Hamilton-Wentworth." 1993. MOEE West Central Region.

3.7 Conclusions

1. Data and resources exist within Hamilton-Wentworth to estimate the health burden of air pollution.

2. The process of review of the literature for the most recent (or most valid) dose-response relationships is a substantial task. This as well as estimation of air-pollution related health outcomes requires the work of experienced scientists from the academic and/or government community.

3. In Hamilton-Wentworth, close to 1% of all non-trauma hospital admissions and up to 9% of all non-trauma deaths are attributable to air pollution.

4. In Hamilton-Wentworth, fine particle pollution (and especially its sulphate component) represents the greatest air pollution problem, being responsible for nearly half of the air pollution related hospitalization and premature mortality.

3.8 Recommendations

1. To estimate the health burden of air pollution in a community it is necessary to have access to three kinds of information: community specific health resource utilization (e.g., hospital admissions); community specific air quality; and dose-response relationships.

2. In order to develop community-related estimates of health burden, the dose-response estimates (for events) should be in the form of risk ratios for a quantified change in pollutant level.

3. A monitoring network maintained to the highest standards is essential to the delivery of air quality data for the purpose of estimating health effects.

4. Access to the health information must be facilitated through a suitable local agency, such as a University research uuit, or the Public Health Department.

5. Data interchange formats with respect to air quality and health outcomes should be discussed at the outset of an estimation exercise, and a standard format agreed upon.

6. The resource commitment of a risk assessment exercise should not be underestimated.

7. The HAQI model could be used to develop a risk assessment protocol for the health effects of air pollution in other communities.

8. Sulphur-species pollution in Hamilton-Wentworth should be reduced significantly from its current level.

PART FOUR: Assessment of the Potential Impact on Human Health of Long-Term Exposure to Air Pollutants - Group #4 (The Air Toxics).

4.1 Introduction

This section summarizes the potential human health impact of long-term exposure to selected environmental contaminants in Hamilton-Wentworth air. The goal of the study was to get a better understanding of the health risks involved and to determine which of the contaminants have the greatest potential to cause an adverse impact on human health.

Most of the airborne compounds considered were carcinogens; however lead and manganese were assessed primarily for their adverse effect on the nervous system, and cadmium was assessed taking into account both its cancer-causing properties and its ability to induce kidney damage (see Table 4.1).

The compounds for the analysis were selected based on their proven toxicity, on their likely presence in the environment and on the availability of adequate monitoring data. Due to the constraints of time and the availability of data, only the risk from *inhalation* of the selected toxicants was assessed. This assessment may therefore underestimate the risk from the exposure to the selected toxicants from all routes of exposure.

Table 4.1. List of Airborne Contaminants - Group #4, "The Air Toxics".

Compound	Endpoint
Cadmium	lung cancer and kidney toxicity
Chromium (VI)	lung cancer
Manganese	neurotoxicity
Lead	neurotoxicity in children
Benzene	cancer (leukemia)
1,3-Butadiene	cancer (leukemia)
PAHs (as benzo[a]pyrene)	lung cancer

People living in Hamilton-Wentworth are likely to be exposed to the toxicants to different degrees depending on the location of their home and workplace and on individual and family lifestyles. While lifestyle factors have an effect on human health risk, the main exposure scenario evaluated for this report emphasized exposure to outdoor ambient air in Hamilton-Wentworth. This is in keeping with the mandate of the Human Health Working Group report of the Hamilton Air Quality Initiative (HAQI).

The potencies of the selected contaminants were obtained from recent MOEE assessments whenever available. The remainder of the toxicity assessments were obtained from the U.S. Environmental Protection Agency's Integrated Risk Information System database (IRIS, 1994) or the World Health Organisation (WHO, 1987).

The estimated health risks of the carcinogens were expressed both as the *lifetime individual cancer risk* and as *annual incidences* (cancer cases per 100,000 population). The lifetime cancer risk estimate is a calculation of the probability that an individual may develop cancer after a lifetime of exposure to a specific compound or group of compounds (see *risk characterization* for further details). Annual incidences are expressed as the annual number of cancer cases per 100,000 population. This calculation permits comparison of the estimated annual incidence with Canadian cancer statistics on a national, provincial or regional basis. It should be noted that lifetime cancer risk estimates for the airborne contaminants in this study are based, in most part, on animal studies and are upper estimates of risk, whereas the Canadian cancer statistics are based on actual medical records.

The lifetime cancer risks to adults with primarily outdoor exposure to non-threshold toxicants are illustrated in Figure 2.

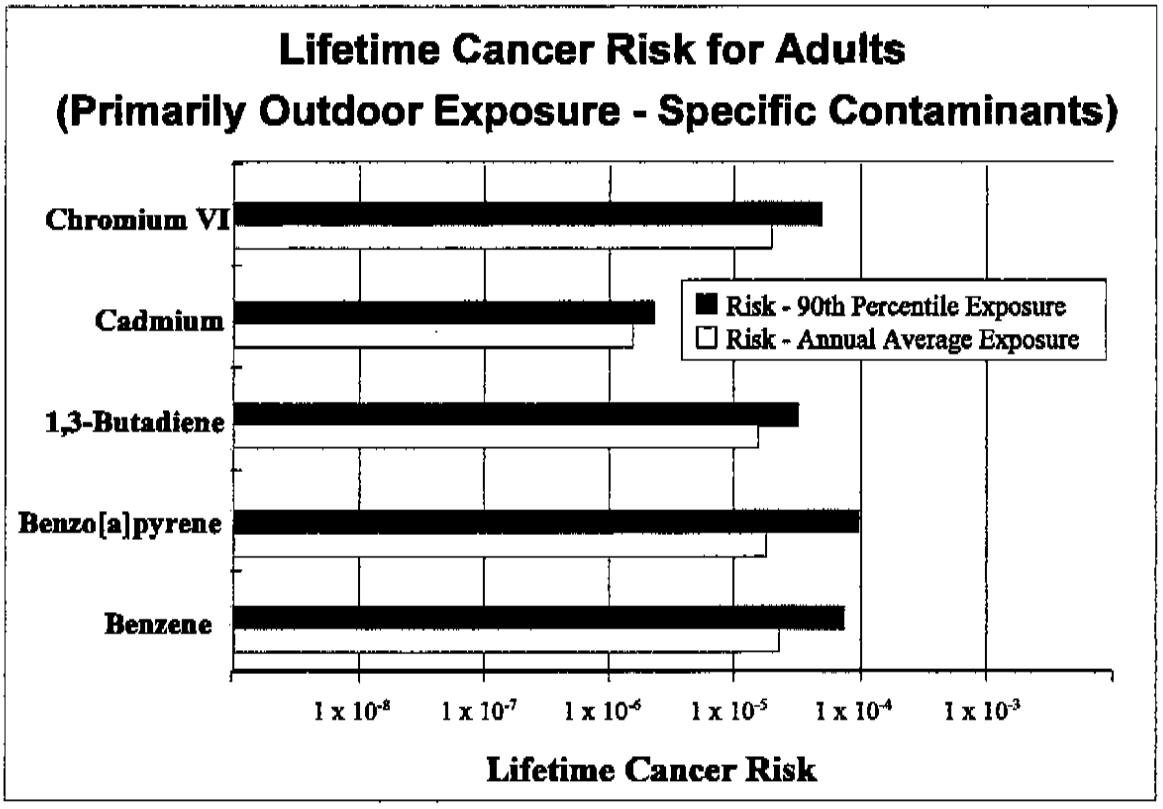
The average lifetime risk of all cancer (lung cancer and leukemia) from the inhalation of the 5 selected cancer-cansing compounds in outdoor Hamilton-Wentworth air is 1.1×10^{-4} assuming average levels of exposure and 3.1×10^{-4} assuming 90th percentile lifetime levels of exposure. Based on inhalation exposure to the selected airborne carcinogens causing lung cancer (cadmium, chromium (VI) and benzo[a]pyrene), the average lifetime lung cancer risk for an individual is estimated to be 5.7×10^{-5} (90th percentile: 1.7×10^{-4}). The estimated lifetime leukemia risk (due to inhalation of benzene and 1,3-butadiene) for an individual also averages 5.7×10^{-5} (90th percentile: 1.4×10^{-4}).

As an annual incidence, the average number of cases of lung cancer due to these selected airborne contaminants is estimated to be 0.08 cases per 100,000 (90th percentile: 0.24 cases per 100,000). This is less than one percent of the expected annual lung cancer incidence of 51 cases per 100,000 for Hamilton-Wentworth based on cancer statistics for Ontario. As an annual incidence, the number of cases of leukemia due to the selected airborne contaminants is estimated to average 0.08 cases per 100,000 (90th percentile: 0.2 cases per 100,000). This is about one percent of the expected annual leukemia incidence of 12 cases per 100,000 for Hamilton-Wentworth based on annual leukemia incidence rates for the province

Based on provincial cancer statistics, the expected annual incidence of all cancer cases for Hamilton-Wentworth (population 467,900) is about 414 cases per 100,000 (or a total of approximately 1940, compare with Table 3.8). In this study, the estimated average annual incidence for all cancers due to inhalation of the selected contaminants (lung cancer and leukemia only) is 0.16 cases per 100,000 (90th percentile: 0.44 cases per 100,000). For the 1994 Hamilton-Wentworth population, this annual incidence rate would mean an average of 0.7 additional cases (90th percentile - 2 cases). In other

words, less than 0.2% of the expected number of cases for Hamilton-Wentworth can be attributed to the inhalation of the air toxics selected for this initiative.

Figure 2. Lifetime Cancer Risk for Outdoor Adults.



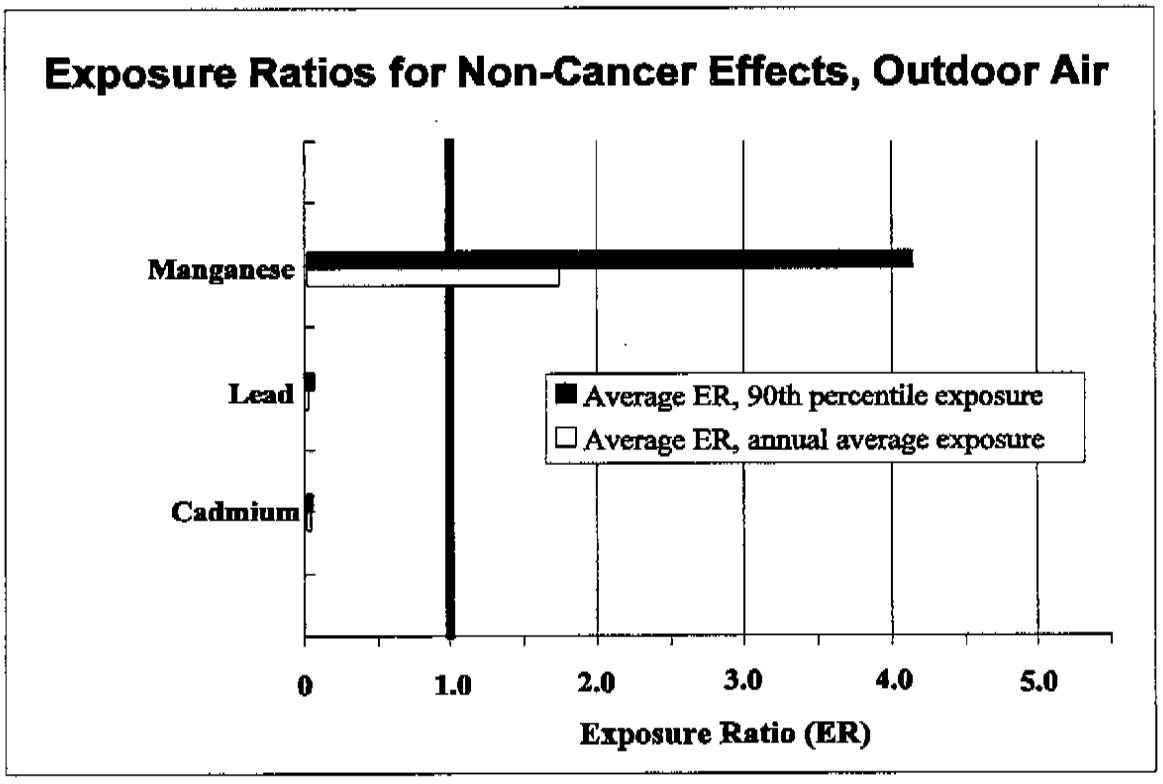
The estimated health hazards of the noncarcinogens were expressed as the exposure ratio (ER), which is the ratio of estimated exposures calculated from Ministry air monitoring data to provincial air quality criteria or other stringent exposure limits (see *risk characterization* for further details). An ER value of less than one indicates that exposure limits are not being exceeded and no adverse health effects are anticipated. An ER greater than one indicates that an exposure limit has been exceeded. At this time, due to the way exposure limits for noncarcinogens are developed, it is difficult to predict the severity of the adverse effect or the number of cases that might occur in the population. The ERs for the noncarcinogens in this study are listed in Figure 3. Note that cadmium has been evaluated as both a carcinogen and as a noncarcinogen as it may cause lung cancer or kidney damage, respectively, at similar exposure levels.

Based on the ERs, it appears that inhalation of lead in Hamilton-Wentworth air should not impact on health. Note, however, that a major concern with lead is the exposure of children through oral intake of contaminated soil and dust (MOEE, 1994a). The deposition of lead from the air will

contribute to its concentration in soil and dust. The assessment of this route of exposure is outside of the scope of this study. As such, the hazard posed by airborne lead may be greater than that estimated using the ERs based solely on inhalation.

The ERs for manganese exceed one. However, a large uncertainty factor was incorporated into the derivation of the USEPA air quality criterion for this compound used in this study suggesting that this exceedance is unlikely to be associated with adverse effects.

Figure 3. Exposure Ratios for Noncancer Effects.



4.2 Background

This study assesses the risk or potential impact on human health of selected toxic airborne contaminants present in Hamilton-Wentworth. Only adverse health effects potentially arising from long-term (chronic) exposure are assessed. The short-term (acute) effects of air contaminants on the respiratory tract are evaluated elsewhere in the Human Health Working Group report. This study consists of three parts:

- Description of the assessment process
- Summary of results and discussion
- Contaminant-specific assessments

4.3 Description of the Risk Assessment Process

This assessment follows accepted risk assessment procedures, similar to those described by USEPA 1989, Summerhays, 1989 and MOEE, 1994c. In principle, estimating health risks consisted of the following four major components:

- hazard identification
- dose-response assessment
- exposure assessment
- risk characterization.

4.3.1 Hazard Identification

The first component, called *hazard identification*, is used to determine how the rest of the assessment should be conducted. It describes the chemical, physical and toxicological properties of the pollutants and their distribution in the environment. This component is described separately for each selected compound (Section 4.5).

4.3.2 Dose - Response Assessment

Dose-response assessment is the step of the risk assessment process where the relationship between the level of exposure and the magnitude of the risk is estimated. The toxicity of a contaminant is often expressed as a lifetime risk per unit of exposure (e.g., risk per $\eta g/m^3$). It may be also expressed as a threshold level of exposure, below which no adverse effect is expected to occur. In order to get a better understanding of dose-response assessment, it is useful to understand the concept of risk. This concept is described below.

Different toxicants have different mechanisms by which they induce their effects, and this can be reflected in the shape of their dose-effect relationship curves and subsequent assessment. Based on the shape of the dose-response curve, the toxicants and their effects fall into two general categories:

- threshold
- non-threshold

Threshold *versus* non-threshold dose-response effects

There are compounds which are believed to be able to cause adverse effects even at minute doses; although with a low probability of this occurring. As the level of exposure increases, so does the risk. Many carcinogenic compounds and some other toxicants fall into this category, and as a result, they are referred to as *non-threshold* toxicants (see Table 4.2).

In contrast, most noncarcinogenic compounds and some carcinogens are thought to cause no adverse effects unless a specific minimum exposure (threshold exposure) is achieved. Such substances are referred to as *threshold* toxicants.

Table 4.2 List of Air Contaminants Considered as Threshold and/or Non-Threshold Toxicants

Substance	Non-threshold/ Threshold	Unit Inhalation Risk	Reference Concentration (RfC)	Agency
Cadmium	non-threshold and threshold	1.8×10^{-6} $(\eta g/m^3)^{-1}$	20 $\eta g/m^3$	IRIS, 1994 WHO, 1987
Chromium (VI)	non-threshold	1.2×10^{-5} $(\eta g/m^3)^{-1}$		IRIS, 1994
Manganese	threshold		50 $\eta g/m^3$	IRIS, 1994
Lead	threshold		700 $\eta g/m^3$	MOEE, 1994a
Benzene	non-threshold	8.3×10^{-9} $(\eta g/m^3)^{-1}$		IRIS, 1994
1,3-Butadiene	non-threshold	1.1×10^{-7} $(\eta g/m^3)^{-1}$		IRIS, 1994
Benzo[a]pyrene *	non-threshold	2.3×10^{-5} $(\eta g/m^3)^{-1}$		MOEE, 1997

** "Benzo[a]pyrene" toxicity as assessed in this report represents the integrated toxicity of not only benzo[a]pyrene but also of other polycyclic aromatic hydrocarbons found in the Hamilton-Wentworth air. For further details, see section 4.3.*

Some compounds may exert *both* threshold and non-threshold effects. For example, inhaled cadmium may pose a lung cancer risk at low levels. However, there is also the potential for inhaled cadmium to cause renal toxicity at low levels; a threshold effect. In such circumstances, it is possible to assess the contaminant as a threshold toxicant, a non-threshold toxicant or both.

Methods for assessment of threshold and non-threshold effects

The distinction between threshold and non-threshold effects is necessary because the assessment of toxicity for the two groups of compounds is done differently. For the chemicals with threshold toxicity, the purpose of the dose-response assessment is to estimate the threshold concentration at which *no observable adverse effect concentration* (NOAEC) occurs. For threshold toxicants, NOAEC is a measure of toxic potency. The more potent the threshold toxicant, the lower the concentration at which no adverse effect is detected. By applying an appropriate *uncertainty factor* which accounts for the uncertainties in the estimate of this threshold, the *reference concentration* (RfC) is determined. The RfC is the recommended maximum daily intake and assumes a lifetime exposure.

Since there is no concentration at which a non-threshold contaminant will elicit no adverse effect, it is necessary to estimate the inhalation risk. The potency of non-threshold toxicants is generally expressed as the upper bound (95th upper confidence interval) of the initial slope of the dose-

response curve obtained from animal studies and extrapolated to humans. This slope estimates the increment of risk as the exposure is incrementally increased. This slope of the dose-response curve is usually used to derive the unit cancer risk which can be used to estimate the lifetime cancer risk associated with inhaling the contaminant in ambient air. For the most part, the potencies are expressed as unit risk (risk/ηg/m^3). This value is the slope of a line representing the relationship between the concentration level of a given toxicant in the air and the extra risk to a population exposed to this level of the toxicant over their lifetime.

The inhalation risk factors and RfCs used in this study are shown in Table 4.2.

4.3.3 Exposure assessment

The purpose of *exposure assessment* is to estimate the dose for the people exposed, usually personified as an individual receptor who exhibits typical exposure patterns. In this case, only the risk from inhalation of the selected toxicants was estimated. This assessment may therefore underestimate the risk from the exposure to the selected toxicants from all sources. The assessment may also underestimate the risk from air sources of contamination, because of the risk from areal deposition of the toxicants on local and home-grown foodstuffs and onto soil has not been evaluated. However, except for lead, the compounds selected are considered to exert most of their toxicity via inhalation route and it is assumed that the main source of risk for the selected compounds has been evaluated.

The breathing activity patterns used in the report are defined in Table 4.3. The patterns are based on published breathing exposure factors (USEPA, 1995), and the time spent exposed to different air sources selected from Ontario demographic statistics (Ontario Ministry of Treasury and Economics, 1986, MOEE, 1994d, Muller and Leece, unpublished). The outdoor adult receptor scenario used in this exposure assessment integrates the breathing activity patterns for different age ranges over the receptor's lifetime. Both personal exposures (mainly indoor) and outdoor exposures are incorporated into this scenario with an emphasis on outdoor activity.

Ambient air levels for any toxicant are subject to change with time and site where the measurements are taken. The levels for a toxicant may vary with season. For example, levels of benzo[a]pyrene and other PAH are higher in winter than in summer, at least in part due to the emissions from heating residences. The levels may also differ according to the cycles in industrial activity. Furthermore, the levels in different parts of Hamilton-Wentworth will depend on the proximity to industrial sources and traffic and to various meteorological and geographical characteristics. Air samples were taken in downtown, suburban and industrial areas in Hamilton-Wentworth (see Figure 4).

The concentrations in air for each air toxicant used to estimate the inhalation exposures reported here are presented in Tables 4.4 and 4.5. It should be noted that the values reported in these tables are based on early summaries of 1992 to 1994 monitoring and may differ slightly from more comprehensive summaries based on data obtained after this period reported elsewhere in these documents. These concentrations came from two types of monitoring programs; the Ministry's

regular outdoor air quality monitoring program (these "network" concentrations are based on year round monitoring) and personal exposure air quality studies conducted in 1993 and 1994 (these personal exposure concentrations are based on short term monitoring during the summer months to provide "snapshots" of the local air quality). Brief summaries of the two programs are provided below.

Outdoor Monitoring

Most outdoor measurements were obtained from the routine monitoring network in Hamilton-Wentworth. Details on how samples are collected and the data reported are provided in the annual air quality data summary for the Regional Municipality of Hamilton-Wentworth (MOEE, 1996a) and in an annual provincial air quality report produced by the MOEE (MOEE, 1995).

In order to account for the variation in the levels of toxicants in air, the *annual average* and the *90th percentile levels* were estimated for all sites. Annual average concentrations were calculated for each contaminant at each site by pooling the 1992 - 1995 annual average concentrations within each site and across those sites to be included in the above categories. Outdoor "90th percentile" levels were also determined, i.e., the levels below which 90% of the samples lie. It is unlikely that any person is exposed to these high levels on a regular basis. The 90th percentile level thus represents the highest level of toxicants to which Hamiltonians are exposed; although such exposure is unlikely on a consistent basis.

The selected airborne metals, including lead, manganese and cadmium, were determined in the total suspended particulate (TSP) fraction over a 24-hour sampling period, every sixth day, using the standard Ministry Hi-Vol network samplers and pre-weighed glass-fibre filters. Although the Hi-Vol pulls air across the filters at a flow rate of 28 lpm (litres per minute or 40 cfm (cubic feet per minute)), the resulting TSP accounts for airborne material in the sub-micron to 50 micron (μm; micrometre or one-millionth of a metre) aerodynamic size fraction. In the Hamilton area, there were 19 Hi-Vol stations in operation in 1994 with each reporting the TSP atmospheric loadings. However, only the filters from 6 of these stations were analyzed routinely for specific metals by using either XRF (X-Ray Fluoresence for manganese and lead) or ICP-MS (Ion Coupled Plasma - Mass Spectrometry for cadmium) methodologies.

Hexavalent chromium is not measured routinely in any Ministry ambient air monitoring network, but is assumed to be 20% of the total chromium detected on the Hi-Vol filters (MOEE 1994c).

For benzo[*a*]pyrene, the polycyclic aromatic hydrocarbon of interest, samples were initially acquired at 3 stations in the Hamilton area. The number of stations was later increased to four. The sampling began in 1992, operating on a 24-hour basis with a 12 day sampling period. The samplers were modified Anderson Hi-Vol samplers containing both filter (Pall Pallflex filters) and adsorbent material (Rohm and Haas Amberlite XAD-2 resin) in order to trap the particulate and vapour phase PAH material. After soxhlet extraction of the filter and adsorbent material, a scan of 34 different PAHs, including benzo[a]pyrene, was routinely analyzed by GC-MS (gas chromatography - mass spectrometry).

Table 4.3. The Exposure Scenarios Used in this Study

Scenario	Age Range (years)	Source	Average Respiration Rate (m^3/day)
Child	0 - 4	Outdoor	2.60
		Personal Exposure*	6.40
Student	5 - 15	Outdoor	2.90
		Personal Exposure	10.70
"Outdoor" Adult	16 - 65	Outdoor	11.40
		Personal Exposure	8.20
Retired	65 - 75	Outdoor	2.90
		Personal Exposure	10.70

** Personal Exposure includes time spent in offices, public buildings and travel as appropriate, but mainly represents time spent at home.*

Volatile organic compounds (VOCs) such as benzene and 1,3-butadiene were analyzed from cartridge samples exposed for 24-hours on a 12-day cycle at 5 different monitoring sites in the Hamilton area. The network sampling started in 1991 and as with the PAHs, the Ministry reports on a scan of approximately 35 different VOCs on a routine basis of which 21 have 24-hour AAQCs (ambient air quality criteria). The sampling rate is usually in the 5 to 10 ml / min (millilitres per minute) range and the adsorbent material is a 3-stage activated carbon mesh. The analysis is done by thermal desorption followed by GC-MS.

Table 4.4 Levels of Selected Toxicants in Outdoor Hamilton-Wentworth Air. (all levels presented as $\eta g/m^3$)

Contaminant	Mean Network Level - annual average	Standard Deviation	Mean Network Level - 90th %le	Standard Deviation
Benzene	3400	1114	6933	1960
1,3-Butadiene	100	na	167	116
Benzo[a]pyrene	1.4	0.2	4.9	1.8
Cadmium	1	na	1.7	1.2
Chromium (VI)	3.5	2	8.3	3.9
Lead	44.7	28	100	62.5
Manganese	146	94.7	320	170

ua - not available

Personal Exposure Studies

Many of the airborne concentration values used in the exposure assessment were acquired through special studies. Exposure assessments require detailed knowledge of air quality in the various environments in which people find themselves on a routine basis. For example, the airborne concentrations for the selected compounds of interest (such as benzene, manganese and benzo[a]pyrene) were needed in microenvironments such as indoors at home, indoors at the office, inside the car, etc.. Since this information is not contained in the routine Ministry network, the following special studies were conducted; the first as part of the 1993 Hamilton "Home Study", the second as a special size-fraction ambient study at the Ministry's network site located in downtown Hamilton (1994), and the third as a stand-alone benzo[a]pyrene exposure assessment study in northeast Hamilton (1996).

a) Airborne metals and volatile organic compounds (VOCs)

The Hamilton Home Study was conducted during the summer of 1993 during which concurrent indoor and outdoor 24-hour air quality sampling was conducted at 57 randomly chosen homes throughout the city of Hamilton. The objective of the study was to measure a wide variety of airborne compounds such as metals, VOCs and carbonyls (a special group of VOCs) both indoors and outdoors at different homes scattered throughout Hamilton. The metal and carbonyl data were analyzed from 24-hour samples whereas the VOC data were acquired from personal and area samplers acquired over various time periods ranging from 1 hour to several hours. The outdoor sampling was usually conducted in the backyard whereas the indoor sampling was done in the "people" place within the home. Usually one or two homes were sampled each day resulting in a 24-hour air quality snapshot. Since these 24-hour samples were gathered on different days, these data do not lend themselves to determining realistic long-time averages. Rather, the results were used to compare exposures /concentrations in three areas (mountain, downtown and industrial) of the city.

b) Personal PAH exposures

For personal exposure assessment studies, small portable PAH samplers were used in various microenvironments and a time diary was kept for each participant. Each sampler consists of a noiseless pump capable of drawing air across a 47 mm teflon-coated glass fibre filter (Zefluor 47 mm, Gelman Sciences) at a controlled flow rate set between 15 and 25 lpm for 24 hours. Since benzo[a]pyrene has a very low vapour pressure, the use of downstream resins or PUFs to collect vapour phase PAH was found to be unnecessary.

In 1996, a special personal exposure assessment study was conducted in the industrial area of northeast Hamilton. After monitoring indoors and outdoors at eleven different homes, a shopping mall and a union hall, the overall average benzo[a]pyrene concentration indoors was found to be 1.2 $\eta g/m^3$, whereas the paired outdoor concentration was 2.6 $\eta g/m^3$.

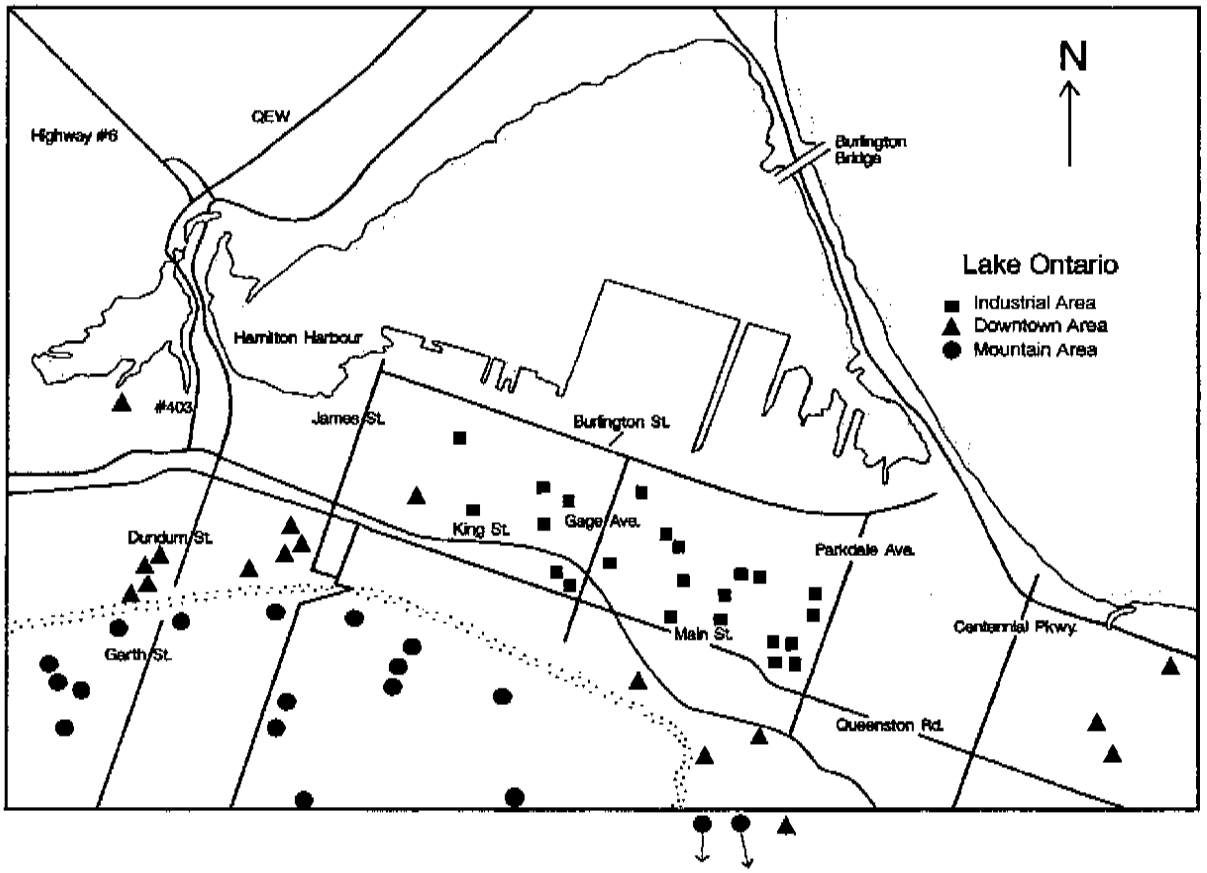
c) Hexavalent Chromium

The monitoring and analytical methodologies for detecting airborne hexavalent chromium at concentrations of less than 1 $\eta g/m^3$ (nanograms per cubic metre) were developed in the mid 1980s. Starting in 1992, a small pilot feasibility study testing these new methodologies was carried out in Windsor and the results were presented in the personal exposure assessment technical report comprising the *Windsor Air Quality Survey* (MOEE, 1994d).

This new sampling methodology consists of pulling air at a flow rate of 15 lpm for 24 hours through a Greenberg-Schmidt impinger filled with 250 ml of a sodium carbonate and sodium bicarbonate solution. The analytical method consisted of cleaning and preconcentrating a small aliquot of the solution on a Dionex AG4A anion exchange cartridge and then determining the hexavalent chromium concentration using ion chromatography.

For the Hamilton "Home" Study, concurrent 24-hour samples were acquired indoors and outdoors at 33 different homes. The average concentration of hexavalent chromium determined indoors was 0.23 $\eta g/m^3$ whereas outdoors, it was 0.65 $\eta g/m^3$. Further statistical analysis revealed that there was little or no relationship between the indoor and outdoor sample sets ($r^2 < 0.02$). Furthermore, the results from the size-fraction study (1994) showed that the majority of the airborne hexavalent chromium was in the inhalable fraction.

Figure 4: The Hamilton "Home" Study



As with the outdoor monitoring, *annual average* and the *90th percentile* levels were estimated for each of the personal exposure media (see above and Table 4.5) .

Table 4.5 Levels of Selected Toxicants in Personal Exposure Studies of Hamilton-Wentworth Air (units: ηg/m³)

Contaminant	Mean Level Annual Average	Standard Deviation	Mean Level 90th %le	Standard Deviation
Benzene				
Indoor	3900	691	8213	2818
Outdoor	5425	7054	26750	45515
1,3-Butadiene				
Indoor	300	278	675	826
Outdoor	350	500	1650	3100
Benzo[a]pyrene				
Indoor	1.1	1.1	4	4.6
Outdoor	1.9	2.1	6.7	8.2
Cadmium				
Indoor	1	0.7	2.1	1.8
Outdoor	1	0.1	2.7	2
Chromium (VI)				
Indoor	0.4	0.3	0.7	0.7
Outdoor	1.2	1	3	2.9
Lead				
Indoor	11.1	3	22.8	10.7
Outdoor	26.3	7.1	57.5	16.1
Manganese				
Indoor	13.2	7.8	29.3	18.6
Outdoor	65.5	44.3	142	120

4.3.4 Risk characterization

In this stage of the assessment, the information about toxicity of the contaminants from the *dose-response assessment* and the exposure information for the selected outdoor adult receptor from the *exposure assessment* are combined to determine the health impact.

Non-threshold Risks

For non-threshold coutaminants (carcinogens), it is possible to estimate risk from exposures to the population. The summary of results of the analysis is presented in Section 4.4. The results of individual assessments can be found in Section 4.5. The health risks of carcinogens are expressed as the *lifetime individual cancer risk* and as *annual incidences* (cases per 100, 000 population). The lifetime cancer risk estimate is a calculation of the probability that an individual may develop cancer after a lifetime of exposure.

Defining a tolerable level of risk can be controversial and may depend not only on the nature of the substance of concern but also on the circumstances of exposure, background concentrations and jurisdiction/organisation. The definition of "tolerable" depends on one's perspective. However, for exposure to environmental contaminants, most regulatory agencies and advisory organisations have generally defined tolerable risks as ranging from a one-in-a-million to a one-in-one hundred-thousand probability of an adverse event, often abbreviated as "$1x10^{-6}$" and "$1x10^{-5}$", respectively (see Section 4.6 - Appendix for some examples of risks).

Annual incidences are expressed as the annual number of cancer cases per 100,000 population (this calculation permits comparison of the estimated annual incidence with Canadian cancer statistics on a national, provincial or regional basis). In this study, two types of annual incidences are reported; annual incidence *estimates* calculated from inhalation of selected air contaminants, and, annual incidences reported in Canadian cancer statistics. It should be noted that lifetime cancer risk estimates (and annual incidence estimates) for the airborne contaminants in this study are based, in most part, on animal studies and are upper estimates of risk, whereas, the Canadian cancer statistics are based on actual medical records. The estimated annual incidences were calculated based on the 1991 population estimate of 467,900 for Hamilton-Wentworth, and, the individual lifetime cancer risk estimates (currently based on a 70 year lifetime). The 1994 Canadian cancer statistics (NCIC, 1995) were used as these were the most recent data available when the study began.

Threshold Risks

For threshold contaminants (noncancer effects), it is useful to calculate the ER, the ratio of the estimated exposure to an exposure limit. The exposure limit may be an RfC determined in the *dose-response assessment*, or a standard, guideline or other criterion set by a regulatory or advisory agency. If the ER is approximately 1, it signifies that the exposure is approximately equal to the RfC, guideline, etc. A large ER indicates possible health concern because the exposure levels exceed those at which no adverse effects are likely. An ER of ≤ 1 indicates little or no health concern. The RfCs for ERs used this report are shown in Table 4.2.

In interpreting the results, it is necessary to exercise caution. There are data gaps and other sources of uncertainty in any risk assessment.

4.3.5 Uncertainty in risk assessment

Ideally, environmental risk assessment would be based on human toxicological data where the subjects were exposed to the low environmental levels of toxicants. The exposures of assessed population could be accurately determined. The interaction between toxicants in the environment would be well understood and readily quantifiable. However, every risk assessment is conducted under much less favourable circumstances and the uncertainty stems from the gaps of knowledge due to incomplete and imperfect data. It is useful to distinguish between two sources of uncertainty.

The first type is the mathematical uncertainty which is associated with variability of the data and numerical processing of the data. This is the type of uncertainty that has long been used in risk assessments, but is probably less important than the second source of uncertainty.

This second source of uncertainty arises in every risk assessment and is caused by the necessity of compensating for and filling gaps in data using judgement in combination with missing, incomplete or unreliable data. It is this second type of uncertainty, the uncertainty associated with professional judgements, that is usually the largest source of error. Unlike the uncertainty related to the variability of data, the judgement-related uncertainty is mnch harder to express in numbers. For example, it is difficult to predict how reliable the estimate of potency is in humans when it is extrapolated from the potency in rodents. Humans and rodents often differ in metabolism and in sensitivity to a given contaminant; but how much these differences will affect the potency is often hard to estimate.

Exposure assessment is associated with less uncertainty. The sources of uncertainty include the uncertainty about the concentration levels at the sites of exposure, the lifestyle characteristics and the time-activity information of potentially exposed populations. The uncertainty associated with the exposure assessment is to a large extent captured in the range of exposures estimated for different exposure scenarios.

4.4. Summary of Results and Discussion

4.4.1 Risk from exposure to the selected contaminants

Non-Threshold Risks

The health risks of carcinogens were expressed as the *lifetime individual cancer risk* and as *annual incidences* (cancer cases per 100,000 population) (see Section 4.3.4). It should be noted that lifetime cancer risk estimates for the airborne contaminants in this study are based, in most part, on animal studies and are upper estimates of risk, whereas the Canadian cancer statistics are based on actual medical records. Cancer risks and annual incidences for chemicals with similar endpoints (e.g., lung cancer, leukemia) were pooled.

Lifetime individual cancer risks

The lifetime cancer risks to adults with a mainly outdoor exposure to non-threshold toxicants are illustrated in Table 4.6 and Figures 5 and 6.

Table 4.6 Lifetime Cancer Risks for "Outdoor" Adults

Lifetime risk (all cancer)						
Contaminant	Average Risk	Std Dev average	Risk, 90th %le	Std Dev 90th %le	% Contribution average	% Contribution 90th %le
Benzene	0.00004	0	0.00009	0	32.7	28.4
Benzo[a]pyrene	0.00003	0	0.0001	0	24.2	31.4
1,3-Butadiene	0.00002	0	0.00006	0	19.5	17.7
Cadmium	0	0	0	0	1.5	1
Chromium (VI)	0.00003	0	0.00007	0	26.4	22.3
Total Lung Cancer[1]	0.00006		0.00017			
Total Leukemia[2]	0.00006		0.00014			
Total (All cancer)[3]	0.00011		0.00031			

1. Sum of benzo[a]pyrene, Chromium (VI) and cadmium risks.
2. Sum of benzene and 1,3-butadiene risks.
3. Lung cancer and leukemia only.

Figure 5. Lifetime Cancer Risk for Adults.

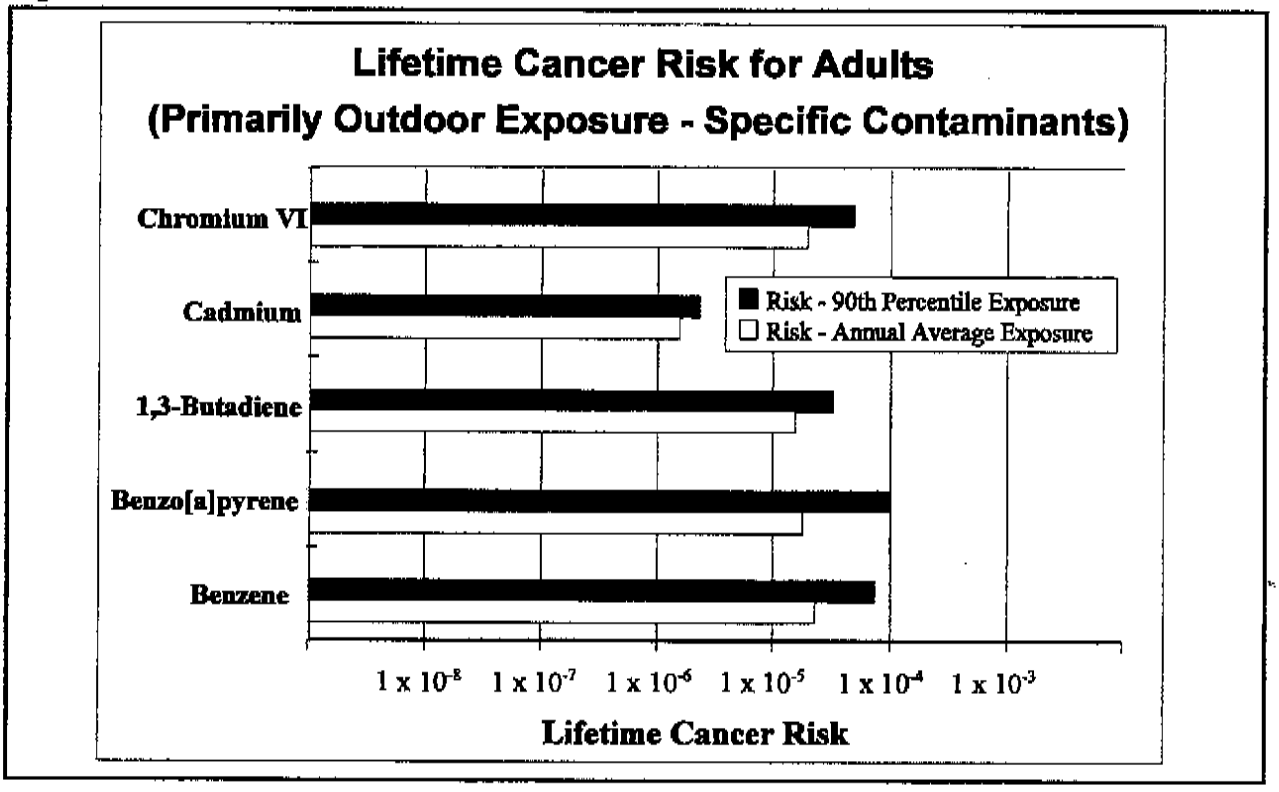
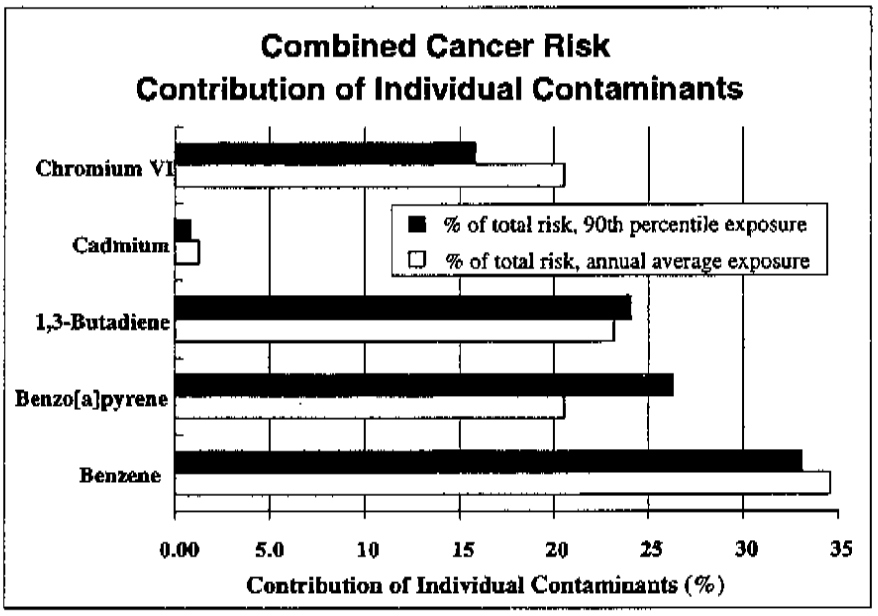


Figure 6. Lifetime Cancer Risk (% of Total)



The estimated lifetime lung cancer risk (benzo[a]pyrene, chromium (VI) and cadmium risks) for an individual averages 5.7 x 10^{-5} (90^{th} percentile: 1.7 x 10^{-4}). The estimated lifetime leukemia risk (benzene and 1,3-butadiene risks) for an individual also averages 5.7 x 10^{-5} (90^{th} percentile: 1.4 x 10^{-4}). The average lifetime risk of all cancer (lung cancer and leukemia) from the inhalation of the 5 selected cancer-causing compounds in outdoor Hamilton-Wentworth air is 1.1 x 10^{-4} assuming average levels of exposure and 3.1 x 10^{-4} assuming 90^{th} percentile lifetime levels of exposure.

Annual Incidences

As an annual incidence, the number of cases of lung cancer due to the selected airborne contaminants is estimated to average 0.08 cases per 100,000 (90^{th} percentile: 0.24 cases per 100,000). This is less than one percent of the expected annual lung cancer incidence of 68 cases per 100,000 for Hamilton-Wentworth based on 1994 cancer statistics for Ontario.

As an annual incidence, the number of cases of leukemia due to the selected airborne contaminants is also estimated to average 0.08 cases per 100,000 (90^{th} percentile: 0.2 cases per 100,000). This is about one percent of the expected annual lenkemia incidence of 13 cases per 100,000 for Hamilton-Wentworth based on annual leukemia incidence rates (1994) for the province.

The expected annual incidence of all cancer cases for Hamilton-Wentworth (population 467,900 in 1991) is about 414 cases per 100,000 (or a total of 1937 cases). As a comparison, refer to Table 3.8 in Section 3.4.1.3 *Cancer Incidence and Mortality*. The estimated average annual incidence for all

cancers due to inhalation of the 5 selected contaminants in this study is 0.16 cases per 100,000 (90th percentile: 0.44 cases per 100,000). For the 1991 Hamilton-Wentworth population, this annual incidence rate would mean an average of 0.7 cases (90th percentile - 2 cases).

Threshold Risks

The threshold (noncancer) health impacts of the specific contaminants expressed as ERs are provided in Table 4.7 and Figure 7. The figure shows that ERs greater than 1 have been estimated for manganese. For the 90th percentile exposure level, the ERs tend to be about double those for the average level of exposure. The advantage of the ER is that it allows rapid identification of compounds that exceed exposure limits. An ER of ≤ 1 indicates little or no health concern, whereas an ER >1 indicates that the likely no-adverse-effect exposure limit has been exceeded. At this time, due to the way exposure limits for noncarcinogens are developed, there is no way of predicting the severity of the adverse effect or the number of cases that might occur in the population.

Note however that ERs greater than 1 do not necessarily indicate relatively larger health impacts. For example, if the health impact of a contaminant rises slowly with dose, the health concern may be less than with another contaminant where the health impact increases steeply with increasing dose. Or, depending on the confidence in the dose-response assessment, the uncertainty factor may be quite large; a fact which would also suggest that minor exceedances would not result in adverse effects. The ERs for manganese exceed one. However, a large uncertainty factor was used in the derivation of the USEPA air quality criterion for this compound used to evaluate potential manganese effects in this study. This suggests that this exceedance is unlikely to be associated with adverse effects.

Table 4.7. Lifetime Exposure Ratios (ERs) - all noncancer

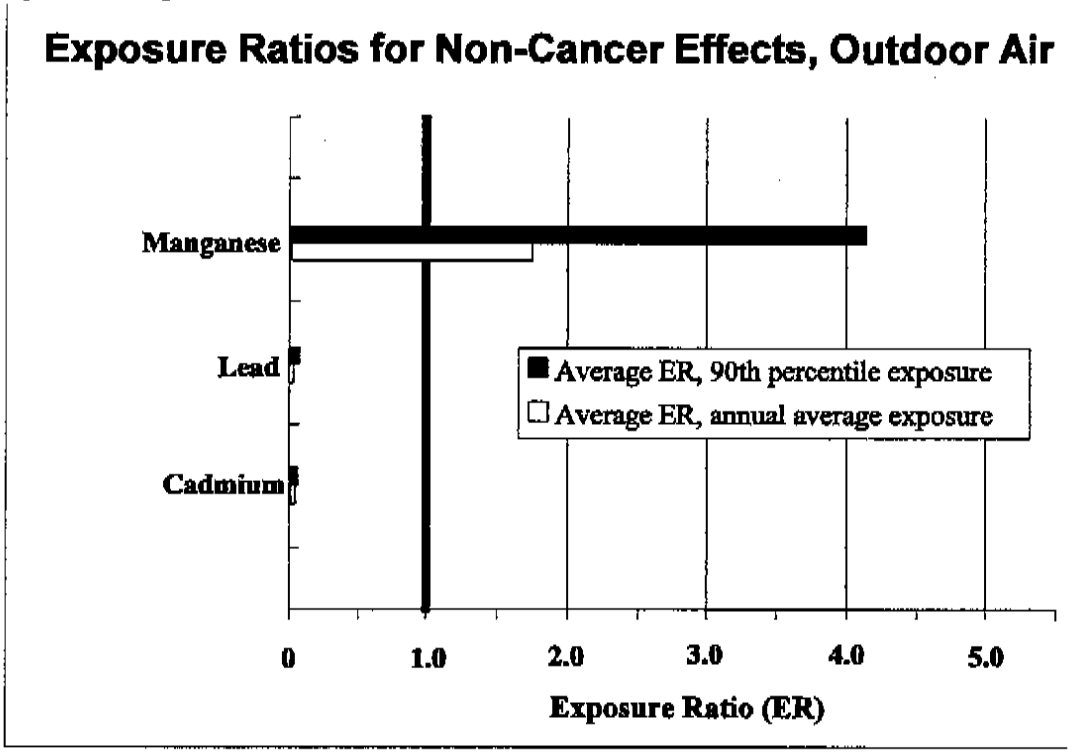
Lifetime ER (all noncancer)						
Contaminant	ER, average exposure	Std Dev, average	ER, 90th percentile	StdDev, 90th %le	% Contribution average	% Contribution 90th %le
Cadmium	0.04	0.01	0.08	0.07	2.2	1.9
Lead	0.04	0.02	0.09	0.04	2.2	2.1
Manganese	1.76	0.76	4.1	1.38	95.6	96
Total	1.84		4.27			

Two assessments were made for cadmium based on two different health effect endpoints. One was based on the carcinogenic potential of cadmium as estimated by U.S. EPA (IRIS 1992). The other was based on cadmium's renal toxicity (i.e., ability to cause kidney damage) using the Ministry's proposed (MOEEb, 1996) ambient air quality standard for comparison in calculating an ER.

Based on the results summarized in Figure 6, it appears that benzene, benzo[a]pyrene, 1,3-butadiene and hexavalent chromium contribute about equally to the overall cancer risk, while cadmium contributes relatively little. The numerical differences in risk estimates attributable to the compounds other than cadmium are relatively small and are likely to be a reflection of different assessment methods rather than significant differences in risk. However, 1,3- butadiene levels were frequently below the detection limit and for the purposes of this assessment it was assumed that the levels are *at* the detection limit. This tended to overestimate the apparent risk from 1,3-butadiene.

Based on the ERs, it appears that exposure to airborne lead does not have a large impact on health at the levels measured in Hamilton-Wentworth. Note however, that the main concern is the oral exposure in children (MOEE, 1994a) and the deposition of lead from the air will contribute to soil and dust levels. The assessment of this oral route of exposure was outside of the scope of this study.

Figure 7 Exposure Ratios (ERs) for Noncancer Effects.



4.5 Assessment of Health Impact of Individual Compounds

4.5.1 Benzene

Hazard identification

The following is a brief review of benzene's properties. Further details can be obtained from Agency for Toxic Substances and Disease Registry (ATSDR, 1995). Benzene is a volatile, flammable colourless liquid. Most people can smell it at 1.5 to 4.7 parts of benzene per million parts of air (ppm) and taste it at 0.5 to 4.5 ppm in water.

Industrial processes are the main source of benzene in the environment. Benzene is used to manufacture a wide range of plastics as well as lubricants, dyes, detergents and drugs and pesticides. Emissions from waste and storage operations and from gasoline service stations are also an important source of exposure to benzene. Benzene is also released during combustion of coal, oil and oil products (for example, gasoline engine exhaust) and cigarette smoke is an important source of indoor exposure to benzene. Natural sources of benzene include volcanoes and forest fires.

In the air, benzene breaks down within a few days. In water and soil, it breaks down more slowly. Benzene is distributed between air, water and soil media. It volatilizes from water and soil and is in turn carried back to the ground by rain or snow. Benzene does not accumulate in plants or animals.

Most people are exposed to a small amount of benzene on a daily basis, primarily from the air. In general, the biggest source of exposure to humans is tobacco smoke, followed by automobile exhaust and industrial sources. Of these, tobacco smoke accounts for most of the exposure, even though more benzene is produced by the other two sources. The reason is that tobacco smoke contributes heavily to the benzene levels in indoor air and most people spend the majority of their time indoors.

The exposure from food, beverages or drinking water is generally much lower than the exposure from the air, but the exposure from drinking water can rise if it is drawn from a groundwater source (i.e., a well) which has become contaminated from leaking underground gasoline storage tanks, contaminated soil or a leaking landfill site.

The main route of entry of benzene to the body is by the lungs into the bloodstream. Benzene is then metabolised and the products of this degradation (metabolites) are eliminated from the body within about 48 hours. Much of the toxicity of benzene may be due to the compounds formed by its metabolism, rather than to benzene itself.

Short-term exposure to very high levels of benzene in air or food can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, unconsciousness and even death. However the levels at which such symptoms are observed are at levels much higher (700 to 20,000 ppm) than those likely to be found in the environment. High levels of benzene may also cause eye or skin irritation.

More relevant are the effects of long-term exposures to lower levels of benzene. Both human and animal studies provide supportive evidence that benzene damages both the humoral and cellular components of the immune system. It appears that benzene lowers the levels of antibodies in the bloodstream, and thus lowers the ability of the body to fight infections. It also lowers the level of a type of white blood cells called lymphocytes, which are involved in cellular immune reactions. These effects are demonstrable at levels of benzene in air of about 1 to 200 ppm, or about 100 times less than the lower range for the short-term effects described above.

Benzene also induces chromosomal abnormalities both in somatic cells which form the body tissues and in the reproductive cells, which are responsible for the maintenance of the hereditary characteristics carried over from parents to children. Benzene also induces cancer. The most characteristic is the *acute myeloid leukemia* (AML) which is associated with lower levels of normal red blood cells, a type of white blood cells called granulocytes and platelets which are an important component in the clotting mechanism. The end result is death from anaemia, infection or bleeding.

These effects were found to occur at the levels roughly comparable to those at which damage to the immune system and blood have also been reported. However, since benzene appears to interfere with the genetic material directly, it is likely that there is no threshold for its toxicity. As a result some damage, not necessarily demonstrable by the available methodology, could occur at lower environmental levels (see the discussion of threshold and non-threshold types of toxicity in the Introduction). For that reason, cancer was selected as an endpoint for the quantitative assessment. USEPA (IRIS, 1994) proposed an initial slope of the dose-response curve (unit risk) to be 8.3×10^{-6} / μg of benzene / m^3. This is the potency used in this report.

Results and Discussion of the Quantitative Assessment

The average lifetime cancer risk from inhalation of benzene is estimated to be 3.6×10^{-5} (90^{th} percentile: 8.8×10^{-5}) (see Table 4.8).

Table 4.8. Lifetime Cancer Risk from Inhalation Exposure to Benzene

	Risk, Average Exposure	Risk, 90^{th} Percentile Exposure
Average	0.000036	0.000088
Standard Deviation	0.000007	0.000019

4.5.2 Benzo[a]pyrene (B[a]P) and Polycyclic Aromatic Hydrocarbons (PAHs)

Hazard identification

Benzo[a]pyrene is one of a family of hundreds of compounds called polycyclic aromatic hydrocarbons (PAHs). Benzo[a]pyrene is often selected as surrogate for PAHs because the combination of relatively high environmental levels and relatively high level of toxicity result in it having a larger health impact than any other PAH identified in the environment. PAHs are organic

molecules consisting of at least three rings and usually at least two are fused benzene rings. Any two neighbouring rings share two adjacent carbon atoms. In some of the rings, carbon may he substituted for some other element, such as nitrogen, oxygen, sulphur, or a halogen. Benzo[a]pyrene itself is a relatively large ring unsubstituted compound. PAH are a large and heterogeneous group, but the most toxic members of this family known to date are the four- to seven-ring PAH. In this report, the discussion therefore focuses on the properties of this four- to seven-ring subset of the PAH family.

PAHs are generally insoluble in water but can be readily solubilized in organic solvents or organic acids. This means that in aqueous environments, PAHs are generally found adsorbed on particulates and on humic matter, or solubilized in any oily matter which may contaminate water, sediments and soil. The solubility of PAHs in water is inversely proportional to the number of rings it contains. Thus, three-ring PAHs tend to be more soluble in water than the five-ring compounds. PAHs are solid in room temperature.

Since PAHs tend to have low vapour pressures, they are usually adsorbed on the particulate matter in the atmosphere. The vapour pressure of PAHs is inversely proportional to the number of rings it contains. Thus, almost all five-ring compounds are found adsorbed to the air particulate, while three-ring PAHs are also found in vapour phase in the atmosphere.

In the atmosphere and in the presence of sunlight, PAHs undergo photo oxidation. Photo oxidation occurs much faster on particle-free PAHs than particle bound compounds. For example about half of benzo[a]pyrene will be oxidized in a matter of hours to days. PAHs in the air can also be oxidized by ozone, reactive compounds adsorbed on the particles, NOx and SOx.

The exposure to PAHs is primarily through ingestion and inhalation. Under normal circumstances, dermal contact with PAHs is relatively unimportant. Similar to other oil-soluble compounds, PAHs are generally well absorbed in the body, but are stored in the body only briefly, primarily in the kidney, liver and spleen. Most of the absorbed dose is then excreted into bile and eventually faeces and to a much lesser extent urine. Most of the PAHs are excreted in a metabolized form and only very small amounts of the parent compound find its way into faeces and urine. PAHs are highly soluble in fats. In this form they can rapidly enter cells and become virtually unavailable for excretion. Metabolic processes tend to make PAHs more water soluble, which facilitates excretion.

A number of metabolic processes compete to produce a variety of different metabolites. In general, we recognize phase I reactions, which add one or more hydroxyl groups to the parent core and phase II reactions, which attach highly water-soluble groups to the PAH molecule. The phase I reactions are controlled by enzymes epoxide hydrolase and a subset of cytochrome P-450 mixed-function oxidases called AHH. The structures of PAHs vary greatly, but the metabolism of these compounds is similar and leads to the formation of homologous metabolites.

The differences in tumorigenicity of these compounds are due to the differences in the location of the metabolic modifications and the activities of the intermediate metabolites formed. Some of the metabolites formed are diol epoxides, and some of these diol epoxides are in turn converted into

carbonium ions. It is these alkylating compouuds that are thought to be the primary carcinogens, acting as initiators. Initiation is the first step in the development of cancer.

These carbonium ions also react with DNA and proteins to form adducts, and induce genotoxic damage as well. The enzymes required for the conversion of parent PAH compounds into the reactive diol epoxides are found mainly in the liver, but also in the lung, skin basal cell layer, intestinal mucosa and other tissues.

PAHs have been shown to induce a number of toxic effects. Some of them, however, are unlikely to be a cause for concern at environmental levels. Several PAHs have been shown to cause death in rodents, for example, after short-term exposure to high doses. On the other hand, no deaths have been reported from short-term occupational exposures to PAHs. Since environmental levels are generally much lower than some of the occupational ones, it is extremely unlikely that short-term exposure to PAHs in the environment would be a cause of death. On the other hand, eye irritation, photophobia and skin toxicity such as dermatitis and keratosis, have been demonstrated to be caused by occupational exposures to PAHs. Extreme environmental conditions (e.g., heavy exposure to a forest fire smoke) may also trigger the above effects.

Adverse respiratory effects have also beeu demonstrated experimentally and include acute and subacute inflammation, and fibrosis. With benzo[a]pyrene, severe and long-lasting hyperplasia and metaplasia were observed. These exhibit themselves as precancerous lesions and are consistent with the general assertion that one of the main targets of PAH toxicity is the respiratory tract. Available data are insufficient to assess the effects of PAHs at the environmentally relevant concentrations.

Carcinogenic PAHs, but not the noncarcinogenic ones, have been reported to suppress immune reaction in rodents. A number of authors have reported immunosuppressive effects in a dose-range similar to that at which carcinogenicity has beeu observed. Furthermore, there appears to be a rough correlation between the potency of PAHs as immunosuppressors and carcinogens. Immunosuppression may therefore be an important endpoint by which PAHs induce their toxic effects. Some authors also suggest that immunosuppression may be involved in the mechanisms by which PAHs induce cancer. At present, however, the data on immunosuppression are not sufficient for quantitative assessment.

Exposure to PAHs can have a number of effects on both female and male reproductive systems and on fetal development. The largest amount of data is available for rodent fetal development, where reported effects include malformations, stillbirths, resorptions, immunosuppression, clastogenicity and tumorigenicity. The doses required to produce the reported effects are generally similar or somewhat higher than those required to elicit a carcinogenic response. Although no human data are available, this may be an important toxic effect in humans. Unfortunately, there is insufficient data to assess this effect quantitatively either.

Genotoxic effects for some PAHs have been repeatedly demonstrated both in *in vivo* tests in rodents and *in vitro* tests using mammalian (including human) cell lines, as well as in prokaryotes. On the

other hand, some PAHs appear not to be genotoxic. Most of the unsubstituted PAHs which are categorized as genotoxic are not genotoxic in themselves, but need to be metabolized first by the aryl hydrocarbon hydroxylase (AHH) system. The diol epoxides that are formed then react with DNA to form DNA adducts and thus induce genotoxic damage. A genotoxic event is postulated as a required step in the carcinogenicity process and may play a role in some forms of developmental toxicity.

The tumorigenicity and carcinogenicity of individual PAHs and PAH-containing mixtures have been well studied in experimental animals. Virtually no data exist regarding the carcinogenicity of individual PAHs in humans, and only a limited amount of data on the human carcinogenicity of PAH-containing mixtures is available. There is evidence that a number of individual PAHs are carcinogenic in experimental animals, while others have been found to be non-carcinogenic. There is solid evidence that some PAH-containing complex mixtures are carcinogenic both to humans and experimental animals. Based on the available evidence, both the IARC, 1983 and USEPA (IRIS, 1994) classified a number of PAHs as carcinogenic to animals and some PAH-rich mixtures as carcinogenic to humans. For other PAHs, there is insufficient data to determine whether the compounds are carcinogenic or not.

Effects such as skin, eye and respiratory mucosa irritation are more likely to be seen with high occupational exposures rather than the characteristically lower environmental exposures. There may be exceptions to this conclusion; exposure to heavily contaminated soils or sediments, for example, may trigger these effects. Nevertheless, these effects were not studied well enough to conduct a quantitative assessment. PAH-induced immunotoxicity and developmental toxicity may be important toxic endpoints. Unlike carcinogenicity, there is a paucity of human data for immunotoxicity and developmental toxicity. Furthermore, animal data in support of the carcinogenicity endpoint are far more extensive than for immunotoxicity and developmental toxicity. In this report it is assumed that there is no threshold for the tumor-initiating effects of PAHs. This means that these toxicants are likely to pose a finite risk of cancer even at extremely low doses. In contrast, it is not clear whether or not there is a threshold for PAHs immunotoxicity and developmental toxicity. It is plausible that at environmentally relevant (low) levels, PAHs do not exhibit appreciable immunotoxicity and developmental toxicity. The dose-response assessment was therefore conducted on the tumorigenicity/carcinogenicity endpoint.

For the purpose of this report, the slope factor of 2.3×10^{-3} /μg of B[a]P/m^3 was used. This potency was developed by MOEE (MOEE, 1997). The potency does not apply to B[a]P alone. Instead B[a]P serves as a surrogate for all unsubstituted PAHs found in the PAHs fraction of airborne organic material. The potency for B[a]P alone would be significantly lower. The estimate developed by MOEE (MOEE 1997) takes into consideration the presence in the air of many PAHs besides B[a]P and the contribution of these other PAHs to the overall risk.

Results and Discussion of the Quantitative Assessment

The lifetime cancer risk from inhalation of PAHs is estimated to be 2.7×10^{-5} (90^{th} percentile: 9.7×10^{-5}) (see Table 4.9).

Table 4.9. Lifetime cancer risk from inhalation exposure to PAHs

	Risk, Average Exposure	Risk, 90th Percentile Exposure
Average	2.7×10^{-5}	9.7×10^{-5}
Standard Deviation	4.9×10^{-6}	3.4×10^{-5}

4.5.3 1,3-Butadiene

Hazard identification

Detailed review of the properties of this compound can be found in ATSDR, (1992a). In the environment, 1,3-butadiene is a colourless gas with odour similar to that of gasoline.

1,3-butadiene is released into the environment in large quantities. Most of it is released into the air, although liquid spills during transportation are also possible. It is produced from petroleum and it is used to produce mainly rubber and rubber products such as tires and some plastics. It is released into the air both intentionally and as fugitive emissions during its production, use, storage transport or disposal. 1,3-butadiene is also present in gasoline vapours, in the exhaust from cars and trucks and in the smoke from forest fires. Cigarette smoke also contains 1,3-butadiene.

Detailed information about soil and water levels of 1,3-butadiene is missing. However, based on the physical and chemical properties of this compound, it is expected that the levels will be low, because of rapid evaporation. In the air, this gas breaks down rapidly, within hours to days, depending on the amount of sunlight. It is also not expected that 1,3-butadiene is accumulated by plants or fish, but again, the detailed information is not available. Breakdown by water-based and soil-based microorganisms has been demonstrated experimentally.

The human exposure to 1,3-butadiene is primarily by inhalation, although low levels of this toxicant have been found in the drinking water and in plastic food containers, which may contaminate food with low amounts of this toxicant. Most of the general population is exposed to at least low levels of this compound as direct emissions from cars and trucks. In the indoor air, exposure from second-hand tobacco smoke may also be significant.

1,3 Butadiene is absorbed from the lungs by passive diffusion. Once absorbed, it distributes rapidly to many tissues, particularly those with relatively high fat content. The toxicant is rapidly metabolised (hours). Based on animal studies, the most important route of elimination is the exhalation of CO_2 and at higher levels of exposure, by formation of the epoxide metabolites. Urinary excretion plays lesser role and elimination in faeces is negligible.

Epidemiological studies suggested possible risk of heart, blood and respiratory diseases as well as cancer. Short-term exposures to 1,3-butadiene cause eye, nose and throat irritation. However, the exact levels which produce these effects are not known. Longer term exposures may cause heart,

blood and respiratory diseases as well as cancer. These findings are based on occupational studies. Until recently, the occupational studies were inconclusive, because of the number of technical difficulties which needed to be overcome in the study design. For example, workers have been exposed to a number of toxicants besides 1,3-butadiene (especially styrene) during the course of their work. It is difficult to determine whether 1,3-butadiene or styrene are the primary cause for the observed toxicity. Smoking habits, which affect the likelihood of occurrence of many of the above health problems were not taken into consideration. Furthermore, the studies may have been of too short of duration to observe the carcinogenic effect of 1,3-butadiene. However, there has been a progress in recent years . Santos-Burgoa et al., (1992) provided evidence, that the leukemias are associated with 1,3-butadiene rather than styrene. Delzell et al., (1995) demonstrated significant increases in the occurrence of leukemia in the synthetic rubber workers which had occupational exposure to 1,3-butadiene. The frequency of tumors increased with the years of exposure and with duration after the occupational exposure started. Furthermore, the workers which died of leukemia tended to have higher total lifetime exposures than the workers which did not die of leukemia.

Both USEPA (IRIS, 1994) and IARC, (1985) concluded that 1,3-butadiene is carcinogenic to animals and is a possible or probable carcinogen to humans. At the time these assessments were made, the above epidemiological studies were not available. It is quite possible, that upon re-evaluation, 1,3-butadiene will be ranked as human carcinogen. The existing ranking is based primarily on the animal data.

Genotoxic evidence for 1,3-butadiene comes primarily from mouse studies. Mice are particularly vulnerable to the effects of 1,3-butadiene. In contrast, the rat studies seem to be negative. The one human study which looked for significant change in the number of chromosomal aberrations in rubber workers was negative. In contrast, in a pilot study, exposed workers in a butadiene extraction plant (all nonsmokers) displayed a significant increase in the mutation in the *hprt* locus in the blood T-cells relative to their unexposed coworkers (Ward et al., 1994). The salmonella *in vitro* studies with metabolic activation were positive for two strains and negative for the TA 100 strain. Without activation, all three tests were negative.

On the whole, this information is consistent with a possibility, that 1,3 butadiene may be genotoxic in humans, although the evidence is not strong. Genotoxic carcinogens are generally considered to be non-threshold toxicants. This is the way 1,3-butadiene was treated in this report. The report utilizes the unit risk prepared by USEPA (IRIS, 1994) of 2.8×10^{-4} / μg of 1,3-butadiene / m^3. This potency estimate is based on animal data and may in the future be modified in the light of good new epidemiological data discussed above.

Results and discussion of the quantitative assessment

The lifetime cancer risk from inhalation of 1,3-butadiene is estimated to be 2.1×10^{-5} (90^{th} percentile: 5.5×10^{-5}) (see Table 4.10).

Table 4.10 Lifetime Cancer Risk from Inhalation Exposure to 1,3-Butadiene

	Risk, Average Exposure	Risk, 90th percentile exposure
Average	0.000021	0.000055
Standard Deviation	0.000008	0.000025

4.5.4 Cadmium

Hazard identification

Cadmium has been reviewed by ATSDR, (1992b). Most of the cadmium in the environment comes from man-made sources, especially from non-ferrous smelting, fossil fuel combustion, disposal of cadmium-containing products and the application of phosphate fertilizer. Other contributing sources include metal and phosphate fertilizer production and wood and waste incineration.

Cadmium is an element usually found as an oxide, chloride sulphate or sulfide. In these forms, it may be soluble. In air, cadmium is found as a component of fine particulate and is usually in the form of cadmium oxide. This form is stable and undergoes little atmospheric transformation. Cadmium particulate is either settled out by rain or snow or eventually dry deposited on land or surface water.

In water, cadmium is partially solubilized and partially a part of insoluble complexes. In sediment and soils, soluble cadmium is quite mobile, while the insoluble cadmium form is not very mobile. Cadmium is taken up and retained by aquatic and terrestrial plants and is concentrated in the liver and kidney of animals that eat the plants.

Non-smokers are exposed to cadmium primarily from food (about 94%) and most of the rest of the exposure comes from air. However, it is assumed, that only 5% to 10% of the ingested dose is absorbed, while about 25% of the inhaled dose is absorbed. Correcting for the differences in absorption, about 80% of uptake of cadmium comes from the food and most of the remaining cadmium is contributed by ambient air. Near major point sources of cadmium, the inhaled contribution may increase further. Smoking is a very important factor. Smoking of a pack of cigarettes a day contributes about the same amount of cadmium as the diet. Second hand tobacco smoke does not appear to increase the exposure to cadmium appreciably.

Cadmium can be absorbed, to different degrees from the respiratory tract, the digestive tract and the skin surface. Large particles (greater than 10 μm) tend to be deposited in the upper respiratory tree, while the finer particulate of about 0.1 μm tends to get deposited in the lungs. Only about 5% of this large particulate remains deposited in the upper respiratory tract. Much of the particulate that is not exhaled is transported up the respiratory tree and eventnally swallowed. In contrast about 50% the finer particulate is deposited into lung tissue where it remains available for absorption. About 50 to 100 % of the delivered dose is eventually absorbed. It is therefore estimated for the purposes of this report that about 25% of cadmium on fine particulate is absorbed.

The absorption of the cadmium from the digestive tract is approximately 3 to 5%, but may be higher (about double) in individuals with iron deficiency. Other dietary factors and physiological factors such as high fat or protein content in the diet, or zinc deficiency may also influence oral absorption of cadmium. The dermal absorption of cadmium is very low.

The distribution of cadmium in the body is relatively independent of the route of exposure. Cadmium is distributed throughout the body, with particularly high levels in the liver and kidneys. In the body most of the cadmium is bound to metallothionein. This small molecular weight protein is rich with cysteine, which is capable of binding cadmium efficiently. It is inducible in most tissues by the presence of cadmium. In this form, cadmium is relatively non-toxic and relatively large amounts of cadmium can be retained in the body in this manner. Cadmium bound to metallothionein is available for filtration in the kidney. There the metallothionein may be metabolised and the filtered cadmium may be reabsorbed again by the kidney and bound to metallothionein synthesized in that organ. It is the free cadmium which is thought-to account for the damage caused by this toxicant.

Faeces generally contain much higher levels of cadmium than urine, but that is because most ingested cadmium is never absorbed. The absorbed cadmium is eliminated slowly and about equally in urine and faeces. It is estimated that about 0.007% of body burden is elimiuated daily in faeces and about 0.009% in urine.

Cadmium is a cumulative toxicant and most of the concerns come from long-term exposure to that compound. The kidney is the main target of cadmium toxicity after intermediate to chronic exposure to this toxicant by either inhalation or oral route. The early sign of kidney damage from cadmium is the decreased reabsorption of filtered low molecular weight proteins. With longer exposures or at higher doses, there is a reduction in filtration of high molecular weight proteins and eveu necrosis (cell death). The impact of the early kidney damage on the overall health of affected individuals is not clear. However, the damage affects the vitamin D metabolism in the kidney. This in turn leads to calcium imbalance and the reduction in bone density. To account for kidney effects, a RfC for air exposure of 0.02 μg/m3 (WHO, 1987) was used..

The evidence that inhalation of cadmium is cancer-inducing in humans is weak, but it is strong for rats. Since the mechanism of action of cadmium across the species seems to be similar, the rodent data may be applicable to humans. The evidence suggests that inhalation of cadmium may cause the development of cancer in the lung. There is no evidence to suggest that cadmium is cancer-inducing by oral route. Both USEPA (IRIS, 1992) and IARC, (1982) classified cadmium as probable human carcinogen.

Cadmium has been shown to be genotoxic in some studies of humans exposed to cadmium but not in others. Similarly, cadmium was shown to be genotoxic in some of the experimental tests, both *in vivo* and *in vitro*. Compounds which exhibit genotoxic behaviour are generally treated as non-threshold toxicants (see Section 2.2 for details). USEPA proposed 1.8 x 10^{-3} / μg of cadmium / m^3 of air as the unit lifetime risk of respiratory cancer. This is the unit risk used in the calculations in this report.

Results and discussion of the quantitative assessment

The average cancer risk from inhalation of cadmium was estimated to be 1.6×10^{-6} (90^{th} percentile: 3.1×10^{-6}) (see Table 4.11). The lifetime noncancer ER due to inhalation of cadmium is estimated to be 0.04 (^{90th} percentile: 0.08) (see Table 4.12).

Table 4.11 Lifetime Cancer Risk from Inhalation Exposure to Cadmium

	Risk, Average exposure	Risk, 90^{th} percentile exposure
Average	0.000002	0.000003
Standard Deviation	0	0.000003

Table 4.12 Lifetime ER for Inhalation Exposure to Cadmium; Kidney Toxicity

	ER, Average exposure	ER, 90^{th} percentile exposure
Average	0.04	0.08
Standard Deviation	0.01	0.07

4.5.5 Chromium

Hazard identification

A good description of chromium properties are available from ATSDR, (1993a) . Chromium is a metal, a naturally occurring element found in rock and soil as well as in the tissues of animals and plants. For the purpose of health impact, it is important to distinguish between three forms of chromium. The forms are:

- **metal chromium** (chromium (0)) is used mainly for making steel and other alloys. Little is known about the health effects of this form of chromium. However there is no reason to believe that chromium (0) is a major cause for concern.

- **trivalent chromium (chromium (III))** is the form of chromium found naturally in the environment. It is used for brick lining for high-temperature industrial furnaces and for making alloys, chrome plating, dye manufacture, leather tanning and wood preserving. In air, most of the chromium is from man-made sources and most of it is in the form of chromium (III). This form is also an essential nutrient and intake of 50 to 200 µg of chromium (III) per day is recommended for adults. Chromium (III) is required to utilize sugars, proteins and fat properly. Insufficient levels of chromium (III) may cause weight loss impacting on growth, a diabetes-like condition and affect the nervous system. Chromium (III) appears to enhance sensitivity to insulin by facilitating the interaction of insulin with its receptor site. The main concern with the exposure to chromium (III) appears to be allergic reactions causing skin rashes as well as redness and swelling of the skin in sensitive people.

- **hexavalent chromium (chromium (VI))** is released into the environment primarily as a result of industrial activity. Chromium (VI) is not an essential nutrient. High air concentration levels (2 $\mu g/m^3$) may cause irritation of nasal mucosa, nose bleeds, ulcers and holes in the nasal septum. High exposure levels may also cause skin nlcers. Sensitive people may develop skin allergies similar to those caused by chromium (III). Very high ingested doses may cause stomach upset, ulcers, convulsion, liver or kidney damage, or death. Such effects are observed only with high doses which are much larger than would be encountered in food or drinking water. The main cause for concern with chromium (VI) is induction of lung cancer after long-term exposure. Because chromium (VI) is the most toxic of the three forms of chromium, the focus of the assessment is on this compound.

The largest source of chromium in air is the combustion of fossil fuels (oil-based and coal-based). However, most of the chromium from this source is chromium (III). The largest sources of chromium (VI) are chemical manufacturing processes. Chrome plating, steel welding and chromium (VI) manufacturers and industrial users (textile industry, manufacturers of dyes and pigments etc.) can discharge waste into the waterways. The soil levels of chromium (VI) are increased primarily by disposal of commercial products containing chromium, industrial waste containing chromium and by coal ash from electric utilities.

In air, chromium is present mostly in a form of dust particulate (median diameter of about 1 μm) and rain or snow may help settle chromium to the ground or waterways. Airborne chromium (VI) may be reduced to chromium (III) by V^{2+}, V^{3+}, VO^{2+}, Fe^{2+}, HSO^{3-} and As^{3+}). The particles of chromium remain in air for only a short time (days) before settling.

In the water, most chromium is insoluble and it is found mostly as a part of sediment. In sediment, chromium (VI) tends to get reduced to chromium (III), but relatively slowly. Some chromium is in a water soluble form and may persist in a water column. Under anaerobic conditions, chromium (VI) is reduced quickly (hours to days) to chromium (III). Under aerobic conditions, slow oxidation (years) from chromium (III) to chromium (VI) may take place. Chromium is not accumulated by fish.

In soils chromium is primarily in the water-insoluble form. In this form, it will be firmly attached to soil and display little mobility within the soil strata. A small proportion of chromium in soils is water-soluble. This form may be mobile and contaminate ground water. Most soil conditions favour chromium (III) over chromium (VI).

The population is normally exposed to chromium primarily from food (about 96%) and to a lesser degree from drinking water (about 3%) and air (about 1%). Dermal exposure is possible from chrome-treated consumer goods, such as wood treated with copper dichromate or leather tanned with chromic sulphate. In addition, subpopulations exposed to tobacco products may also be exposed to higher levels of chromium, since these products contain chromium.

Chromium (VI) is more readily absorbed across the body barriers (lung tissue, gastrointestinal tract and skin) than trivalent chromium. However in the stomach, hexavalent chromium (Chromium (VI)) is largely reduced to chromium (III). As a result, chromium (VI) is less readily absorbed by oral route than by the other routes. In order to exert its toxicity, chromium (VI) needs to cross the cellular membranes to move into the cells. It does so much more readily than chromium (III). Inside the cells, chromium (VI) is reduced (mostly to chromium (V). During the reduction process, highly reactive molecules (radicals) are formed, which may react with surrounding cellular material and thus cause tissue damage. For example, after exposure to chromium(VI) in an animal model, chromium (V) has been shown to react with DNA, forming chromium (V) - glutathione DNA adducts. This may be the cause for the tumor-initiating capability of chromium (VI) and some of its other of its toxic effects (see below).

Chromium is distributed to most tissues to some degree, but the highest levels tend to be found consistently in lung, liver, spleen and kidneys. The lung levels are particularly high when the exposure takes place via the respiratory tract. With other routes of exposure, lung levels are relatively lower.

Inorganic chromium (III) can be incorporated into as yet not fully characterized complex called dinicotinato chromium (III) glutathione-like complex (GTF). This is the biologically active form of chromium which facilitates the action of insulin. Chromium (VI) can be reduced to chromium (III) before it enters the tissues in the stomach by the gastric acid and ascorbate and also in the lungs by the epithelial lining fluid (ELF). Once it moves into the cells, chromium (VI) can also be reduced to chromium (III). This appears to be a process requiring enzymes cytochrome P450 and an endogenous reducing agent NADPH. Alternatively, chromium (VI) can be reduced by another reducing glutathione to chrominm (V)-glutathione complex. Chromium (VI) is ultimately reduced to chromium (III) in the cell and eliminated from the cell in a form of chromium (III)-glutathione complex. Chromium (III) is eventually eliminated in the urine.

Chromium can induce, in high enough doses a range of systemic effects. However, most of these effects only occur at very high exposures and are thus unlikely to be pose significant human health risk as a result of normal low environmental exposures. However, inhaled chromium (VI) at air levels above 20 $\eta g/m^3$ may cause irritation of nasal mucosa, nose bleeds, ulcers and holes in the nasal septum. This compares to typical ambient air levels of total chromium of about 10 to 30 $\eta g/m^3$. Note however, that most of the chromium in the ambient air is chromium (III) and not the more toxic chromium (VI). It is estimated that only 20 to 25% of airborne total chromium is Chromium (VI). USEPA defined the RfC (MRL or minimal risk level) for the systemic respiratory effects described above for chromium (VI) as 20 $\eta g/m^3$ (IRIS, 1994).

Exposure to low doses of chromium induces allergic reactions causing skin rashes as well as redness and swelling of the skin in sensitive people. This applies to all forms of chromium. Exposure to chromium (VI) and perhaps chromium (III) may also cause reproductive effects. However, there are no human data which could be used to estimate the potency in humans directly and it is difficult to

estimate the potency of chromium in humans based on animal data. It is therefore not clear what the health impact may be at environmentally relevant levels.

Probably the most important effect of chromium is its cancer-inducing potential. Both IARC, (1990) and USEPA (IRIS, 1992) classified chromium (VI) as a human carcinogen based on the studies of workers exposed to chromium (VI) by the respiratory route. The tumors were found primarily in the respiratory tract (in bronchi and nose). Most experiments suggest, that chromium (VI) is cancer-inducing in animals. In contrast, neither IARC, nor USEPA treats chromium (III) as a cancer-inducing compound. Chromium (VI) is genotoxic in humans and in a number of experimental tests. Compounds which exhibit genotoxic behaviour are generally treated as non-threshold toxicants (see section 2.2 for details). USEPA proposed 1.2 x 10^{-2} / μg of chromium (VI) / m^3 of air as the unit lifetime risk of respiratory cancer. This is the unit risk used in the calculations in this report.

Results and discussion of the quantitative assessment

The lifetime cancer risk from inhalation of chromium (VI) is estimated to be 2.9 x 10^{-5} (90^{th} percentile - 6.9 x 10^{-5}) (see Table 4.13).

Table 4.13. Lifetime Risk from Inhalation Exposure to Chromium (VI)

	Risk, Average exposure	Risk, 90^{th} percentile exposure
Average	0.000029	0.000069
Std. Dev	0.000014	0.000028

4.5.6 Lead

Hazard identification

The properties of lead are summarized in MOEE, (1994a) and in ATSDR, (1993b). Lead is a metal which is found naturally in the earth crust in small quantities. It is mined either by itself or as a byproduct of the copper mining. Copper mines are often open pits, while primary lead mines are usually underground operations. Lead is also recovered from secondary sources, such as battery scrap.

The primary use is in the lead acid batteries and gasoline additives. Construction, ammunition, electrical uses, television glass and paint are some of the other major applications. Besides batteries and ammunition, lead is used in consumer-oriented products such as brass and bronze, cable covering, shield lead and solder. Most of the lead is recycled, bnt some lead finds its way into landfills.

Most of the lead released to the environment comes from anthropogenic sources initially emitted into the air. In the ambient air, sotue lead forms a part of particulates, while complexes of lead with organic molecules are formed, which are often volatile. Historically, emissions from combustion of leaded gasolines predominated as the main source of lead in the atmosphere. But with the restriction

of the lead in gasoline, the contribution of lead from mobile sources has dropped dramatically. The emissions from point sources have also declined substantially after the installation of emission - control devices. But some of these industrial operations are now the major sources of lead in air. The main point sources are smelters and non-ferrous foundries. Some lead may be released (volatilized) from lead paints, although modern paints contain little or no lead. Elevated lead concentration levels are likely to be localized near the vicinity of old painted structures.

The major anthropogenic sources of lead in the water are steel and iron foundries and lead production and processing operations. Urban runoff, atmospheric deposition and perhaps leachates from hazardous waste sites are the other major sources of lead in water.

The primary source of lead in soils are solid wastes from ore production and from ammunition use. Other sources include solder, weights, metal bearings and iron and steel production. In some areas, remnants of lead-based paint may be an important source. In the areas without the above major point sources, the atmospheric deposition of lead is the major source of lead in soils.

Most airborne lead is in a particulate form and the particles settle to the ground by both dry and wet deposition. In water, some lead will be present in a soluble form, while the rest will be present in a form of suspended particulate or colloidal form or as a part of sediment. Lead is more soluble in soft or acidic (low pH) water. The ratio of undissolved to dissolved lead was found to vary from 4:1 to 27:1 in surface waters.

In soils, most lead is retained strongly and only a small fraction is mobile or available to enter surface or ground water. High pH (low acidity) and significant content of organic matter in soils further retard lead solubility and mobility in soils. Lead in surface soils can become airborne and transported to other locations as air particulate as a result of weathering and wind erosion.

Plants and animals may bioconcentrate lead but there is no evidence for biomagnification in the upper levels of the food chain. In general, the highest lead concentrations are found in the organisms living near major sources of lead. Older organisms tend to contain higher levels of lead.

The main sources of exposure to lead is about equally from contaminated food and from inhalation of contaminated (airborne) soil and dust. Intake from drinking water plays much smaller role and the exposure from direct inhalation of lead or from dermal absorption are negligible.

Children may be exposed to relatively higher doses of lead because they tend to ingest greater amount of soil than adults. Some of this increased exposure may be due to ingestion of soil from contaminated hands. In addition, some young children ingest soil directly and in some children, exposure to lead by direct ingestion of soil may be significant. Furthermore, children may swallow lead-paint chips in the houses with old paint surfaces.

The old houses with lead-paint may be a source of elevated exposure for adults as well as children. The paint may degrade and become a part of dust which may be inhaled or swallowed by the

residents. Furthermore attempts to remove the old paint may lead to increased exposure of the residents during renovations. Paint-stripping with flame or scraping or sanding has been found to lead to temporarily increased of exposure of the affected residents.

Intake and uptake of airborne lead is somewhat different for inorganic and organic lead. The lead-containing particulate which is deposited into the lungs is fully absorbed. However, typically only 30 to 50% of the particulate is deposited into the lungs. Some is deposited into the npper respiratory tract, transported up the tract by the retrograde transport and eventually swallowed. The remainder of the particulate is exhaled. The exact proportion of the particulate deposited in the lungs depends on the size distribution of the particulate and on the respiration rate. Vapours of organic lead appear to be well absorbed from the lungs but a large proportion is exhaled.

In adults, oral absorption of lead appears to be lower that absorption by inhalation. Only about 10% of lead is absorbed if administered with food. However the absorption in fasting adults can by around 50%. It appears, that children absorb lead more readily than adults and about 50% of lead ingested with diet is absorbed.

Dermal absorption of inorganic lead is negligible, but organic lead may be absorbed more readily. The data are too limited to provide a quantitative estimate of the absorption of organic lead through the skin.

The distribution of inorganic lead after absorption has been well characterized. It appears that the distribution is independent of the route of exposure. Lead is distributed by blood to different body tissues. In blood, lead is found primarily inside the red blood cells. Most of it is bound to haemoglobin. About ½ of the lead will clear from red blood cells in about 36 days into urine. The remaining lead will deposit initially in tissues other than bone. However lead is relatively qnickly eliminated from these tissues either via blood into the bone or to bile, hair sweat or nails. Bone has by far the largest capacity to store lead of any tissue in the body . About 94% of the total dose in adults is stored in the bone and 73% stored in the bones of children. The elimination from bone is measured in decades (about ½ of the lead will be eliminated from the bones in 27 years) and the slow release of lead from the bone may be responsible for the presence of low level of lead in other body tissues long after the exposure to lead has ended. Transplacental transfer of lead has been demonstrated. There are animal data which indicate that lead finds its way into mother's milk if the exposure took place before or during lactation.

The distribution of organic lead is less well studied. In general, the highest levels have been found consistently in the liver, kidney, pancreas and brain.

Inorganic lead may be conjugated by Phase II enzymes (e.g. to form glutathione conjugate) before it is excreted. Organic lead is dealkylated in liver by the P-450 enzymes.

Inorganic lead is excreted in both urine and faeces. The relative contribution of the two routes of elimination in humans is not clear and it may be dose-dependent. Of course, faeces will also contain

any lead not absorbed after oral exposure. In adults, about 50 to 60% of the adult dose is eliminated relatively quickly and half of this fraction will be eliminated in about 19 days. The rate of elimination in children is much slower. Bone represents the major deposit for absorbed lead. Lead is eliminated from the bone depot at a slow rate.

Organic lead such as tetraethyl lead is eliminated rapidly by exhalation route (days), although other routes may also contribute.

Lead has a number of toxic effects which affect most organs and/ or systems. The effects on blood-forming, cardiovascular, kidney and immune system may be important, but the most sensitive seems to be the nervous system, particularly in children. As a result, this assessment focuses on neurotoxicity in children.

USEPA has not developed a RfD for lead, because there does not appear to be a discernible threshold to the neurotoxicity of this metal. MOEE, (1994a) came to the same conclusion. However, they concluded that total uptake of 1.85 μg of lead /kg/day would offer a degree of protection to children. Most of the lead is taken up from soil, dust and food and air contributes only less than 1% to the overall uptake. Based on this information, MOEE, 1994b developed a 30 day average ambient air quality criteria for lead of 0.7 $\mu g/ m^3$. This number takes into account varions factors including economic achievability, detection limit, etc..

Results and discussion of the quantitative assessment

The lifetime ER due to inhalation of lead is estimated to be 0.04 (90^{th} percentile: 0.08) (see Table 4.14).

Table 4.14. Lifetime ER from Inhalation Exposure to Lead.

	ER, Average exposure	ER, 90^{th} percentile exposure
Average	0.04	0.09
Standard Deviation	0.02	0.04

4.5.7 Manganese

Hazard Identification

Manganese is present naturally in the environment in the form of sulphides, oxides, carbonates, silicates, phosphates and borates. Most common forms are dioxide, carbonate and silicate. Metallic manganese is used primarily in the steel production. The other compounds containing manganese are used for the production of dry cell batteries, and production of glass and porcelain, food additives, fertilizers fungicides, disinfectants, for cleaning metal, tanning bleaching and in the antiknocking additive methylcyclopentadienyl manganese tricarbonyl (MMT).

In the air, manganese is present in particulate form. The largest point source of airborne manganese are ferroalloy production and iron and steel foundries. The next most important source are the combustion emissions including power plants and coking operations. MMT in automotive fuel may be another source of manganese.

In water, manganese exists partially in a solubilized form and partially as a part of sediment. The levels of manganese are typically considerably higher in marine organisms than in the surrounding water (100 times to 40 000 times), but there does not appear to be any biomagnification of manganese in the food chain. Thus the levels in the fish are equal or lower than the levels plants and invertebrates on which the fish feed. In both water and soil, manganese may be naturally present or it may be found there as a result of human activities. Important sources of manganese in water are industrial discharges and leachates from landfills. In soils, disposal of manganese - containing wastes is the principal source of manganese releases to the soil.

Dry deposition is the main mechanism of removing manganese from air, while rain and snow washouts are less important. Re-entrainment of manganese-rich soils as airborne dust is also an important source of manganese in air.

Humans are exposed to manganese primarily from food. The levels ingested are typically less than 10,000 times higher than the levels inhaled. It is not clear however, what proportion of the inhaled manganese is absorbed. Only about 3% to 5% of ingested manganese is absorbed. Manganese is probably competing for the same absorption system with iron and thus if there is less iron to be absorbed, the levels of absorbed manganese rise. There are no data to assess absorption of manganese through the skin, but the absorption by this route is assumed to be negligible.

Manganese distributes into most tissues about equally, with liver, pancreas and kidney having somewhat higher levels. In a monkey study, manganese was found to be preferentially accumulating in the specific parts of the brain (caudate nucleus, globus pallidum and substantia nigra). This may be important factor in accounting for the neurological deficits observed after long-term exposure to high levels of manganese (see below). Some of the inhaled manganese is eliminated relatively quickly in the faeces (days to weeks). After inhalation, some is retained in the lungs for periods of weeks to months.

Humans are exposed to manganese in the course of their daily lives, because it is a naturally occurring element. Indeed, animal studies indicate that manganese is required for good health and the growth of the animals deprived of this element was impaired. Skeletal abnormalities, impaired reproductive function and deficiencies in the metabolism of carbohydrates and lipids have been reported. However, no cases of manganese deficiencies in humans have been reported.

The most important toxic effect of manganese is a disease induced by high levels of inhaled manganese called manganism. The motor impairment associated with this disease is similar in its symptoms to Parkinsonism and the effects either persist for a long time or become irreversible even if the exposure to manganese has been terminated. The first symptoms are usually a sense of

weakness and lethargy, which eventually progresses to muscle rigidity and tremor. Hallucinations or psychosis may also be present. Just as in the Parkinson's disease, manganism is probably associated with the damage of the nerve cells in the brain which communicate with other cells by controlled release of dopamine neurotransmitter. These cells have their cell body located in the substantia nigra and their dopamine-releasing terminals in the caudate nucleus and putamen. It is in these brain areas that manganese tends to accumulate. Just as in Parkinson's disease, the level of the neurotransmitter dopamine is reduced in patients with manganism and the replacement of dopamine by administering a precursor of dopamine (L-dopa) tends to alleviate some of the symptoms of Parkinson's disease and manganism. It is important to note, that manganism has been observed only in the individuals exposed to high levels of airborne manganese. Manganism has not been reported after high exposure to manganese by the oral route.

Since humans are normally exposed to manganese from the air and no manganism cases were reported as a result of environmental exposures, it is assumed tbat there is a threshold for the neurological damage induced by manganese which leads to manganism. USEPA has derived an RfC of 0.05 $\mu g/m^3$ based on data obtained for occupational exposures (IRIS, 1994). This RfC incorporates an uncertainty factor of 1000.This is the value adopted in this study.

Results and Discussion of the Quantitative Assessment

The lifetime ER due to inhalation of manganese is estimated to be 1.76 (90^{th} percentile: 4.1) (see Table 4.15).

Table 4.15. Lifetime ER from Inhalation Exposure to Manganese.

	ER, Average exposure	ER, 90^{th} percentile exposure
Average	1.76	4.1
Standard Deviation	0.76	1.38

4.6. Appendix

Description of risk

Risk is defined as the *probability* of an adverse event occurring. For example, the risk of an average person dying of heart disease is roughly 3 in 10. This does not mean that if the cause of death is examined for 100 people chosen at random, exactly 30 would have died of the heart disease. The actual number may be higher or lower, but based on past experience, 30 would be expected to have died of heart disease "on average".

Most lifetime risks from exposure to toxic chemicals in the environment are very small; typically less than 1 in 100,000. To put this in perspective, Table 4.16 below shows the probabilities of a person dying under various conditions or contracting cancer. These examples are not intended to

trivialise potential risks from environmental exposures, rather the intent is to give the reader a useful yardstick for visualizing relative probabilities.

Table 4.16. Perspectives on Risks. Table Adapted from MOEE, 1994c

Situation	Probability	
Struck by lightning	$1x10^{-7}$	1 in 10,000,000
Fatal accident while travelling by commercial aircraft, rail and bus	$1x10^{-6}$	1 in 1,000,000
Fatal accident as a pedestrian	$1x10^{-5}$	1 in 100,000
Fatal accident at work (manufacturing)	$1x10^{-4}$	1 in 10,000
Contracting cancer if smoking 1 pack of cigarettes a day	$1x10^{-2}$	1 in 100
Cancer as cause of death in Ontario (1995)	$2.7x10^{-1}$	greater than 1 in 4

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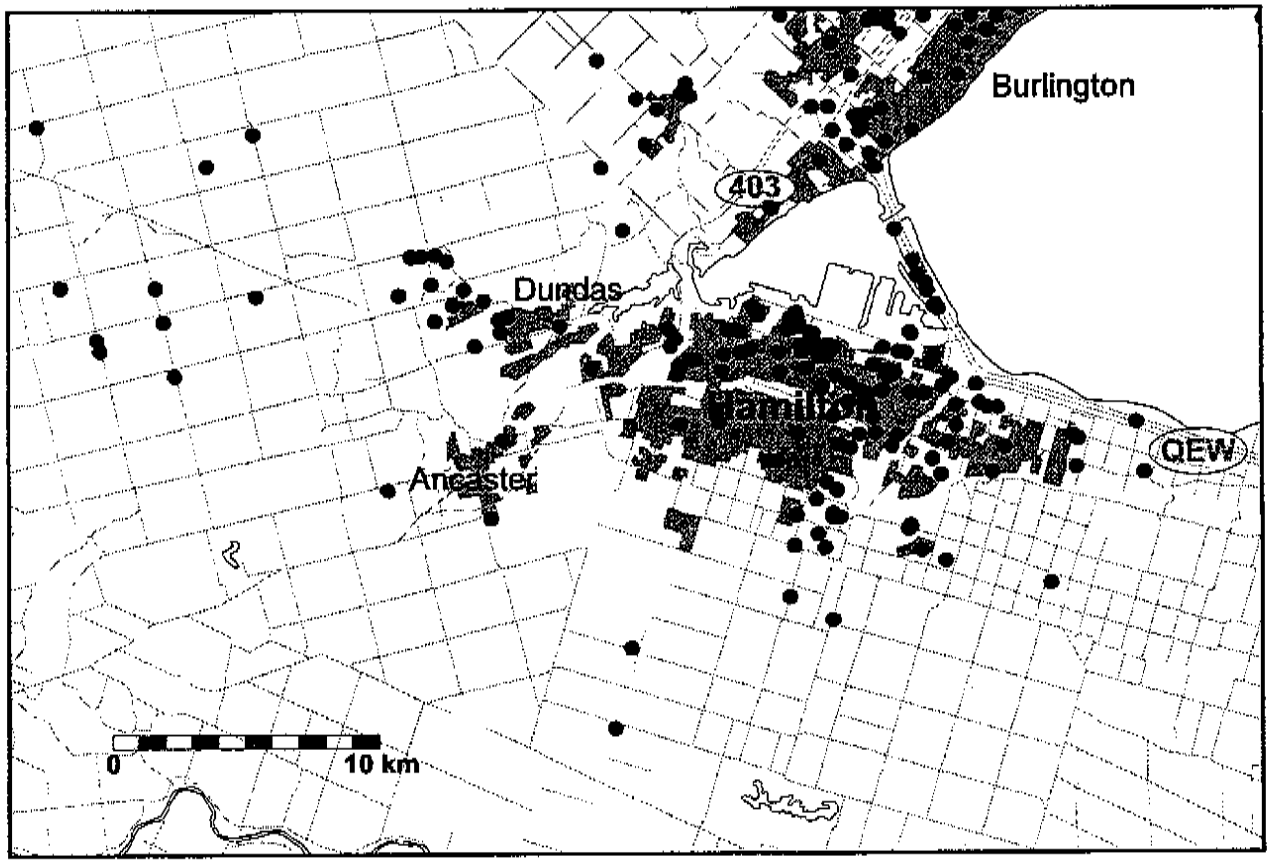
PART FIVE: Assessment of the Potential Human Health Effects Due to Exposure to Total Reduced Sulphur Compounds (TRS) and Black Particulate Fallout - Group #5.

5.1. Total Reduced Sulphur (TRS) Compounds

5.1.1. Background - Odours

Odour has always been a concern for people living and working in the Hamilton area. As an example, of the 1,266 environmental complaints originating from the Hamilton-Burlington area and logged with the Ontario Ministry of Environment and Energy in 1994, almost 30% dealt with odour. Of course, many of the complaints came from the same people within the same neighbourhood and most likely within the same short periods of time (such as when there was a spill of some nature into the immediate area), but nevertheless, a composite map of where the odour complaints originated throughout 1994 shows that the majority originated in areas either immediately downwind of the industrial area of Hamilton or in the downtown sectors of Hamilton and Burlington.

Figure 8 : Odour Complaints - 1994 (Burlington and Hamilton)



The ability to detect odours at very low concentrations is a sense which has evolved to help us detect immediate changes in our surroundings. Odours can be pleasant, as in those associated with freshly prepared food, or unpleasant, as in those associated with spoilage. The sense of smell is extremely sensitive, capable of detecting minute quantities of an odourant. Because of this, unpleasant odours associated with industrial emissions can be one of the more difficult air pollution problems to address.

Odours are characterised as having four basic properties; character (what the odour smells like), tone (is it pleasant or unpleasant), intensity (the degree to which the strength of the odour changes with concentration) and detectability (the minimum concentration which can be detected). TRS compounds are considered intense odourants with an extremely unpleasant character. Because of their intensity, relatively large changes in the concentration of TRS may only lead to small changes in the perceived strength of the odour. It has been estimated that a 2-fold change in the concentration of hydrogen sulphide (one of the major TRS compounds) will only result in a 20% change in the perceived intensity (CARB, 1985). The intensity of the odours associated with TRS compounds coupled with their extremely low detection thresholds makes the control of TRS emissions very difficult.

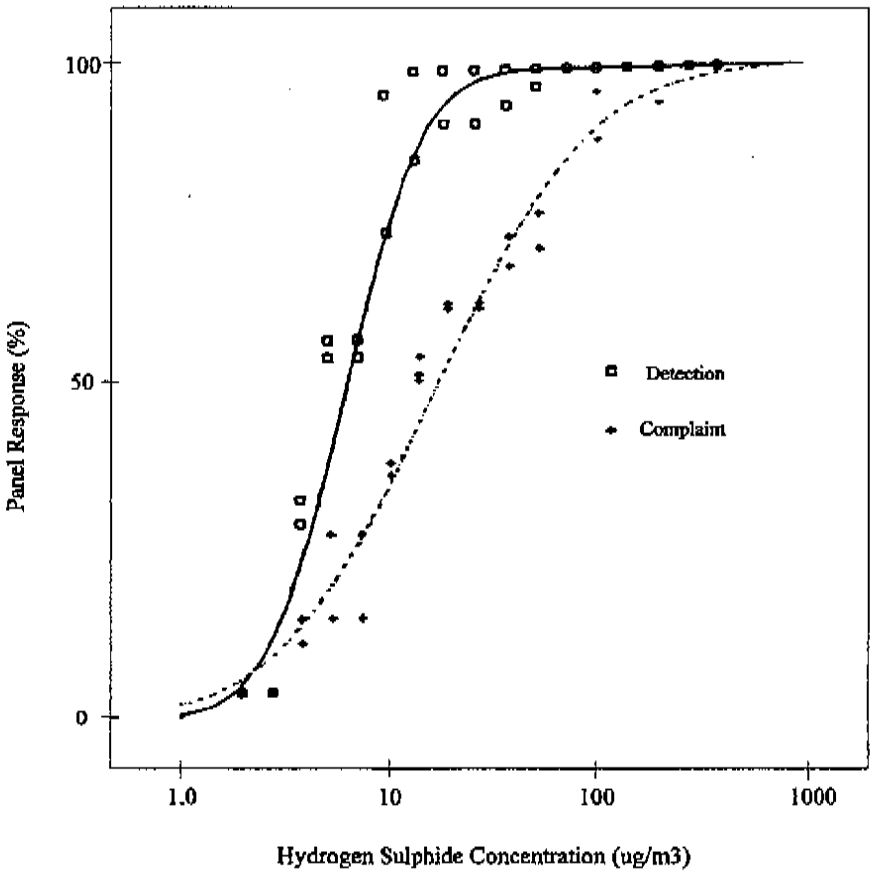
The concentration at which specific odours can be detected and recognized is termed the odour threshold. Odour thresholds are normally determined by panels of volunteers who are exposed to specific concentrations of a chemical in a controlled environment and asked if they can detect or recognize an odour. The concentration at which 50% of the panel members can recognize the odour is commonly referred to as the odour threshold. Threshold values are therefore not absolute values but rather reflect the estimated concentration at which 50% of the population would be likely to recognize the presence of an odourant (reviewed in NRC, 1979; USEPA, 1990b). At a concentration of TRS equivalent to the odour threshold, approximately half of the people exposed will be able to detect the presence of an odour whereas half will not.

TRS usually appears as a mixture of compounds in our natural environment. These compounds consists primarily of hydrogen sulphide, methyl mercaptan, dimethyl sulphide and dimethyl disulphide.

Results from an odour panel study for hydrogen sulphide conducted by ORTECH International under contract to the Ministry are presented in Figure 2. The study involved a panel of nine members who were presented with a series of increasing coucentrations of hydrogen sulphide or odour-free air in two separate sampling ports. Respondents were then asked in which of the ports, if any, they could detect and identify an odour. If an odour was detected, panel members were also asked if they would take the trouble to contact regulatory officials if forced to smell the odour at that concentration for 8 hours. The percent response and the hydrogen sulphide concentration were used to generate the curves shown in Figure 2. The variability in the ability of panel members to detect the presence of hydrogen sulphide is clear from the figure. The detection threshold for individual panel members spans a concentration range of approximately 10 fold (2-22 $\mu g/m^3$). Similarly, the complaint threshold for individuals spans a concentration range of approximately 100 fold (2-200 $\mu g/m^3$). From

this study, the odour threshold for hydrogen sulphide was estimated to be 7 $\mu g/m^3$ or 5 ppb. Although, this is the level at which 50% of respondents could detect the presence of hydrogen sulphide, approximately 25% of panel members would already consider complaining to regulatory authorities at this level. The 50% complaint threshold is estimated to be 20 $\mu g/m^3$ (14.3 ppb). A similar panel response is obtained with other odorous compounds including methyl mercaptan, dimethyl sulphide and dimethyl disulphide. The 50% complaint threshold is generally 2.5-3 times higher than the detection threshold.

Figure 9 : Odour Panel Response to Hydrogen Sulphide



Published odour thresholds for the constituents of TRS are summarized in Table 1. The variability between studies reflect differences in experimental design, the quality of individual studies and the variability in the population. An individual's ability to detect and respond to an odour can be influenced by age, occupation, genetics, gender and personal habits such as smoking. Individuals can also respond differently to odours at different times of the day. Within a particular community, there

will be persons that are extremely sensitive to odours and are thus likely to be bothered by their presence and persons relatively insensitive to odours.

Table 5.1: Odour Thresholds for TRS Compounds *

TRS Compounds	*Source*
Hydrogen Sulfide	
0.072 - 1400 ppb (mean 7.45 ppb)	Reviewed in CARB, 1985
5 ppb (detection), 14.5 ppb (complaint)	MOEE, 1994
0.5 - 32 ppb	USEPA, 1979
4.5 ppb (detection), 9.4 ppb (recognition)	AIHA, 1989
0.5 - 10 ppb	Ruth, 1986
Methyl Mercaptan	
2.1 ppb	USEPA, 1979
1.25 ppb (detection), 5 ppb (complaint)	MOEE, 1989
0.54 ppb (detection), 1 ppb (recognition)	AIHA, 1989
0.02 - 41 ppb	Ruth, 1986
Dimethyl Sulphide	
1 ppb	USEPA, 1979
17.2 ppb (detection), 34.5 ppb (complaint)	MOEE, 1989
0.6 - 17 ppb	Ruth, 1986
0.3 - 3.0 ppb (detection), 1 ppb (recognition)	ASTM, 1978
Dimethyl Disulphide	
5.6 ppb	USEPA, 1979
15.6 ppb (detection), 46 ppb (complaint)	MOEE, 1989
0.02 - 82 ppb	Ruth, 1986

* Odour thresholds include detection, recognition and complaint thresholds

One of the difficulties of experimental determinations of odour thresholds is how these valnes translate into real world situations. There are some suggestions that odour thresholds of single compounds obtained in a well controlled laboratory overestimate an individual's ability to perceive

odours nnder more realistic conditions (NRC, 1979). In addition, there is a perception that a mixture of odourants can be different than the individual constituents of the mixture. For most mixtures involving similar compounds, the perceived intensity of the mixture is approximately equivalent to or slightly less than the sum of the intensity of the individual components of the mixture (CARB, 1985; NRC, 1979). This would suggest that for typical mixtures of TRS, the odour thresholds would be in the range of 1-5 ppb. However, studies which examined the odour threshold of emissions from kraft pulp mills indicate that the odour threshold based on the concentration of TRS measured in the emission stream was approximately 0.02-0.03 ppb (NCASI 1971, cited in CARB, 1985).

5.1.2. Acute and Chronic Health Effects

Although there are several occupational and community studies which have examined the health effects of exposure to TRS, most studies that have examined the toxicity of TRS have concentrated on the individual constituents of the mixture. A summary of this information is provided below.

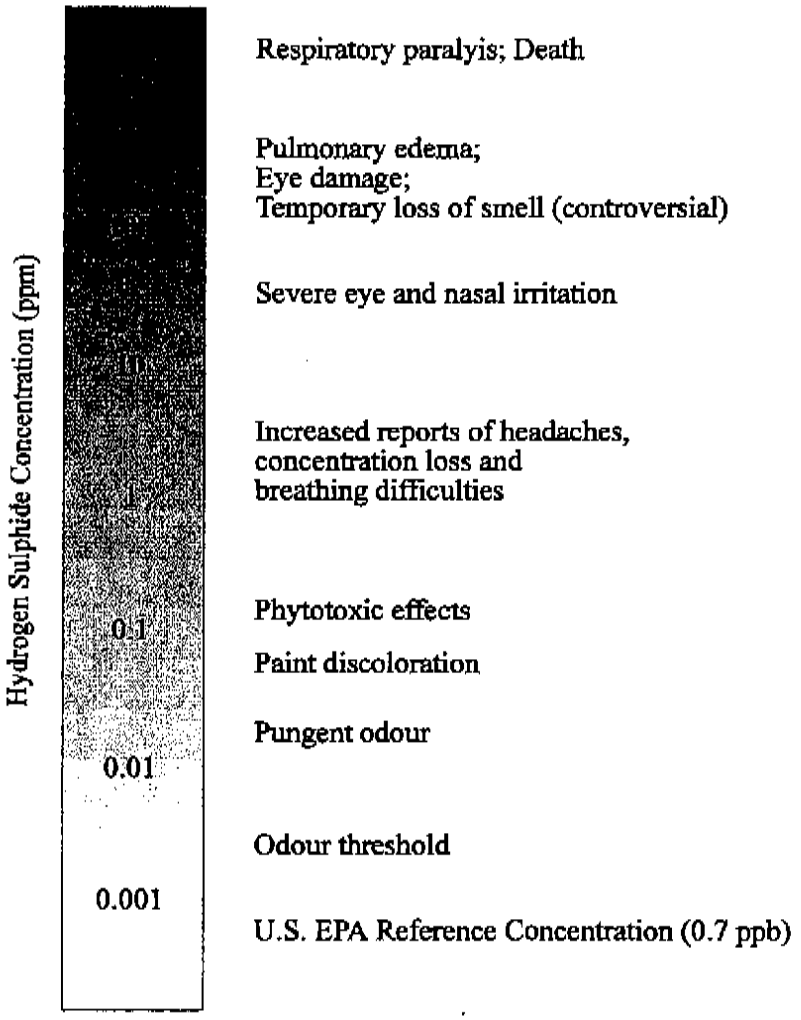
5.1.2.1 *Hydrogen Sulphide*

The toxic gas hydrogen sulphide is the most extensively studied of the TRS compounds. The adverse effects which result from exposure to hydrogen sulphide occur over a concentration range of over a million-fold and are summarized in Figure 3. At high concentrations (greater than 1000 ppm), even brief periods of exposure can result in respiratory paralysis and death. Exposure to lower concentrations can cause eye irritation (10-20 ppm), permanent eye damage (50-100 ppm), temporary loss of the sense of smell (150 ppm) and pulmonary edema (300-500 ppm) (WHO, 1987). Exposures in occupational settings to concentrations less than 10 ppm have been reported to lead to headaches, loss of concentration, eye irritation and a feeling of restlessness. In a volunteer study using men and women suffering from asthma, exposure to 2 ppm for 30 minutes did not have a significant effect on respiratory function although participants complained of dryness of the nose and throat (Jappinen, Vilkka, Marttila, & Haahtela, 1990). In another experiment, male volunteers were asked to exercise while being exposed to varying concentrations of hydrogen sulphide by mouth breathing. The only effects noted were increased lactate production and oxygen demand and were present only at the highest level of exposure (5 ppm).

A nnmber of epidemiological studies have examined the effects on community health from exposure to hydrogen sulphide. Community residents exposed to hydrogen sulphide at coucentrations greater than approximately 50 ppb (24 hour average) complain of a number of symptoms including headaches, loss of sleep, eye and nose irritation, sore throat and nausea. Based on an animal study in which mice were exposed to various concentrations of hydrogen sulphide for a period of 90 days, the United States Environmental Protection Agency (USEPA) has recommended an inhaled reference concentration (RfC) for hydrogen sulphide of 1 $\mu g/m^3$ (0.71 ppb) (USEPA, 1995). An RfC is an estimate of daily inhalation concentration which would not be expected to lead to any adverse effects in the human population including sensitive sub-populations based on a lifetime of exposure. The RfC for hydrogen sulphide is based on a *N*o *O*bserved *A*dverse *E*ffect *L*evel (NOAEL) in mice of 30.5 ppm (nasal irritation was observed in mice exposed to 80 ppm hydrogen sulphide). The

NOAEL in mice was adjusted for differences in dosing regimes and respiratory physiology between mice and humans to derive a human NOAEL equivalent of 710 ppb (1 mg/m^3). The human equivalent NOAEL was then adjusted with a safety factor of 1000 to yield the RfC of 1 µg/m^3.

Figure 10 : Effects of Hydrogen Sulphide



As there are a number of studies which have evaluated the effects of hydrogen sulphide on humans, it can be argued that the use of animal studies to establish an RfC does not utilize all of the available information on hydrogen sulphide toxicity. Occupational and volunteer studies which have examined human exposure directly, suggest that the lowest concentration at which acute exposure to hydrogen sulphide results in direct irritancy of nasal, respiratory and eye mucosa occurs in the range of 2 - 20 ppm.

5.1.2.2. *Dimethyl Sulphide (DMS) and Dimethyl Disulphide (DMDS)*

The acute toxicity of DMS and DMDS has been assessed experimentally in both mice and rats. One study reports an LC_{50} (the concentration at which death occurs in 50% of the animals exposed) in rats for DMDS as being approximately 800 ppm for a 4-hour exposure while another study reports a value of 4.1 ppm (Tansy, Kendall, Fantasia, Landin, Oberly, & Sherman, 1981). In long-term studies in which groups of rats were exposed to differing concentrations of DMDS (10 to 250 ppm) for 6 hours per day, 5 days per week, breathing difficulties and lethargy could be observed at 250 ppm (Gage, 1970; Elf Atochem, 1992). Decreases in weight gain could be observed at 50 ppm and alterations in the nasal mucosa (squamous metaplasia) could be observed at all exposure levels (Elf Atochem, 1992).

Much less information is available for DMS. Similar to DMDS, reported LC_{50} values in rats are varied (20 ppm vs 40,000 ppm for 4-hour exposure). In mice a single LC_{50} of 12.5 ppm has been reported (Tansy, et al., 1981). In addition, short duration exposures to concentrations above approximately 40,000 ppm are acutely lethal to mice (Tansy, et al., 1981). No information is available on the effects of longer-term exposure in animal studies.

The only data available on acute human exposure comes from one industrial accident where a worker died after being exposed to a number of sulphur containing gases. Although the concentration of DMDS, DMS and methyl mercaptan were all below 10 ppm, the gases were measured several hours after the accident occurred. DMS was attributed as the cause of death due to the presence of this compound in the tissue of the victim (Terazawa, Mizukami, Wu, & Takatori, 1991). Beyond this one industrial accident, occupational exposure has been examined in pulp mill workers. Workers were surveyed to assess the health impacts resulting from exposure to levels of DMS and DMDS between 0-15 ppm. An increase in the reported number of headaches was the only statistically significant symptom noted (Kangas, Jappinen, & Savolainen, 1984). As with many occupational studies, it is difficult to attribute these affects solely to DMS and DMDS as the workers were also exposed to other malodorous sulphur compounds.

5.1.2.3 *Methyl Mercaptan*

The toxicity of methyl mercaptan has been studied only to a limited extent. Similar to DMS and DMDS, the reports on the acute toxicity in rats are varied. One study reports an LC_{50} of 675 ppm while another reports a value of 4.5 ppm (Tansy, et al., 1981). Fifteen minutes of exposure to concentration of 1600 ppm induced unconsciousness in rats (Zieve, Doizaki, & Zieve, 1974). In mice, there is a single report of an LC_{50} of 3 ppm (Tansy, et al., 1981).

In long-term studies, rats exposed to various concentrations of methyl mercaptan (0-57 ppm) for a period of 7 hours per day, 5 days per week for 90 days only showed adverse effects (reduced weight gain) at the highest levels of exposure. No adverse effects were noted in rats exposed to concentrations of 17 ppm and less. Similar to the studies with DMDS, this observation is difficult to reconcile with the report of an LC_{50} of 4.5 ppm. Continuous exposure to 50 ppm for a period of

90 days resulted in minor lung and unspecified brain effects in monkeys, minor lung and haematological effects in rats and haematological and hepatic effects in mice (Sandmeyer, 1981).

In humans, similar to all malodorous sulphur containing compounds, individuals exposed to mercaptans in general report irritation of the eyes, nose and respiratory system, an increase in the frequency of headaches and a general feeling of nausea. Only in one industrial accident has the exposure to methyl mercaptan resulted in death. This occurred as a worker was emptying tanks containing methyl mercaptan. The actual levels the worker was exposed to were not known (ATSDR, 1990).

While there is little documented information on the chronic and acute health effects associated with exposure to TRS gases other than hydrogen sulphide, it is generally accepted that the odour thresholds for these compounds are well below the concentrations required to induce symptoms via known toxicological mechanisms (Neutra, Lipscomb, Satin, & Shusterman, 1991; NRC, 1979; Shusterman, 1992a).

5.1.3 Health Effects of Odours

A number of studies have examined the relationship between exposure to odours and community health effects (Goldsmith, 1973; Haahtela, Marttila, Vikka, Jappinen, & Jaakkola, 1992; Jaakkola, Vilkka, Marttila, Jappinen, & Haahtela, 1990; Persson, Skog, & Hasenson, 1987; Shusterman, Lipscomb, Neutra, & Kenneth, 1991; Sider, Nosal, Willmott, Delmore, Stronach, Streiner, et al., 1994). For many communities impacted by odorous emissions, there is an increase in the reporting of symptoms such as headaches, nausea, eye and throat irritation, sleep disturbances and stomach upset, as well as a decreased sense of well-being (reviewed in NRC, 1979; Shusterman, 1992b). It has also been reported that exposure to odours can trigger asthma attacks in patients suffering from bronchial asthma (Shusterman, 1992b). Studies that have examined the impact of emissions from kraft pulp mills (the primary air pollutants are reduced sulphur compounds) on community health have revealed a systematic increase in the self-reporting of symptoms such as headaches, nausea, eye and throat irritation and breathlessness. Although the increase in symptom reporting could be correlated with measured or modelled increases in airborne emissions of sulphur compounds (Haahtela, et al., 1992; Jaakkola, et al., 1990), the levels to which residents were exposed (highest daily average was approximately 65 ppb) were below those that would be expected to lead to irritancy or adverse health effects through known toxicological mechanisms.

Similar findings linking adverse health impacts resulting from exposure to odours have been observed around petroleum refineries (Persson, et al., 1987; Sider, et al., 1994), sour gas plants (Schechter, et al., 1990) and hazardous waste sites (Shusterman, et al., 1991). In the latter study, a strong correlation was noted between odour perception, environmental worry and the self- reporting of symptoms. Those individuals who noticed odours frequently (greater than 4 times per month) were 4-5 times more likely to report symptoms such as headaches, nausea, and throat and eye irritation than those individuals who only noticed odours infrequently. Those residents who characterized themselves as having a high degree of environmental worry (approximately 37% of

respondents) were 5-12 times more likely to report increased symptoms than those residents who had a low degree of environmental worry. However, those residents who frequently notice odours and characterized themselves as having a high degree of environmental worry were 12-38 times more likely to report the presence of symptoms. Based on these observations, the authors suggested that the increase in symptom reporting could best be explained by a stress-induced mechanism which is triggered by exposure to odour. Odours from industrial sites are thought to enhance the level of anxiety felt in individuals in neighbouring communities worried about health impacts from industrial emissions. The increased anxiety can then trigger symptoms such as headaches and nausea through stress related mechanisms. The increased anxiety may also heighten the awareness and thus the reporting of other symptoms not thought to be directly induced by a stress-related mechanism (Neutra, et al., 1991; Shusterman, et al., 1991). Others have suggested however, that the increased symptom reporting could be the result of reaction to the toxic properties of various contaminants at extremely low levels in sensitive individuals, a condition often termed multiple chemical sensitivity (Ziem & Davidoff, 1992). As this condition is not well understood clinically, it is difficult to state with any certainty how plausible an explanation this is. However, the consistent association between those who perceive odours and those who report the onset of specific symptoms suggests that the symptoms are triggered by exposure to odours rather than the toxic properties of the contaminants themselves.

One of the difficulties with the studies similar to those discussed above is the obvious reliance on study participants to recall and report the frequency of occurrence of specific symptoms. In many studies which rely on self-reporting, participants concerned about an issue may overstate their case in order to force corrective action (Goldsmith, 1973). Although most of the studies described above attempted to eliminate participant bias, in studies where the source of odours is obvious, complete elimination of bias is often difficult to achieve. While it might be argued that participant bias may, in part, be responsible for the increased reporting of certain symptoms, the consistent association between the perception of odours and the reporting of adverse health symptoms strongly suggests that exposure to unpleasant odours can have a negative impact on health and the quality of life in a community.

5.1.4 Non-Health Effects of TRS

There are a number of other effects which are caused by TRS, primarily attributable to the hydrogen sulphide in the mixture. Hydrogen sulphide is toxic to plants although reports on the sensitivity of various species are quite varied. Most plants can tolerate relatively high concentrations (greater than 1000 ppb) of hydrogen sulphide without severe injury (USEPA, 1979). However, depending on the species, prolonged exposure to concentrations of hydrogen sulphide in excess of 100 ppb can cause spotting and discolouration of leaves (Stern, Boubel, Turner, & Fox, 1984). Rapidly growing tissues such as new leaves and shoots appear to be most sensitive to the effects of hydrogen sulphide (USEPA, 1979).

Hydrogen sulphide can also react with heavy metals in paints to form precipitates which can discolour painted surfaces. In the presence of moisture, the reaction between hydrogen sulphide and

lead, mercury, cobalt, iron or tin salts present in pigments or drying agents can lead to formation of a grey or blackish precipitate. The degree of discolouration is concentration and time dependent and can occur after several hours exposure at 50 ppb. While discolouration of painted surfaces by hydrogen sulphide emissions was at one time quite commonplace, newer paint formulations are more resistant (Stern, et al., 1984; USEPA, 1979).

5.1.5 TRS and Air Quality

The level of TRS monitored in ambient air is used as one of the indicators of general air quality in Ontario's Air Quality Index (AQI). The AQI is based on monitoring information for six pollutants (carbon monoxide, ground level ozone, nitrogen dioxide, suspended particulate, sulphur dioxide and TRS) and provides a numerical indicator of air quality. The Ministry currently has 29 AQI sites of which 12 include measurements for TRS. At concentrations of TRS between 6 and 10 ppb (considered slightly odorous) air quality is considered good (AQI 16-31); levels between 10 and 27 ppb (considered odorous) correspond to an air quality of moderate (AQI 32-49) whereas levels between 27 and 1000 ppb air quality is considered poor (AQI 50-99). Locations in 1993 with moderate air quality as a result of TRS levels included Fort Frances (1117 hours); Cornwall (43 hours); Hamilton Downtown (9 hours); Hamilton Mountain (13 hours); Sault Ste. Marie (15 hours); Thunder Bay (3 hours) and Windsor (College & Prince St. location 5 hours) (MOEE, 1994).

Although odour complaints continue to be the single largest category of complaints received by the Ministry, there tends to be a poor correlation between TRS levels and formal complaints in different communities in Ontario. The acceptability of odour in a community depends very much on the character of the odour, its intensity, duration and the attitudes of the residents within the community (NRC, 1979). In some communities, complaints attributable to TRS are registered at levels as low as 5 and 10 ppb whereas in communities where there is a large kraft pulp mill operation, there are very few complaints even though maximum TRS levels often exceed 100 ppb. The lack of a correlation between odour complaints and TRS levels may be attributable to a number of factors including the communities familiarity with the source of the odour or a perceived lack of action on the part of regulatory officials in response to complaints. In contrast to odour complaints, information from community surveys suggests there is a strong correlation between odour perception, odour bother and the proximity of the resident to the source of the odour (Goldsmith, 1973, Sider, et al., 1994).

5.1.6 Sources of TRS from Iron and Steel Production

Information on the sources of TRS in the iron and steel industry is limited. TRS, primarily in the form of hydrogen sulphide, is emitted from steel mills from the manufacture of coke and the quenching of iron slag. Depending on its design, TRS may also be released from the by-products recovery plant. Coke used in the manufacture of steel is made by heating coal in the absence of oxygen in large batteries of ovens. Heating the coal in this way removes the volatile components of the coal (up to 25% by weight) which are collected as coke oven gas. Because of the oxygen starved conditions, coking generates hydrogen sulphide which originates from the sulphur contained within

the coal. Coke oven gas is normally processed to recover saleable by-products and the heat energy present in the gas. Hydrogen sulphide present in coke oven gas may be recovered as elemental sulphur in the by-products recovery step.

Coke is burned in the blast furnace to provide the heat energy and reducing conditions required to convert iron oxide to molten iron. Limestone is added to the blast furnace along with iron ore and coke in order to facilitate the removal of impurities in the form of slag. The composition of the slag is important in order to maximize the removal of sulphur from the iron. Molten iron is separated from the slag and is further refined to make steel. The molten slag is cooled with water (quenched) and sold as an aggregate for use in the manufacture of cement or cinder blocks. The addition of quench water during slagging results in the formation and release of large quantities of steam, sulphur dioxide and hydrogen sulphide. Hydrogen sulphide is produced from the reaction between water and the calcium sulphide present in the slag. While slag quenching occurs intermittently during the refining of iron, estimated TRS emission rates from this source are very high. Limited source testing suggests that TRS emission rates can be as high as 14-18 g/s (MOEE, 1995).

While other processes, such as the by-products recovery plant, can also be sources of TRS emissions, there is little information available on emission rates from these sources.

5.1.7 Summary of the Effects of TRS

The major concern associated with TRS in air is considered to be odour detection which occurs in the range of 1-5 ppb. This range is approximately 10 times lower than concentrations which lead to paint discolouration and at least 100 times lower than concentrations that lead to adverse health effects due to toxicological mechanisms.

While odour detection is considered to be the critical effect of TRS, to characterize this end-point as simply a nuisance or annoyance effect understates its impact on individual and community health. The consistent self-reporting of adverse health outcomes ranging from headaches to nausea as well as a decrease in a sense of well-being in communities impacted by odours is sufficient to suggest that persistent exposure to unpleasant odours can have a significant impact on community health. Persistent unpleasant odours can also have a negative impact on urban and industrial development and tourism (Doty, 1972; Goldsmith, 1973).

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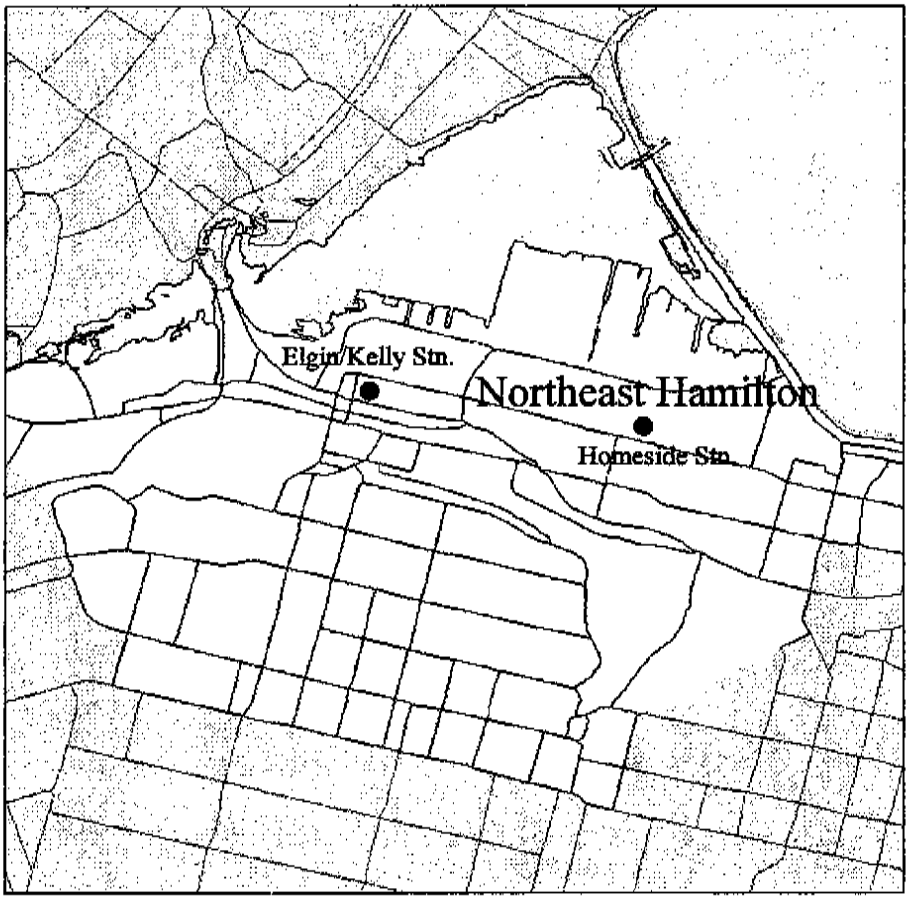
5.2 Black Particulate Fallout in Northeast Hamilton

5.2.1 Background:

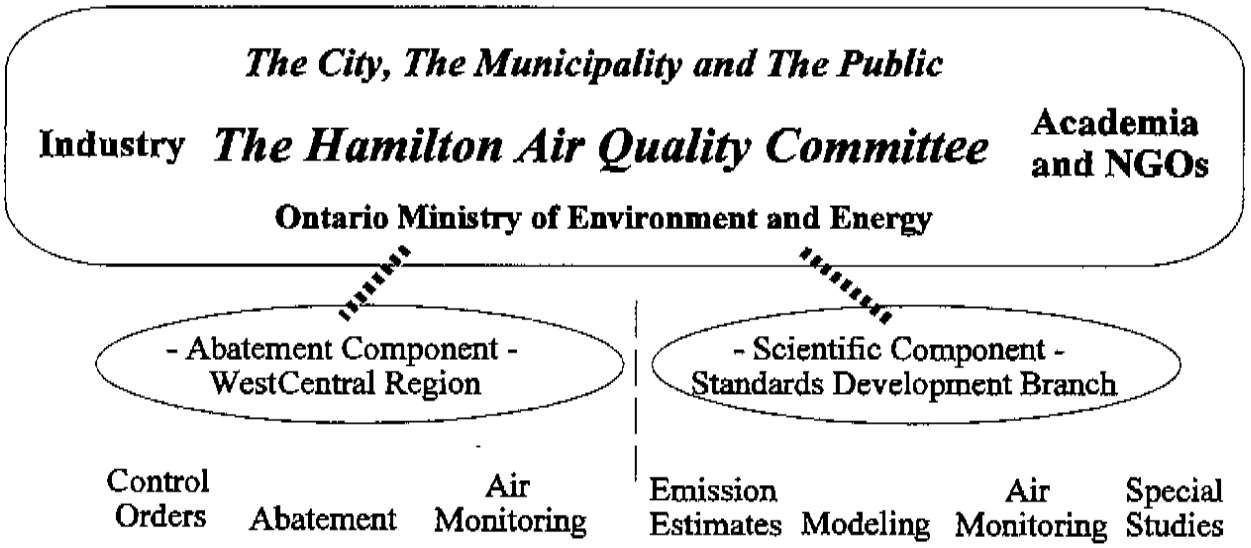
For many years, the residents of northeast Hamilton have been concerned about fine black airborne particles that have been falling in their area. Their original concern dealt with soiling (property damage) and the enjoyment of their property. From the 1994 MOEE environmental complaint data base recorded by the Hamilton regional office, almost 40% of all registered complaints dealt with airborne particles and smoke. In a recent telephone study conducted by the Regional Public Health Department of over 400 residents in northeast Hamilton, almost 75% of the respondents described disruptions in lifestyle due to deposits of black sooty material and approximately 70% believed air pollution in their area would likely lead to adverse health effects for themselves and their family (Elliott S. et. al, 1997).

Figure 11 Northeast Hamilton



In January of 1995, the Black Fallout Stakeholder Committee was formed. Members of the Committee include representatives from the City of Hamilton, the Municipality, the Public, the Homeside Neighbourhood and other local environmental groups, non-governmental organizations

Figure 12



(NGOs), industry (Columbian Chemical, Dofasco and Stelco), the academia (McMaster University) and the MOEE. As directed by this Committee, the MOEE serves as a resource for proposed monitoring, abatement and remediation programs, and for the gathering and distribution of scientific and engineering information. The Committee has been given the task of investigating this problem in detail: including determining its composition, its impact area; its source; its health effects; and, if necessary, recommending and supporting appropriate amelioration activities.

Historically, analytical investigations of this black particulate fallout material have been limited. Definitive cause and effect conclusions implicating specific source(s) have usually not been possible. New sampling and analytical methodologies are being developed and several special programs are currently underway. These include

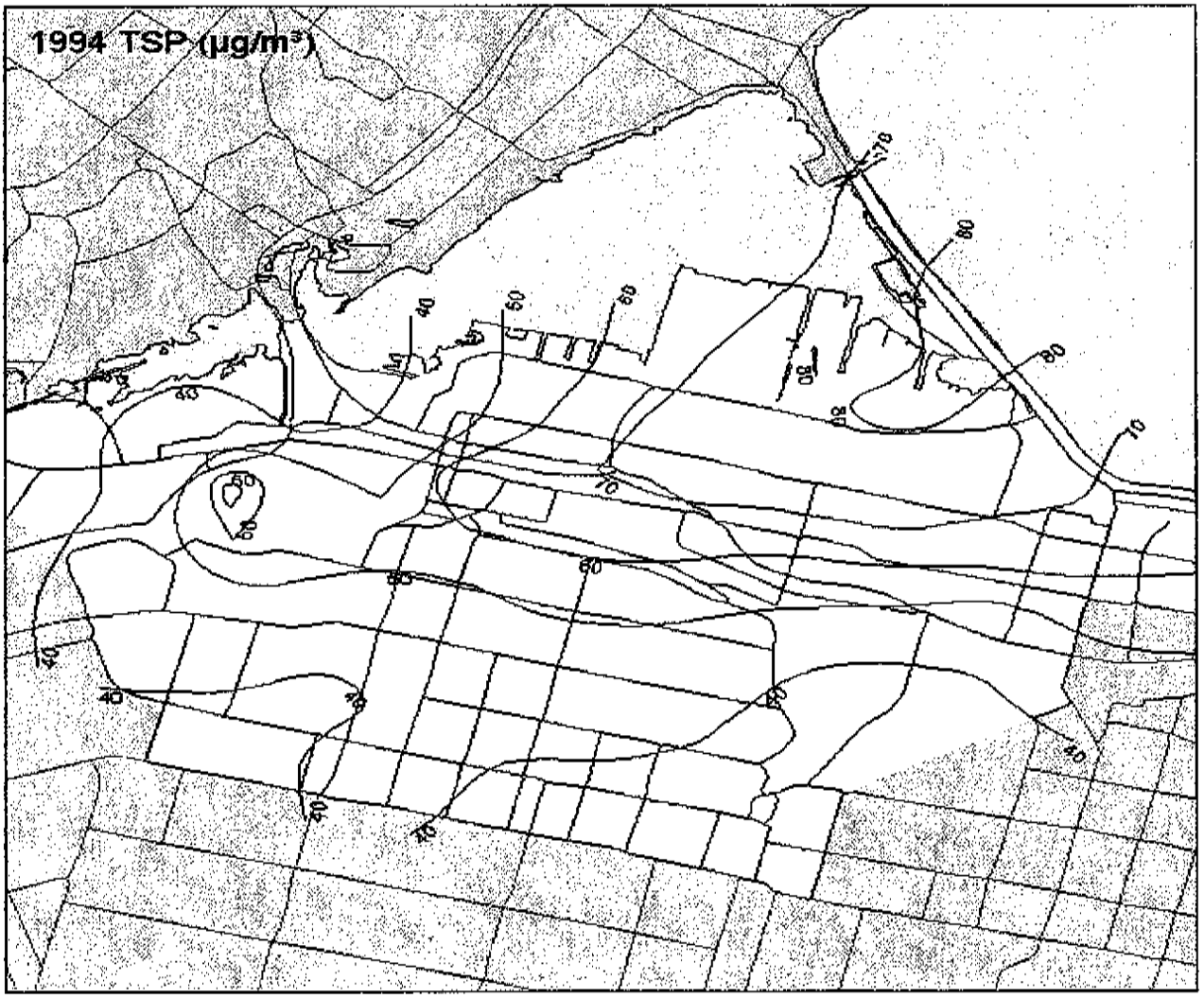
- a video surveillance computerized monitoring system,
- several applied scientific research programs with McMaster University,
- the re-alignment of the MOEE's regional ambient network monitoring stations,
- engineering, process and emissions reviews of Columbian Chemical, Dofasco and Stelco,
- proactive operation, maintenance and housekeeping practices by Columbian Chemical,
- enhanced cooperation with the Committee by Columbian Chemical, Dofasco and Stelco, and
- an optical remote sensing demonstration study using Lidar (Laser radar).

5.2.2 Health Aspects

The possible/probable health effects of the black airborne particulate material falling in northeast Hamilton is a very difficult question to answer as the composition, texture and source(s) of this material are not well defined. Some residents think it's a fine smeary sooty oily type of black material whereas others think it's a dry coarser type of material. Others think it's ubiquitous

throughout the area and that it's a result of gradual or long-term deposition; others think it's a result of an upset or spill at one of the plants; and others think it's a result of a combination of both coupled poor weather conditions. As an example of the complexity of this issue, optical and spectral analysis of many air samples acquired in this area have revealed over 20 different compounds of which at least half are black; these include coal, coke, kish, coal soot, flyash, biological material, slag, tire rubber, asphalt, carbon black and mineral dust. As suggested by this analysis, there are many steady-state sources of black fallout material including the steel-making operations, the carbon black industry, traffic (especially diesel vehicles), and product (both raw and finished) handling. In addition, there are accidents, spills, fugitive emissions, drag-out operations and long-range atmospheric transport from upwind locations.

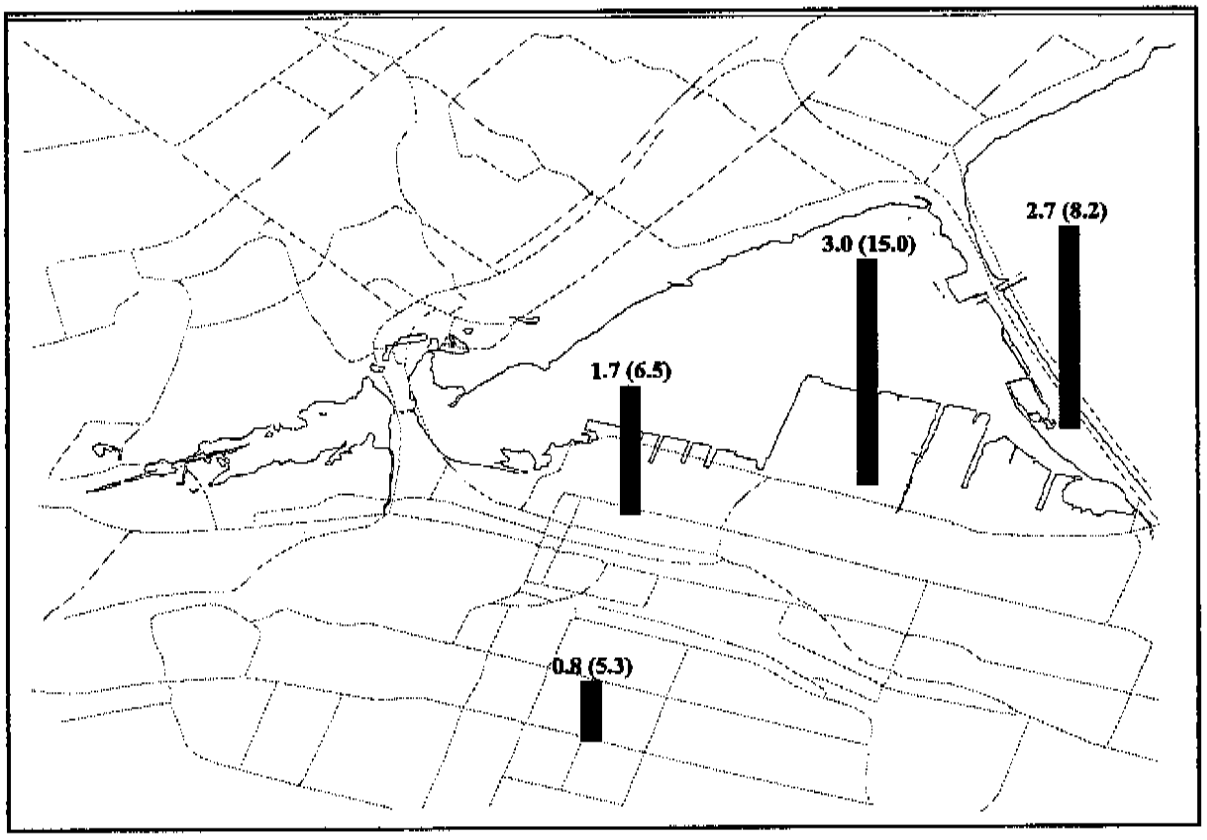
Figure 13
Annual Average TSP Isopleths - 1994



Although there are many individual components of black fallout, many come from a combustion process. Not only are particles a product of combustion but also polycyclic aromatic hydrocarbons (PAHs) and the most carcinogenic of the PAHs is benzo[a]pyrene (B[a]P).

The Ministry's Ambient Air Quality Criteria (AAQC) for both particulate matter and B[a]P continues to be exceeded in northeast Hamilton. In 1994 and as shown in Figure 11, the 60$\mu g/m^3$ total suspended particulate (TSP) AAQC isopleth covers most of northeast Hamilton below the escarpment. The maximum annual average ground level concentration isopleth is 80$\mu g/m^3$.

Figure 14 - Urban Toxics Network in Hamilton - PAHs: 1994
Annual Average B[a]P Concentrations
(Maximum 24-Hour B[a]P Concentrations)
Concentration Units : $\eta g/m^3$



In 1994, the MOEE's annual AAQC for B[a]P from all sources (i.e. 0.3 $\eta g/m^3$) was exceeded at all 4 PAH Urban Air Toxics monitoring stations in Hamilton. Recently, much analytical work is being done by McMaster University on the identification of the various PAHs (not just B[a]P) on collected filter media; but no health assessment has been done on these specific results.

The inhalable particulate (i.e. the PM_{10} fraction) and B[a]P components of the black fallout are probably the most important elements with respect to human health. Both PM_{10} and B[a]P have been treated in previous sections of this report and these health risk assessments would be a reasonable starting point in the interpretation of the human health effects of black fallout. Due to the importance of this issue, a special air monitoring station was installed in the Homeside area. It was very interesting to note that from the analysis of the acquired data, no statistical significant difference was found between the airborne PM_{10} concentrations measured at this monitoring station and that measured in downtown Hamilton (Homeside versus Elgin/Kelly - Figure 11).

Since B[a]P is adsorbed onto particulates, the reduction of any particulate emissions from the many combustion sources in the industrial sector of Hamilton should also reduce the airborne B[a]P concentrations. In fact, this is the current abatement strategy being followed by the Ministry. But before we go onto the interpretation and health risk assessment step, an agreement as to what exactly constitutes black fallout has to be established.

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