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Canadian Environmental Protection Act, 1999

PRIORITY SUBSTANCES LIST ASSESSMENT REPORT

Respirable Particulate Matter Less Than or Equal to 10 Microns

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PRIORITY SUBSTANCES LIST ASSESSMENT REPORT

Respirable Particulate Matter Less Than or Equal to 10 Microns

Environment Canada
Health Canada

May 2000

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List of Acronyms and Abbreviations

CEPA 1999	*Canadian Environmental Protection Act, 1999*
COPD	chronic obstructive pulmonary disease
CVD	cardiovascular disease
ERDA	Energy Research and Development Authority
FAM	fixed ambient monitor
MMAD	mass median aerodynamic diameter
NAAQO	National Ambient Air Quality Objective
NAPS	National Air Pollution Surveillance
PM	particulate matter
$PM_{2.5}$	particles ≤2.5 µm in diameter
PM_{10}	particles ≤10 µm in diameter
$PM_{10–2.5}$	"coarse" particulate matter, particles >2.5 µm but ≤10 µm in diameter
PSL	Priority Substances List
pTEAM	Particle Total Exposure Assessment Methodology
RR	relative risk
SD	standard deviation
SSI	size selective inlet
TEOM	Tapered Element Oscillation Microbalance
TSP	total suspended particulate
VOC	volatile organic compound

SYNOPSIS

Particulate matter (PM) refers in this assessment to particles of less than or equal to 10 µm mass median aerodynamic diameter (PM_{10}). PM_{10} generally includes a fine fraction of particles 2.5 µm or less ($PM_{2.5}$) and a coarse fraction of particles larger than 2.5 µm ($PM_{10–2.5}$). Particulate matter can be emitted directly into the atmosphere or formed secondarily from precursor gases as a result of physical and chemical transformations. Particulate matter may include a broad range of chemical species, such as elemental carbon and organic carbon compounds, oxides of silicon, aluminum and iron, trace metals, sulphates, nitrates and ammonia.

Particulate matter is ubiquitous, being emitted from both natural and anthropogenic sources. The fine fraction of particulate matter and its precursor gases originate typically from combustion processes — motor vehicles, industrial processes and vegetative burning. In contrast, the coarse fraction of PM_{10} is associated with mechanical processes, such as wind erosion, breaking ocean waves and grinding operations. The available data indicate that source contributions to primary particulate matter emissions and precursor gases in Canada vary by province/territory and by region. Industrial sources provide a major contribution in most provinces, followed by non-industrial fuel combustion and the transportation sector. Forest fires and prescribed burning are the largest estimated sources of particulate matter in some provinces and in the territories. Long-range transport from industrial regions of the United States makes a major contribution to levels of particulate matter in some regions of Canada.

Concentrations of particulate matter typically vary by time of day, day of the week, season and year. Based on fixed-site monitoring of 24-hour concentrations in ambient air, long-term mean PM_{10} concentrations during the mid-1980s to mid-1990s ranged from 11 to 42 µg/m^3 at urban sites and during the mid-1990s ranged from 11 to 17 µg/m^3 at rural sites. The corresponding values for $PM_{2.5}$ were 6.9–20.2 µg/m^3 and 7.0–10.5 µg/m^3, respectively. The values for both PM_{10} and $PM_{2.5}$ are above estimated background levels, indicating that anthropogenic activities make an important contribution to ambient particulate matter loadings. On a national scale, average particulate matter concentrations decreased by approximately 2–3% annually between 1984 and 1995.

In numerous epidemiological studies from around the world, including Canada, positive associations have been observed between ambient levels of particulate matter (as PM_{10}, $PM_{2.5}$ or other particle metrics) and a range of health outcomes, including daily mortality, respiratory and cardiovascular hospitalizations, impaired lung function, adverse respiratory symptoms and medication use, restricted activity days and the frequency of reported chronic respiratory disease. These associations could not be explained by the influence of weather, season, yearly trends, day-to-day variations or variations due to holidays, epidemics or other non-pollutant factors. While the populations studied were always exposed to other air pollutants in addition to particulate matter, associations of a similar magnitude were observed across numerous locations with differing air pollutant mixtures, and the association with particulate matter remained in analyses that adjusted for the effects of various other pollutants. These particulate matter-related health effects were observed at ambient concentrations that currently occur in Canada.

Therefore, the epidemiological evidence for mortality and morbidity in response to current levels of particulate air pollution meets a number of the criteria for causality, including consistency, dose–response relationship, coherence, temporal relationship and specificity of both outcome and agent. With respect to the biological plausibility

of the association, the results of experimental studies in animals and humans provide some limited support for the epidemiological findings. However, both animal and experimental human work are constrained by the technological difficulties in reproducing environmentally relevant particulate matter, and this work has generally been conducted at high levels with artificial particles. Some of this work, specifically the most recent work with concentrated ambient particles, has provided initial evidence of the particulate matter-induced effects on the cardiorespiratory system, particularly in individuals with pre-existing respiratory and cardiovascular disease, and has provided preliminary indications of possible mechanisms. The database supports, therefore, a causal relation between current ambient PM_{10} and $PM_{2.5}$ exposure and adverse health effects and provides a reasonable basis for preventive action.

Based principally on the sufficient weight of evidence of mortality and morbidity in the general population exposed to ambient concentrations of PM_{10} and $PM_{2.5}$ examined in recent extensive epidemiological analyses in Canada and in other countries (at ambient concentrations currently occurring in Canada), as well as on some limited supporting data in experimental animal and controlled human exposure studies, PM_{10} and particularly $PM_{2.5}$ are considered to be entering the environment in a quantity or concentration or under conditions that constitute or may constitute a danger in Canada to human life or health. Therefore, PM_{10} and particularly $PM_{2.5}$ are considered to be "toxic" as defined in Section 64 of the *Canadian Environmental Protection Act, 1999* (CEPA 1999).

Based on available data on the health effects of particulate matter, investigations of options to reduce exposure to particulate matter should be focussed on the fine fraction ($PM_{2.5}$). They should also be designed to reduce mid-range (24-hour average) rather than peak (i.e., <24-hour periods) exposures, since, on the basis of available data, 24-hour average exposure is associated with increases in mortality and morbidity.

The available data clearly indicate that relative source contributions to PM_{10} and $PM_{2.5}$ vary by province/territory and by region. There are ongoing initiatives in risk management designed to accommodate these regional variations. Under the Canada-wide Standards subagreement of the Harmonization Accord signed by the Environment Ministers in January 1998, federal and provincial/territorial governments will develop numerical air quality standards for PM_{10} and $PM_{2.5}$, with each jurisdiction developing a plan of action to achieve the standards in a specified time frame. Any investigations of options to reduce exposure as a result of the assessment of particulate matter as a Priority Substance under CEPA 1999 will complement those for this ongoing initiative.

The conclusion of this assessment is based on estimated and measured ambient levels of PM_{10}. However, the assessment acknowledges that this substance can be emitted directly into the atmosphere or formed secondarily from precursors as a result of physical or chemical transformations. It is recommended that stakeholders be consulted on the need to add precursors to PM_{10} to the List of Toxic Substances in Schedule 1 and on the form of the Schedule 1 listing.

1.0 INTRODUCTION

The *Canadian Environmental Protection Act, 1999* (CEPA 1999) requires the federal Ministers of the Environment and of Health to prepare and publish a Priority Substances List (PSL) that identifies substances, including chemicals, groups of chemicals, effluents and wastes, that may be harmful to the environment or constitute a danger to human health. The Act also requires both Ministers to assess these substances and determine whether they are "toxic" or are capable of becoming "toxic" as defined in Section 64 of the Act, which states:

> …a substance is toxic if it is entering or may enter the environment in a quantity or concentration or under conditions that
> (a) have or may have an immediate or long-term harmful effect on the environment or its biological diversity;
> (b) constitute or may constitute a danger to the environment on which life depends; or
> (c) constitute or may constitute a danger in Canada to human life or health.

Substances that are assessed as "toxic" as defined in Section 64 may be placed on Schedule 1 of the Act and considered for possible risk management measures, such as regulations, guidelines, pollution prevention plans or codes of practice to control any aspect of their life cycle, from the research and development stage through manufacture, use, storage, transport and ultimate disposal.

Based on an initial screening of readily accessible information, the rationale for assessing "respirable particulate matter less than or equal to 10 microns" provided by the Ministers' Expert Advisory Panel on the Second Priority Substances List (Ministers' Expert Advisory Panel, 1995) was as follows:

> Exposure to respirable particulate matter in the Canadian environment is widespread. Sources include vehicle exhaust, construction, industrial air pollution and the bulk shipping of minerals. Small particles, irrespective of their origins, are trapped in the lungs. Effects associated with ambient exposure to respirable particulate matter include respiratory and pulmonary health dysfunction, which can lead to school absenteeism and increased hospital admissions. An assessment is needed to evaluate health risks.

The potential impacts of ambient exposure to respirable particulate matter on the environment and on human health have recently been evaluated by the Federal–Provincial Working Group on Air Quality Objectives and Guidelines as part of the development of National Ambient Air Quality Objectives (NAAQO) for Particulate Matter. The relevant background material was reviewed and the potential risks from ambient PM_{10} and $PM_{2.5}$ were assessed in the Science Assessment Document produced in support of the NAAQO (WGAQOG, 1998). This Assessment Report simply summarizes the critical information from that document in the context of the PSL program. Only the risks posed by ambient particulate matter to human health (i.e., whether respirable particulate matter ≤10 µm [as PM_{10} and $PM_{2.5}$] is "toxic" as defined in Paragraph 64(c) of CEPA 1999) are addressed in this Assessment Report, since these were the critical effects identified in the NAAQO Science Assessment Document.

The Science Assessment Document on which this Assessment Report is based was prepared by the following individuals:

R. Bailey, Environment Canada
M. Bourgeau, Health Canada
E. Bush, Environment Canada
T. Dann, Environment Canada
L. Liu, Health Canada
S. MacDonald, Health Canada
S. Pryor, Indiana University
M. Shepherd, Environment Canada
N. Suzuki, British Columbia Environment
K. Timoffee, Health Canada
G. Wood, Health Canada

Selected sections of the NAAQO Science Assessment Document were prepared based on background contract reports by G. Oberdörster, University of Rochester (toxicokinetics, animal studies) and by M. Utell and J. Samet, University of Rochester and Johns Hopkins University, respectively (controlled human studies).

In January 1997, the draft NAAQO Science Assessment Document, incorporating information on fine particles, was submitted to the Federal–Provincial Working Group on Air Quality Objectives and Guidelines for technical review. In March 1997, the draft document was sent to the following external peer reviewers, who provided written comments with respect to the accuracy of reporting, adequacy of coverage and defensibility of conclusions:

G. Pengelly, McMaster University
J. Samet, Johns Hopkins University
C. Shy, University of North Carolina at Chapel Hill
M. Williams, U.K. Department of Environment, Transport and the Regions
W. Wilson, U.S. Environmental Protection Agency

Information acquired up to the end of the period of peer review (i.e., April/May 1997) was considered in revising the NAAQO Science Assessment Document. Following revisions, technical editing and translation, the final Science Assessment Document was released in 1999. Staff of the Priority Substances Section of Health Canada (R. Newhook and M.E. Meek) assisted in the preparation of this Assessment Report, primarily to provide context for presentation to meet the needs of the Priority Substances program.

For additional information on the preparation process for or the content of the NAAQO Science Assessment Document, please contact B. Jessiman of the Air and Waste Section of Health Canada (health-related sections) or M. Shepherd of the Atmospheric Environment Service of Environment Canada (environmental sections).

The health-related sections of the Assessment Report were reviewed and approved by the Health Protection Branch Risk Management meeting of Health Canada.

The entire Assessment Report was reviewed and approved by the Environment Canada/Health Canada CEPA Management Committee.

A draft of the Assessment Report was made available for a 60-day public comment period from May 15 to July 14, 1999 (Environment Canada and Health Canada, 1999). Following consideration of comments received, the Assessment Report was revised as appropriate. A summary of the comments and their responses is available on the Internet at:

www.ec.gc.ca/cceb1/eng/final/index_e.html

Copies of this Assessment Report are available upon request from:

Inquiry Centre
Environment Canada
Main Floor, Place Vincent Massey
351 St. Joseph Blvd.
Hull, Quebec
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or on the Internet at:

www.ec.gc.ca/cceb1/eng/final/index_e.html

The NAAQO Science Assessment Document on which this Assessment Report is based is available upon request from:

Environmental Health Centre
Room 104
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2.0 SUMMARY OF INFORMATION CRITICAL TO ASSESSMENT OF "TOXIC" UNDER CEPA 1999

2.1 Identity and physical/chemical properties

2.1.1 Identity

Particulate matter is defined for the purposes of this assessment as particles of less than or equal to 10 µm mass median aerodynamic diameter (MMAD) (PM_{10}) that are emitted directly into the atmosphere or formed secondarily from precursor gases as a result of physical and chemical transformations.

Particles may range from approximately 0.005 µm to 100 µm in diameter, although the suspended portion is generally less than 40 µm. PM_{10} is generally subdivided into a fine fraction of particles 2.5 µm or less ($PM_{2.5}$) and a coarse fraction of particles larger than 2.5 µm ($PM_{10–2.5}$). It is further classified as primary (emitted directly into the atmosphere) or secondary (formed in the atmosphere through chemical and physical transformations). The principal gases involved in secondary particulate formation are sulphur dioxide (SO_2), nitrogen oxides (NOx), volatile organic compounds (VOCs) and ammonia. Primary particles are present in both the fine and coarse fractions, whereas secondary particles, such as sulphates and nitrates, are present predominantly in the fine fraction. Both primary and secondary particulate matter can result from either natural or anthropogenic (human-made) sources.

Particulate matter is unique among atmospheric constituents in that it is not defined on the basis of its chemical composition. It may include a broad range of chemical species, including elemental carbon and organic carbon compounds, oxides of silicon, aluminum and iron, trace metals, sulphates, nitrates and ammonia.

The evaluation of the scientific information on particulate matter in this document focusses on particle size because the evidence indicates that particle size is important in influencing the site of deposition in the respiratory tract and the degree of toxicity. Particle size also reflects origin and formation of airborne particles, the larger sizes being often of crustal origin and the smaller sizes originating from combustion processes.

2.1.2 Physical/chemical properties

2.1.2.1 Particle size

Particle size is considered to be one of the most relevant parameters in characterizing the physical behaviour of particulate matter in the atmosphere. Extremely small ("ultrafine") particles less than 0.1 µm in diameter (the nuclei mode) are formed primarily from the condensation of hot vapours during high-temperature combustion processes and from the nucleation of atmospheric species to form new particles. While the greatest concentration of airborne particles is found in the nuclei mode, these particles contribute little to overall particle mass loading due to their tiny size. They are subject to random motion and to coagulation processes in which particles collide to quickly yield larger particles. Consequently, these tiny particles have short atmospheric residence times.

Particles in the size range of 0.1–2.0 µm (the accumulation mode) result from the coagulation of particles in the nuclei mode and from the condensation of vapours onto existing particles, which then grow into this size range. These particles account for most of the particle surface area and much of the particle mass in the atmosphere. The accumulation mode is so-named since atmospheric removal processes are least

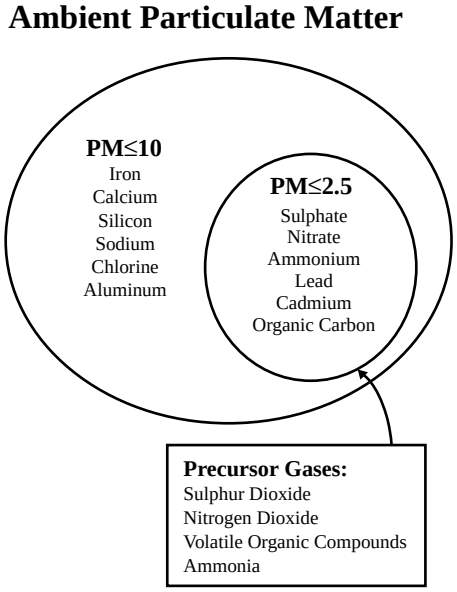
efficient in this size range. These fine particles can remain in the atmosphere for days to weeks. Dry deposition and precipitation scavenging are the primary processes by which these fine particles are eventually removed from the atmosphere. It is calculated that precipitation scavenging accounts for about 80–90% of the mass of particles in the accumulation mode removed from the atmosphere (Wallace and Hobbs, 1977).

Particles larger than 2.0 µm (the sedimentation or coarse mode) are typically associated with mechanical processes, such as wind erosion, breaking ocean waves and grinding operations. Grinding operations result in the physical breakdown of larger particles into smaller ones, such as windblown soil, sea salt spray and dust from quarrying operations. These particles are efficiently removed by gravitational settling and therefore remain in the atmosphere for shorter periods of a few hours to a few days. They contribute little to particle number concentrations but significantly to total particle mass.

2.1.2.2 Chemical composition

As a consequence of their different sources and mechanisms of production, fine and coarse particles have markedly different chemical composition also (Figure 1). Coarse particles consist primarily of particles derived from the earth's crust and are therefore rich in oxides of iron, calcium, silicon and aluminum and are typically basic in nature. Particles in coastal regions are enriched with sodium chloride from sea salt. Fine particles are composed mainly of sulphate, nitrate, ammonium, inorganic and organic carbon compounds, and heavy metals such as lead and cadmium, all of which are indicators of anthropogenic production processes (Seinfeld, 1986). Fine particles tend to be acidic in nature. Sulphate has repeatedly been shown to be the most abundant single component of fine particles (Keeler *et al.*, 1990; Environment Canada, 1994). However, only a few of the numerous organic carbon compounds have been identified, and together these may comprise approximately 50% of the fine particle mass (van Houdt, 1990; Lowenthal *et al.*, 1994).

FIGURE 1 Generalized chemical composition of particulate matter



Comparisons of urban and rural sites in close proximity to one another demonstrate that urban particulate matter concentrations are higher than rural ones, particularly for coarse particulate matter. This is mirrored by an enrichment in urban areas in the concentration of all inorganic elements and ions assayed. There are several elements/ions for which the urban–rural difference is disproportionately greater than the total mass difference, however, indicating that these constituents are particularly enriched in urban areas (calcium, silicon, nitrate, iron, aluminum, magnesium, zinc, titanium, manganese, vanadium, lead, nickel). This pattern is most likely attributed to the greater suspension of road dust and more intensive industrial and combustion activity in urban areas.

Estimates of the amount of fine and coarse particle mass attributable to carbonaceous material (organic and elemental carbon) were made using a mass reconstruction technique and data on inorganic species (Brook *et al.*, 1997).

Depending upon site, only about 37–61% of the $PM_{2.5}$ could be explained given the measured concentrations of several inorganic ions and elements. Thus, carbonaceous material, which was likely to have been predominantly organic in nature, was responsible for about half of the overall fine particle mass. This fraction was higher in Alberta and British Columbia (~65%) than it was on the east coast (40–45%). Sulphate, nitrate and ammonium dominate the identifiable components of the fine particulate matter mass, consistent with the results of many studies. Because of the increased importance of crustal material, a greater portion of the coarse particle mass (~70%) was explained by the inorganic constituents.

2.1.2.3 Other properties

Other physical characteristics that affect particle behaviour include particle shape and density and bulk properties such as chemical composition, vapour pressure, hygroscopicity (water-attracting nature), deliquescence and refractive index. Surface properties such as electrostatic charge, the presence of surface films and surface irregularities may also influence particle behaviour. Small particles are characterized by a large surface area relative to their mass, which, when combined with surface irregularities and internal pores, leads to greater reactivity of fine particles compared with coarse particles.

2.2 Sources of particulate matter

Particulate matter is a ubiquitous pollutant, reflecting the fact that it has both natural and anthropogenic sources.

Natural sources of primary particulate matter include windblown soil and mineral particles, volcanic dust, sea salt spray, biological material such as pollen, spores and bacteria, and debris from forest fires. By and large, these natural sources produce coarse particles, although high-temperature combustion sources such as wildfires will generate fine particulate matter. Secondary particulate matter can be formed through reactions involving natural sources of the precursor gases. For example, VOCs are released from trees, and nitrogen oxides are released from soils.

Anthropogenic sources also produce both primary and secondary particulate matter and both coarse and fine particles. Windblown agricultural soil and dust from roads, construction sites and quarrying operations all contribute primarily to the coarse fraction. Smaller particles of more complex chemical composition are generated as a result of many industrial processes and through fossil fuel combustion (electrical power plants, gasoline and diesel vehicles, industrial boilers, residential heating, etc.), both directly and via the release of precursor gases (VOCs, sulphur dioxide and nitrogen oxides) (Table 1).

Current estimates of the magnitude of primary particulate matter emissions have been compiled as part of the Environment Canada Criteria Air Contaminants 1995 emission inventory. This inventory includes emission estimates for primary particulate matter designated as PART (total particulate matter), PM_{10} (particles sized from 0 to 10 μm) and $PM_{2.5}$ (particles sized from 0 to 2.5 μm), as well as emissions of the principal precursor gases that contribute to the formation of particulate matter.

Environment Canada compiles emission inventories based upon three source types: point, area and mobile. Provinces and territories provide the point source information. Emissions from all three source types are estimated by applying some emission factor to a base quantity related to activity or production. Minimal information is available for directly measured emissions. For point sources, the U.S. Environmental Protection Agency PMCALC model was used to estimate emissions based upon Standard Classification Codes for each source type. Area and mobile source emissions are modelled using source-specific emission factors (principally from the U.S. Environmental Protection Agency AP42 Compilation of Air Pollutant Emission Factors)

TABLE 1 Sources of particulate matter

Natural		Anthropogenic	
Primary	**Secondary**	**Primary**	**Secondary**
$PM_{2.5}$			
• wildfire (elemental carbon and organic carbons)	• organic carbons from biogenic VOCs • nitrates from natural NOx	• fossil fuel combustion (industrial, residential, autos) (elemental carbon and organic carbons) • residential wood combustion (elemental carbon and organic carbons)	• organic carbons from anthropogenic sources of VOCs (autos, industrial processes, solvents) • sulphates and nitrates from anthropogenic sources of SO_x and NOx (autos, power plants, etc.)
PM_{10}			
• windblown dust • sea salt spray • pollen, spores		• mineral dust from mining and extraction industries • windblown agricultural soil • road dust • tire and brake wear • dust from construction sites	

and information regarding activity level from Environment Canada, Statistics Canada, Natural Resources Canada and provincial/territorial government publications.

Table 2 provides the 1995 national Criteria Air Contaminants emission inventory by category. The provincial source sector breakdown is provided in Table 3. The sources included in each category in Table 3 are as listed in Table 2.

The available data clearly indicate that source contributions of primary particulate matter vary by province/territory and by region. In Yukon, the Northwest Territories, British Columbia and Saskatchewan, forest fires and prescribed burning are the largest estimated sources of particulate matter. Industrial sources are a major contributor to particulate matter emissions including precursor gases in all provinces except Prince Edward Island. In Prince Edward Island, non-industrial fuel combustion (primarily residential wood combustion) is a major source. The transportation sector is also a large contributor in Ontario and British Columbia, while non-industrial fuel combustion is significant in Alberta, Manitoba, Ontario, Quebec, New Brunswick and Nova Scotia. A further consideration is the seasonality of particulate emissions. Residential wood combustion is most prevalent during the winter months, while forest fires are generally limited to the summer months. Emissions from the industrial and transportation sectors occur year-round, although they are subject to many fluctuations.

Characterization of the sources and atmospheric processes that contribute to ambient particulate matter levels is a complex process due to the contribution of secondary formation from diverse biogenic and anthropogenic precursor sources. Primary particles, those emitted directly from sources, undergo few physical or chemical changes between source and receptor. Consequently, atmospheric concentrations are approximately proportional to their emission concentrations. Secondary particles undergo physical and chemical transformations, masking the chemical composition of the original source. Most sulphate and nitrate particles are of

Table 2 The 1995 Criteria Air Contaminants emission inventory for Canada (tonnes)[1]

Category/sector	PART[2]	PM_{10}	$PM_{2.5}$	SO_x	NOx	VOCs
Industrial sources						
Abrasives manufacture	784	361	254	2 827	187	1 481
Aluminum industry	11 758	7 787	5 331	46 236	1 058	963
Asbestos industry	80	48	25	763	240	1
Asphalt paving industry	32 930	5 460	1 950	2 384	2 014	3 318
Bakeries						6 005
Cement and concrete industry	21 079	8 486	3 769	33 984	32 168	438
Chemicals industry	4 495	2 611	1 391	6 430	24 118	9 403
Clay products industry	2 576	622	181	34	128	3
Coal mining industry	11 663	8 849	6 265	5 321	3 232	1 762
Ferrous foundries	667	448	362	1 673	28	1 807
Grain industries	58 274	11 729	1 742	1	31	2
Iron and steel industries	20 672	10 813	7 085	62 801	25 490	28 277
Iron ore mining industry	39 412	21 290	7 625	54 650	7 767	839
Mining and rock quarrying	86 016	11 508	3 223	20 770	14 578	688
Non-ferrous mining and smelting industry	15 630	13 159	9 845	891 720	3 532	75
Oil sands	3 937	1 787	1 407	160 948	16 542	81
Other petroleum and coal products industry	324	121	57	578	418	88
Paint and varnish manufacturing	124	99	35		18	1 957
Petrochemical industry	1 310	660	265	1 275	11 598	16 523
Petroleum refining	6 522	5 012	3 268	141 086	26 923	47 655
Plastics and synthetic resins fabrication	162	90	62	272	382	6 684
Pulp and paper industry	74 384	50 835	39 337	77 030	58 064	23 283
Upstream oil and gas industry	2 053	2 005	1 938	387 261	314 905	689 393
Wood industry	153 697	86 002	52 594	2 621	16 025	47 100
Other industries	72 612	37 474	23 833	48 953	60 893	52 988
Total industrial sources	621 160	287 255	171 847	1 949 617	620 343	940 814
Non-industrial fuel combustion						
Commercial fuel combustion	3 360	2 963	2 683	13 014	29 291	1 787
Electric power generation (utilities)	78 797	34 874	18 633	534 323	254 985	2 980
Residential fuel combustion	4 787	3 956	3 692	17 270	36 699	2 353
Residential fuel wood combustion	137 840	137 268	131 797	1 837	12 176	400 092
Total non-industrial fuel combustion	224 784	179 060	156 806	566 445	333 152	407 211

Table 2 (continued)

Category/sector	PART[2]	PM_{10}	$PM_{2.5}$	SO_x	NOx	VOCs
Transportation						
Air transportation	2 018	1 115	787	2 263	34 026	11 636
Heavy-duty diesel vehicles	32 075	32 075	29 498	32 807	378 300	48 540
Heavy-duty gasoline trucks	545	528	414	588	15 073	11 814
Light-duty diesel trucks	1 304	1 304	1 203	1 535	5 567	2 600
Light-duty diesel vehicles	379	379	347	632	1 978	747
Light-duty gasoline trucks	2 586	2 509	1 986	4 399	112 437	142 425
Light-duty gasoline vehicles	4 870	4 717	3 256	11 048	273 396	355 873
Marine transportation	8 438	8 129	7 379	58 000	118 578	37 449
Motorcycles	16	16	11	34	630	2 027
Off-road use of diesel	17 081	17 081	15 714	16 149	209 231	22 581
Off-road use of gasoline	4 414	3 867	3 393	1 005	25 395	93 111
Rail transportation	19 492	19 492	17 933	7 226	115 604	5 608
Tire wear and brake lining	4 362	4 313	1 353			
Total transportation	97 580	95 524	83 276	135 686	1 290 214	734 412
Incineration						
Crematorium	3	2	1	3	19	
Industrial and commercial incineration	70	51	38	603	752	690
Municipal incineration	435	370	355	457	1 298	703
Wood waste incineration	1 846	1 015	738	42	318	4 568
Other incineration and utilities	157	38	16	149	163	294
Total incineration	2 510	1 476	1 149	1 253	2 550	6 255
Miscellaneous						
Cigarette smoking	962	962	962		8	8
Dry cleaning					1	7 832
Fuel marketing	30	30	30	2	256	98 498
General solvent use						274 926
Marine cargo handling industry	3 074	1 385	416			1
Meat cooking	1 594	1 594	1 583			
Pesticides and fertilizer application	10 516	5 153	1 472		792	66
Printing						29 058
Structural fires	5 297	5 244	4 768		10	5 147

Table 2 (continued)

Category/sector	PART[2]	PM_{10}	$PM_{2.5}$	SO_x	NOx	VOCs
Surface coatings						134 194
Total miscellaneous	21 472	14 368	9 232	2	1 068	549 731
Open sources						
Agriculture (animals)	248 734	141 041	22 280			12 982
Agriculture tilling and wind erosion	1 754 440	848 408	18 037			
Construction operations	2 402 115	528 449	10 707			
Dust from paved roads[3]	2 549 526	511 159	129 517			
Dust from unpaved roads[3]	6 833 650	2 020 663	300 644			
Forest fires	835 391	706 095	585 048	478	211 027	902 444
Landfill sites	4 735	379	94			5 139
Mine tailings	46 858	3 749	937			
Prescribed burning	41 415	32 986	26 872	92	5 551	16 306
Total open sources	14 716 862	4 792 926	1 094 136	569	216 578	936 871
National total	15 684 370	5 370 610	1 516 445	2 653 571	2 463 904	3 575 293

[1] Notes:

1. Table source is Deslauriers (2000).
2. Numbers may not add to totals, due to rounding.
3. The 1995 emission inventory was compiled with the latest technical and statistical information available; only the sulphur oxide emissions can be compared with previous emission inventories.
4. The estimates for PART, PM_{10} and $PM_{2.5}$ refer to primary particulate matter emissions only.

[2] PART = total particulate matter.

[3] Work is in progress to improve the road dust emission estimates, using the road dust study in British Columbia and additional measurements and statistics for selected provinces. These estimates provide an update to the mid-1998 emission figures released in Ontario and other provinces.

Table 3 Provincial/territorial distribution of particulate matter and precursor emissions (tonnes), 1995[1]

Category/sector	Nfld.	PEI	NS	NB	Que.	Ont.	Man.	Sask.	Alta.	BC	Yukon	NWT	Canada
PART[2]													
Total industrial sources	44 882	1 142	19 352	25 161	85 375	162 747	20 108	39 193	73 549	144 872	1 626	3 154	621 160
Total non-industrial fuel combustion	12 559	1 717	16 523	13 943	41 307	37 330	6 072	61 676	18 876	13 875	244	663	224 784
Total transportation	1 796	478	2 061	2 267	19 343	23 497	3 687	6 033	13 282	24 775	207	154	97 580
Total incineration	252	5	118	610	58	929	65	69	21	381	0	1	2 510
Total miscellaneous	107	201	352	345	2 562	4 481	2 577	3 033	3 166	4 583	35	32	21 472
Total open sources	274 219	86 735	378 251	411 940	2 006 207	3 279 005	951 942	1 534 015	4 946 513	458 158	67 784	322 093	14 716 862
PM_{10}													
Total industrial sources	25 116	305	7 296	10 338	38 902	67 203	8 129	10 796	38 478	80 120	192	380	287 255
Total non-industrial fuel combustion	12 138	1 693	14 838	13 227	41 041	32 419	5 375	25 807	17 922	13 752	237	613	179 060
Total transportation	1 743	473	2 009	2 241	19 019	22 528	3 592	5 921	13 023	24 631	204	140	95 524
Total incineration	138	3	59	336	28	472	36	38	16	350	0	0	1 476
Total miscellaneous	102	127	308	284	2 070	3 592	1 610	1 655	1 937	2 626	30	27	14 368
Total open sources	62 952	21 714	90 303	98 063	545 424	918 300	388 569	689 666	1 509 094	181 286	32 287	255 268	4 792 926
$PM_{2.5}$													
Total industrial sources	10 198	106	3 972	5 733	22 322	38 284	4 223	4 154	25 939	56 811	40	66	171 847
Total non-industrial fuel combustion	11 306	1 601	13 844	12 444	38 932	31 358	4 928	11 253	17 270	13 064	224	581	156 806
Total transportation	1 528	411	1 716	1 929	16 331	18 972	3 120	5 251	11 420	22 297	179	122	83 276
Total incineration	101	3	41	244	21	334	26	28	11	340	0	0	1 149
Total miscellaneous	93	73	260	227	1 640	2 807	894	686	1 045	1 461	25	22	9 232
Total open sources	7 636	2 728	14 736	14 591	98 053	168 265	119 824	151 226	213 278	78 512	19 648	205 640	1 094 136
SO_x													
Total industrial sources	43 156	916	20 722	40 695	336 373	498 220	361 261	18 368	467 270	147 749	10	14 877	1 949 617
Total non-industrial fuel combustion	17 507	887	142 798	71 247	8 058	82 312	1 566	108 833	131 285	1 224	147	580	566 445
Total transportation	4 319	712	3 449	3 590	28 696	51 777	2 568	3 797	9 453	26 948	239	138	135 686
Total incineration	31	32	103	11	498	403	1	1	17	133	0	23	1 253
Total miscellaneous	0	0	0	0	0	0	0	0	0	2	0	0	2
Total open sources	1	0	0	0	22	50	79	102	75	53	16	172	569
NOx													
Total industrial sources	10 498	163	10 085	6 306	48 027	97 802	10 573	38 088	327 444	69 876	1 044	437	620 343
Total non-industrial fuel combustion	5 543	687	28 125	19 114	14 160	89 285	3 898	50 838	103 211	11 380	695	6 216	333 152
Total transportation	26 488	7 096	34 749	37 111	310 192	349 511	59 184	80 226	206 326	173 903	2 979	2 449	1 290 214
Total incineration	74	29	84	105	1 101	586	12	13	15	508	0	23	2 550
Total miscellaneous	0	0	0	0	0	1	0	0	0	1 067	0	0	1 068
Total open sources	56	2	18	21	9 664	18 699	35 405	45 327	16 323	7 113	7 030	76 922	216 578

TABLE 3 (continued)

Category/sector	Nfld.	PEI	NS	NB	Que.	Ont.	Man.	Sask.	Alta.	BC	Yukon	NWT	Canada
VOCs													
Total industrial sources	6 443	4	12 804	2 813	40 230	136 946	7 912	140 636	497 497	83 558	496	11 475	940 814
Total non-industrial fuel combustion	20 418	3 263	29 419	25 784	113 794	116 254	12 653	11 877	37 905	33 674	635	1 534	407 211
Total transportation	16 347	4 284	21 974	22 846	166 532	230 723	42 144	48 727	107 946	70 059	1 242	1 588	734 412
Total incineration	514	20	445	1 157	559	2 070	124	131	1 071	127	0	38	6 255
Total miscellaneous	8 928	2 248	14 141	12 679	123 634	255 375	22 263	19 660	50 840	38 942	373	649	549 731
Total open sources	245	17	150	157	42 327	80 755	152 538	195 255	67 473	36 539	30 266	331 150	936 871

[1] Notes:

1. Table source is Deslauriers (2000).
2. Numbers may not add to totals, due to rounding.
3. The 1995 emission inventory was compiled with the latest technical and statistical information available. Only the sulphur oxide emissions can be compared with previous emission inventories.
4. Work is in progress to improve the road dust emission estimates using the road dust study in British Columbia and additional measurements and statistics for selected provinces. These estimates provide an update to the mid-1998 emission figures released in Ontario and other provinces.

[2] PART = total particulate matter.

secondary origin, resulting from sulphur dioxide, nitrogen dioxide (NO_2) and ammonia emissions. Some organic carbon is also of secondary origin, resulting from volatile organic gas emissions. There are limitations in the current ability to comprehensively and accurately assess particulate matter emissions due to a lack of any Canadian source apportionment information — i.e., information with which to estimate the magnitude of secondary particulate matter formed from precursor gases.

Source apportionment, or source attribution, allows for the identification (qualitatively and quantitatively) of contributing sources to support the development of air quality management strategies. Source apportionment techniques use mathematical models that model the precursor gas emissions and complex atmospheric chemistry involved in the formation of secondary particulate matter. There are a variety of techniques of various degrees of complexity, from which several common features emerge:

1) In the coarse fraction, which in some studies has been defined as PM_{10} or the range from $PM_{2.5}$ to PM_{15}, there is a predominance of crustal material (or road dust, which has components of crustal material). The exact contribution, of course, depends on the location, the season, etc. The contribution of crustal sources greatly diminishes in $PM_{2.5}$ samples and is generally less than 5–15%.

2) The greatest contributions to $PM_{2.5}$ come from organic compounds and secondary sulphates and nitrates. In urban areas, the sources of these compounds and their precursor gases (sulphur and nitrogen oxides and VOCs) are typically combustion processes — motor vehicles, industrial processes and vegetative burning. Even remote areas may be impacted by these sources.

3) The early estimates for Canadian cities (EAG, 1984) are consistent with more recent analyses. The estimated motor vehicle source contribution ranges from 9 to 39% of $PM_{2.5}$ from both REVEAL (Lowenthal *et al.,* 1996) in the Lower Fraser Valley of British Columbia and EAG (1984) in a national overview. Chemical mass balance motor vehicle source apportionments within the Vancouver area were highly consistent, at about 40% of $PM_{2.5}$. REVEAL (Pryor and Steyn, 1994) indicates a motor vehicle contribution of 15–20% to $PM_{2.5}$. In Toronto, the motor vehicle contribution is approximately 50% (Lowenthal, 1997).

Long-range transport of particulate matter is also an important source of particulate matter in some regions of Canada. Back-trajectory analysis based on ambient monitoring provides insight into the portion of particulate matter contributing to local concentrations via long-range transport on a continental scale. Ambient concentrations of particulate matter for days on which back-trajectories identified air masses as originating from the north (Canada) or from the south (United States) are summarized in Table 4 for Kejimkujik, Nova Scotia, and Sutton, Quebec. Considering the average values, air masses originating from the north contain approximately two-thirds the PM_{10}, half the $PM_{2.5}$ and one-third the sulphate of air masses originating from the south. These results indicate the substantial contribution (50% or more for fine particles) from long-range transport events from industrial regions of the United States to ambient levels of particulate matter and are consistent with the fact that it is the smaller particles that are most likely to be transported long distances. Further analysis is required to determine how often long-range transport from the United States impacts Canada, and thus the total contribution of long-range transport to particulate matter concentrations in Canada.

2.3 Exposure characterization

2.3.1 Monitoring technology

Measurements of particulate matter for the purpose of current compliance monitoring are generally expressed in terms of mass. Mass measurements may be made directly or indirectly. Direct (or manual) measurements of particulate matter

TABLE 4 Average, median and standard deviation of the 24-hour PM_{10}, $PM_{2.5}$ and sulphate measurements sorted by trajectory group

Site	Trajectory group[1]	n[2]	PM_{10} (µg/m³)			$PM_{2.5}$ (µg/m³)			Sulphate (µg/m³)		
			Average	Median	SD	Average	Median	SD	Average	Median	SD
Sutton	N	100	10.3	8.1	7.6	6.6	5.9	4.3	1.8	1.3	2.0
Sutton	S	27	15.6	15.9	6.3	12.0	11.5	6.1	4.4	4.3	2.8
Kejimkujik	N	145	8.7	7.0	6.7	5.3	4.0	4.5	1.4	1.0	1.9
Kejimkujik	S	56	13.8	11.0	9.8	8.9	7.0	7.3	3.5	2.1	4.5

[1] N = North and S = South.

[2] n corresponds to the number of $PM_{2.5}$ samples used in the calculation.

concentrations in the ambient air are made by collecting particles on a pre-weighed filter over a specified period of time, weighing the soiled filter and then dividing the gain in mass by the volume of air sampled. Samples are typically collected for a 24-hour period. Sampling inlets that remove particles larger than 10 µm may be selected so that particles in the PM_{10} size range are selectively retained on the filter. Particles may also be fractionated into a fine fraction (≤2.5 µm) and a coarse fraction (>2.5–10 µm), which are collected on separate filters for measurement and analysis using dichotomous samplers. Examples of manual samplers include high-volume (hi-vol) and dichotomous (dichot) samplers.

Indirect measurements are made using parameters other than mass, generally optical properties, which can then be converted to units of mass concentration based on known relationships between the two parameters. Examples of indirect methods include British Smoke, Tapered Element Oscillation Microbalance (TEOM) and beta-attenuation monitors; the latter two methods can provide near real-time measurements of particle concentrations.

Intercomparison studies indicate that size selective inlet (SSI) high-volume samplers and dichotomous samplers yield comparable results (Dann, 1994), although there are few data for $PM_{2.5}$ samplers. With respect to indirect samplers, some data from co-located TEOMs and PM_{10} high-volume samplers have shown good agreement, with correlation coefficients of 0.977 for 24-hour averages (Meyer, 1993). However, there are indications that TEOM data are consistently lower (on the order of 25%) than data from manual samplers (Moore and Barthelmie, 1995).

2.3.2 Ambient levels of particulate matter

2.3.2.1 Background concentrations

"Background" particulate matter is generally defined as the distribution of particulate matter concentrations that would be observed in the absence of anthropogenic emissions of particulate matter and of VOC, nitrogen oxide and sulphur oxide (SO_x) precursors. The actual magnitude of background concentrations of particulate matter for a given location is difficult to determine because of the influence of long-range transport of anthropogenic particles and precursor gases. The range of expected background concentrations on an annual or long-term basis is from 4 to 11 µg/m³ for PM_{10} and from 1 to 5 µg/m³ for $PM_{2.5}$ for remote sites in North America (Trijonis, 1982; NAPAP, 1991; Malm *et al.*, 1994). The range of expected background concentrations on a short-term basis is much broader given the episodic nature of such natural events as wildfires and prairie dust storms, which can result in short-term particulate matter levels comparable to those in polluted urban atmospheres.

2.3.2.2 Rural and urban concentrations

A national PM_{10} and $PM_{2.5}$ monitoring program has been in operation since 1984 under the auspices of the National Air Pollution Surveillance (NAPS) network. This is primarily an urban network with few rural sites. Measurements are obtained from SSI high-volume samplers (PM_{10}) and dichotomous samplers (PM_{10} and $PM_{2.5}$). Since 1994, hourly PM_{10} data from TEOM instruments at NAPS sites have also been reported to the database. In addition to the national network, British Columbia, Ontario and Quebec operate particulate matter monitors. Particulate matter data are typically collected over a 24-hour sampling period on a one-day-in-six sampling regime. By operating on this schedule, given a long enough sampling period, each day of the week is equally well sampled, and hence all conditions during the week are represented. It should be noted, however, that this sampling frequency does not permit the extremes of the concentration distribution to be accurately quantified. The one-in-six-day schedule has the likelihood of underestimating the frequency and magnitude of high-concentration PM_{10} events (by 20–30%), because the nearest days to the event day, and/or the event day itself, may be excluded by the sampling schedule.

2.3.2.2.1 Twenty-four-hour average PM_{10} levels

Particulate matter data typically exhibit a skewed distribution dominated by a large number of low values. Particulate matter concentrations also typically exhibit variation on a number of temporal scales: diurnal, hebdomadal (day of week), seasonal and annual. The causes of these variations are multifaceted and are related both to emission variability and to variations in geophysical variables, such as mixed layer depth, wind speed and humidity levels.

Mean PM_{10} concentrations across Canada range from 11 to 42 µg/m^3, with most sites in the range of 20–30 µg/m^3 (Table 5). These levels are substantially above estimated background levels, indicating that anthropogenic activities make a significant contribution to ambient PM_{10} loadings. The highest PM_{10} concentrations recorded by the NAPS monitoring network were observed in Quebec (at a site in Montréal), Ontario (at sites in Windsor, Hamilton and Walpole Island) and Alberta (at a site in Calgary). However, even within cities, there may be sites that experience comparatively low ambient PM_{10} levels, as is the case in Montréal and Calgary. The three rural sites of Kejimkujik (Nova Scotia; 1992–1995), Sutton (Quebec; May–September 1993) and Egbert (Ontario; 1992–1995) recorded mean 24-hour PM_{10} concentrations of 11, 11 and 17 µg/m^3, respectively.

The season of maximum PM_{10} concentrations is regionally variable, reflecting variations in dominant sources of PM_{10} (especially secondary aerosols) and synoptic meteorology. The sites that exhibit the highest degree of seasonality are in Windsor (a summertime maximum) and Victoria (a wintertime maximum). Many of the sites in British Columbia seem to exhibit a late winter/early spring maximum of both mean and median PM_{10} concentrations and the upper quartile of the distribution, indicating that both average and extreme PM_{10} concentrations are typically higher during the months of January, February and March. Sites in Ontario seem to exhibit summertime maximum PM_{10} concentrations, which may reflect the greater abundance of secondary aerosols in the Windsor–Québec corridor, where precursor concentrations are known to be high.

A hebdomadal cycle of PM_{10} concentrations is evident at most urban sites. Typically, weekend concentrations of PM_{10} are lower than those observed during the work week. This difference is magnified for roadway sites, where up to a 50% increase in PM_{10} was noted midweek relative to Sunday (all sites). This suggests a substantial contribution to PM_{10} concentrations from transportation sources.

There has been an apparent decrease in yearly variations in PM_{10} concentrations during the 1984–1995 sampling period at

Table 5 Frequency distributions of 24-hour average PM_{10} concentrations (µg/m^3) from dichotomous sampler sites (1984–1995) and SSI sites (1988–1994) — national network

NAPS ID	City	n	Minimum	Percentiles					Maximum	Arithmetic mean	SD
				10	30	50	70	90			
Dichotomous sampler sites											
10101	St. John's, Nfld.	39	2	5	12	16	22	29	34	17	8
40203	Saint John, N.B.	819	1	5	8	12	18	28	70	15	10
30101	Halifax, N.S.	315	6	12	18	23	29	40	81	25	11
30118	Halifax, N.S.	272	3	8	11	13	16	22	52	15	7
30119	Dartmouth, N.S.	83	2	7	9	11	14	19	36	12	6
30501	Kejimkujik, N.S.	506	1	4	6	8	11	21	60	11	9
50104	Montréal, Que.	815	3	11	17	23	30	45	99	26	14
50109	Montréal, Que.	367	2	18	28	36	49	74	175	42	24
50307	Québec, Que.	265	2	9	15	18	27	40	71	22	13
50308	Québec, Que.	40	1	5	12	16	20	33	70	18	13
54101	Sutton, Que.	136	2	5	7	9	13	21	42	11	7
60104	Ottawa, Ont.	470	3	8	14	18	24	38	76	21	12
60204	Windsor, Ont.	360	4	13	20	27	36	55	110	31	18
60211	Windsor, Ont.	615	5	14	21	27	35	52	105	31	16
60417	Toronto, Ont.	272	3	13	19	24	32	48	96	28	15
60424	Toronto, Ont.	597	3	11	16	23	30	47	102	26	15
60403	Toronto, Ont.	96	6	11	18	25	32	44	63	26	12
60512	Hamilton, Ont.	329	4	12	19	26	37	54	177	31	19
61901	Walpole Island, Ont.	329	3	9	16	24	35	61	149	30	23
64401	Egbert, Ont.	304	1	5	9	13	21	32	77	17	12
70119	Winnipeg, Man.	550	3	12	18	24	32	46	112	27	17
90130	Edmonton, Alta.	497	4	10	16	23	30	45	86	25	14
90204	Calgary, Alta.	144	6	14	20	27	35	59	114	32	19
90227	Calgary, Alta.	472	5	10	16	21	29	43	84	24	13
100106	Vancouver, B.C.	108	8	13	18	25	30	51	84	28	15
100111	Vancouver, B.C.	442	3	11	17	22	29	43	82	25	13
100118	Vancouver, B.C.	296	4	9	14	19	23	36	69	21	11
100303	Victoria, B.C.	508	2	8	11	15	20	30	71	17	10

Table 5 (continued)

NAPS ID	City	n	Minimum	Percentiles					Maximum	Arithmetic mean	SD
				10	30	50	70	90			
SSI sites											
40201	Saint John, N.B.	268	1	9	15	19	26	38	64	21	11
40203	Saint John, N.B.	246	1	6	11	14	19	28	59	16	10
40204	Saint John, N.B.	106	1	3	5	8	11	16	50	9	7
40205	Saint John, N.B.	85	3	6	9	12	16	25	77	14	10
30311	Sydney, N.S.	269	5	11	14	18	24	39	111	22	15
60104	Ottawa, Ont.	405	3	8	12	15	22	32	77	19	11
80110	Regina, Sask.	409	4	8	13	19	28	48	367	25	24
80209	Saskatoon, Sask.	217	2	8	13	17	23	35	76	20	13
80211	Saskatoon, Sask.	121	4	8	12	15	20	28	75	18	11
90130	Edmonton, Alta.	404	5	12	17	23	33	49	132	28	16
100111	Vancouver, B.C.	249	5	9	14	17	22	34	53	19	9
100118	Vancouver, B.C.	183	4	9	12	15	21	30	58	18	10
100109	Vancouver, B.C.	246	5	12	17	22	28	40	70	24	11

most sites with a complete data record. The largest percent decreases occurred at the Montréal–Duncan/Decarie, Edmonton and Vancouver sites. A trend analysis of annual PM_{10} data for 1984 through 1993 showed a statistically significant ($p < 0.001$) decreasing trend in PM_{10} concentrations on a national basis, averaging 2% per year (Dann, 1994).

2.3.2.2.2 Twenty-four-hour average $PM_{2.5}$ levels

Mean concentrations at the NAPS urban sites ranged from 6.9 to 20.2 µg/m³ (Table 6). $PM_{2.5}$ concentrations are more spatially homogeneous than PM_{10} concentrations, but there are still significant site-to-site differences, even within the same urban area. The highest $PM_{2.5}$ concentrations (in terms of means and 90th percentiles) were measured at sites in Montréal, Toronto, Hamilton, Windsor, Walpole Island and Vancouver. These were almost the same sites that recorded the highest PM_{10} concentrations. The three rural sites of Kejimkujik (1992–1995), Sutton (May–September 1993) and Egbert (1992–1995) recorded mean $PM_{2.5}$ 24-hour concentrations of 7.0, 7.7 and 10.5 µg/m³, respectively.

The seasonal variability of $PM_{2.5}$ is more pronounced than that of PM_{10}; however, the seasonal patterns vary for different regions. The Montréal, Ottawa, Edmonton, Calgary and Vancouver/Victoria sites record higher $PM_{2.5}$ concentrations in the winter months, particularly during January and February. Other Ontario sites record the highest daily concentrations in the summer months, with a peak median in August, reflecting long-range transport from the U.S. Midwest. For Maritime sites, there is variable seasonal variation in $PM_{2.5}$ concentrations, with Saint John and Kejimkujik having a summer maximum (as a consequence of long-range transport from the east coast of the United States) and Halifax a winter maximum.

Minimum $PM_{2.5}$ concentrations occur on Sunday and maximum concentrations during the middle of the week at most urban sites. Again, this difference is magnified for roadway sites, where up to a 60% increase in $PM_{2.5}$ midweek relative to Sunday was noted for all sites. This indicates that there are large day-of-week differences in anthropogenic emissions and significant contributions from motor vehicles.

A trend analysis of $PM_{2.5}$ data for the period 1984–1993 showed a statistically significant ($p < 0.001$) decreasing trend in $PM_{2.5}$ on a national basis, averaging 3.3% per year (Dann, 1994). For the Ontario sites, there was no significant change in $PM_{2.5}$ between 1987 and 1993.

2.3.2.2.3 One-hour average PM_{10} levels

In 1994, 10 sites (all but two in the Lower Fraser Valley) reported hourly PM_{10} concentrations to the NAPS network using TEOM instruments. A maximum one-hour PM_{10} concentration of 255 µg/m³ was measured at the Abbotsford site (in the Lower Fraser Valley), and a maximum one-hour concentration of 204 µg/m³ was recorded at the Edmonton site. Analysis of diurnal variations in PM_{10} has shown that a substantial increase in PM_{10} levels occurs during the morning rush hour, with a secondary peak during the late evening. Minimum values occur during the mid-afternoon and in the early hours of the morning (12:00–6:00 a.m.).

2.3.2.3 Relationships among TSP, PM_{10} and $PM_{2.5}$ and inorganic constituents of particulate matter

Fourteen urban sites in the NAPS dichotomous sampler network operating from 1986 to 1994 made simultaneous measurements of total suspended particulates (TSP), PM_{10}, $PM_{2.5}$ and sulphate. This data set is valuable in that it allows comparison of the composition of these different particulate matter fractions at the 14 sites (Table 7). On average across the 14 sites, PM_{10} accounted for approximately 50% of TSP, while $PM_{2.5}$ accounted for approximately 25% of TSP. Both fine and coarse particles accounted for approximately equal portions (about 50%) of the PM_{10}. Most of the sulphate was present in fine

Table 6 Frequency distributions of 24-hour average $PM_{2.5}$ concentrations (µg/m³) from dichotomous sampler sites (1984–1995) — national network

NAPS ID	City	n	Minimum	Percentiles					Maximum	Arithmetic mean	SD
				10	30	50	70	90			
10101	St. John's, Nfld.	39	1.0	3.0	6.0	9.0	11.0	18.0	26.0	9.8	5.7
40203	Saint John, N.B.	821	0.6	2.5	4.8	7.2	10.0	16.2	53.2	8.5	6.2
30101	Halifax, N.S.	335	2.8	6.0	10.0	12.0	15.6	25.0	45.5	14.0	7.1
30118	Halifax, N.S.	273	0.8	4.6	6.0	7.4	9.6	15.1	43.4	9.0	5.5
30119	Dartmouth, N.S.	83	0.7	3.1	4.5	6.3	7.7	10.9	23.5	6.9	4.0
30501	Kejimkujik, N.S.	506	0.4	2.0	3.3	4.8	7.3	13.6	46.7	7.0	7.1
50104	Montréal, Que.	849	1.4	5.3	8.9	12.5	17.8	29.0	69.6	15.2	10.3
50109	Montréal, Que.	373	0.6	7.4	12.4	17.0	23.0	36.0	89.0	20.2	13.0
50307	Québec, Que.	267	0.4	4.0	6.9	9.0	13.0	23.0	49.0	11.5	8.1
50308	Québec, Que.	40	0.1	3.0	4.5	7.3	9.1	19.1	32.9	9.0	7.2
54101	Sutton, Que.	136	1.0	2.4	4.1	6.1	9.1	15.6	33.2	7.7	5.5
60104	Ottawa, Ont.	569	1.2	3.9	6.0	9.0	14.0	24.0	53.8	12.0	8.8
60204	Windsor, Ont.	367	1.8	6.5	11.0	15.5	21.6	32.2	70.6	17.9	11.1
60211	Windsor, Ont.	615	1.8	6.7	10.3	15.0	19.7	31.2	85.6	17.4	11.3
60417	Toronto, Ont.	282	1.4	6.4	10.0	14.0	19.0	29.0	71.0	16.4	10.5
60424	Toronto, Ont.	599	1.3	5.2	8.1	12.6	18.4	30.4	66.4	15.5	10.6
60403	Toronto, Ont.	96	3.1	5.8	9.5	14.8	20.1	28.0	43.0	15.9	9.0
60512	Hamilton, Ont.	329	1.9	5.6	10.4	15.2	22.2	33.4	74.1	18.1	12.1
61901	Walpole Island, Ont.	330	2.0	4.4	8.7	13.3	21.0	36.6	126.6	17.9	14.8
64401	Egbert, Ont.	304	0.7	2.6	4.8	7.7	12.6	21.9	47.7	10.5	8.5
70119	Winnipeg, Man.	567	0.9	4.0	6.3	8.4	11.1	17.0	81.0	10.1	7.4
90130	Edmonton, Alta.	588	0.4	4.2	6.4	8.7	11.6	21.0	56.3	10.8	7.6
90204	Calgary, Alta.	145	2.0	6.0	9.0	11.0	15.0	28.0	52.0	14.3	9.8
90227	Calgary, Alta.	472	1.8	4.0	6.0	8.1	11.0	17.0	42.0	9.8	6.4
100106	Vancouver, B.C.	109	3.0	7.0	9.0	13.0	18.0	39.0	72.0	17.3	12.6
100111	Vancouver, B.C.	597	2.1	6.0	9.0	12.0	16.0	24.6	62.0	14.1	8.8
100118	Vancouver, B.C.	343	1.3	5.2	8.0	11.0	14.0	24.0	49.0	12.9	8.0
100303	Victoria, B.C.	508	0.5	3.6	5.7	8.1	12.0	22.0	59.8	10.7	8.0

TABLE 7 Combined summary statistics for 14 urban NAPS sites in operation between 1986 and 1994[1] (from Brook *et al.*, 1997)

Ratios	Percentiles					Statistics				
	10	**50**	**70**	**90**	**95**	**Max.**	**Mean[2]**	**SD[2]**	**Geo. mean[3]**	**Geo. SD[3]**
PM_{10}/TSP	0.3	0.5	0.6	0.9	1.0	2.404	0.56	0.24	0.51	1.52
$PM_{2.5}$/TSP	0.1	0.3	0.4	0.5	0.6	1.414	0.3	0.19	0.25	1.91
$PM_{2.5}/PM_{10}$	0.3	0.5	0.6	0.7	0.8	1.0	0.51	0.17	0.48	1.45
$PM_{10-2.5}/PM_{10}$	0.26	0.50	0.58	0.71	0.76	1.00	0.49	0.17	0.46	1.51
FSO_4[5]$/PM_{2.5}$	0.0	0.2	0.3	0.4	0.4	0.95	0.2	0.11	0.17	1.89

[1] Sample size, n = 2831, is based upon the number of 24-hour periods with valid measurements for TSP, PM_{10}, $PM_{2.5}$ and sulphate.

[2] Arithmetic average and arithmetic standard deviation (SD).

[3] Geometric mean and geometric standard deviation (SD).

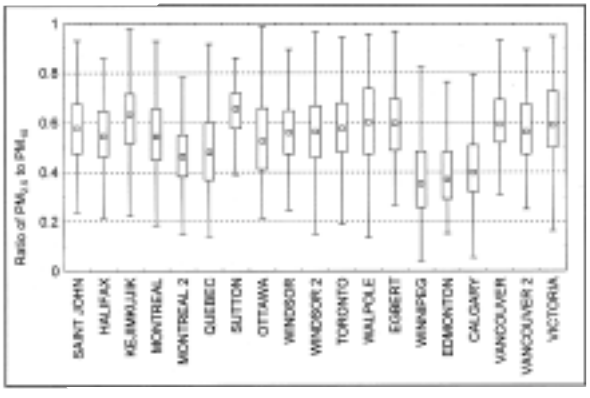
[4] Values greater than 1.0 are assumed to arise from instrument error.

[5] FSO_4 corresponds to fine sulphate.

particles, where it comprised on average approximately 17% of the fine particulate matter. However, there was considerable variation within and among sites for these ratios. The relationships between TSP, PM_{10} and $PM_{2.5}$ are dependent on concentration, with ratios of PM_{10} and $PM_{2.5}$ to TSP decreasing with increasing TSP concentration (i.e., more of the TSP mass is composed of very coarse particulate matter) (Brook *et al.*, 1997).

Other data from the NAPS network corroborate both the variability in $PM_{2.5}/PM_{10}$ ratios and the overall finding that on average across Canada, approximately 50% of PM_{10} is made up of fine particles (Figure 2). Based on data collected from 1984 to 1993 from 19 sites (16 locales), the median $PM_{2.5}/PM_{10}$ ratios for most sites fall within a fairly narrow range of 0.4–0.6; that is, at least half of the time, 40–60% of PM_{10} at a site is composed of fine particles. Although there is clearly temporal variability in $PM_{2.5}/PM_{10}$ ratios at a site, about 50% of the time the ratios do not vary by much more than ±10%, as indicated by the interquartile ranges (25th–75th percentiles) (Brook *et al.*, 1997).

FIGURE 2 Distributions of the ratio of $PM_{2.5}$ to PM_{10} mass at the NAPS dichotomous sampler sites. The box plots indicate the median, 5th and 95th and 25th and 75th percentiles (from Brook *et al.*, 1997).



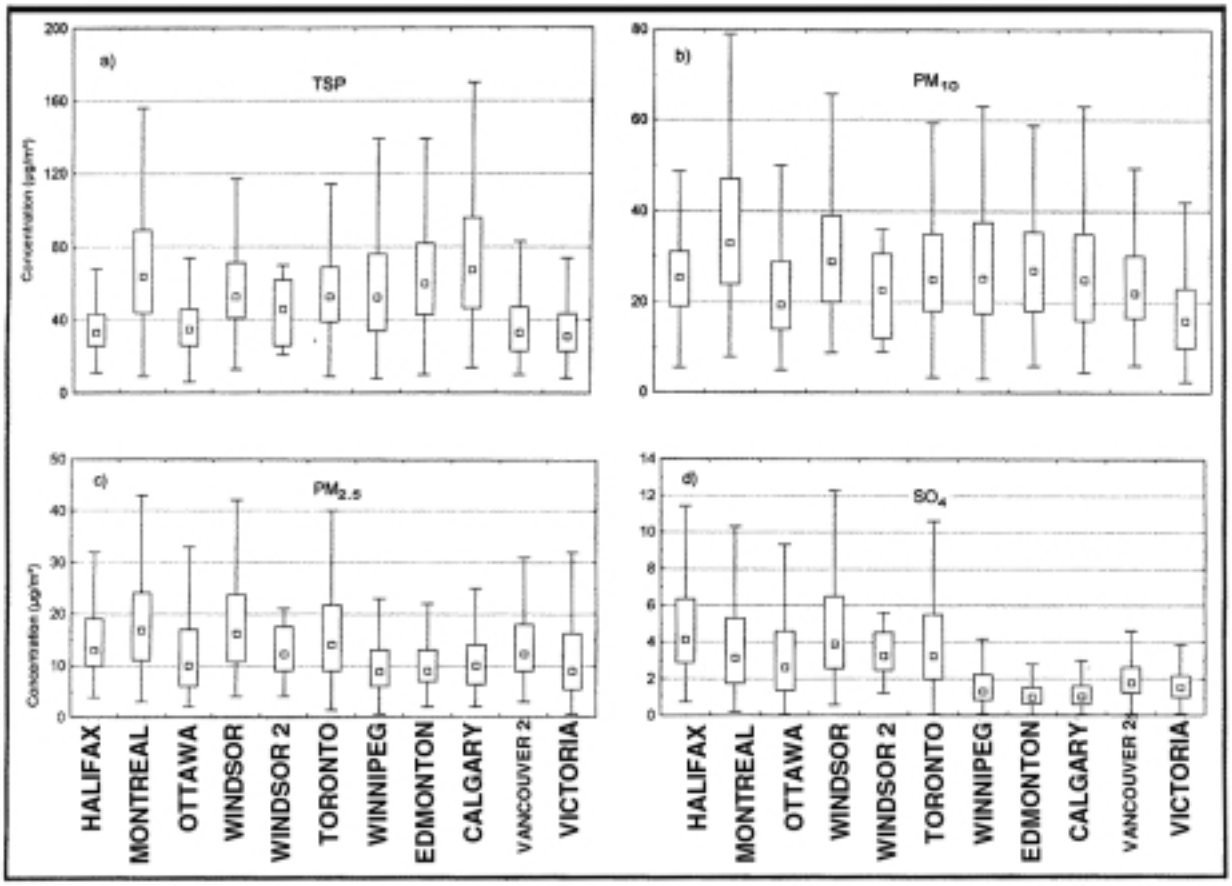
There are relatively strong correlations (r^2) between PM_{10} and $PM_{2.5}$ at each of the 19 sites, which is consistent with the belief that temporal variations in fine particles have a significant influence on the observed variability in PM_{10}. At a majority of the sites, the daily variability in fine particle mass had a stronger influence on the variations in PM_{10} than did the daily variability in coarse particle mass. This was most evident at the rural locations and at sites not heavily impacted by urbanization (i.e., traffic and construction). The exceptions to this pattern were the Prairie sites, where coarse mass dominated PM_{10}, and a site in Montréal that is heavily impacted by traffic (Brook *et al.*, 1997).

There are two key trends based on comparisons of TSP, PM_{10}, $PM_{2.5}$ and sulphate mass distributions at sites across Canada (Figure 3). First of all, sites in the three Prairie cities of Winnipeg, Calgary and Edmonton have large and variable TSP concentrations, but their $PM_{2.5}$ and sulphate concentrations are small relative to the other sites and exhibit less variability. Much of the airborne particulate matter observed in these areas is considered to be mechanically derived and likely consists of local crustal material. Secondly, there is an obvious decrease in sulphate levels from the sites located east of the upper Great Lakes to those located west of the lakes. This pattern has been repeatedly observed and is a direct reflection of the magnitude and spatial density of sulphur dioxide emissions within and upwind of these two areas (Brook *et al.*, 1997).

2.3.3 Indoor and personal air

The vast majority of particulate monitoring data are available for ambient air. However, North Americans spend, on average, less than 10% of their time outdoors (U.S. EPA, 1995; Leech *et al.*, 1996). In addition, individuals can spend time in a number of microenvironments that differ in their particulate matter concentrations during the course of a day. Consequently, indoor air and personal air levels of airborne particles have been examined in a number of studies.

FIGURE 3 Comparison of the distributions of TSP, PM_{10}, $PM_{2.5}$ and sulphate at 11 urban sites (1984–1993). The box plots indicate the median, 5th and 95th and 25th and 75th percentiles (from Brook *et al.*, 1997).



2.3.3.1 Indoor air

Levels of particulate matter in indoor air are a function of indoor sources, outdoor particle levels, the fraction of ambient air penetrating indoors, filtration, air exchange rates, decay rates and resuspension rates (U.S. EPA, 1982, 1996; Clayton *et al.*, 1993; Wallace *et al.*, 1993; Thatcher and Layton, 1995). Fine particles readily penetrate buildings; penetration factors of 0.6–1 have been reported for $PM_{2.5}$, with the coarser fraction ($PM_{10-2.5}$) probably penetrating less effectively (Dockery and Spengler, 1981; Yocom, 1982; Lioy *et al.*, 1990; Colome *et al.*, 1992; Koutrakis *et al.*, 1992; Özkaynak *et al.*, 1993; Thatcher and Layton, 1995; U.S. EPA, 1996). Once indoors, ambient particles settle out quickly by gravity or electrostatic forces. Average decay rates, due to diffusion or sedimentation, were calculated as part of the Particle Total Exposure Assessment Methodology (pTEAM) study (Özkaynak *et al.*, 1993) for sulphate (0.16 per hour), $PM_{2.5}$ (0.39 ± 0.16 per hour), PM_{10} (0.65 ± 0.28 per hour) and the coarse fraction, $PM_{10-2.5}$ (1.01 ± 0.43 per hour). Particles of size range 0.1–1 µm have negligible setting velocities, while particles >10 µm normally settle out of the air. Once deposited, particles greater than 5 µm are easily resuspended during indoor activities of occupants, while smaller particles (<1 µm) are "not" resuspended, and particles 1–5 µm in size can be resuspended with vigorous activity (Thatcher and Layton, 1995).

In three large-scale studies conducted in the United States — the Harvard Air Pollution Health Effects Study (also called the Six Cities Study), the New York State Energy Research and Development Authority (ERDA) Study, and the pTEAM study — the range of mean values for $PM_{2.5}$ or $PM_{3.5}$ in various cities across the country was 20–47 µg/m³ in indoor air, compared with 13–50 µg/m³ in outdoor air (Dockery and Spengler, 1981a; Sexton *et al.*, 1984; Spengler *et al.*, 1985; Sheldon *et al.*, 1989; Pellizzari *et al.*, 1992). Concentrations in indoor air generally exceeded those outdoors at sites with low outdoor levels and were slightly less at sites with high outdoor concentrations (Wallace, 1996). Levels in indoor air were, however, within a factor of two of those outdoors in all of the available studies.

Source contributions to levels of respirable particles in indoor air were estimated from the pTEAM results using elemental analyses and a non-linear method of solving a mass balance model. Averaged over 244 homes containing no known indoor sources, outdoor sources accounted for 75% of indoor $PM_{2.5}$ and 65% of PM_{10}. In homes where there was tobacco smoking or cooking (the two main identified indoor sources of particles), each accounted for approximately 20–30% of the indoor particle concentrations on average; however, outdoor air still remained the largest source of indoor PM_{10} or $PM_{2.5}$, accounting for approximately 60% of indoor air levels (Özkaynak *et al.*, 1993, 1995a). Source apportionment analyses from the Harvard Six Cities and ERDA studies confirmed the importance of outdoor air and (in homes with smokers) tobacco smoking as sources of indoor particles (Santanam *et al.*, 1990; Koutrakis *et al.*, 1992). In Canada, where building construction emphasizes energy efficiency and, therefore, low air exchange rates, the fractions of fine and coarse particles of ambient origin that are found indoors under equilibrium should tend towards 50% or less, particularly in winter.

2.3.3.2 Personal air

Personal exposures to airborne particles have been measured in a small number of studies. In non-smoking households, mean levels of $PM_{2.5}$, $PM_{3.5}$ or PM_{10} in personal air were roughly 1.5–2.5 times higher than particle concentrations in indoor air (Spengler *et al.*, 1985; Lioy *et al.*, 1990; Özkaynak *et al.*, 1993; Neas *et al.*, 1994; Thatcher and Layton, 1994; Wallace, 1996). The source of the difference between personal exposures and indoor concentrations, the "personal cloud," has not yet been determined. However, most of the excess personal exposure is likely due to generation or re-entrainment of particles during personal activities. In the pTEAM study, the increment in personal exposure levels over the corresponding concentrations of particles in indoor air was restricted to daytime monitoring, whereas levels were similar at night, when subjects were less active. In addition, personal exposure levels were significantly greater in subjects who were engaged in activities such as cooking, cleaning the house and smoking during the monitoring period (Pellizzari *et al.*, 1992). The "personal cloud" may be made up mostly of coarse particles ($PM_{10-2.5}$) (Bahadori *et al.*, 1995), which is consistent with the evidence that coarse particles are more easily resuspended than fine particles (Thatcher and Layton, 1995).

Correlations between ambient particulate matter data obtained from fixed ambient monitors (FAMs) and personal exposure data obtained from personal exposure monitors have been examined in a number of studies. Most of these reveal poor correlations for data collected at one point in time, particularly during the day, and indicate, not surprisingly, that personal exposures are usually greater than indoor or outdoor ambient concentrations. When personal exposure is longitudinally regressed against levels at the nearest outdoor site, the correlations improve (Lioy *et al.*, 1990; Wallace, 1996). The improvement in correlation coefficients suggests that, for individuals who are not exposed to important microenvironmental sources of particles (e.g., smoking) and whose day-to-day activities

are fairly repetitive, ambient levels of particles may more directly reflect their exposure to particles. The correlation also increases when the mean of the personal exposures from various studies is related to the FAM (Mage and Buckley, 1995). Thus, ambient fine particles measured at the FAM can serve as an indicator of community (population) exposure.

Personal and population exposure models have been developed that combine ambient measurements of pollutants with information on age-specific time–activity relationships and estimates of microenvironmental pollutant concentrations. A probabilistic PM_{10} exposure model was applied to Canadian ambient PM_{10} measurements, demographics and smoking prevalences by region to produce estimated distributions of 24-hour average personal, indoor, outdoor and in-transit PM_{10} concentrations (Özkaynak *et al.*, 1995b). The model reproduced the empirical findings that personal exposures to PM_{10} are substantially greater than ambient concentrations at FAMs (the predicted mean personal air concentration was 39 µg/m³, compared with an average of 28 µg/m³ measured at the ambient sites used as input to the model), and that personal exposures were quite variable (e.g., the 95th percentile, at 93 µg/m³, was nearly 2.5 times greater than the mean).

2.4 Effects characterization

2.4.1 Humans

2.4.1.1 Controlled human exposure studies

Almost all of the human clinical studies have been based on observations of pulmonary function changes and reports of subjective symptoms. All of the exposures were conducted for very short periods of time (i.e., most of them between 40 and 120 minutes). Controlled human exposures to acidic and inert particles have not caused significant alterations in respiratory function and symptoms in healthy individuals, even at levels higher than those in the environment (450–1000 µg/m³) (Utell *et al.*, 1983a, 1984). The clinical studies have identified asthmatics as a susceptible population for acidic aerosols (Utell *et al.*, 1983b), but not persons with chronic obstructive pulmonary disease (COPD) (Morrow *et al.*, 1994) or the elderly (Koenig *et al.*, 1992, 1993; Morrow *et al.*, 1994). In controlled studies, asthmatics, especially children and adolescents, have experienced adverse effects on airway function at concentrations encountered on occasion in ambient air (exposure to sulphuric acid at ~35 µg/m³ for 40 minutes) (Koenig *et al.*, 1989, 1992; Hanley *et al.*, 1992).

Few data are available on particle-induced airway inflammatory responses in humans. In one study (Frampton *et al.*, 1992), brief exposures to sulphuric acid aerosols at 1000 µg/m³ had only minor effects on the airway defence system in healthy subjects — i.e., increased antibody-mediated cytotoxicity of alveolar macrophages, a trend (not statistically significant) to a decreased percentage of T lymphocytes in bronchoalveolar lavage fluid, and no effects on the number of polymorphonuclear leukocytes in the bronchoalveolar lavage fluid or on the release of superoxide anion or inactivation of influenza virus *in vitro* when compared with sodium chloride.

No data on changes to the cardiovascular system in controlled human studies were identified.

There has been very little work comparing the effects of different particle sizes on human health. In a panel study, Peters *et al.* (1997) reported that symptoms (cough) and decrements of peak expiratory flow in asthmatic subjects (n = 27) were more strongly associated with the five-day mean of the number or mass of ultrafine particles (MMAD 0.01–0.1 µm) than with other discrete fractions of the fine particles (MMAD 0.1–0.5, 0.5–2.5 or 0.1–2.5 µm).

The particles to which volunteers were exposed in the human clinical studies do not adequately reflect the complexity of ambient particles. Based on the extremely limited clinical database available, acidic aerosols produce the most

significant bronchoconstriction, while the toxicity of sulphate is related to acidity *per se* (Utell *et al.*, 1983b, 1989). Nitrates did not exert effects on lung function at concentrations below 1000 µg/m^3 in clinical studies (U.S. EPA, 1989; Aris *et al.*, 1993), while inert particles appear to have no effect on lung function in either healthy or asthmatic volunteers (Anderson *et al.*, 1992). In one study (Sandstrom and Rudell, 1991), very fine particle diesel exhaust (3×10^6 particles/cm^3) affected neutrophil production and macrophage clearance of microorganisms from the lung, but exposure to formaldehyde and other combustion gases (nitrogen dioxide, nitric oxide and carbon monoxide [CO]) was also elevated, which might confound any effects from particulate matter.

Few clinical studies have been conducted to investigate the effects of air pollutant mixtures on humans. Pre-exposure of normal and asthmatic adults to sulphuric acid aerosol potentiated reductions in lung function and increases in airway resistance induced by exposure to ozone (O_3) in the asthmatic group only (Frampton *et al.*, 1995). Respiratory symptoms were increased in healthy and asthmatic children by a mixture of sulphuric acid, ozone and sulphur dioxide (Linn *et al.*, 1997). Concurrent exposure to ozone and sulphuric acid at concentrations typically observed in air pollution episodes produced no changes in lung function or symptoms greater than those associated with ozone alone in normal or asthmatic adults and children (Linn *et al.*, 1994, 1995).

It should be noted that decrements in pulmonary function measured in most clinical studies may not be a sensitive indicator for particle-induced lung injury. Relatively high levels of acidic aerosols produce only small decrements in lung function, even in susceptible subpopulations. Moreover, if reductions in pulmonary function serve to protect the lungs from receiving further insults in the deep airways, failure of certain subjects (such as COPD patients) to respond to particles in this manner might render them more vulnerable to pulmonary injury.

2.4.1.2 Epidemiological studies

That high levels of ambient particulate matter from combustion sources could have severe adverse effects on health was noted in the air pollution episodes of the 1940s to 1960s. Indeed, one such episode in London, England, in 1952 was responsible for several thousand premature deaths within a week. However, until the publication of new studies beginning in the early 1990s, there were no data to suggest that relatively low concentrations of particulate matter, as currently experienced in urban areas of North America and Western Europe, had effects on human health.

Many of these recent studies on air pollution, including all the mortality and hospitalization studies, have been based on the time-series analysis of associations between daily variations in ambient concentrations and daily variations in adverse health outcomes, with data obtained from large administrative databases. In this longitudinal type of study, confounding due to population differences is much less likely than in a cross-sectional analysis, since the population remains the same over a short time (typically one or a few days) and acts as its own "control." However, the potential for confounding by seasonal variations and weather remains a problem in the time-series analysis, and much recent effort has been directed to addressing this, with considerable success. A major advantage of the time-series study is that it usually provides many more units of observation (typically 1000 days and 10 000 adverse effects) than the cross-sectional study (2–150 communities), and thus its power to detect effects of low magnitude is usually greater than that of even the most sensitive cross-sectional study. While time-series studies do not provide information on exposures at the individual level (i.e., they are considered to be "ecological" with respect to their exposure component), they can provide important evidence for the assessment of relationships between levels of air pollution and health at a community level.

Several cohort studies, in which the effects of longer-term exposures to particulate matter were examined and where potentially confounding risk factors were taken into account in the analysis, have also been completed. However, exposure remained on a community basis in the available studies, which limits the inferences to be drawn from these studies to groups of individuals with a given set of measured risk factors.

Studies on adverse effects other than premature mortality and hospitalizations, such as effects on lung function, have been completed using a variety of methods.

2.4.1.2.1 Acute effects: mortality

Nineteen time-series studies in which the relationship of daily or short-term variations in particulate matter with mortality was investigated are summarized in Table 8. The studies were conducted in cities across North and South America and Europe, and almost all of them demonstrated associations between particulate air pollution and acute mortality. The associations of mortality with particulate matter could not be explained by the influence of weather, season, yearly trends, day-to-day variations or variations due to holidays, epidemics or other non-pollutant factors, since all the studies investigated these potential biases and adjusted for them in various ways in the analyses.

For 21 out of 23 cities, the observed increases in relative risk (RR) in relation to elevated PM_{10} concentrations were statistically significant or close to significance, as measured by the 95% confidence interval (CI). The exceptions were Salt Lake County, Utah (Styer *et al.*, 1995) and Kingston-Harriman, Tennessee (Dockery *et al.*, 1992). Negative results in the latter were explicable on the basis of a combination of poor exposure assessment and inaccurate methodology (Dockery *et al.*, 1992). The negative results for Salt Lake County (Styer *et al.*, 1995) are not easily explained except as a lack of statistical power to detect an effect due to small population and possibly also due to overcompensation for weather and seasonal factors in the method used to analyse the results.

For PM_{10} at ambient concentrations averaging 18–115 µg/m^3 (from 23 reported cities), a 100 µg/m^3 increase was associated with a mean (unweighted) RR of mortality from all causes except accidents of 1.082 ± 0.056 [mean ± standard deviation (SD)] and a median of 1.08 (Table 8). The results indicate that each 10 µg/m^3 of daily increase in PM_{10} is associated with an unweighted and weighted mean increase in daily mortality of 0.8% and 0.5%, respectively. The weighted RRs were calculated based on the standard deviation of each study; RRs with smaller standard deviations were weighted more heavily than those with large standard deviations (see Schwartz, 1994e, for detailed method of weighting).

For Black Smoke (a finer particle, approximately PM_5) with average concentrations of 12–84 µg/m^3 in six locations, the observed increase in RR with increasing Black Smoke concentration was statistically significant in all but one of the six cities (Paris, France), where it was marginal (Dab *et al.*, 1996). Across all studies, the unweighted mean RR for mortality was 1.096 ± 0.050 (mean ± SD), with a median at 1.08, for a 100 µg/m^3 increase. This indicates that a 10 µg/m^3 increase in Black Smoke is associated with a daily increase in mortality of approximately 1% on average, with the median being 0.8%.

For the fine fraction of particles ($PM_{2.5}$), at concentrations averaging 11.2–21 µg/m^3, RRs for mortality and $PM_{2.5}$ were increased in all nine cities, although the increase was marginally significant in three cities (St. Louis, Missouri, in Dockery *et al.*, 1992; Steubenville, Ohio, and Portage, Wisconsin, in Schwartz *et al.*, 1996) and was not significant in two cities (Kingston-Harriman, Tennessee, in Dockery *et al.*, 1992; Topeka, Kansas, in Schwartz *et al.*, 1996). The

Table 8 Summary of relative risks for particulate matter from time-series studies: Total mortality

Location and references	PM concentration (µg/m³) (mean; range)	RR (100 µg/m³ increase) (95% CI)	RR + other pollutant in model (95% CI with other pollutants in model)
PM_{10}			
St. Louis, Missouri, USA (Dockery *et al.*, 1992)	28; max. 97	1.16 (1.01–1.30)	1.12 (0.96–1.15) (+ O_3) (U.S. EPA, 1996)
Kingston-Harriman, Tennessee, USA (Dockery *et al.*, 1992)	30; max. 67	1.17 (0.88–1.50)	1.17 (0.88–1.52) (+ O_3) (U.S. EPA, 1996)
Utah Valley, Utah, USA (Pope *et al.*, 1992)	47; 1–365	1.16 (1.10–1.22)	1.14 (1.04–1.24) (+ O_3) (Pope and Kalkstein, 1996)
Philadelphia, Pennsylvania, USA (Dockery *et al.*, 1996b)	not given	1.12 (0.99–?; upper CL not given) (p = 0.06)	—
Birmingham, Alabama, USA (Schwartz, 1993)	48; 21–80, max. 163	1.11 (1.02–1.20)	—
Toronto, Ontario, Canada (Özkaynak *et al.*, 1995c)	40; range not given in text	1.07 (1.05–1.09)	1.05 (1.03–1.07) (+ "other pollutants") (Thurston, 1996)
Los Angeles, California, USA (Kinney *et al.*, 1995)	58; 15–177	1.05 (1.00–1.11)	1.05 (1.00–1.11) (+ O_3) 1.04 (0.98–1.09) (+ CO)
Chicago (Cook County), Illinois, USA (Styer *et al.*, 1995)	38 (median); 28–51 (IQR)	1.055 (1.01–1.10) (≥65 years)	—
Chicago (Cook County), Illinois, USA (Ito and Thurston, 1996)	41 ± 19	1.05 (1.03–1.08)	1.04 (1.01–1.07) (+ O_3)
Utah Valley, Utah, USA (Lyon *et al.*, 1995)	47; 1–365	1.04 (0.98–1.10)	—
Salt Lake County, Utah, USA (Styer *et al.*, 1995)	48; 9–194	1.008 (~0.8–1.08) (≥65 years)	—
Six Cities Study (USA) (combined)	25; 8–68	1.08 (1.05–1.11)	—
- St. Louis, Missouri	30.6 ± 16.2 (mean ± SD)	1.06 (1.01–1.10)	
- Kingston-Harriman, Tennessee	32.0 ± 14.5	1.09 (1.01–1.18)	
- Boston, Massachusetts	24.5 ± 12.8	1.12 (1.07–1.17)	
- Steubenville, Ohio	45.6 ± 32.3	1.09 (1.01–1.16)	
- Topeka, Kansas	26.7 ± 16.1	0.95 (0.80–1.09)	
- Portage, Wisconsin	17.8 ± 11.7	1.08 (0.96–1.17)	
(Schwartz *et al.*, 1996)			
Sao Paulo, Brazil (Saldiva *et al.*, 1995)	82 ± 39	1.13 (1.07–1.19) (≥65 years)	1.13 (1.03–1.23) (+ O_3, SO_2, NO_2, CO)
Santiago, Chile (Ostro *et al.*, 1996)	115; 32–367	1.07 (0.99–1.11) (OLS, summer), 1.08 (1.06–1.12) (Poisson, all year)	1.06 (1.00–1.09) (+ O_3, OLS, summer) 1.04 (1.01–1.08) (+ SO_2, Poisson, all year) 1.07 (1.03–1.11) (+ NO_2, Poisson, all year)
Paris, France (Dab *et al.*, 1996)	51; 19–137 (PM_{13})	1.17 (1.04–1.31)	—
Lyon, France (Zmirou *et al.*, 1996)	38; 5–98	1.02 (0.94–l.10) total deaths – accidents, 1.04 (1.02–1.09) respiratory deaths	RR for PM, not given with SO_2
Athens, Greece (Touloumi *et al.*, 1996)	78; 8–300	[1.063 (1.033–1.09) — estimate from BS]	[1.03 (1.01–1.06) (SO_2 < 100) — estimate from BS] [1.02 (0.97–1.07) (SO_2 > 100) — estimate from BS]

TABLE 8 (continued)

Location and references	PM concentration (µg/m³) (mean; range)	RR (100 µg/m³ increase) (95% CI)	RR + other pollutant in model (95% CI with other pollutants in model)
Amsterdam, Netherlands (Verhoeff *et al.*, 1996)	38; max. 163	1.06 (0.99–1.14)	1.03 (0.94–1.13) (+ O_3) 1.02 (0.93–1.13) (+ SO_2) 1.10 (1.01–1.20) (+ CO)
Unweighted RR: PM alone (n = 23) mean 1.082 ± 0.056 (95% CI 0.80–1.50), median 1.08; bivariate analysis, PM + other gases (n = 16): mean 1.069 ± 0.047; median 1.05			
Black Smoke			
Amsterdam, Netherlands (Verhoeff *et al.*, 1996)	12; max. 81	1.19 (1.02–1.38)	1.18 (1.01–1.37) (+ O_3) 1.27 (1.07–1.49) (+ SO_2) 1.20 (1.01–1.44) (+ CO)
London, U.K. (Anderson *et al.*, 1996)	15; 8–23; 10th–90th %	1.11 (1.05–1.17)	1.19 (1.11–1.27) (+ O_3)
Barcelona, Spain (Sunyer *et al.*, 1996)	50, 35; winter, summer	1.07 (1.03–1.11)	—
Valencia, Spain (Ballester *et al.*, 1996)	68 ± 29	1.09 (1.03–1.15)	—
Paris, France (Dab *et al.*, 1996)	32; 11–123; 5th–95th %	1.07 (0.98–1.18)	—
Athens, Greece (Touloumi *et al.*, 1996)	84; 9–333	1.05 (1.03–1.08)	1.03 (1.01–1.06) (SO_2 < 100) 1.02 (0.97–1.08) (SO_2 > 100)
Unweighted RR: PM alone (n = 6) mean 1.096 ± 0.050 (95% CI 0.98–1.38), median 1.08 (95% CI 1.03–1.16)			
$PM_{2.5}$			
St. Louis, Missouri, USA (Dockery *et al.*, 1992)	17.7; max. 75	1.19 (p = 0.075) (95% CI not given)	—
Kingston-Harriman, Tennessee, USA (Dockery *et al.*, 1992)	21; max. 58	1.17 (p = 0.28) (95% CI not given)	—
Philadelphia, Pennsylvania, USA (Dockery *et al.*, 1996b)	not given	1.20 (1.04–1.35)	RR for PM + O_3 not stated (both independent)
Six Cities Study (USA) (combined)	14.7; 4.3–431 (IQR 14.0)	1.15 (1.11–1.19)	"same" (+ CP or $PM_{10-2.5}$)
- St. Louis, Missouri	18.7 ± 10.5	1.11 (1.04–1.17)	
- Kingston-Harriman, Tennessee	20.8 ± 9.6	1.14 (1.02–1.26)	
- Boston, Massachusetts	15.7 ± 9.2	1.22 (1.15–1.29)	
- Steubenville, Ohio	29.6 ± 21.9	1.10 (0.99–1.21)	CP: RR = 1.24 (1.05–1.43); correlation = 0.69
- Topeka, Kansas	12.2 ± 7.4	1.08 (0.80–1.36)	
- Portage, Wisconsin (Schwartz *et al.*, 1996)	11.2 ± 7.8	1.12 (0.97–1.28)	
Unweighted RR (6 individual cities + 3 cities) mean 1.148 ± 0.049; median 1.14 (95% CI 0.80–1.36)			

TABLE 8 (continued)

Location and references	PM concentration (µg/m³) (mean; range)	RR (100 µg/m³ increase) (95% CI)	RR + other pollutant in model (95% CI with other pollutants in model)
Sulphate			
St. Louis, Missouri, USA (Dockery *et al.*, 1992)	8.0; max. 38	weak positive, not significant	—
Kingston-Harriman, Tennessee, USA (Dockery *et al.*, 1992)	8.7; max. 27	not significant	—
Six Cities Study, USA (combined) (Schwartz *et al.*, 1996)	5.8; 1.5–22.3	1.22; 1.13–1.33	SO_4 RR strong (t = 4.66) but less than $PM_{2.5}$ (t = 7.41)
Unweighted RR: Too few data points for comparison			

Abbreviations: BS = Black Smoke; CI = confidence interval; CL = confidence limit; CP = coarse particle; IQR = interquartile range; OLS = Ordinary Least Squares regression; PM = particulate matter; RR = relative risk; SD = standard deviation.

weak or negative results from St. Louis and Kingston-Harriman were considered likely due to the poor placement of monitors and generally less developed methodology in this relatively early study (Dockery *et al.*, 1992). In Portage and Topeka, $PM_{2.5}$ concentrations were very low and the population was small, which resulted in relatively few "events" and lack of statistical power. The marginal increase in RR in Steubenville was considered to be due to a high correlation with coarse particulate (see below) (Schwartz *et al.*, 1996). Across all studies, the unweighted RRs were also elevated, with a mean ± SD of 1.15 ± 0.05 (n = 9) and a median of 1.14, for a 100 µg/m³ increase. The results indicate an average increase in daily mortality of 1.5% (unweighted) for each 10 µg/m³ daily increase in $PM_{2.5}$. No quantitative results have been reported for multivariate analyses of weighted data, except the combined (weighted) RR of 1.15 (95% CI 1.11–1.19) for the Six Cities Study (Schwartz *et al.*, 1996). The increase in $PM_{2.5}$-related risk of mortality is thus about twice that for PM_{10}.

The relationship between sulphate concentrations and mortality was investigated in only two studies (Dockery *et al.*, 1992; Schwartz *et al.*, 1996). In only one study (Schwartz *et al.*, 1996) was there a significant association, with a mean RR of 1.22 for six cities combined (95% CI 1.13–1.33) for a 100 µg/m³ increase, which can be interpreted as an average 2.2% increase in mortality per 10 µg/m³ increase in sulphate. There was no association of mortality with sulphate concentrations in two other cities (Dockery *et al.*, 1992), although, as noted above, the negative results in these cities (St. Louis, Missouri, and Kingston-Harriman, Tennessee) may have been due to poor placement of monitors and limitations in the methodology in this early study.

The coarse fraction of PM_{10} above 2.5 µm in diameter was not associated with mortality in the overall analysis of six U.S. cities (0.4% increase in mortality; 95% CI 0.01–1.0%) or in five individual cities (Schwartz *et al.*, 1996). In the sixth city (Steubenville, Ohio), the positive association was explained by the high correlation coefficient between coarse particles and $PM_{2.5}$ (r = 0.7), which resulted in attribution of the increased risk of mortality to the coarse fraction instead of to $PM_{2.5}$.

There is no clear evidence of a level of particulate matter that is without effect on mortality; instead, analyses suggest some increase at even the lowest ambient levels studied. The RR of mortality was observed to increase monotonically with increasing concentration of PM_{10} in the concentration range below 80–100 µg/m³, in both non-parametric analyses (Schwartz, 1993; Ostro *et al.*, 1996; Pope and Kalkstein, 1996) and quartile or quintile analyses (Pope *et al.*, 1992; Schwartz, 1993; Saldiva *et al.*, 1995). The quintile results for St. Louis, Missouri, and Kingston-Harriman, Tennessee (Dockery *et al.*, 1992) were also roughly monotonic when analysed together, but not separately, due to relatively poor information on exposure and (in Tennessee) to a small study population. A curvilinear response was observed in Santiago, Chile, with high slope at low PM_{10} concentrations and levelling off at concentrations above 100 µg/m³ (Ostro *et al.*, 1996). There is also an apparent curvilinear response in the Birmingham, Alabama, data at PM_{10} concentrations approximately above 50 µg/m³ (Schwartz, 1993). The quintile analysis of Utah Valley data by Lyon *et al.* (1995) indicated no effects at the mean concentration of 47 µg/m³; however, discrepancies were evident in the quintile means, and caution was indicated in interpretation of these data.

A number of investigators examined the specificity of effect for the causes of mortality. In six out of nine studies, there was a moderate to strong elevation of mortality from respiratory disease when compared with death from all causes (Schwartz, 1993; Ostro *et al.*, 1996; Schwartz *et al.*, 1996; Sunyer *et al.*, 1996; Zmirou *et al.*, 1996). In three studies (Lyon *et al.*, 1995; Anderson *et al.*, 1996; Ballester *et al.*, 1996), there was a lower RR for respiratory compared with total mortality. In the Six Cities Study

(Schwartz *et al.*, 1996), RRs for both COPD and pneumonia mortality were higher than for total mortality. Compared with total mortality, the RR for COPD was almost doubled, while the RR for pneumonia was 2.7 times the RR for total mortality for a 10 μg/m^3 increase in $PM_{2.5}$, which suggests an increased risk for people with pre-existing respiratory diseases.

The association between particulate matter and cardiovascular disease (CVD) was also examined in time-series analyses of mortality. In seven out of nine studies, there was a modestly (13–33%) (Ballester *et al.*, 1996; Schwartz *et al.*, 1996; Sunyer *et al.*, 1996) to substantially (up to 300%) (Schwartz, 1993; Lyon *et al.*, 1995) higher RR for CVD than for total mortality. Nevertheless, the association with CVD was often of lesser magnitude than the association with respiratory disease.

Elderly people have been suggested to be at increased risk from exposure to particulate matter (Schwartz, 1993, 1994d; Lipfert, 1994), but, overall, there is only a modest increase in RR for the elderly compared with the whole population.

Because of the strong intercorrelations often demonstrated between co-pollutants, such as particulate matter, sulphur dioxide, nitrogen dioxide, carbon monoxide and ozone, it has been difficult to attribute effects of air pollution to any single one of these agents to the exclusion of others. Overall, particulate matter retained its association with acute mortality in analyses that adjusted for other pollutants, although the RRs for particulate matter were slightly reduced during bivariate analyses (Dockery *et al.*, 1992; Kinney *et al.*, 1995; Saldiva *et al.*, 1995; Ito and Thurston, 1996; Ostro *et al.*, 1996; Pope and Kalkstein, 1996; Touloumi *et al.*, 1996; Verhoeff *et al.*, 1996; Zmirou *et al.*, 1996) and in one multivariate analysis for Toronto (Özkaynak *et al.*, 1995c). The relative risk for mortality and PM_{10} in nine bivariate analyses (unweighted) was 1.07 ± 0.05 (mean ± SD), with a median of 1.05 (Table 8).

In all of the analyses that examined one or more air pollutants together in the same model with particulate matter, the association of particulate matter with daily mortality was remarkably robust. This was the case for all four of the normally considered gaseous pollutants — sulphur dioxide, nitrogen dioxide, carbon monoxide and ozone. Moreover, in most locations, the magnitude of the particulate matter association was greater than for any of the other air pollutants considered, the exceptions being ozone in London, U.K. (Anderson *et al.*, 1996) and sulphur dioxide in Lyon, France (Zmirou *et al.*, 1996) and Barcelona, Spain (Sunyer *et al.*, 1996). The magnitude, robustness and consistency of this association across so many locations with differing air pollutant mixtures indicate that particulate matter, especially PM_{10} and $PM_{2.5}$, is the best indicator of the effects of air pollution on mortality, possibly acting together with other air pollutants.

2.4.1.2.2 Acute effects: hospitalizations and emergency department visits

A summary of the findings for particulate matter from the time-series analyses of hospitalizations for cardiorespiratory disease is presented in Table 9.

In all of the 16 studies using univariate analyses, there were significant associations with PM_{10} after adjustment for potential confounders and covariates, at concentrations averaging 25–55 μg/m^3. Each 10 μg/m^3 increase in PM_{10} was associated with elevated respiratory and/or cardiac hospitalizations or emergency department visits, ranging between 0.35% and 7.3%, with a median of 1.7%.

Studies varied in quality. There were methodological limitations in handling of potential confounders and covariates in the majority of the older studies (1994 and earlier). In the most reliable studies, results for larger cities over a period of several to many years were reported, thereby increasing the number of data points and the statistical power to detect

TABLE 9 Summary of results of time-series studies of hospital admissions and emergency department visits in relation to particulate matter

Location and references	PM concentration (µg/m³) (mean; range)	Endpoints	% increase of effects per 10 µg/m³ (95% CI)	Other pollutants considered
PM_{10}				
Utah Valley Cache Valley Salt Lake Valley (Pope *et al.*, 1991)	53 55 <40 [3 locations (max. 365)]	Respiratory, all ages (total 4144 admissions) - pneumonia - bronchitis - asthma	Rates doubled in Utah Valley compared with Salt Lake or Cache Valley when steel mill open or when PM_{10} > 50 1.7% 4.0% 1.4% (not significant)	No other pollutants considered (SO_2 was low, near detection limit)
Seattle, Washington (Schwartz *et al.*, 1993)	29.6; 6–103	ED visits, all ages - asthma (7.1/day)	3.7% (1.2–6.4%) (< 65 years) (not significant, ≥65 years)	SO_2, O_3 not significant (RR = 0.99 for SO_2, RR = 0.97 for O_3); extinction coefficient also associated, RR = 1.11 (1.04–1.18); main source of PM was wood smoke
Birmingham, Alabama (Schwartz, 1994a)	45 (19, 77; 10th, 90th %)	(≥65 years) - pneumonia (5.9/day) - COPD (2.2/day)	 1.9% (0.7–3.2%) 2.7% (0.8–5.0%)	O_3 (24-hour) pneumonia RR = 1.14 (0.94–1.38); O_3 (1-hour) COPD RR = 1.07 (0.96–1.20); no bivariate regression (PM_{10}–O_3 correlation = 0.29)
Detroit, Michigan (Schwartz, 1994b)	48 (22, 90; 10th, 90th %)	(≥65 years) - pneumonia (15.7/day) - COPD (5.8/day)	 1.2% (0.4–1.9%) (+ O_3) 2.0% (0.9–3.2%) (+ O_3)	O_3 (24-hour) positive; pneumonia RR = 1.052, COPD RR = 1.054 (per 20 µg/m³); bivariate regression did not alter coefficients for PM_{10} or O_3, i.e., both independently associated (PM_{10}–O_3 correlation = 0.35)
Detroit, Michigan (Schwartz and Morris, 1995)	48 (22, 90; 10th, 90th %)	cardiovascular, ≥65 years - ischemic heart disease (44/day) - congestive heart failure (27/day)	 0.56% (0.2–1.0%) 1.00% (0.38–1.62%)	Congestive heart failure: PM_{10} RR 0.75% (0.13–1.4%) + CO; O_3 not significant; CO RR = 1.022 (1.01–1.03) but not significant in bivariate regression; SO_2 RR = 1.014 (1.003–1.026 for IQR) but not significant + PM_{10} (i.e., CO, SO_2 not independent of PM_{10}) Ischemic heart disease: PM_{10} RR 0.5% (0.06–0.93%)

TABLE 9 (continued)

Location and references	PM concentration (µg/m³) (mean; range)	Endpoints	% increase of effects per 10 µg/m³ (95% CI)	Other pollutants considered
Minneapolis, Minnesota (Schwartz, 1994c)	36 (18, 58; 10th, 90th %)	(≥65 years) - pneumonia (6/day) - COPD (2/day)	 1.7% (0.3–3.3%) 5.7% (2.0–10.6%)	PM_{10} RR same or higher + O_3; O_3 independently associated (RR = 1.15) with pneumonia in co-regression with PM_{10} (i.e., both independently associated)
New Haven, Connecticut (Schwartz, 1995)	41 (67; 90th %)	(≥65 years) respiratory (8.1/day)	1.2% (0.0–2.6%; $p < 0.05$)	PM_{10} + O_3: PM 1.8%, O_3 1.4% (20 µg/m³); PM + SO_2: PM_{10} same, SO_2 0.8%, PM_{10} and O_3 independently associated, SO_2 probably not (SO_2 not significant with O_3)
Tacoma, Washington (Schwartz, 1995)	37 (67; 90th %)	(≥65 years) respiratory (4.2/day)	2.0% (0.6–3.4%)	PM_{10} + O_3: PM not significant ($p > 0.1$), O_3 RR same; PM_{10} + SO_2: PM_{10} RR same, SO_2 not significant (RR = 0.99); O_3 dominates over PM_{10} and SO_2
Spokane, Washington (Schwartz, 1996)	46 (83; 90th %)	- all respiratory (≥65 years) (3.9/day) - pneumonia (1.9/day) - COPD (1.0/day)	1.7% (0.7–2.7%) 1.1% (–0.03 to 2.5%) 3.4% (1.6–5.4%)	SO_2 negligible in Spokane; 1-hour O_3 also associated, 9.5% for 20 µg/m³; not co-regressed because only 115 days measured together
Anchorage, Alaska (Gordian *et al*., 1996)	45.5; 5–565	(doctors' visits) - asthma - upper respiratory tract infections	 3.5% ($p < 0.01$) 1.2% ($p < 0.05$)	CO associated with upper respiratory tract infections, bronchitis; benzene high; limited data for other pollutants; O_3 max. = 80 µg/m³ (2 years); SO_2, NO_2 low
East Washington State (Hefflin *et al*., 1994)	40; 3–1700	- bronchitis - asthma	0.35% ($p = 0.03$) not significant, data not shown	No other pollutants considered; dust storms resulted in increased otitis, upper respiratory tract infections
Toronto, Ontario (Thurston *et al*., 1994)	30, 30, 39 (3 years)	- respiratory - asthma	4.7% ($p = 0.015$) 0.44% (not significant)	O_3 also associated in bivariate regression with all PM metrics; H^+ remained significant, SO_4 marginally so

TABLE 9 (continued)

Location and references	PM concentration (µg/m³) (mean; range)	Endpoints	% increase of effects per 10 µg/m³ (95% CI)	Other pollutants considered
Toronto, Ontario (Burnett *et al.*, 1997)	25; 10–58	- respiratory - cardiac	2.5% (t = 3.42) 2.3% (t = 2.24)	PM_{10} coefficient retained value when regressed with all gases except NO_2 (but r high); strength of association: COH > $PM_{2.5}$ > SO_4 > PM_{10} > H^+ > CP; O_3 was independently associated in bivariate regressions with all PM
Montréal, Quebec (Delfino *et al.*, 1994)	31.5 (July–August)	- asthma - respiratory non-asthma	2.1% (0.6–4.0%) 1.0% (t = 1.83; not significant)	PM_{10} RR for asthma reduced to 1.8% (marginally significant) in bivariate regression with O_3 and O_3 also reduced (not significant), but r = 0.63, SO_4 included
Montréal, Quebec (Delfino *et al.*, 1997)	30.1, 21.7 (2 years)	(ED visits) - respiratory	Elderly (>64 years) only: 7.3% (1.95–12.7%)	PM_{10} reduced to 4.5% (not significant) with O_3; O_3 significant with all PM (H^+, SO_4, $PM_{2.5}$, PM_{10}); limited evidence for independent associations of O_3 and 3 of 4 PM, but PM–O_3 and O_3–temperature correlations high
Paris, France (Dab *et al.*, 1996)	50.8 (max. 138) (PM_{13})	- all respiratory - COPD - asthma	0.45% (0.04–0.87%) –0.5% not significant –0.025% not significant	SO_2 significant, all 3 endpoints; NO_2 significant for asthma; BS ≈ PM_{13}
Black Smoke				
London, England (Ponce de Leon *et al.*, 1996)	14.6; 6–27	- respiratory disease	–2 to 2% (not significant)	O_3 RR unchanged (independent) in bivariate regression with BS; correlations for BS–O_3: 0.3–0.45; for BS–NO_2: 0.4–0.8; NO_2, SO_2 not significant
Birmingham, England (Walters *et al.*, 1994)	12.7 (10%–90%, 4–25; max. 188) 12.3 (winter)	- respiratory disease - asthma	12.3% (5.8–18.2%) — winter 2.8% (0.3–5.3%) — winter	BS + SO_2: BS significant, respiratory disease + asthma, SO_2 negative; alone, SO_2 significant, respiratory disease; marginal for asthma
Amsterdam, Netherlands (Schouten *et al.*, 1996)	11; 1–37	- respiratory disease - COPD - asthma	1.3% (–3.1 to 8.7% — 65+) 4.7% (–1.9 to 16.5%) –2.0% (–5.6 to 4.8%)	No correlations or co-regressions; O_3, SO_2 and NO_2 also not significant

TABLE 9 (continued)

Location and references	PM concentration (µg/m³) (mean; range)	Endpoints	% increase of effects per 10 µg/m³ (95% CI)	Other pollutants considered
Rotterdam, Netherlands (Schouten *et al.*, 1996)	26; 6–61	- respiratory disease - COPD - asthma	3.7% (0.9–7.3%) 2.4% (–1.2 to 7.6%) not significant (not given)	No correlations or co-regressions; O_3 marginal, respiratory disease (adults), NO_2 significant, COPD (all ages); SO_2 not significant
Paris, France (Dab *et al.*, 1996)	31.9 (max. 268) 40 (winter) 25 (summer)	- respiratory - COPD - asthma	0.41% (0.07–0.75%) –0.05% (not significant) 0.43% (not significant)	No correlations or co-regressions; PM_{13} similar to BS; O_3 not significant; SO_2 positive all endpoints; NO_2 positive, asthma
Athens, Greece (Pantazopoulou *et al.*, 1995)	75 (winter) 55 (summer)	(ED visits) - respiratory disease - cardiac	 1.2% (winter) 0.96% (winter)	High collinearity (values not given); no co-regressions; CO, NO_2 also significant in winter
Barcelona, Spain (Sunyer *et al.*, 1993)	68 (IQR 45–84) winter 48 (IQR 31–63) summer	(ED visits) - COPD	2.28% (t = 5.0) (winter) 0.94% (t = 2.7) + SO_2	In bivariate regression, both BS and SO_2 coefficients reduced, but BS remained significant in winter; in summer, SO_2 significant, BS not; BS–SO_2 r = 0.7 (high)
Barcelona, Spain (Castellsague *et al.*, 1995)	68 (IQR 45–84) winter 48 (IQR 31–63) summer	(ED visits) - asthma	0.9% (–0.8 to 2.6%) winter 3.3% (lag 0 days) summer 4.6% (lag 0–3 days) summer	No co-regression; BS r = 0.32–0.73 for O_3, NO_2, SO_2; NO_2 significant, but O_3, SO_2 not significant
$PM_{2.5}$ and other fine PM				
Toronto, Ontario 3 summers, June–September 1992–1994 (388 days) (Burnett *et al.*, 1997)	(mean; 5th–95th) $PM_{2.5}$ (16.8; 4–40) SO_4 (5.6; 0.5–22.4) H^+ (5; 0–23 nmol/m³) COH (0.8 × 10³ ft)	respiratory admissions cardiac admissions	RRs for interquartile range: respiratory; cardiac; IQR $PM_{2.5}$, 1.037; 1.031; 11.0 SO_4, 1.029; 1.017; 3.9 H^+, 1.024; 1.024; 5.3 COH, 1.037; 1.062; 0.25 % increase in admissions per 10 µg/m³ of $PM_{2.5}$: Respiratory: 3.3% (95% CI 1.3–5.2%, significant) Cardiac: 2.8% (95% CI –0.2 to 5.8%, not significant) % increase in admissions per 10 µg/m³ of sulphate: Respiratory: 7.3% (95% CI 3.8–10.8%) Cardiac: 0.4% (95% CI –1.1 to 0.9%)	RR, regression with all gases respiratory; cardiac 0.999; 0.993 1.000; 0.984 1.006; 1.005 1.023; 1.059 % increase in admissions per 10 µg/m³ of $PM_{2.5}$ with O_3: Respiratory: 2.4% (95% CI 0.3–4.4%) Cardiac: 1.3% (95% CI –2.0 to 4.6%) % increase in admissions per 10 µg/m³ of sulphate with O_3: Respiratory: 3.8% (95% CI –0.1 to 7.7%) Cardiac: –1.0% (95% CI –6.4 to 4.4%)

TABLE 9 (continued)

Location and references	PM concentration (µg/m³) (mean; range)	Endpoints	% increase of effects per 10 µg/m³ (95% CI)	Other pollutants considered
Southern Ontario (including Algoma, Sudbury) daily time series, 6 years 1983–1988 (April–September) (Burnett *et al.*, 1994)	SO_4 (5.3) (Toronto 6.7; 95th % 20.1)	respiratory admissions (May–August calculated)	4.15% (single regression)	2.08% (model + O_3) 2.05% (model + O_3 lag 1, 3 days, + SO_4 lag 1 day) O_3 was more important predictor of respiratory admissions than SO_4, but SO_4 independently associated ($p < 0.0001$); O_3–SO_4 r = 0.38–0.65, various monitors
Southern Ontario (including Algoma, Sudbury) daily time series, 6 years 1983–1988 (2192 days) (Burnett *et al.*, 1995)	SO_4: 4.37 (95th % 13) [$PM_{2.5}$: 15.3 (95th % 31.9), based on site-specific conversion factor: $PM_{2.5} = 6.97 + 1.92\ SO_4$] [$PM_{10}$: 26.4, based on site-specific conversion factor: $PM_{10} = 15.5 + 2.49\ SO_4$]	respiratory - asthma - COPD - infections cardiac - coronary disease - dysrhythmias - heart failure	SO_4 2.7% (1.8–3.6%) 2.5% (1.1–4.3%) 3.7% (1.6–5.3%) 2.5% (1.3–3.7%) 2.5% (1.3–3.7%) 1.8% (0.54–2.9%) 1.0% (–1.5 to 3.5%) 2.3% (0.46–4.1%)	Figures included adjustment for O_3; results of single pollutant regression virtually the same as results for bivariate regression with O_3; PM and O_3 independently associated
Montréal, Quebec daily time series, 2 summers, June–September 1992 (66 days) 1993 (98 days) (Delfino *et al.*, 1997)	$PM_{2.5}$: 18.5 (90th % 44); 12.2 (90th % 22) SO_4: 4.96 (year 1992; 90th % 17.0); 3.34 (year 1993; 90th % 81) H^+: 11.3; 4.0 (nmol/m³)	ED visits for respiratory disease, age >64 years	Results for 1992 not significant 1993 results (elderly, ≥65 years): $PM_{2.5}$: 9.6% (1.9–17.3%) SO_4: 18.2% (2.4–34.3%) H^+ (infants, <2 years): 12.6% (1.0–24%)	H^+ significant only in infants in preliminary results, but judged inconsistent; in adults, $PM_{2.5} + O_3$: 4.5% (not significant, but $PM_{2.5}$–O_3 correlation coefficient very high, r = 0.62); PM and O_3 likely independently associated
Montréal, Quebec daily time series 5 years, 1984–1988 (May–October, 911 days) (July–August, 310 days) (Delfino *et al.*, 1994)	SO_4: May–October: 4.2 (90th % 8.2) July–August: 4.9 (90th % 9.7)	respiratory admissions asthma and non-asthma	For respiratory non-asthma admissions: 9.6% for days ≥8.2 µg/m³ (no continuous data given)	O_3, temperature included in model; correlations high between PM_{10}, SO_4 (r = 0.90); O_3–SO_4 (r = 0.59); PM_{10}–O_3 (r = 0.63); suggested that PM_{10} effect was really FP
Toronto, Ontario 3 summers, July–August 1986–1988 (123 days) (Thurston *et al.*, 1994)	$PM_{2.5}$: 18.6, 3-year mean (17.7, 15.8, 22.3 for individual year) SO_4: 7.1, 3.6, 11.8 for individual year H^+: 21, 13, 52 nmol/m³ for individual year	respiratory admissions asthma admissions	respiratory, asthma $PM_{2.5}$: 9.3% (1.3–17.3%), 3.7% (–1.6 to 9.3%) SO_4: 0.8%, 0.7% H^+: 1.65%, 2.1%	H^+ significant alone, marginally with O_3; all other PM fell 50–66% when co-regressed + O_3; correlation coefficients with O_3: r = 0.7 with $PM_{2.5}$; r = 0.8 with SO_4; r = 0.5 with H^+; r = 0.65 with CP; r = 0.7 with IP; r = 0.6 with TSP

TABLE 9 (continued)

Location and references	PM concentration (µg/m³) (mean; range)	Endpoints	% increase of effects per 10 µg/m³ (95% CI)	Other pollutants considered
Buffalo, New York June–August, 2 years, 1988–89 (Thurston *et al.*, 1992)	SO_4 (9.0; max. 37.4) H^+ (2.2; max. 18.7)	respiratory admissions	SO_4: 9.0% ($p < 0.01$) H^+: 28% ($p < 0.01$)	O_3 had highest mean effect; O_3 correlation coefficient $r = 0.51$ (H^+); 0.67 (SO_4); H^+–SO_4 $r = 0.77$ (can't be distinguished)
Anchorage, Alaska 22 months, May 1992 – March 1994 (Gordian *et al.*, 1996)	$PM_{2.5}$ was unusually low fraction of PM_{10}; limited monitoring gave average ratio 0.26, as low as 0.14	outpatient visits to ED or doctors' offices, for respiratory diseases	No data reported	CO also examined; coarse fraction of PM larger than usual; PM significant, but high PM during eruption not significant for asthma, respiratory infections (CP not as important as fine fraction PM_{10})
Eastern Washington State 3 towns, October 1990, all of 1991 (Hefflin *et al.*, 1994)	Area subject to blowing dust, thus $PM_{2.5}$ fraction of PM_{10} was smaller than average	ED visits for respiratory diseases (various)	No data reported	Although the odds ratio for ED visits was raised slightly in dust storms, this was less than expected on the basis of high PM_{10} levels; concluded that crustal dust not significant

Abbreviations: BS = Black Smoke; CI = confidence interval; COH = coefficient of haze; COPD = chronic obstructive pulmonary disease; CP = coarse particles ($PM_{10-2.5}$); ED = emergency department; FP = fine particles ($PM_{2.5}$); IP = inhalable particles; IQR = interquartile range; PM = particulate matter; RR = relative risk; SO_4 = sulphate; TSP = total suspended particulate.

adverse effects. In several studies, data from only one city for a limited time, as short as one summer, were examined (Delfino *et al.*, 1997), which limits the statistical power. Certain studies involving larger cities with directly measured PM_{10} were carefully done and included data over several years (Schwartz, 1994a,b,c, 1996; Dab *et al.*, 1996; Burnett *et al.*, 1997); however, all were lacking in one or more respects. For example, only 388 days of data over three years were considered in the Toronto, Ontario, study by Burnett *et al.* (1997). Schwartz and co-workers (Schwartz *et al.*, 1994a,b,c, 1995, 1996; Schwartz and Morris, 1995) included only the elderly, aged 65 or more (due to limitations in the U.S. medicare database), and did not always consider the possibility of effects from other co-occurring gaseous air pollutants. The most reliable results with respect to estimation of increased hospitalization risks associated with PM_{10} are considered to be those of Burnett *et al.* (1995) for sulphate pollution for all of southern Ontario (population 8.7 million), over a period of six years (2192 days). Although PM_{10} itself was not directly measured, site-specific conversion factors were available for estimation of PM_{10} from measured sulphate. An increase in respiratory hospitalizations of 0.7% (95% CI 0.5–1.0%) per 10 µg/m³ increase in PM_{10} was calculated based on the results of this study. This figure is within the same range as the risk observed from the combined studies in which PM_{10} was directly measured.

The results for Black Smoke are also presented in Table 9. In six of eight studies, there was a significant association between concentrations of Black Smoke and respiratory hospitalizations. The increase in respiratory hospitalizations in the positive studies varied widely from 0.4% (95% CI 0.07–0.75%) to 12.3% (95% CI 5.8–18.2%) per 10 µg/m³ increase in Black Smoke, at the mean concentrations ranging between 12.7 and 75 µg/m³. In the two studies in which there was no association (Schouten *et al.*, 1996 in Amsterdam, Netherlands; Ponce de Leon *et al.*, 1996 in London, U.K.), the ambient Black Smoke concentrations were quite low (<15 µg/m³ versus >26 µg/m³). The authors of the London study (Ponce de Leon *et al.*, 1996) explained their negative results with respect to a Black Smoke–respiratory association as misclassification of exposure due to poor placement of Black Smoke monitors in the city. A strong and consistent association with ozone was also noted in this study, as well as a somewhat less consistent association with nitrogen dioxide, with which Black Smoke was highly correlated. In the case of the Amsterdam results, there were only 6.5 respiratory admissions per day. The authors repeated their analysis for the entire population of the Netherlands, and there was an increased risk of 1.01% (95% CI 0.2–1.8%) for a 10 µg/m³ increase in Black Smoke. Thus, these two studies do not detract from the consistency of the positive association observed between increases in Black Smoke and increased hospitalizations.

A summary of findings for $PM_{2.5}$ and other fine particle metrics is presented in Table 9. Only three hospitalization studies, two in Toronto (Thurston *et al.*, 1994; Burnett *et al.*, 1997) and one in Montréal (Delfino *et al.*, 1997), directly examined the association between $PM_{2.5}$ and hospitalizations or emergency department visits. Significantly positive associations of $PM_{2.5}$ (mean concentrations 12.2–18.6 µg/m³) with respiratory effects were seen in univariate analyses in all three studies. Each 10 µg/m³ increase in $PM_{2.5}$ was associated with a 3.3–9.6% increase in respiratory hospitalizations and emergency department visits and a 2.8% increase (not statistically significant) for cardiac hospitalizations. The association was slightly reduced in Toronto (Burnett *et al.*, 1997) when ozone was included in a bivariate regression analysis and was no longer significant in an earlier Toronto study (Thurston *et al.*, 1994) or in Montréal (Delfino *et al.*, 1997) after inclusion of ozone. The high correlation between ozone and $PM_{2.5}$ in these two studies ($r = 0.62–0.7$) makes it difficult to single out the individual effects of $PM_{2.5}$ from ozone. However, in the larger Toronto study by Burnett *et al.* (1997) in which the correlation coefficient was relatively low ($r < 0.34$), the association with $PM_{2.5}$ remained significant after including ozone. A study by

Burnett *et al.* (1995) also provided additional information on the magnitude of the risk of respiratory hospitalizations associated with $PM_{2.5}$ based on measurements of sulphate in southern Ontario. An overall site-specific conversion factor for sulphate to $PM_{2.5}$ was determined using the equation $PM_{2.5} = 6.973897 + 1.917519$ sulphate. The data indicate that a 1.1% (95% CI 0.7–1.4%) increase in respiratory hospitalization and a 1.0% (95% CI 0.5–1.5%) increase in cardiac hospitalization were associated with an estimated 10 µg/m³ increase in $PM_{2.5}$ (adjusted for ozone).

In all seven studies in which sulphate was examined, there were positive associations with respiratory endpoints (Table 9). Increases ranged from 0.8% to 9.6% in respiratory hospitalizations (n = 5), and up to 18% for emergency department visits (n = 1), associated with every 10 µg/m³ increase in ambient sulphate, at mean concentrations of 4.4–11.8 µg/m³. As noted previously, reliance has been placed on the study by Burnett *et al.* (1995) for southern Ontario, because of the very large database drawn from a population of 8.7 million over 2192 days and the advanced methodological treatment of confounders and covariates. In two studies (Burnett *et al.*, 1995, 1997), associations between cardiac effects and sulphate air pollution were also reported. The mean ozone-adjusted increase in cardiac hospitalizations was 2.5% (95% CI 1.3–3.7%) per 10 µg/m³ elevation of sulphate in the large southern Ontario yearly study (Burnett *et al.*, 1995). However, in the much smaller summertime study in Toronto (Burnett *et al.*, 1997), the increase in RR was not significant (mean 0.4%; 95% CI –1.1% to 0.9%). The associations were weakened (not statistically significant) in both studies when ozone was included in the regression with sulphate. Sulphate forms a part of the fine particle fraction and is usually <1.0 µm in size. Because it correlates well with $PM_{2.5}$ (correlation coefficients >0.7 in southern Ontario), it appears to serve well as a fine particle indicator in the absence of direct measurements of $PM_{2.5}$. However, the respective roles of sulphate and non-sulphate fine particles remain unclear.

While the results of the available studies suggest that it is largely the fine fraction of particulate matter that is associated with cardiorespiratory morbidity, significant associations were observed between the coarse fraction ($PM_{10–2.5}$) and respiratory and/or cardiac admissions in two studies in Toronto (Thurston *et al.*, 1994; Burnett *et al.*, 1997) and between PM_{10} and increased respiratory emergency department visits in two U.S. studies in which $PM_{2.5}$ was an unusually small fraction of PM_{10} (Hefflin *et al.*, 1994; Gordian *et al.*, 1996). In those studies in which account was taken of the effects of co-occurring pollutants, the association of the coarse fraction with hospitalizations was robust to the inclusion of gaseous pollutants in some instances (Burnett *et al.*, 1997, for respiratory admissions), but not in others (Thurston *et al.*, 1994; Burnett *et al.*, 1997, for cardiac admissions with sulphur dioxide or nitrogen dioxide); however, the high correlations with one or more gaseous pollutants, and among the particle metrics, complicated attribution of the observed effects to the coarse fraction.

Overall increases in respiratory hospitalizations were associated with particulate matter even at the low concentration ranges (10–100 µg PM_{10}/m³) examined. Curves appeared to increase monotonically, with steep slopes at low concentrations and some suggestion of curvilinear responses (lower slopes) at higher concentrations. The curve for COPD admissions in Detroit, Michigan (Schwartz, 1994b) was the only one showing an anomalous response, since hospitalizations for quartile 2 (at 30 µg/m³) were higher than for quartile 3 (at 50 µg/m³).

The specificity of the association between particulate matter and the causes of disease was investigated. In a series of studies on the elderly in four U.S. cities (Schwartz, 1994a,b,c, 1996), the association between PM_{10} and COPD was strong, 2.0–5.7% per 10 µg/m³ increase. However, in these studies, the authors did not compare COPD admissions for the elderly with total respiratory admissions or admissions of any other age categories. The study conducted in southern

Ontario (Burnett *et al.*, 1995) on the entire age range indicates that the increased hospitalization risk for COPD (4.8%) was somewhat higher than that for total respiratory admissions (excess admissions 3.7%) per 13 μg/m³ increase, using sulphate as the particle metric. The southern Ontario studies also demonstrate an effect of sulphate pollution on asthma onset (Burnett *et al.*, 1994, 1995). The excess asthma admissions were higher than the total excess respiratory admissions for all ages (7.1% versus 5.8% for a 5.3 μg/m³ increase in sulphate + 100 μg/m³ increase in ozone), the highest rate being for infants ≤1 year (13%).

There was a positive, but less strong, association of CVD with particulate matter pollution in southern Ontario, with an excess hospitalization rate of 2.5% (95% CI 1.3–3.7%; adjusted for ozone) for a 10 μg/m³ increase in sulphate, in comparison with a rate of 2.7% (95% CI 1.8–3.6%) for respiratory disease (Burnett *et al.*, 1995). In a much smaller study carried out in Toronto with a mean $PM_{2.5}$ concentration of 16.8 μg/m³, the risks for CVD hospitalizations were lower by up to one-half the RR for respiratory disease and were not significant (2.8% increase in CVD versus 3.3% increase in respiratory disease per 10 μg/m³ increase in $PM_{2.5}$ without ozone adjustment) (Burnett *et al.*, 1997). Similarly, in Detroit, Michigan (Schwartz, 1994b; Schwartz and Morris, 1995), admission rates were higher for respiratory diseases (2.0% for COPD and 1.2% for pneumonia per 10 μg/m³ increase in PM_{10}) than for CVD (0.56% for ischemic heart disease and 1.0% for heart failure) at PM_{10} concentrations averaging 48 μg/m³.

The effects of age on hospitalizations or emergency department visits were examined in several locations. For cardiovascular hospitalizations, there was a 3.5% increase (95% CI 1.9–5.0%) in the elderly compared with a 2.5% increase (95% CI 0.5–4.8%) for those less than 65 years of age for a 13 μg/m³ increase in sulphate (Burnett *et al.*, 1995). In several studies, elderly people (>64 years) were also at higher risk of respiratory hospitalizations due to particulate matter pollution than were younger people (Schouten *et al.*, 1996; Delfino *et al.*, 1997). In Amsterdam, Netherlands, the elderly appeared to be at greatest risk for respiratory disease associated with Black Smoke; however, most risks were not significantly elevated, likely due to the small numbers in subdivided groups (Schouten *et al.*, 1996). In Montréal, Quebec, mean increases of 10 μg/m³ for PM_{10}, $PM_{2.5}$ and sulphate were associated with increases of emergency department visits in the elderly of 7.3% (95% CI 1.95–12.7%), 9.6% (95% CI 1.9–17.3%) and 18.2% (95% CI 2.4–34.3%), respectively (Delfino *et al.*, 1997). Results of other studies are not consistent with the above observations. In the southern Ontario studies, after adjustment for ozone and temperature, warm-season sulphate was associated with smaller increases in respiratory hospitalizations in the elderly (4.3% increase per 5.1 μg sulphate/m³ increase) than in those between ages 2 and 64 (5.5–7.2%) (Burnett *et al.*, 1994), whereas increases for all-year sulphate were similar for younger (3.7% increase) and older people (65+ years) (3.8% increase per 13 μg/m³ increase in sulphate) (Burnett *et al.*, 1995). In Rotterdam, Netherlands, the association between Black Smoke and all respiratory admissions was strongest for the 15- to 64-year age group, with an RR of 1.37 compared with an RR of 0.97 for those aged 65 or older (Schouten *et al.*, 1996). In London, U.K., there were no consistent differences in RRs of respiratory hospitalizations for those 65 and over compared with those between 15 and 64 years (Ponce de Leon *et al.*, 1996). With asthma as the endpoint, the elderly were at less risk than those under age 65 years in Seattle, Washington (Schwartz *et al.*, 1993), Anchorage, Alaska (Gordian *et al.*, 1996) and southern Ontario (Burnett *et al.*, 1994).

Young children appear to be a high-risk group with respect to respiratory hospitalizations. In the Utah Valley study, bronchitis and asthma admissions were twice as high for children (1–5 years) when a local steel mill was open as when it was closed; for all ages combined, the rates were only 1.4 times as high for bronchitis

and 1.2 times as high for pneumonia (Pope, 1991). In Anchorage, Alaska (mean PM_{10} 45.5 µg/m³), upper respiratory tract infections were most strongly associated with PM_{10} in children (<10 years) and in older adults (≥45 years) compared with adolescents and younger adults (Gordian *et al.*, 1996). In southern Ontario, the association between summertime ozone (mean one-hour maximum 64–140 µg/m³) and sulphate (mean 3.1–8.2 µg/m³) combined and respiratory hospitalizations in infants and children was greater (14.8%) than for adults (4.3–7.2%) (Burnett *et al.*, 1994), whereas associations between all-year sulphate (2.0–7.7 µg/m³) and respiratory hospitalizations in children (mean excess admission 2.7%) was less than in adults (3.7–3.8% for 13 µg/m³ increases in sulphate) after adjusting for the effects of ozone (Burnett *et al.*, 1995). In the London, U.K., respiratory hospitalization study (Ponce de Leon *et al.*, 1996), children (aged 0–14 years) were not at increased risk for respiratory admissions due to Black Smoke or other air pollutants, at least not at the concentrations experienced in the 1980s and early 1990s.

In most of the studies, potential confounding by temperature and season was well addressed using a variety of methods. However, there has been concern that the effects observed might be due to the fluctuation of other unmeasured pollutants in concert with particulate matter. Most of the hospitalization studies on particulate matter examined at least one or two other air pollutants as potential covariates (Table 9). Indeed, in many studies, there was a strong correlation between particulate matter and gaseous pollutants; yet for respiratory hospitalizations or emergency department visits, gaseous pollutants in bivariate or multivariate analyses reduced but did not abolish the significance of the risk for particulate matter. With respect to cardiac hospitalizations, the results are inconsistent. In studies conducted by Schwartz and Morris (1995) in Detroit, Michigan, and by Burnett *et al.* (1997) in Toronto, Ontario, the association between PM_{10} and cardiac hospitalizations remained significant in the bivariate or multivariate regression with ozone, carbon monoxide and sulphur dioxide. In the southern Ontario study (Burnett *et al.*, 1997), co-regression with ozone also did not reduce the significance of the risk of sulphate on cardiac hospitalizations. However, in the Toronto study (Burnett *et al.*, 1997), co-regression of $PM_{2.5}$ or sulphate with ozone reduced the cardiac risk to insignificance.

2.4.1.2.3 Acute effects: respiratory health (lung function, symptoms, restricted activity and days absent from work or school)

Increases in ambient particulate matter have been associated with small, reversible decrements in lung function in normal asymptomatic children and in both adults and children who have some form of pre-existing respiratory conditions, particularly asthma, at PM_{10} concentrations ranging from 10 to 174 µg/m³ (Pope *et al.*, 1991, 1992; Spektor *et al.*, 1991; Hoek and Brunekreef, 1993, 1994; Roemer *et al.*, 1993; Peters *et al.*, 1996a,b). For every 10 µg/m³ increase in PM_{10}, average decrements in peak expiratory flow rate were 0.09–0.4%. These changes were often accompanied, especially in adults, by increases in, for example, chronic bronchitis or cough. Each 10 µg/m³ increase in PM_{10} was associated with increases of 0.6–2.2% in respiratory symptoms.

Based on limited data, associations between $PM_{2.5}$ pollution and increased respiratory symptoms have been observed at concentrations averaging 18–22 µg/m³ (Ostro and Rothschild, 1989; Ostro *et al.*, 1991; Schwartz *et al.*, 1994; Peters *et al.*, 1996a,b). Each 10 µg/m³ increase in $PM_{2.5}$ was associated with increases of 0.9–2.2% in respiratory symptoms. For every 10 µg/m³ increase in sulphate, average decrements in peak expiratory flow rate were 1.2%, while the risk for increased respiratory symptoms was 4.6–16.4% (Ostro *et al.*, 1991; Schwartz *et al.*, 1994; Peters *et al.*, 1996a,b).

Respiratory-related restrictions in activity severe enough to result in an increased number of days lost to work in adult workers and in school

absences in children were also associated with elevated ambient PM_{10} (mean concentrations 41–51 µg/m³) (Ransom and Pope, 1992) and $PM_{2.5}$ (mean concentrations 20–25 µg/m³) (Ostro, 1987, 1990; Ostro and Rothschild, 1989) or with other fine particulate components such as sulphate (Ostro, 1990). Each 10 µg/m³ increase in PM_{10}, $PM_{2.5}$ and sulphate was associated with increases in respiratory symptom-related activity restriction of 9.0%, 12.9–15.8% and 17.4%, respectively.

2.4.1.2.4 Long-term and chronic effects

Relatively few studies are available in which the effect of long-term or chronic exposure to particulate matter on health outcomes has been examined. Such exposures, varying in duration between one and 16–20 years, were associated with increases in mortality, respiratory disease symptoms, decrements in lung function and, possibly, increases in lung cancer in both cross-sectional and prospective cohort studies.

In these studies, the concentrations of PM_{10}, $PM_{2.5}$ and sulphate were 18–47 µg/m³, 11–30 µg/m³ and 4.8–13 µg/m³, respectively. In two prospective cohort studies (Dockery *et al.*, 1993; Pope *et al.*, 1995), average increases in mortality from all causes of 10%, 7–14% and 7.5–32% were observed for each 10 µg/m³ increase in PM_{10}, $PM_{2.5}$ and sulphate, respectively, after adjustment for a number of potential confounders and covariates. In two descriptive studies (Özkaynak and Thurston, 1987; Pope *et al.*, 1995), the odds for annual mortality were increased by 4.3–9.8% and 8.2–12.6% for each 10 µg/m³ increase in $PM_{2.5}$ and sulphate, respectively. Based on the mean particulate matter levels across six cities, lifespan was estimated to have been reduced by about two years over a 14-year period (Dockery *et al.*, 1993), an observation incompatible with suggestions that most or all of the observed deaths were due to "harvesting" or accelerating the death of persons already ill by a few days or a few weeks.

In several cross-sectional studies, significant decrements in lung function and/or increased odds ratios for bronchitis were associated with chronic exposure of children to particulate matter for all or most of their lives. In the most powerful of the available studies, each 10 µg/m³ increase in PM_{10}, $PM_{2.5}$ and sulphate was associated with decrements of forced vital capacity of 1.4%, 2.2% and 4.5%, respectively (Raizenne *et al.*, 1996). The odds ratios for bronchitis ranged from non-significant to 4.3% for each 10 µg/m³ increase in $PM_{2.5}$ and from non-significant to 9.3% for each 10 µg/m³ increase in sulphate (Özkaynak and Thurston, 1987; Dockery *et al.*, 1996a).

In a prospective study, an 18% increase in new cases of chronic bronchitis in older adults was associated with each 10 µg/m³ increase in $PM_{2.5}$ for the 10 years of the observation period (Abbey *et al.*, 1995a). Development of new cases of chronic bronchitis in association with exposure to particulate matter has also been observed in children (Dockery *et al.*, 1989, 1996a). Increases in the severity of respiratory symptoms of airway obstructive disease, chronic bronchitis and asthma have been associated with exposure to TSP, PM_{10}, $PM_{2.5}$ and sulphate for 10 years (Abbey *et al.*, 1995a).

There is also some evidence for an association between long-term exposure to fine particulate air pollution and lung cancer. In two recent cohort studies, exposure to average annual concentrations of 11–29.6 µg/m³ for $PM_{2.5}$ (Dockery *et al.*, 1993) or 3.6–23.5 µg/m³ for sulphate (Pope *et al.*, 1995a) was associated with an increased risk of lung cancer mortality (19.9% or 18.1% for an increase of 10 µg/m³ of $PM_{2.5}$ or sulphate, respectively), after adjustment for potential confounders or covariates. The associations were weak compared with those for other lifestyle factors such as smoking. In a cohort study conducted by Abbey *et al.* (1995b) on non-smoking Californian Seventh-Day Adventists (n = 6340), exposure to PM_{10} concentrations above 100 µg/m³ for 42 days per year was associated with a marginally increased incidence of all cancers combined in females (RR 1.15, 95% CI 0.97–1.38) and a non-

significant increase in respiratory cancers, but these results were based on relatively few cases of total (n = 175) and respiratory (n = 17) cancers. The association with lung cancer at this time is inconclusive.

2.4.2 Experimental animals and in vitro

Given the extensive epidemiological evidence of an association between acute health effects and ambient exposure to particulate matter (Section 2.4.1.2), information on particulate matter-induced effects in animals is primarily of interest with respect to the extent to which it provides insight into the biological plausibility for the association. Consequently, this section only briefly summarizes the results of studies in animals exposed to particulate matter, with emphasis on the target tissues, susceptible subpopulations, the toxicity of various particle size fractions and compositions, and plausible biological mechanisms for particulate matter-induced effects identified in these studies. These include severe damage of the alveolar interstitium tissue by the highly reactive surfaces of ultrafine particles and metal components, causing acute pulmonary edema and inflammation and/or particulate matter-induced alteration (enhancement or impairment) of the respiratory immune system.

2.4.2.1 Acute and short-term exposure

Acute exposures (four- to six-hour single exposures) of laboratory animals to a variety of types of particles, almost always at concentrations well above those occurring in the ambient environment (particles >1 mg/m^3, acid aerosols >50 µg/m^3), confirm that the cardiorespiratory system is a target for particle-induced effects.

Effects on the respiratory system include decreased ventilatory function and airway hyperresponsiveness (a hallmark of human asthma) in guinea pigs or rabbits (Chen *et al.*, 1990, 1991b, 1992a; El-Fawal and Schlesinger, 1994), altered mucociliary clearance in rabbits (Chen and Schlesinger, 1983; Grose *et al.*, 1985; Naumann and Schlesinger, 1986) and a range of histological and cellular (Callis *et al.*, 1985; Wiessner *et al.*, 1989, 1990; Guilianelli *et al.*, 1993) and biochemical (Lindenschmidt *et al.*, 1990; Chen *et al.*, 1991a,b,c, 1992b; Mohr *et al.*, 1992; Kobzik *et al.*, 1993; Kodavanti *et al.*, 1997) alterations in the lung, including the production of proinflammatory cytokines and other mediators by pulmonary alveolar macrophages.

The pulmonary immune system is also affected by exposure to particulate matter, seen as increased numbers of alveolar macrophages and polymorphonuclear leukocytes in the alveoli of mice, hamsters and rats (Brain and Cockery, 1977; Adamson and Bowden, 1981; Lehnert *et al.*, 1985), decreased mobility of alveolar macrophages in rabbits (Schlesinger, 1987) and altered (increased or decreased) ability of macrophages to phagocytize particles in mice and rats (Fisher and Wilson, 1980; Tabata and Ikada, 1988; Schlesinger *et al.*, 1990; Warheit *et al.*, 1991; Chen *et al.*, 1992b). (The latter effect is often related to particle composition, with silica and quartz causing a decrease in macrophage activity and iron oxide causing an increase in macrophage activity.) Particulate matter also modifies immunological responses, including airway defence mechanisms against microbial infections. This effect appears to be related to composition and not the particle effect, since particles with known cytotoxic properties, such as metals, affect the immune system to a significantly greater degree than other particles (Hatch *et al.*, 1985; Chen *et al.*, 1989, 1992b; Oberdörster *et al.*, 1992a, 1994b; Zelikoff and Schlesinger, 1992; Berg *et al.*, 1993; Nadeau *et al.*, 1995, 1996).

Finally, the cardiovascular system can also be affected; acute exposure to particulate matter induces electrocardiographic abnormalities in rats and dogs (Sakakibara *et al.*, 1994; Campen *et al.*, 1996; Nearing *et al.*, 1996).

The results of studies in animals also confirm that adverse effects of exposure to particulate matter, including mortality, morbidity and bronchial hypersensitivity to non-specific stimuli, are much more pronounced in individuals

with pre-existing cardiorespiratory diseases (Slauson *et al.*, 1989; Raabe *et al.*, 1994; Godleski *et al.*, 1996; Gilmour *et al.*, 1997; Killingsworth *et al.*, 1997). For example, mortality was increased following exposure of rats with acute pulmonary inflammation or chronic bronchitis to 250 µg $PM_{2.5}/m^3$ (three days, six hours per day), while no deaths occurred in healthy rats (Godleski *et al.*, 1996). Similarly, Killingsworth *et al.* (1997) reported that inhalation of fuel oil ash (approximately 580 $\mu g/m^3$, six hours per day for two days) caused effects only in the rats with pre-existing cardiorespiratory injury induced by monocrotaline, including acute mortality (40%), inflammatory cell infiltration in pulmonary interstitium and blood vessel walls, and increases in expression in lung and heart of proteins and messenger RNA of several chemokines involved in inflammatory cell recruitment.

The particle types most likely to induce acute adverse effects include metals, organics, acids and acidic sulphates of the fine particle mode, possibly occurring as coatings on fine or even ultrafine carrier particles (Chen *et al.*, 1991a,b, 1992a). It appears that the ultrafine particle mode (≤0.1 µm in size) may be of significant toxicological importance due to its large number and slow clearance rate from pulmonary interstitium (Oberdörster *et al.*, 1992b, 1994d). Ultrafine particles were shown to result in pulmonary inflammation and death after about 30 minutes in rats or guinea pigs exposed to a number concentration of 700 000 to 1 million particles (median diameter 26 nm) at near-ambient concentrations of ultrafine particles (9–60 $\mu g/m^3$) (Warheit *et al.*, 1990; Chen *et al.*, 1992b; Oberdörster *et al.*, 1995). There are indications that surface-complexed iron and other metals on particles are involved in pulmonary injury (Berg *et al.*, 1993; Guilianelli *et al.*, 1993; Carter *et al.*, 1997; Kodavanti *et al.*, 1997).

While the mechanisms for the cardiorespiratory effects observed following exposure to particulate matter are not clear, there is emerging evidence from the studies in animals that may ultimately explain the acute effects observed in the epidemiological studies. It is possible that the increase in respiratory diseases associated with particulate matter in the epidemiological studies is the result of severe damage of the alveolar interstitium tissue by the highly reactive surfaces of ultrafine particles (Ferin *et al.*, 1991) and metal components (Berg *et al.*, 1993; Guilianelli *et al.*, 1993; Carter *et al.*, 1997; Kodavanti *et al.*, 1997), causing acute pulmonary edema and inflammation (Oberdörster *et al.*, 1992a, 1994c); alternatively, these outcomes may reflect the particulate matter-induced alteration (enhancement or impairment) of the respiratory immune system (Brain and Cockery, 1977; Adamson and Bowden, 1981; Lehnert *et al.*, 1985; Killingsworth *et al.*, 1997). With respect to particulate matter-initiated acute cardiovascular effects, it has been postulated that oxidized low-density lipoprotein may play a pathological role, since diesel exhaust particles have been reported to cause oxidative modification of low-density lipoprotein *in vitro* (Ikeda *et al.*, 1995). Oxidized low-density protein has been found to cause endothelial damage, the proliferation of smooth muscle cells, monocyte–endothelial interactions, platelet aggregation and inhibition of endothelial-derived relaxation of vascular smooth muscles (Morel *et al.*, 1983; Ocana, 1989; Berliner *et al.*, 1990; Cushing *et al.*, 1990; Ezaki *et al.*, 1994; Ichinose *et al.*, 1995; Ikeda *et al.*, 1995). These biochemical and pathological changes may all lead to coronary vasospasm, lesion and blockade, and hypertension. There are also reports that chemokines, which are among the mediators induced by exposure to particulate matter, are involved in myocardial dysfunction, decreased contractility and vasoconstriction *in vitro* and *in vivo* (DeMeules *et al.*, 1992; Abe *et al.*, 1993; Yokoyama *et al.*, 1993; Mann and Young, 1994).

2.4.2.2 Subchronic and chronic exposure

In the available subchronic and chronic exposure studies, animals were repeatedly exposed to very high (>1 mg/m^3) concentrations of a wide variety of types of particles, often on a schedule that mimicked workplace conditions (e.g., six hours

per day, five days per week). These exposures resulted in a wide range of effects on the lung, including compromised lung functions in guinea pigs and rats (Wiester *et al.*, 1980; Ellakkani *et al.*, 1987; Mauderly *et al.*, 1988; Begin *et al.*, 1989; Heinrich *et al.*, 1989), hyperresponsiveness in rabbits (Gearhart and Schlesinger, 1986), impaired airway clearance function in guinea pigs, donkeys and rabbits (Gearhart and Schlesinger, 1989; Nagai *et al.*, 1991; Schlesinger *et al.*, 1992; Samet and Cheng, 1994) and impairment of immune functions in the lungs of rats and mice by reducing macrophage phagocytosis and bactericidal capability (Spiegelberg *et al.*, 1984; Gilmour *et al.*, 1989a,b; Kleinman *et al.*, 1995).

Long-term exposure to particulate matter results in histopathological and cytological changes in the lung, including chronic pulmonary inflammation, hyperplasia of the alveolar epithelium and pulmonary fibrosis, regardless of particle type, mass concentration, duration of exposure or species examined (Shami *et al.*, 1984; Henderson *et al.*, 1988; Gearhart and Schlesinger, 1989; Schlesinger *et al.*, 1992; Kawabata *et al.*, 1993; Kleinman *et al.*, 1995). The development of lung tumours has also been observed following chronic exposure of rats to high levels of a wide variety of particle types (Heinrich *et al.*, 1986a,b; Kawabata *et al.*, 1993; Pott *et al.*, 1994).

A particularly relevant subchronic study was conducted by Kleinman *et al.* (1995), in which rats were exposed to ammonium sulphate (20 or 70 µg/m^3, 0.2 µm MMAD), ammonium nitrate (90 or 350 µg/m^3, 0.6 µm MMAD), resuspended road dust (300 or 900 µg/m^3, 4 µm MMAD) or purified air, four hours per day, four days per week, for eight weeks. Decreases in alveolar macrophage function and increased pulmonary permeability were observed following exposure to nitrate and sulphate (low and high doses) and road dust (high dose only). Based on quantitative histopathological analyses, there were moderate to substantial changes following exposure to particles, in the order of sulphate > nitrate > road dust. Thus, in this study, the lungs were adversely affected by repeated exposure to particles of a size, mass concentration and composition relevant to ambient exposure conditions, and the fine fraction of PM_{10} was more toxic than the coarse fraction.

2.5 Toxicokinetics

Particle size is believed to be the most important characteristic influencing deposition in the three anatomical regions of the respiratory system (Lippmann, 1977; Anderson *et al.*, 1990; Dockery and Pope, 1994). In the extrathoracic region (nose and mouth) in human airways, virtually all particles >10 µm in diameter, when inhaled through the nose, are deposited in the nasal region, whereas during mouth breathing this drops to approximately 65% (U.S. EPA, 1982). In the tracheobronchial region, only particles ≤10 µm in diameter are deposited. However, owing to the bypass of the nasal cavity during oral breathing, up to 10% deposition of particles up to 15 µm in diameter can occur in the tracheobronchial region (Miller *et al.*, 1979). The deposition of particles in the pulmonary region in humans is probably the most critical with regard to the health effects associated with particulate matter, since this appears to be the target tissue. Lung deposition in this region is highest for small particles of submicrometre size and is markedly reduced for particles of 2 µm and above (ICRP, 1994). Churg and Brauer (1997) examined the upper lobe apical segment parenchyma of autopsy lung tissue for long-term residents of Vancouver, British Columbia, using analytical electron microscopy and found that 96% of the particles deposited had aerodynamic diameters less than 2.5 µm.

While deposition of fine particles on a mass per unit alveolar surface area is not different between rats and humans, based on the calculations per ventilatory unit or per alveolus, humans receive much greater numbers of particles than do rats when exposed to the same mass concentration of particulate matter, particularly for particles 0.1–0.3 µm in size. This difference

has been observed to be even more pronounced for individuals with compromised lungs (smokers and patients with respiratory diseases) than for normal subjects (Kim and Kang, 1997). It was estimated that rats exposed to 1–1.5 mg particles/m^3 may actually have received a level of particles equivalent to 120–150 µg/m^3 in humans (Miller *et al.*, 1995).

Clearance of particles from the extrathoracic region occurs by mechanical processes; in the nasal area, by blowing, wiping or sneezing; and in the more anterior regions, either through swallowing (in mucus) or by expectorating. Clearance from the extrathoracic region may take up to one or more days in humans (Proctor and Wagner, 1965). In the conducting airways of the tracheobronchial region, the most prominent mechanism for elimination of particles is via the action of the mucociliary escalator into the gastrointestinal tract. This is a very fast clearance pathway, which is mainly completed within 24 hours after deposition of particles in this region. The half-time of clearance in humans ranges from half an hour in the larger airways to five hours in the smaller airways (U.S. EPA, 1982), although there is some evidence of a long-term component to tracheobronchial retention (approximately 500 days) (Stahlofen *et al.*, 1986a,b; Smaldone *et al.*, 1988). In the pulmonary region (the site of effects in the epidemiological studies), insoluble particles are rapidly cleared through phagocytosis by alveolar macrophages. Following phagocytosis, the macrophages can migrate such that they are cleared via mucociliary flow; however, particle-laden alveolar macrophages are found in the lung up to many hundreds of days post-exposure, indicating that some macrophages do not migrate and that particles are reingested by other generations of alveolar macrophages. Particles may also enter the interstitium via endocytosis by alveolar epithelial cells, specifically Type I cells. Ultrafine particles (<~50 nm) have a much greater propensity to penetrate into the pulmonary interstitium and escape phagocytosis by alveolar macrophages (Ferin *et al.*, 1991).

3.0 ASSESSMENT OF "TOXIC" UNDER CEPA 1999

3.1 CEPA 1999 64(c): Human health

3.1.1 Exposure

Data on particulate matter in Canada are almost entirely from fixed-site monitoring of 24-hour concentrations in ambient air. Within the national network, long-term mean PM_{10} concentrations during the mid-1980s to mid-1990s ranged from 11 to 42 µg/m^3 at urban sites and during the mid-1990s ranged from 11 to 17 µg/m^3 at rural sites. The corresponding values for $PM_{2.5}$ were 6.9–20.2 µg/m^3 and 7.0–10.5 µg/m^3, respectively. The values for PM_{10} and $PM_{2.5}$ are above estimated background levels, indicating that anthropogenic activities make an important contribution to ambient particulate matter loadings. On a national scale, average particulate matter concentrations decreased approximately 2–3% annually between 1984 and 1995.

The distribution of ambient particulate matter data at a given site is typically strongly skewed, with a large number of low values and relatively few greater ones. Short-term levels can be substantially greater than mean values; for example, the 90th percentile of 24-hour concentrations from the various sites in the national network is approximately twice the mean. Typically, weekend concentrations are lower than those during the work week, and there are diurnal variations in particulate matter, with peaks occurring during the morning rush hour and late evening. There are relatively strong correlations between 24-hour concentrations of PM_{10} and $PM_{2.5}$ at most sites in the national network, consistent with the belief that the daily variations in PM_{10} generally reflect fluctuations in fine particles, rather than coarse particles.

There has been much discussion of the adequacy of particulate matter levels measured at fixed-area monitors as surrogates for human exposure to particulate matter. While mean personal air levels of fine particles are generally poorly correlated with ambient monitoring data collected at the same time, the correlations are stronger when individual (longitudinal) regressions of personal exposure with the nearest outdoor site are calculated and when the mean of the personal exposures from a number of studies is related to the fixed ambient monitoring. Results from studies in the United States have also indicated that outdoor air is the largest source of indoor PM_{10} or $PM_{2.5}$, even in homes where tobacco smoking or cooking (the two main identified indoor sources of particles) took place. This should also be the case in Canada, although "tighter" building construction will lower the contribution from this source in winter. Together, these considerations suggest that ambient fine particles measured at a fixed point in the community will be related to exposure, particularly for individuals who are not exposed to important indoor sources of particles such as smoking, and that such monitoring can serve as an adequate surrogate for community (population) exposure.

3.1.2 Effects

The strongest evidence demonstrating an association between particulate matter and cardiorespiratory illness is provided by the mass of epidemiological data. These point to a "pyramid of effects" headed by increases in mortality due to cardiorespiratory diseases, increases in hospitalizations for cardiorespiratory diseases, decreases in lung function in children and in asthmatic adults, increases in respiratory symptoms, which can lead to increases in respiratory-related activity restrictions and days lost from work or school, and long-term or chronic effects, including reduced survival, reduced lung function and capacity in children, and increases in the development of chronic bronchitis and asthma in some adults (Table 10).

Table 10 Summary of adverse health effects associated with particulate matter (epidemiological studies)

Endpoints	% increase of risk per 10 µg/m³ of PM_{10} [average concentrations (µg/m³)]	% increase of risk per 10 µg/m³ of $PM_{2.5}$ [average concentrations (µg/m³)]	% increase of risk per 10 µg/m³ of sulphate [average concentrations (µg/m³)]
Acute increase in mortality	0.8% (unweighted); 0.5% (weighted) [18–115 µg/m³]	1.5% [11.2–21 µg/m³]	2.2% [5.8–8.7 µg/m³]
Acute increase in respiratory hospitalizations and emergency department visits	0.35–7.3% [25–55 µg/m³]	3.3–9.6% [16.8 µg/m³]	0.8–18.2% [4.4–11.8 µg/m³]
Acute increase in cardiovascular hospitalizations	0.56–1% [48 µg/m³]	1% [15.3 µg/m³]	2.5% [4.4 µg/m³]
Acute pulmonary function decrements	0.09–0.4% [10–174 µg/m³]	No data	1.2%
Acute increase in symptoms	0.6–2.2% [10–174 µg/m³]	0.9–2.2% [18–22 µg/m³]	4.6–16.4% [2.5 µg/m³]
Acute increase in respiratory symptom-related activity restriction	9.0% [41–51 µg/m³]	12.9–15.8% [20–25 µg/m³]	17.4% [2.1 µg/m³]
Long-term increase in mortality	10% from cohort studies [18–47 µg/m³]	7–14% from cohort studies; 4.3–9.8% increase in odds from cross-sectional studies [11–30 µg/m³]	7.5–32% from cohort studies; 8.2–12.6% increase in odds from cross-sectional studies [4.8–13 µg/m³]
Long-term pulmonary function decrements	1.4% increase in odds from cross-sectional studies [24–58 µg/m³]	2.2% increase in odds from cross-sectional studies [8–46 µg/m³]	4.5% increase in odds from cross-sectional studies [4.7–13 µg/m³]
Long-term increase in symptoms	From non-significant to 39% increase in odds from cross-sectional studies [20–59 µg/m³]	18% from a cohort study; from non-significant to 4.3% increase in odds from cross-sectional studies [11.8–37 µg/m³]	From non-significant to 9.3% increase in odds from cross-sectional studies [3.2–14 µg/m³]
Increase in lung cancer	No data	19.9% from cohort studies [11–29.6 µg/m³]	18.1% from cohort studies [3.6–23.5 µg/m³]

Although the epidemiological studies are observational rather than experimental, they have been weighted more heavily than the toxicological or controlled human exposure studies for several reasons: (1) they are the most direct way of assessing the adverse health outcomes of "real-world" complex mixtures of pollutants to which people are exposed; (2) human populations, unlike laboratory animals, are highly heterogeneous, including individuals who encompass a large range of susceptibilities, disease status and exposures and whose responses cannot be predicted from animal toxicology studies or are not available from controlled human exposure studies due to ethical reasons; and (3) no extrapolation is necessary when assessing the effects on public health of a particular concentration of air pollutant or of an ambient air objective, as measured by the ambient compliance monitoring network, despite our lack of knowledge about the exposures of each individual in the population.

The results of the time-series epidemiological studies of air pollution and health need to be interpreted within the context of mechanisms of disease pathogenesis, relevant toxicological evidence and the findings of analytical epidemiological studies such as cohort studies. The results of these investigations (i.e., the association between ambient exposure to particulate matter and cardiorespiratory mortality and morbidity) are considered, therefore, in the context of traditional criteria for causality for epidemiological studies.

3.1.2.1 Consistency

The results of the time-series studies of mortality have been remarkably consistent in indicating a positive association between PM_{10} and $PM_{2.5}$ pollution and daily mortality. These associations were seen in 43 analyses in 20 cities across North America, South America, England and Europe. The Canadian and U.S. cities included in the analyses range from large metropolitan areas, such as Toronto and New York City, to smaller cities with polluting industry, such as Steubenville and Utah Valley. The estimates of the risks have been similar, in spite of the potentials of misclassification of personal exposure to ambient particles, different combinations of co-pollutants and different health care systems. While most of the studies used the time-series analysis, widely differing analytical applications were employed, with similar results. The results of two long-term cohort studies indicated similar trends for PM_{10}- and $PM_{2.5}$-related mortality (Dockery *et al.*, 1993; Pope *et al.*, 1995).

With respect to studies of morbidity, diverse study designs have been used, including time-series studies, cross-sectional studies, short-term cohort or "panel" studies and longer-term cohort studies. The array of health outcomes considered in these studies is equally diverse. Significant association of PM_{10} and $PM_{2.5}$ with respiratory hospitalizations was demonstrated in most time-series studies examined. Associations between particulate matter pollution, including PM_{10}, $PM_{2.5}$ and sulphate, and increased hospital admissions due to CVD have also been reported in Canada and the United States. Regression modelling indicates adverse effects of particle exposure on lung function in children and adults, respiratory symptoms and medication use, restricted activity days and frequency of reported chronic respiratory disease.

Thus, epidemiological studies of morbidity and mortality have provided consistent evidence of an association between exposure to particulate matter and several of these critical health outcomes, in areas with different pollutant sources, different combinations of co-pollutants and different health care systems.

3.1.2.2 Strength of association

The increased RRs for mortality and morbidity are summarized in Table 10. The magnitudes of the estimates of increased risks are seemingly small, although they represent a substantial impact on public health. Thus, the magnitude of the associations is relatively weak, but statistically significant.

3.1.2.3 Dose–response relationship

Responses increased monotonically from very low ambient concentrations of particulate matter up to much higher levels with remarkable consistency in many epidemiological studies on acute and chronic mortality (Pope *et al.*, 1992, 1995; Dockery *et al.*, 1993; Schwartz, 1993; Saldiva *et al.*, 1995; Ostro *et al.*, 1996; Pope and Kalkstein, 1996) and hospitalizations (Sunyer *et al.*, 1993; Burnett *et al.*, 1994, 1997; Schwartz, 1994a,b,c; Castellsague *et al.*, 1995; Schwartz and Morris, 1995). The dose–response curve for mortality and morbidity versus concentrations of PM_{10} and fine fractions ($PM_{2.5}$ and sulphate) appears to be linear in the majority of mortality and hospitalization analyses based on PM_{10} concentration in locations including St. Louis, Missouri, six eastern and central U.S. cities, the Utah Valley and Sao Paulo, Brazil. This linear relationship is supported by studies utilizing TSP as the metric in Steubenville, Ohio, Philadelphia, Pennsylvania, and Detroit, Michigan. Non-parametric smoothing techniques applied to data from several of these locations, at least in the lower exposure range, for mortality in Philadelphia and the Utah Valley and for hospitalizations in Toronto, Ontario, Birmingham, Alabama, New Haven, Connecticut, Tacoma, Washington and Spokane, Washington, have generally confirmed the approximately monotonic dose–response.

The data from several studies are consistent with curvilinear models. In several European locations that included PM_{10} and $PM_{2.5}$ metrics, the dose–response curve showed a steeper linear component at lower concentrations and a slight flattening (lower slope) at high concentrations. This curvilinear response was seen in mortality studies from Amsterdam, Netherlands, for both Black Smoke and PM_{10} (Verhoeff *et al.*, 1996) and from Athens, Greece, for Black Smoke (Touloumi *et al.*, 1994). In the study by Burnett *et al.* (1995) on the association between sulphates and respiratory hospitalizations in southern Ontario, the decile curve appears to be slightly curvilinear and rises monotonically from 0 to 20 µg sulphate/m^3 (lagged one day), with a slightly reduced slope at the higher concentrations above about 8–10 µg/m^3.

In a reanalysis (by quintiles) and extension of the Utah Valley mortality data by two years, Lyon *et al.* (1995) suggested a threshold for PM_{10} effects at a concentration of 50 µg/m^3. However, this analysis was carried out by subdivision of the data by year, season, age, location and cause of death and by dichotomizing PM_{10} data at a 50 µg/m^3 cutoff, resulting in substantial losses of PM_{10} information and statistical power.

Thus, a dose–response relationship has consistently been observed, with risks increased even at very low ambient concentrations of particulate matter.

3.1.2.4 Coherence

A "pyramid of effects" of varying severity of outcome has been observed in the epidemiological studies. Particulate matter has been associated in many studies with cardiorespiratory mortality. One would expect that hospital admissions would also be elevated, and to a greater degree than mortality, since not all affected people would die. Similarly, emergency department and doctors' visits, respiratory symptoms, lung function, respiratory-related reduced activity and days absent from work or school due to respiratory illnesses would be expected to be elevated. All of these have in fact been observed, providing a strongly coherent picture (Table 10). The slopes of the response curves also tend to increase as the effects go down the scale of seriousness on the "pyramid of effects," which adds to the evidence for coherence.

A strong pattern of coherence between endpoints is hence provided both qualitatively and quantitatively by the associations shown between particulate matter and a broad range of endpoints from the least to the most serious, i.e., mortality.

3.1.2.5 Temporal relationship

In many epidemiological studies, the lag time between pollution peaks and onset of effects was investigated. In some, "reverse lag," or effects several days prior to the episode, were analysed to ensure that the time sequence was correct. Daily peaks of particulate matter were followed within 24 hours to several days by mortality, hospitalizations, lung function changes and respiratory symptoms. Thus, the criterion of temporality was satisfied by the study results. However, for mortality, this lag period was surprisingly short, being less than 24 hours in a number of studies. This has created some difficulties in trying to explain what mechanism could be responsible for these sudden deaths, since not enough time would have elapsed for sufficient tissue damage to occur to account for mortality or for infections to have progressed to this stage so rapidly, unless there was an acute coronary artery spasm and a subsequent massive myocardial infarction or a malignant arrhythmia. Alternatively, this short lag time might be due to the susceptibility of certain subpopulations whose health had already been compromised by cardiorespiratory diseases and who were particularly vulnerable to environmental changes.

The time pattern of exposure and effect adds to the weight of evidence for causality, with the exception of the rapidity of the effects on mortality.

3.1.2.6 Specificity

Evidence shows that particulate pollution-related increases in mortality and hospitalizations were associated with cardiorespiratory causes, but not with other diseases (Thurston *et al.*, 1994; Burnett *et al.*, 1995; Schwartz and Morris, 1995).

With respect to specificity of the agent, possible confounders such as temperature, weather, season and (in some studies) epidemics of influenza were controlled for in the analyses, and it seems unlikely that such factors could be responsible for the associations of particulate matter and mortality in such a wide range of locales. The evidence for particulate matter as opposed to certain gaseous pollutants is strong in the majority of studies, although a broad range of air pollutants has been examined in only a few studies. On the other hand, the associations have been observed in a wide range of locations with differing mixtures of air pollutants and have been quite consistently positive for particulate matter. In analyses designed to help separate out effects of one pollutant from another, such as bivariate or multivariate regressions, the associations of PM_{10}, $PM_{2.5}$ and sulphate with adverse health outcomes were remarkably robust to inclusion (one at a time) of all four gaseous air pollutants (sulphur dioxide, nitrogen dioxide, carbon monoxide and ozone). Moreover, the magnitude of this association was often (but not always) greater than that for any of the gaseous pollutants individually or combined.

The evidence is considered to be strong regarding the specificity of effect for respiratory and cardiac outcomes. With respect to specificity of cause, the evidence is harder to judge, but, where possible confounding factors have been examined, they have not explained the observed excesses of adverse health outcomes.

3.1.2.7 Biological plausibility

When evaluating the effects of low-level ambient particles, acute adverse effects — i.e., mortality and morbidity — correlated with daily changes in ambient levels need to be clearly distinguished from chronic effects that are associated with long-term levels of particulate pollution. The association of mortality with daily variations in particulate air pollution presents difficulties in establishing a plausible mechanism that could explain these associations, because of the very short lag period, or in some cases no lag, between recording of elevated particle concentrations and the occurrence of increased mortality. Several hypotheses have been proposed for acute particle-related mortality, including: (1) exacerbation of severe asthma or COPD; (2) progression of an acute respiratory infection; (3) worsening of pulmonary edema, due perhaps to either a

permeability defect or left ventricular dysfunction; and (4) malignant cardiac arrhythmias.

The clinical data lend only limited support to the results of the epidemiological studies. Controlled human exposure studies have shown that asthmatic individuals, especially asthmatic children and adolescents, are responsive to acidic aerosols at concentrations close to ambient levels (~35 µg sulphuric acid/m^3 for 40 minutes) (Koenig *et al.*, 1989, 1992; Hanley *et al.*, 1992). However, there is no conclusive evidence of enhanced responsiveness in other susceptible groups identified in the epidemiological studies (the elderly or individuals with COPD) and little or no support for the observation that daily fluctuations of ambient particulate concentrations are associated with mortality within hours or a few days at most. The discrepancy between clinical and epidemiological findings may be due to one or more of the following: for practical and ethical reasons, the experimental subjects can be exposed to the tested air pollutants only for shorter durations than pollution episodes, and the studies cannot include those people most likely to be affected by air pollutants, such as cardiovascular patients; the pulmonary function parameters that are most often used in clinical studies may not be sensitive enough to indicate particulate matter-induced adverse health effects; artificial particles used in exposure chambers may not reflect the potential synergistic effects of ambient particulate matter and aerosol mixtures; and in most human studies, the sizes of aerosols used are above 0.5 µm, whereas nanometre-sized ultrafine particles have been found in animal studies to induce acute pulmonary inflammation and death at very low concentrations (Oberdörster *et al.*, 1994a).

Experimental animal studies thus far have not corroborated the epidemiological findings of an association between 24-hour ambient levels of PM_{10} and $PM_{2.5}$ below 100 µg/m^3 and mortality, but this may be the result of dosimetric differences between animals and humans. The lowest concentration of $PM_{2.5}$ reported that caused death in rats with acute pulmonary inflammation or chronic bronchitis was 250 µg/m^3 (three days, six hours per day) (Godleski *et al.*, 1996). Based on a recent comparative dosimetric analysis conducted by Miller *et al.* (1995), it was estimated that humans receive approximately a 10-fold higher number of ultrafine particles than do rats exposed to the same mass concentration, when calculated per ventilatory unit or per alveolus. This difference has been observed to be even more pronounced for individuals with compromised lungs (smokers and patients with respiratory diseases) than for normal subjects (Kim and Kang, 1997). Based on this analysis, the results of studies of animals exposed to several hundred µg particulate matter/m^3 appear relevant to ambient exposures for the general population.

Several studies have shown that bronchial hypersensitivity to non-specific stimuli, morbidity and mortality are most likely to occur in animals with pre-existing cardiorespiratory diseases (Slauson *et al.*, 1989; Raabe *et al.*, 1994; Gilmour *et al.*, 1997; Killingsworth *et al.*, 1997), providing further support for the epidemiological findings.

Based on data from toxicological studies in animals, the epidemiological observations of rapid effects of low ambient particle concentrations in cardiorespiratory disease may be attributable to ultrafine particles. The urban particulate cloud may contain up to millions of nanometre-sized particles per millilitre, with a gravimetric concentration of only 100–200 µg/m^3. The particle surface area is therefore greatly enlarged and has been shown to be capable of carrying adsorbed metals, acids and toxic organic molecules down to the deep recesses of the lung. Pulmonary inflammation and mortality in animals have been observed at near-ambient concentrations of ultrafine particles — i.e., 9–60 µg/m^3 (Warheit *et al.*, 1990; Chen *et al.*, 1992b; Oberdörster *et al.*, 1995).

The available data from animal toxicological and controlled human clinical studies have implied a mechanistic basis for PM_{10}- and $PM_{2.5}$-induced cardiorespiratory injury —

namely, by altering the airway immune system and/or causing epithelial cell damage, resulting in respiratory diseases. The mechanism for the cardiovascular effects is not clear but may be the result of lipoprotein peroxidation and modifying blood coagulation, resulting in cardiovascular abnormalities. The effects are most frequently observed in individuals with a compromised cardiorespiratory system.

In summary, available data provide some, although weak, support to satisfy the criterion of biological plausibility.

3.1.3 Human health risk characterization

Based on the weight of evidence presented in this section, the epidemiological evidence for mortality and morbidity in response to current levels of particulate air pollution meets a number of the criteria for causality, including consistency, dose–response relationship, coherence, temporal relationship and specificity (of both outcome and agent). With respect to the biological plausibility of the association, the results of animal studies and, to a lesser extent, controlled human studies provide support for the target tissues and susceptible populations and preliminary indications of possible mechanisms. Although ambient levels of particulate matter in Canada have been decreasing over time, there are clear indications of adverse health effects based on the results of very recent studies in Canada and in other countries at ambient levels similar to those currently occurring in Canada. The database supports, therefore, a causal relation between current ambient PM_{10} and $PM_{2.5}$ exposure and adverse health effects and provides a reasonable basis for preventive action.

The epidemiological evidence is suggestive that adverse health effects occur only in a susceptible subset of the general population. This group appears to include those with pre-existing respiratory or cardiovascular conditions, a group that forms a substantial fraction of the general population. That this group comprises the responders to particulate matter is supported by the results of recent laboratory experiments using appropriate animal models such as bronchitic rats.

There is no clear evidence of a level below which the positive associations between PM_{10} (and probably $PM_{2.5}$ too) and both daily mortality and hospitalization rates are not observed. That is, any increase in ambient particulate matter is associated with a statistical increase in mortality and hospitalization rates. While mortality and hospitalization rates have been emphasized owing to their adequacy as a basis for a quantitative measure of risk, other adverse health effects have also been observed, including exacerbation of respiratory symptoms such as bronchitis and asthma, reduced lung function, restricted activity due to illness, loss of work-days or school-days and increased costs for medication. Effects of particulate matter on respiratory health (lung function, symptoms and absenteeism), although much less serious than hospitalizations and mortality, have the potential to affect much more of the population.

These particulate matter-related adverse health effects are observed at concentrations currently occurring in Canada. The results were highly consistent under the widely varying climatic exposure conditions and pollutant mixtures encountered in the different locations. While the increases in RR are of small magnitude, they signify substantial numbers of deaths due to the very large size of the populations that are impacted by air pollution.

3.1.4 Uncertainties and degree of confidence in the human health risk characterization

There is a fair degree of uncertainty in the exposure assessment for particulate matter. Even though the monitoring data are extensive and national in scope and have been collected using appropriate methods, virtually all of the data are for 24-hour ambient concentrations of particulate matter collected at fixed sites. Hence, there is a lack of individual exposure data; this may be particularly important, since it is likely that the

susceptible subpopulations (i.e., those with pre-existing cardiorespiratory diseases) are relatively inactive and spend more time indoors than average. In addition, the ambient monitoring network does not provide information on diurnal fluctuations in particulate matter, and it is possible that the health effects observed may be the result of peak exposures, rather than those averaged over the monitoring period. There are also no monitoring data for ultrafine particles in Canada, and the results of some studies in animals and humans have suggested that this fraction of the fine particles is extremely toxic. The overall degree of confidence in the exposure assessment is, therefore, moderate, owing principally to the lack of information on personal exposure to particulate matter.

There is more certainty in the effects characterization for particulate matter. As outlined in Section 3.1.2, statistically significant and concentration-related associations between ambient concentrations of particulate matter and a "pyramid" of related cardiorespiratory health effects, including mortality, have been remarkably consistently observed in the available epidemiological studies. These have been observed at ambient concentrations similar to those currently occurring in Canada, obviating the need to extrapolate the results of these studies in assessing the health risks posed by particulate matter. The results of controlled exposures of humans and animals to particulate matter have provided support, albeit limited, for the epidemiological findings in terms of target tissues, susceptible populations and plausible mechanisms of action.

However, there are still some important uncertainties in the available effects-related data. There is concern for possible confounding from exposure to other co-occurring (and often highly correlated) pollutants, in which case the increased risk could be ascribed to the wrong agent (although, as discussed in Section 3.1.2, the weight of evidence suggests that particulate matter is the best indicator for effects of air pollution on health outcomes, and measures to reduce exposure to particulate matter, particularly $PM_{2.5}$, would also reduce exposure to these other pollutants). The strength of the association is also weak, although fairly consistent, particularly with respect to mortality. There are no epidemiological studies that have investigated health outcomes in relation to exposure to ultrafine particles or to personal exposures, and there are few epidemiological data on the health effects of long-term exposure to particulate matter. The available controlled studies of humans exposed to particulate matter are quite limited; there are no studies of cardiovascular outcomes, and sensitive biomarkers of effect have not been identified. With respect to studies in animals, there are few dosimetric data to account for differences in responses observed in animals and humans, and the modes of action for particulate matter-related health effects have still not been elucidated, although there are emerging data from both areas that support the biological plausibility of the epidemiological observations. Overall, the degree of confidence in the effects characterization is considered to be moderate to high, owing principally to the limitations in the available epidemiological studies, although it is noted that this database is far more extensive than is generally the case for environmental pollutants.

3.2 Conclusion

Based principally on the sufficient weight of evidence of mortality and morbidity in the general population exposed to ambient concentrations of PM_{10} and $PM_{2.5}$ examined in recent extensive epidemiological analyses in Canada and in other countries (at ambient concentrations currently occurring in Canada), as well as on some limited supporting data in experimental animal and controlled human exposure studies, PM_{10} and particularly $PM_{2.5}$ are considered to be entering the environment in a quantity or concentration or under conditions that constitute a danger in Canada to human life or health. On this basis, PM_{10} and particularly $PM_{2.5}$ are considered to be "toxic" as defined in Section 64 of CEPA 1999.

3.3 Considerations for follow-up (further action)

The robustness and consistency of the association between respirable particulate matter and adverse health effects across so many locations with differing air pollutant mixtures support the position that PM_{10}, $PM_{2.5}$ and sulphate are the best indicators for the effects of air pollution on adverse health outcomes. In the available time-series analyses, the fine fraction of particulate matter ($PM_{2.5}$) was consistently associated with adverse health effects; moreover, the association was usually of greater magnitude than those with other particle metrics, including PM_{10}, in studies that included several measures of exposure. In a few studies (Dockery *et al.*, 1992; Schwartz *et al.*, 1996; Burnett *et al.*, 1997) in which associations with the coarse fraction of PM_{10} in addition to PM_{10} and/or $PM_{2.5}$ were examined, the coarse fraction was often not associated with adverse health outcomes, while the fine fraction and often the total PM_{10} fraction were. Moreover, approximately 25–60% of $PM_{2.5}$ can be deposited in the human alveolar region, compared with <5% of larger particles (~10 µm) (Lippmann, 1977), which may render the fine fraction more harmful in causing lung injury.

While sulphate has been used as a surrogate for $PM_{2.5}$ in locations where data on concentrations of $PM_{2.5}$ were not available, the magnitude of the sulphate-related association was not as great as that of total $PM_{2.5}$ when both were available in the same location. In addition, sulphate appears to be too area-specific to be used as a general metric for regulatory purposes.

Thus, based on available data on health effects of particulate matter, investigations of options to reduce exposure to particulate matter should be focussed on the fine fraction ($PM_{2.5}$). (However, it is noted that coarse particles [$PM_{10-2.5}$] have not yet been eliminated from consideration, as there is some indication that they may play a role in respiratory and/or cardiovascular disease.) The investigation of management options should also be designed to reduce mid-range (24-hour average) rather than peak exposures (i.e., <24-hour periods), since, on the basis of available data, 24-hour average exposure is associated with increases in mortality and morbidity.

The fine fraction of particulate matter (which can remain in the atmosphere for days to weeks) is composed of organic compounds and secondary sulphates and nitrates. In urban areas, these compounds and their precursor gases (sulphur oxides, nitrogen oxides and VOCs) originate typically from combustion processes — motor vehicles, industrial processes and vegetative burning; 30–82% of $PM_{2.5}$ is estimated to be generated locally. In contrast, the coarse fraction of PM_{10} (particles >2.5 µm but ≤10 µm) is typically associated with mechanical processes, such as wind erosion, breaking ocean waves and grinding operations. These coarse particles, which are efficiently removed by gravitational settling, remain in the atmosphere for shorter periods of a few hours to a few days. Further detail is available in Appendix G of the National Ambient Air Quality Objectives for Particulate Matter, Addendum to the Science Assessment Document (WGAQOG, 1999).

The available data clearly indicate that relative source contributions to PM_{10} and $PM_{2.5}$ vary by province/territory and by region, and there are ongoing initiatives in risk management designed to accommodate these regional variations. Under the Canada-wide Standards subagreement of the Harmonization Accord signed by the Environment Ministers in January 1998, federal and provincial/territorial governments will develop numerical air quality standards for PM_{10} and $PM_{2.5}$, with each jurisdiction developing a plan of action to achieve the standards in a specific time frame. Any investigations of options to reduce exposure as a result of the assessment of particulate matter as a Priority Substance under CEPA 1999 will complement those for this ongoing initiative.

4.0 REFERENCES

Abbey, D.E., B.E. Ostro, F. Petersen and R.J. Burchette. 1995a. Chronic respiratory symptoms associated with estimated long-term ambient concentrations of fine particulates less than 2.5 microns in aerodynamic diameter ($PM_{2.5}$) and other air pollutants. J. Expos. Anal. Environ. Epidemiol. 5(2): 137–159.

Abbey, D.E., M.D. Lebowitz, P.K. Mills, F.F. Petersen, W.L. Beeson and R.J. Burchette. 1995b. Long-term ambient concentrations of particulates and oxidants and development of chronic disease in a cohort of nonsmoking California residents. Inhal. Toxicol. 7: 19–34.

Abe, Y., M. Kawakami, M. Kuroki, T. Yamamoto, M. Fujii, H. Kobayashi, T. Yaginuma, A. Kashii, M. Saito and K. Matsushima. 1993. Transient rise in serum interleukin-8 concentration during acute myocardial infarction. Br. Heart J. 70: 132–134.

Adamson, I.Y.R. and D.H. Bowden. 1981. Dose response of the pulmonary macrophagic system to various particulates and its relationship to transepithelial passage of free particles. Exp. Lung Res. 2: 165–175.

Anderson, H.R., A. Ponce de Leon, J.M. Bland, J.S. Bower and D.P. Strachan. 1996. Air pollution and daily mortality in London: 1987–92. Br. Med. J. 312: 665–669.

Anderson, K.A., E.L. Avol, S.A. Edward, D.A. Shamoo, R.-C. Pang, W.S. Linn and J.D. Hackney. 1992. Controlled exposures of volunteers to respirable carbon and sulfuric acid aerosols. J. Air Waste Manage. Assoc. 42: 770–776.

Anderson, P.J., J.D. Wilson and F.C. Hiller. 1990. Respiratory tract deposition of ultrafine particles in subjects with obstructive or restrictive lung disease. Chest 97: 1115–1120.

Aris, R., D. Christian, I. Tager, L. Ngo, W.E. Finkbeiner and J.R. Balmes. 1993. Effects of nitric acid gas alone or in combination with ozone on healthy volunteers. Am. Rev. Respir. Dis. 148: 965–973.

Bahadori, T., H. Suh and P. Koutrakis. 1995. Personal, indoor, and outdoor concentrations of PM_{10} and $PM_{2.5}$ for ten COPD patients living at home. In preparation [cited in Wallace, 1996].

Ballester, F., D. Corella, S. Perez-Hoyos and A. Hervas. 1996. Air pollution and mortality in Valencia, Spain: a study using the APHEA methodology. J. Epidemiol. Commun. Health 50: 527–533.

Begin, R., A. Dufresne, A. Cantin, F. Possmayer and P. Sebastien. 1989. Quartz exposure, retention, and early silicosis in sheep. Exp. Lung Res. 15: 409–428.

Berg, I., T. Schluter and G. Gercken. 1993. Increase of bovine alveolar macrophage superoxide anion and hydrogen peroxide release by dusts of different origin. J. Toxicol. Environ. Health 39: 341–354.

Berliner, J.A., M.C. Territo, A. Sevanian, S. Ramin, J.A. Kim, B. Bamshad, M. Esterson and A.M. Fogelman. 1990. Minimally modified low density lipoprotein stimulates monocyte endothelial interactions. J. Clin. Invest. 85: 1260–1266.

Brain, J.D. and G.C. Cockery. 1977. The effect of increased particles on the endocytosis of radiocolloids by pulmonary macrophages *in vivo*: Competitive and toxic effects. *In*: W.H. Walton (ed.), Inhaled particles and vapours. Vol. IV. Pergamon Press, London. pp. 551–564.

Brook, J.R. 1997. Personal communication. Atmospheric Environment Service, Environment Canada, Downsview, Ontario. February.

Brook, J.R., T.F. Dann and R. Burnett. 1997. The relationship among TSP, PM_{10}, $PM_{2.5}$ and inorganic constituents of atmospheric particulate matter at multiple Canadian locations. J. Air Waste Manage. Assoc. 47: 2–19.

Burnett, R.T., R.E. Dales, M.E. Raizenne, D. Krewski, P.W. Summer, G.R. Roberts, M. Raad-Young, T. Dann and J. Brook. 1994. Effects of low ambient levels of ozone and sulfates on the frequency of respiratory admissions to Ontario hospitals. Environ. Res. 65: 172–194.

Burnett, R.T., R.E. Dales, D. Krewski, R. Vincent, T. Dann and J.R. Brook. 1995. Associations between ambient particulate sulfate and admissions to Ontario hospitals for cardiac and respiratory diseases. Am. J. Epidemiol. 142(1): 15–22.

Burnett, R.T., J.R. Brook, S. Cakmak and D. Krewski. 1997. The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. Environ. Health Perspect. 105: 614–620.

Callis, A.H., P.G. Sohnle, G.S. Mandel, J. Wiessner and N.S. Mandel. 1985. Kinetics of inflammatory and fibrotic pulmonary changes in a murine model of silicosis. J. Lab. Clin. Med. 105: 547–553.

Campen, M.J., W.P. Watkinson, J.R. Lehmann and D.L. Costa. 1996. Modulation of residual oil fly ash (ROFA) particle toxicity in rats by pulmonary hypertension and ambient temperature (T_a) change. Am. J. Respir. Crit. Care Med. 153: A542.

Carter, J.D., A.J. Ghio, J.M. Samet and R.B. Devlin. 1997. Cytokine production by human airway epithelial cells after exposure to an air pollution particle is metal-dependent. Toxicol. Appl. Pharmacol. 146: 180–188.

Castellsague, J., J. Sunyer, M. Saez and J.M. Anto. 1995. Short-term association between air pollution and emergency room visits for asthma in Barcelona. Thorax 50: 1051–1056.

Chen, L.C. and R.B. Schlesinger. 1983. Response of the bronchial mucociliary clearance system in rabbits to inhaled sulfite and sulfuric acid aerosols. Toxicol. Appl. Pharmacol. 71: 123–131.

Chen, L.C., P.D. Miller and M.O. Amdur. 1989. Effects of sulfur oxides on eicosanoids. J. Toxicol. Environ. Health 28: 99–109.

Chen, L.C., H.F. Lam, E.J. Kim, J. Guty and M.O. Amdur. 1990. Pulmonary effects of ultrafine coal fly ash inhaled by guinea pigs. J. Toxicol. Environ. Health 29: 169–184.

Chen, L.C., P.D. Miller, H.F. Lam, J. Guty and M.O. Amdur. 1991a. Sulfuric acid-layered ultrafine particles potentiate ozone-induced airway injury. J. Toxicol. Environ. Health 34: 337–352.

Chen, L.C., S.M. Peoples and M.O. Amdur. 1991b. Pulmonary effects of sulfur oxides on the surface of copper oxide aerosol. Am. Ind. Hyg. Assoc. J. 52: 187–191.

Chen, L.C., L.C. Armstrong, S. Liu, J.E. Gerriets and J.A. Last. 1991c. Silica increases cytosolic free calcium ion concentration of alveolar macrophages *in vitro*. Toxicol. Appl. Pharmacol. 111: 211–220.

Chen, L.C., P.D. Miller, M.O. Amdur and T. Gordon. 1992a. Airway hyper-responsiveness in guinea pigs exposed to acid-coated ultrafine particles. J. Toxicol. Environ. Health 35: 165–174.

Chen, L.C., J.M. Fine, Q.S. Qu, M.O. Amdur and T. Gordon. 1992b. Effects of fine and ultrafine sulfuric acid aerosols in guinea pigs: Alterations in alveolar macrophage function and intracellular pH. Toxicol. Appl. Pharmacol. 113: 109–117.

Churg, A. and M. Brauer. 1997. Human lung parenchyma retains $PM_{2.5}$. Am. Rev. Crit. Care Med. 155: 2109–2111.

Clayton, C.A., R.L. Perrit, E.D. Pellizzari, K.W. Thomas, R.W. Whitmore, L.A. Wallace, H. Özkaynak and J.D. Spengler. 1993. Particle Total Exposure Assessment Methodology (PTEAM) study. Distributions of aerosol and elemental concentrations in personal, indoor, and outdoor air samples in a southern California community. J. Expos. Anal. Environ. Epidemiol. 3(2): 227–250.

Colome, S.D., N.Y. Kado, P. Jaques and M. Kleinman. 1992. Indoor–outdoor air pollution relations, particulate matter less than 10 µm in aerodynamic diameter (PM_{10}) in homes of asthmatics. Atmos. Environ. 26A: 2173–2178.

Cushing, S.D., J.A. Berliner, A.J. Valente, M.C. Territo, M. Navab, F. Parhami, R. Gerrity, C.J. Schwartz and A.M. Fogelman. 1990. Minimally modified low density lipoprotein induces monocyte chemotactic protein 1 in human endothelial cells and smooth muscle cells. Proc. Natl. Acad. Sci. U.S.A. 87: 5134–5138.

Dab, W., S. Medina, P. Quenel, Y. Le Moullec, A. Le Tertre, B. Thelot, C. Monteil, P. Lameloise, P. Pirard, I. Momas, R.A. Ferry and B. Festy. 1996. Short term respiratory health effects of ambient air pollution: Results of the APHEA project in Paris. J. Epidemiol. Commun. Health 50(Suppl. 1): S42–S46.

Dann, T. 1994. PM_{10} and $PM_{2.5}$ concentrations at Canadian urban sites, 1984–1993. Pollution Measurement Division, Environmental Protection Service, Environment Canada, Ottawa, Ontario (Report No. PMD 94-3).

Delfino, R.J., M.R. Becklake and J.A. Hanley. 1994. The relationship of urgent hospital admissions for respiratory illnesses to photochemical air pollution levels in Montreal. Environ. Res. 67: 1–19.

Delfino, R.J., A.M. Murphy-Moulton, R.T. Burnett, J.R. Brook and M.R. Becklake. 1997. Effects of air pollution on emergency room visits for respiratory illnesses in Montreal, Quebec. Am. J. Respir. Crit. Care Med. 155: 568–576.

DeMeules, J.E., F.A. Pigula, M. Mueller, S.J. Raymond and R.L. Gamelli. 1992. Tumor necrosis factor and cardiac function. J. Trauma 32: 686–692.

Deslauriers, M. 2000. Canadian emissions inventory of Criteria Air Contaminants (1995). Environmental Protection Series. Pollution Data Branch, Environmental Protection Service, Environment Canada, Hull, Quebec (in press).

Dockery, D.W. and C.A. Pope III. 1994. Acute respiratory effects of particulate air pollution. Annu. Rev. Public Health 15: 107–132.

Dockery, D.W. and J.D. Spengler. 1981. Indoor–outdoor relationships of respirable sulfates and particles. Atmos. Environ. 15: 335–343.

Dockery, D.W., F.E. Speizer, D.O. Stram, J.H. Ware, J.D. Spengler and B.G. Ferris. 1989. Effects of inhalable particles on respiratory health of children. Am. Rev. Respir. Dis. 139(3): 587–594.

Dockery, D.W., J. Schwartz and J.D. Spengler. 1992. Air pollution and daily mortality: associations with particulates and acid aerosols. Environ. Res. 59: 362–373.

Dockery, D.W., C.A. Pope, X. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris and F.E. Speizer. 1993. An association between air pollution and mortality in six U.S. cities. N. Engl. J. Med. 329(24): 1753–1759.

Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne and F.E. Speizer. 1996a. Health effects of acid aerosols on North American children: Respiratory symptoms. Environ. Health Perspect. 104: 500–505.

Dockery, D.W., G. Hoek, J. Schwartz and L.M. Neas. 1996b. Specific air pollutants and the Philadelphia mortality associations. *In*: J. Lee and R. Phelan (eds.), Proceedings of the 2nd Colloquium on Particulate Air Pollution and Human Health, December 1996, Park City, Utah. University of Utah, Salt Lake City, and University of California, Irvine. pp. 3–10.

EAG (Environmental Applications Group). 1984. Identification of sources of inhalable particulates in Canadian urban areas. Report prepared for Technical Services Branch, Environmental Protection Service, Environment Canada (Contract 52SS.KKE145-3-0527).

El-Fawal, H.A.N. and R.B. Schlesinger. 1994. Nonspecific airway hyperresponsiveness induced by inhalation exposure to sulfuric acid aerososl: An *in vitro* assessment. Toxicol. Appl. Pharmacol. 125: 70–76.

Ellakkani, M.A., Y. Alarie, D. Weyel and M.H. Karol. 1987. Chronic pulmonary effects in guinea pigs from prolonged inhalation of cotton dust. Toxicol. Appl. Pharmacol. 88: 354–369.

Environment Canada. 1994. The Canadian Acid Aerosol Measurement Program 1992–93 report. Atmospheric Environment Service, Ottawa, Ontario (CARD Report No. 94-013).

Environment Canada and Health Canada. 1999. Notice concerning the assessment of the Priority Substance respirable particulate matter less than or equal to 10 microns under the *Canadian Environmental Protection Act*. *Canada Gazette*, *Part I*, May 15, 1999. pp. 1345–1348.

Ezaki, M., M. Ikeda, I. Tomita and T. Tomita. 1994. Inhibition of EDRE release by native low-density lipoprotein from cultured porcine endothelial cells through intracellular mechanisms. J. Cardiovasc. Pharmacol. 24: 552–558.

Ferin, J., G. Oberdörster, S.C. Soderholm and R. Gelein. 1991. Pulmonary tissue access of ultrafine particles. J. Aerosol Med. 4: 57–68.

Fisher, G.L. and F.D. Wilson. 1980. The effects of coal fly ash and silica inhalation on macrophage function and progenitors. J. Reticuloendothel. Soc. 27: 513–524.

Frampton, M.W., K.Z. Voter, P.E. Morrow, N.J. Roberts, D.J. Culp, C. Cox and M.J. Utell. 1992. Sulfuric acid aerosol exposure in humans assessed by bronchoalveolar lavage. Am. Rev. Respir. Dis. 146: 626–632.

Frampton, M.W., P.E. Morrow, C. Cox, P.C. Levy, J.J. Condemi, D. Speers, F.R. Gibb and M.J. Utell. 1995. Sulfuric acid aerosol followed by ozone exposure in healthy and asthmatic subjects. Environ. Res. 69: 1–14.

Gearhart, J.M. and R.B. Schlesinger. 1986. Sulfuric acid-induced airway hyperresponsiveness. Fundam. Appl. Toxicol. 7: 681–689.

Gearhart, J.M. and R.B. Schlesinger. 1989. Sulfuric acid-induced changes in the physiology and structure of the tracheobronchial airways. Environ. Health Perspect. 79: 127–137.

Gilmour, M.I., F.G.R. Taylor, A. Baskerville and C.M. Wathes. 1989a. The effect of titanium dioxide inhalation on the pulmonary clearance of *Pasteurella haemolytica* in the mouse. Environ. Res. 50: 157–172.

Gilmour, M.I., F.G.R. Taylor and C.M. Wathes. 1989b. Pulmonary clearance of *Pasteurella haemolytica* and immune responses in mice following exposure to titanium dioxide. Environ. Res. 50: 184–194.

Gilmour, M.I., M.J. Daniels, D. Winsett, D.L. Costa and M.J.K. Selgrade. 1997. Oil flyash exposure enhances airway responsiveness and pulmonary inflammation to influenza virus infection in rats. Toxicologist 36: 7.

Godleski, J.J., C. Sioutas, M. Katler and P. Koutrakis. 1996. Death from inhalation of concentrated ambient air particles in animal models of pulmonary disease. Am. J. Respir. Crit. Care Med. 153: A15.

Gordian, M.E., H. Özkaynak, J. Xue, S.S. Morris and J.D. Spengler. 1996. Particulate air pollution and respiratory disease in Anchorage, Alaska. Environ. Health Perspect. 104: 290–297.

Grose, E.C., M.A. Grady, J.W. Illing, M.J. Daniels, M.K. Selgrade and G.E. Hatch. 1985. Inhalation studies of Mt. St. Helens volcanic ash. III. Host defense mechanisms. Environ. Res. 37: 84–92.

Guilianelli, C., A. Baeza-Squiban, E. Boisvieux-Ulrich, O. Houcine, R. Zalma, C. Guennou, H. Pezerat and F. Marano. 1993. Effect of mineral particles containing iron on primary cultures of rabbit tracheal epithelial cells: Possible implication of oxidative stress. Environ. Health Perspect. 101: 436–442.

Hanley, Q.S., J.Q. Koenig, T.V. Larson, T.L. Anderson, G. van Belle, V. Rebolledo, D.S. Covert and W.E. Pierson. 1992. Response of young asthmatic patients to inhaled sulfuric acid. Am. Rev. Respir. Dis. 145: 326–331.

Hatch, G.E., E. Boykin, J.A. Graham, J. Lewtas, F. Pott, K. Mumfor and J.L. Loud. 1985. Inhalable particles and pulmonary host defense: *in vivo* and *in vitro* effect of ambient air and combustion particles. Environ. Res. 36: 67–80.

Hefflin, B.J., B. Jalaludin, E. McClure, N. Cobb, C.A. Johnson, L. Jecha and R.A. Etzel. 1994. Surveillance for dust storms and respiratory diseases in Washington State, 1991. Arch. Environ. Health 49(3): 170–174.

Heinrich, U., H. Muhle, S. Takenaka, H. Ernst, R. Fuhst, U. Mohr, F. Pitt and W. Stober. 1986a. Chronic effects on the respiratory tract of hamsters, mice and rats after long-term inhalation of high concentrations of filtered and unfiltered diesel engine emissions. J. Appl. Toxicol. 6(6): 383–395.

Heinrich, U., F. Pott, U. Mohr, R. Fuhst and J. Donig. 1986b. Lung tumours in rats and mice after inhalation of PAH-rich emissions. Exp. Pathol. 29: 29–34.

Heinrich, U., H. Muhle, H.G. Hoymann and R. Mermelstein. 1989. Pulmonary function changes in rats after chronic and subchronic inhalation exposure to various particulate matter. Exp. Pathol. 37: 248–252.

Henderson, R.F., J.A. Pickrell, R.K. Jones, J.D. Sun, J.M. Benson, J.L. Mauderly and R.O. McClelland. 1988. Response of rodents to inhaled diluted diesel exhaust: biochemical and cytological changes in bronchiolar lavage fluid and in lung tissue. Fundam. Appl. Toxicol. 11: 546–567.

Hoek, G. and B. Brunekreef. 1993. Acute effects of a winter air pollution episode on pulmonary function and respiratory symptoms of children. Arch. Environ. Health 48(5): 328–335.

Hoek, G. and B. Brunekreef. 1994. Effects of low-level winter air pollution concentrations on respiratory health of Dutch children. Environ. Res. 64: 136–150.

Ichinose, T., A. Furuyama and M. Sagai. 1995. Biological effects of diesel exhaust particles (DEP). II. Acute toxicity of DEP introduced into lung by intratracheal instillation. Toxicology 99: 153–167.

ICRP (International Commission on Radiological Protection). 1994. Human respiratory tract model for radiological protection. A report of Committee 2 of the ICRP. Pergamon Press, Oxford, U.K.

Ikeda, M., M. Shitashige, H. Yamasaki, M. Sagai and T. Tomita. 1995. Oxidative modification of low density lipoprotein by diesel exhaust particles. Biol. Pharm. Bull. 18: 866–871.

Ito, K. and G.D. Thurston. 1996. Daily PM_{10} mortality associations: an investigation of at-risk subpopulations. J. Expos. Anal. Environ. Epidemiol. 6(1): 79–95.

Kawabata, Y., T. Udagawa and K. Iwai. 1993. One year exposure to diesel engine exhaust causes lung tumors. *In*: Toxic and carcinogenic effects of solid particles in the respiratory tract. Abstracts of the 4th International Inhalation Symposium, Hannover, Germany. Hannover Medical School. p. 61.

Keeler, G.J., J.D. Spengler, P. Koutrakis and G.A. Allen. 1990. Transported acid aerosols measured in southern Ontario. Atmos. Environ. 24A: 2935–2950.

Killingsworth, C.R., F. Alessandrini, G.G.K. Murthy, P.J. Catalano, J.D. Paulauskis and J. Godleski. 1997. Inflammation, chemokine expression, and death in monocrotaline-treated rats following fuel oil fly ash inhalation. Inhal. Toxicol. 9: 541–565.

Kim, C.S. and T.C. Kang. 1997. Comparative measurement of lung deposition of inhaled fine particles in normal subjects and patients with obstructive airway disease. Am. J. Respir. Crit. Care Med. 155: 899–905.

Kinney, P.L., K. Ito and G.D. Thurston. 1995. A sensitivity analysis of mortality/PM_{10} associations in Los Angeles. Inhal. Toxicol. 7: 59–69.

Kleinman, M.T., D.K. Bhalla, W.J. Mautz and R.F. Phalen. 1995. Cellular and immunologic injury with PM_{10} inhalation. Inhal. Toxicol. 7: 589–602.

Kobzik, L., S. Huang, J.D. Paulauskis and J.J. Godleski. 1993. Particle opsonization and lung macrophage cytokine response. *In vitro* and *in vivo* analysis. J. Immunol. 151(5): 2753–2759.

Kodavanti, U.P., R.H. Jaskot, D.L. Costa and K.L. Dreher. 1997. Pulmonary proinflammatory gene induction following acute exposure to residual oil fly ash: Roles of particle-associated metals. Inhal. Toxicol. 9: 679–701.

Koenig, J.Q., D.S. Covert and W.E. Pierson. 1989. Effects of inhalation of acidic compounds on pulmonary function in allergic adolescent subjects. Environ. Health Perspect. 79: 173–178.

Koenig, J.Q., D.S. Covert, T.V. Larson and W.E. Pierson. 1992. The effect of duration of exposure on sulfuric acid-induced pulmonary function changes in asthmatic adolescent subjects: a dose–response study. Toxicol. Ind. Health 8: 285–296.

Koenig, J.Q., K. Dumler, V. Rebolledo, P.V. Williams and W.E. Pierson. 1993. Respiratory effects of inhaled sulfuric acid on senior asthmatics and nonasthmatics. Arch. Environ. Health 48: 171–175.

Koutrakis, P., S.L.K. Briggs and B.P. Leaderer. 1992. Source apportionment of indoor aerosols in Suffolk and Onondaga counties, New York. Environ. Sci. Technol. 26: 521–527.

Leech, J.A., K. Wilby, E. McMullen and K. Laporte. 1996. The Canadian Human Activity Pattern Survey: report of methods and population surveyed. Chron. Dis. Can. 17(3/4): 118–123.

Lehnert, B.E., Y.E. Valdez and S.H. Bomalaski. 1985. Lung and pleural "free cell responses" to the intrapulmonary deposition of particles in the rat. J. Toxicol. Environ. Health 16: 823–839.

Lindenschmidt, R.C., K.E. Driscoll, M.A. Perkins, J.M. Higgins, J.K. Mauer and K.A. Belfiore. 1990. The comparison of a fibrogenic and two nonfibrogenic dusts by bronchioalveolar lavage. Toxicol. Appl. Pharmacol. 102: 268–281.

Linn, W.S., D.A. Shamoo, K.R. Anderson, R.-C. Peng, E.L. Avol and J.D. Hackney. 1994. Effects of prolonged, repeated exposure to ozone, sulfuric acid, and their combination in healthy and asthmatic volunteers. Am. J. Respir. Crit. Care Med. 150: 431–440.

Linn, W.S., K.R. Anderson, D.A. Shamoo, S.A. Edwards, T.L. Webb, J.D. Hackney and H. Gong, Jr. 1995. Controlled exposures of young asthmatics to mixed oxidant gases and acid aerosol. Am. J. Respir. Crit. Care Med. 152: 885–891.

Linn, W.S., H. Gong, D.A. Shamoo, K.R. Anderson and E.L. Avol. 1997. Chamber exposures of children to mixed ozone, sulfur dioxide, and sulfuric acid. Arch. Environ. Health 52: 179–187.

Lioy, P.J., J.M. Waldman, T. Buckley, J. Butler and C. Pietarinen. 1990. The personal, indoor and outdoor concentrations of PM_{10} measured in an industrial community during the winter. Atmos. Environ. 24B(1): 57–66.

Lipfert, F.W. 1994. Air pollution and community health — a critical review and data sourcebook. Van Nostrand Reinhold, New York, N.Y. 556 pp.

Lippmann, M. 1977. Regional deposition of particles in the human respiratory tract. *In*: D.H.K. Lee, H.L. Falk, S.D. Murphy and S.R. Geiger (eds.), Handbook of physiology. Section 9: Reactions to environmental agents. American Physiological Society, Bethesda, Maryland. pp. 213–232.

Lowenthal, D.H. 1997. Motor vehicle contributions to $PM_{2.5}$ aerosol in Canadian urban areas. Prepared for Atmospheric Science Expert Panel of the Industry/ Government Joint "Sulphur in Gasoline Studies," February.

Lowenthal, D.H., D. Whittorff and A.W. Gertler. 1994. CMB source apportionment during REVEAL. Prepared for the Air Resources Branch, B.C. Ministry of Environment, Lands and Parks (Report No. ENV 484410/10/94).

Lowenthal, D.H., D. Wittorf, A.W. Gertler and S. Sakiyama. 1996. CMB source apportionment during REVEAL. J. Environ. Eng. 123: 80–87.

Lyon, J.L., M. Mori and R. Gao. 1995. Is there a causal association between excess mortality and exposure to PM-10 air pollution? Additional analyses by location, year, season, and cause of death. Inhal. Toxicol. 7: 603–614.

Mage, D.T. and T.J. Buckley. 1995. The relationship between personal exposures and ambient concentrations of particulate matter. Presented at the 88th Annual Meeting of the Air & Waste Management Association, San Antonio, Texas, June. Air & Waste Management Association, Pittsburgh, Pennsylvania (Paper No. 95-MP18.01).

Malm, W.C., J.F. Sisler, D. Huffman, R.A. Eldred and T.A. Cahill. 1994. Spatial and seasonal trends in particle concentration and optical extinction in the United States. J. Geophys. Res. 99: 1346–1370.

Mann, D.L. and J.B. Young. 1994. Basic mechanisms in congestive heart failure. Recognizing the role of proinflammatory cytokines. Chest 105: 897–904.

Mauderly, J.L., N.A. Gillett, R.F. Henderson and R.O. McClelland. 1988. Relationships of lung structural and functional changes to accumulation of diesel exhaust. Am. J. Occup. Hyg. 32: 659–669.

Meyer, M.B. 1993. Applications of continuous PM_{10} measurements. Presented at The Role of Meteorology in Managing the Environment of the 90's, Scotsdale, Arizona, January 26–28.

Miller, F.J., C.E. Gardner, J.A. Graham, R.R. Lee, W.E. Wilson and J.D. Bachman. 1979. Size considerations for establishing a standard for inhalable particles. J. Air Pollut. Control Assoc. 29: 610–615.

Miller, F.J., S. Anjilvel, M.G. Menache, B. Asgharian and T.R. Gerrity. 1995. Dosimetric issues relating to particulate toxicity. Inhal. Toxicol. 7: 615–632.

Ministers' Expert Advisory Panel. 1995. Report of the Ministers' Expert Advisory Panel on the second Priority Substances List under the *Canadian Environmental Protection Act* (CEPA). Government of Canada, Ottawa, Ontario. 26 pp.

Mohr, C., G.S. Davis, C. Graebner, D.R. Hemenway and D. Gemsa. 1992. Enhanced release of prostaglandin E_2 from macrophages of rats with silicosis. Am. J. Respir. Cell Mol. Biol. 6: 390–396.

Moore, C.T. and R.J. Barthelmie. 1995. A review of comparative manual and automated PM_{10} data. Final draft. Western States Air Resources Council (WESTAR), Portland, Oregon.

Morel, D.W., J.R. Hessler and G.M. Chisolm. 1983. Low density lipoprotein cytotoxicity induced by free radical peroxidation of lipid. J. Lipid Res. 24: 1070–1076.

Morrow, P.E., M.J. Utell, M.A. Bauer, D.M. Speers and F.R. Gibb. 1994. Effects of near ambient levels of sulphuric acid aerosol on lung function in exercising subjects with asthma and chronic obstructive pulmonary disease. Ann. Occup. Hyg. 38(1): 933–938.

Nadeau, D., R. Vincent, M. Laviolette and J. Brook. 1995. Ambient air particles inhibit the respiratory burst of alveolar macrophages from rat and human lungs upon secondary challenge. J. Aerosol Med. 8: 112.

Nadeau, D., R. Vincent, P. Kumarathasan, J. Brook and A. Dufresne. 1996. Cytotoxicity of ambient air particles to rat lung macrophages: Comparison of cellular and functional assays. Toxicol. *In Vitro* 10: 161–172.

Nagai, A., K. Sakamoto, T. Takizawa, S. Morinibu, Y. Yamano, N. Nagao, Y. Ishihara and J. Kagawa. 1991. Sulfuric acid submicron aerosols induce morphological changes in the central airways in the guinea pig. Jpn. J. Thorac. Dis. 29: 992–997.

NAPAP (National Acid Precipitation Assessment Program). 1991. Visibility, existing and historical conditions — causes and effects. Office of the Director, Acid Deposition, State of Science and Technology, Washington, D.C. (Report 24).

Naumann, B.D. and R.B. Schlesinger. 1986. Assessment of early alveolar particle clearance and macrophage function following an acute inhalation sulfuric acid mist. Exp. Lung Res. 11: 13–33.

Nearing, B.D., R.L. Verrier, W.A. Skornik, G. Gazula, C.R. Killingsworth, K. Oakberg and J.J. Godleski. 1996. Inhaled fly ash results in alteration in cardiac electrophysiologic function. Am. J. Respir. Crit. Care Med. 153: A543.

Neas, L.M., D.W. Dockery, J.H. Ware, J.D. Spengler, B.G. Ferris, Jr., and F.E. Speizer. 1994. Concentration of indoor particulate matter as a determinant of respiratory health in children. Am. J. Epidemiol. 139(11): 1088–1099.

Oberdörster, G., J. Ferin, R.M. Gelein, S.C. Soderholm and J. Finkelstein. 1992a. Role of the alveolar macrophage in lung injury: Studies with ultrafine particles. Environ. Health Perspect. 97: 193–197.

Oberdörster, G., J. Ferin and P.E. Morrow. 1992b. Volumetric loading of alveolar macrophages (AM): a possible basis for diminished AM-mediated particle clearance. Exp. Lung Res. 18: 87–104.

Oberdörster, G., R.M. Gelein, J. Ferin, N. Corson and P. Wade. 1994a. Association of low level particulate air pollution and acute mortality/morbidity: a potential role of ultrafine particles. Eur. Respir. J. 7 (Suppl. 18): 213.

Oberdörster, G., J. Oberdörster and B.E. Lehnert. 1994b. Particulate air pollution: animal toxicology. Contract report to Health Canada.

Oberdörster, G., J. Ferin and B.E. Lehnert. 1994c. Correlation between particle size, *in vivo* particle persistence and lung injury. Environ. Health Perspect. 102 (Suppl. 5): 173–179.

Oberdörster, G., J. Ferin, S.C. Soderholm, R. Gelein, C. Cox, R. Baggs and P.E. Morrow. 1994d. Increased pulmonary toxicity of inhaled ultrafine particles: Due to lung overload alone? Ann. Occup. Hyg. 38 (Suppl. 1): 295–302.

Oberdörster, G., R.M. Gelein, J. Ferin and B. Weiss. 1995. Association of particulate air pollution and acute mortality: Involvement of ultrafine particles. Inhal. Toxicol. 7: 111–124.

Ocana, A.M. 1989. Low-density lipoprotein and atherogenesis. N. Engl. J. Med. 321: 1196.

Ostro, B.D. 1987. Air pollution and morbidity revisited: a specification test. J. Environ. Econ. Manage. 14: 87–98.

Ostro, B.D. 1990. Associations between morbidity and alternative measures of particulate matter. Risk Anal. 10(3): 421–427.

Ostro, B.D. and S. Rothschild. 1989. Air pollution and acute respiratory morbidity: an observational study of multiple pollutants. Environ. Res. 50: 238–247.

Ostro, B.D., M.J. Lipsett, M.D. Weiner and J.C. Selner. 1991. Asthmatic responses to airborne acid aerosols. Am. J. Public Health 81(6): 694–702.

Ostro, B., J.M. Sanchez, C. Aranda and G. Eskeland. 1996. Air pollution and mortality: results from a study of Santiago, Chile. J. Expos. Anal. Environ. Epidemiol. 6(1): 97–114.

Özkaynak, H. and G.D. Thurston. 1987. Associations between 1980 U.S. mortality rates and alternative measures of airborne particle concentrations. Risk Anal. 7(4): 449–461.

Özkaynak, H., J.D. Spengler, J. Xue, P. Koutrakis, E.D. Pellizzari and L.A. Wallace. 1993. Sources and factors influencing personal and indoor exposures to particles, elements and nicotine, findings from the Particle TEAM pilot study. *In*: Proceedings of Indoor Air '93, Helsinki, Finland, July 3. pp. 447–462.

Özkaynak, H., J. Xue, R. Weker, D. Butler, P. Koutrakis and J.D. Spengler. 1995a. The Particle TEAM (PTEAM) study, analysis of the data. Final report. Vol. III. U.S. Environmental Protection Agency, Research Triangle Park, North Carolina (Contract No. 68-02-4544).

Özkaynak, H., D. MacIntosh, J. Xue and H. Zhou. 1995b. Predicted distribution of personal exposure to PM_{10} in Canada. Health Canada, Ottawa, Ontario (Contract No. 4802).

Özkaynak, H., J. Xue, P. Severance, R. Burnett and M. Raizenne. 1995c. Associations between daily mortality, ozone and particulate air pollution in Toronto, Canada. Inhal. Toxicol. 7(5): 812.

Pantazopoulou, A., K. Katsouyanny, J. Kourea-Kremastinou and K. Trichopoulos. 1995. Short-term effects of air pollution on hospital emergency outpatient visits and admissions in the Greater Athens, Greece area. Environ. Res. 69: 31–36.

Pellizzari, E., K.W. Thomas, C.A. Clayton, R.W. Whitmore, R.C. Shores, H.S. Zelon and R.L. Perritt. 1992. Particle Total Exposure Assessment Methodology (PTEAM), Riverside, California pilot study. Vol. I. U.S. Environmental Protection Agency, Research Triangle Park, North Carolina (Contract No. 68-02-4544; EPA/600/R-93/050).

Peters, A., I.F. Goldstein, U. Beyer, K. Franke, J. Heinrich, D.W. Dockery, J.D. Spengler and H.-E. Wichmann. 1996a. Acute health effects of exposure to high levels of air pollution in Eastern Europe. Am. J. Epidemiol. 144: 570–581.

Peters, A., T. Tuch, P. Brand, J. Heyder and H.-E. Wichmann. 1996b. Size distribution of ambient particles and its relevance to human health. *In*: J. Lee and R. Phalen (eds.), Proceedings of the 2nd Colloquium on Particulate Air Pollution and Human Health, December 1996, Park City, Utah. University of Utah, Salt Lake City, and University of California, Irvine.

Peters, A., H.E. Wichmann, T. Tuch, J. Heinrich and J. Heyder. 1997. Respiratory effects are associated with the number of ultrafine particles. Am. J. Respir. Crit. Care Med. 155: 1376–1383.

Ponce de Leon, A., H.R. Anderson, J.M. Blanc, D.P. Strachan and J. Bower. 1996. Effects of air pollution on daily hospital admissions for respiratory disease in London between 1987–88 and 1991–92. J. Epidemiol. Commun. Health 50 (Suppl. 1): S63–S70.

Pope, C.A. 1991. Respiratory hospital admissions associated with PM_{10} pollution in Utah, Salt Lake, and Cache valleys. Arch. Environ. Health 46(2): 90–97.

Pope, C.A. and L.S. Kalkstein. 1996. Synoptic weather modeling and estimates of the exposure–response relationship between daily mortality and particulate air pollution. Environ. Health Perspect. 104(4): 414–420.

Pope, C.A., D.W. Dockery, J.D. Spengler and M.E. Raizenne. 1991. Respiratory health and PM_{10} pollution. Am. Rev. Respir. Dis. 144(3): 668–674.

Pope, C.A., J. Schwartz and M.R. Ransom. 1992. Daily mortality and PM_{10} pollution in Utah Valley. Arch. Environ. Health 47(3): 211–217.

Pope, C.A., III, M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer and C.W. Heath, Jr. 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am. J. Respir. Crit. Care Med. 151: 669–674.

Pott, F., D.L. Dungworth, U. Heinrich, H. Muhle, K. Kamino, P.-G. Germann, M. Roller, R.M. Rippe and U. Mohr. 1994. Lung tumors in rats after intratracheal instillation of dusts. *In*: J. Dodgson and R.I. McCallum (eds.), Inhaled particles VII. Proceedings of the 7th International Symposium. Pergamon Press, Oxford, U.K. pp. 357–363.

Proctor, D.F. and H.N. Wagner. 1965. Clearance of particles from the human nose. Arch. Environ. Health 11: 366.

Pryor, S. and D. Steyn. 1994. Visibility and ambient aerosols in southwestern British Columbia during REVEAL — Part 2. Report prepared for the Air Resources Branch, B.C. Ministry of Environment, Lands and Parks (Report No. ENV 484415/03/95).

Raabe, O.G., D.W. Wilson, M.A. Al-Bayati, W.J. Hornot and L.S. Rosenblatt. 1994. Biological effects of inhaled pollutant aerosols. Ann. Occup. Hyg. 389 (Suppl. 1): 323–330.

Raizenne, M., L.M. Neas, A.I. Damokosh, D.W. Dockery, J.D. Spengler, P. Koutrakis, J.H. Ware and F.E. Speizer. 1996. Health effects of acid aerosols on North American children: pulmonary function. Environ. Health Perspect. 104: 506–514.

Ransom, M. and C.A. Pope. 1992. Elementary school absences and PM_{10} pollution in Utah Valley. Environ. Res. 58: 204–219.

Roemer, W., G. Hoek and B. Brunekreef. 1993. Effect of ambient winter air pollution on respiratory health of children with chronic respiratory symptoms. Am. Rev. Respir. Dis. 147: 118–124.

Sakakibara, M., M. Minami, T. Endo, M. Hirafuji, S. Murakami, Y. Mori and M. Sagai. 1994. Biological effects of diesel exhaust particles (DEP) on isolated cardiac muscle of guinea pigs. Res. Commun. Mol. Pathol. Pharmacol. 86: 99–110.

Saldiva, P.H.N., D.W. Dockery, C.A. Pope, III, A.J. Lichtenfels, J.M. Salge, I. Barone and G.M. Bohm. 1995. Air pollution and mortality in elderly people: a time-series study in Sao Paulo, Brazil. Arch. Environ. Health 50(2): 159–163.

Samet, J.M. and P.-W. Cheng. 1994. The role of airway mucus in pulmonary toxicology. Environ. Health Perspect. 102 (Suppl. 2): 89–103.

Sandstrom, T. and B. Rudell. 1991. Bronchoalveolar neutrophilia and depressed macrophage phagocytosis following single-exposure to diesel exhaust. Am. Rev. Respir. Dis. 143(4): A96 (abstract).

Santanam, S., J.D. Spengler and P.B. Ryan. 1990. Particulate matter exposures estimated from an indoor–outdoor source apportionment study. *In*: D.S. Walkinshaw (ed.), Indoor Air ’90. Proceedings of the 5th International Conference on Indoor Air Quality and Climate. Vol. 2. Canada Mortgage and Housing Corporation, Ottawa, Ontario. pp. 583–588.

Schlesinger, R.B. 1987. Functional assessment of rabbit alveolar macrophages following intermittent inhalation exposures to sulfuric acid mist. Fundam. Appl. Toxicol. 8: 328–334.

Schlesinger, R.B., L.C. Chen, I. Finkelstein and J.T. Zelikoff. 1990. Comparative potency of inhaled acidic sulfates: Speciation and the role of hydrogen ion. Environ. Res. 52: 210–224.

Schlesinger, R.B., J.E. Gorczynski, J. Dennison, L. Richards, P.L. Kinney and M.C. Bosland. 1992. Long-term intermittent exposure to sulfuric acid aerosol, ozone, and their combination: Alterations in tracheobronchial mucociliary clearance and epithelial secretory cells. Exp. Lung Res. 18: 505–534.

Schouten, J.P., J.M. Vonk and A. de Graaf. 1996. Short term effects of air pollution on emergency hospital admissions for respiratory disease: results of the APHEA project in two major cities in The Netherlands, 1977–89. J. Epidemiol. Commun. Health 50 (Suppl. 1): S22–S29.

Schwartz, J. 1993. Air pollution and daily mortality in Birmingham, Alabama. Am. J. Epidemiol. 137(10): 1136–1147.

Schwartz, J. 1994a. Air pollution and hospital admissions for the elderly in Birmingham, Alabama. Am. J. Epidemiol. 139: 589–598.

Schwartz, J. 1994b. Air pollution and hospital admissions for the elderly in Detroit, Michigan. Am. J. Respir. Care Med. 150: 648–655.

Schwartz, J. 1994c. PM_{10}, ozone, and hospital admissions for the elderly in Minneapolis–St. Paul. Arch. Environ. Health 49: 366–374.

Schwartz, J. 1994d. What are people dying of on high air pollution days? Environ. Res. 64: 26–35.

Schwartz, J. 1994e. Air pollution and daily mortality: a review and meta analysis. Environ. Res. 64: 36–52.

Schwartz, J. 1995. Short term fluctuations in air pollution and hospital admissions of the elderly for respiratory disease. Thorax 50: 531–538.

Schwartz, J. 1996. Air pollution and hospital admissions for respiratory disease. Epidemiology 7: 20–28.

Schwartz, J. and R. Morris. 1995. Air pollution and hospital admissions for cardiovascular disease in Detroit, Michigan. Am. J. Epidemiol. 142: 23–35.

Schwartz, J., D. Slater, T.V. Larson, W.E. Pierson and J.Q. Koenig. 1993. Particulate air pollution and hospital emergency room visits for asthma in Seattle. Am. Rev. Respir. Dis. 147: 826–831.

Schwartz, J., D.W. Dockery, L.M. Neas, D. Wypij, J.H. Ware, J.D. Spengler, P. Koutrakis, F.E. Speizer and B.G. Ferris, Jr. 1994. Acute effects of summer air pollution on respiratory symptom reporting in children. Am. J. Respir. Crit. Care Med. 150: 1234–1242.

Schwartz, J., D.W. Dockery and L.M. Neas. 1996. Is daily mortality associated specifically with fine particles? J. Air Waste Manage. Assoc. 46: 927–939.

Seinfeld, J.H. 1986. Atmospheric chemistry and physics of air pollution. John Wiley & Sons, New York, N.Y.

Sexton, K., J.D. Spengler and R.D. Treitman. 1984. Personal exposure to respirable particles, a case study in Waterbury, Vermont. Atmos. Environ. 18: 1385–1398.

Shami, S.G., S.A. Silbaugh, F.F. Hahn, W.C. Griffith and C.H. Hobbs. 1984. Cytokinetic and morphological changes in the lungs and lung-associated lymph nodes of rats after inhalation of fly ash. Environ. Res. 35: 373–393.

Sheldon, L.S., T.D. Hartwell, B.G. Cox, J.E. Sickles II, E.D. Pellizzari, M.L. Smith, R.L. Perritt and S.M. Jones. 1989. An investigation of infiltration and indoor air quality. Final report. New York State Energy Research and Development Authority, Albany, N.Y. (New York State ERDA Contract No. 736-CON-BCS-85).

Slauson, D.O., J.C. Lay, W.L. Castleman and N.R. Neilsen. 1989. Influence of acute pulmonary interstitial inflammation on kinetics of phagocytosis by alveolar macrophages. Inflammation 13: 429–441.

Smaldone, G.E., R.J. Perry, W.D. Bennett, M.S. Messina, J. Zwang and J. Ilowite. 1988. Interpretation of “24 hour lung retention” in studies of mucociliary clearance. J. Aerosol Med. 1: 11–20.

Spektor, D.M., B.M. Yen and M. Lippmann. 1989. Effect of concentration and cumulative exposure of inhaled sulfuric acid on tracheobronchial particle clearance in healthy humans. Environ. Health Perspect. 79: 167–172.

Spektor, D.M., V.A. Hofmeister, P. Artaxo, J.A.P. Brague, F. Echelar, D.P. Nogueira, C. Hayes, G.D. Thurston and M. Lippmann. 1991. Effects of heavy pollution on respiratory function in the children of Cubatao, Brazil: a preliminary report. Environ. Health Perspect. 94: 51–54.

Spengler, J.D., R.D. Treitman, T.D. Tosteson, D.T. Mage and M.L. Soczek. 1985. Personal exposures to respirable particulates and implications for air pollution epidemiology. Environ. Sci. Technol. 19: 700–707.

Spiegelberg, T., W. Kördel and D. Hochrainer. 1984. Effects of NiO inhalation on alveolar macrophages and the humoral immune systems of rats. Ecotoxicol. Environ. Saf. 8: 516–525.

Stahlofen, W., J. Gebhart, G. Rudolf and G. Scheuch. 1986a. Measurement of lung clearance with pulses of radioactively-labeled particles. J. Aerosol Med. 1: 11–20.

Stahlofen, W., J. Gebhart, G. Rudolf, G. Scheuch and K. Philipson. 1986b. Clearance from the human airways of particles of different sizes deposited from inhaled aerosol boli. *In*: Aerosols, formation and reactivity. Proceedings of the 2nd International Aerosol Conference, Berlin. Pergamon Journals Ltd., U.K. pp. 192–208.

Styer, P., N. McMillan, F. Gao, J. Davis and J. Sacks. 1995. Effect of outdoor airborne particulate matter on daily death counts. Environ. Health Perspect. 103: 490–497.

Sunyer, J., M. Saez, C. Murillo, J. Castellsague, F. Martinez and J.M. Anto. 1993. Air pollution and emergency room admissions for chronic obstructive pulmonary disease: a 5-year study. Am. J. Epidemiol. 137: 701–705.

Sunyer, J., J. Castellsague, M. Saez, A. Tobias and J.M. Anto. 1996. Air pollution and mortality in Barcelona. J. Epidemiol. Commun. Health 50 (Suppl. 1): S76–S80.

Tabata,Y. and Y. Ikada. 1988. Effect of the size and surface charge of polymer microspheres on their phagocytosis by macrophage. Biomaterials 9: 356–362.

Thatcher, T.L. and D.W. Layton. 1994. Deposition, resuspension and penetration within a residence. Riverside, CA. University of California, Lawrence Livermore National Laboratory (Report No. UCLR-JC-116597).

Thatcher, T.L. and D.W. Layton. 1995. Deposition, resuspension and penetration of particles within a residence. Atmos. Environ. 29: 1487–1497.

Thurston, G.D. 1996. A critical review of PM_{10} mortality time-series studies. J. Expos. Anal. Environ. Epidemiol. 6: 3–21.

Thurston, G.D., K. Ito, P.L. Kinney and M. Lippmann. 1992. A multi-year study of air pollution and respiratory hospital admissions in three New York State metropolitan areas: results for 1988 and 1989 summers. J. Expos. Anal. Environ. Epidemiol. 2(4): 429–450.

Thurston, G.D., K. Ito, C.G. Hayes, D.V. Bates and M. Lippmann. 1994. Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols. Environ. Res. 65: 271–290.

Touloumi, G., S.J. Pocock, K. Katsouyanni and D. Trichopoulos. 1994. Short-term effects of air pollution on daily mortality in Athens: a time-series analysis. Int. J. Epidemiol. 23(5): 957–967.

Touloumi, G., E. Samoli and K. Katsouyanni. 1996. Daily mortality and "winter type" air pollution in Athens, Greece — a time series analysis within the APHEA project. J. Epidemiol. Commun. Health 50 (Suppl. 1): S47–S51.

Trijonis, J. 1982. Visibility in California. J. Air Pollut. Control Assoc. 32: 165–169.

U.S. EPA (United States Environmental Protection Agency). 1982. Air quality criteria for particulate matter and sulfur oxides. Office of Air Quality Planning & Standards, Research Triangle Park, North Carolina, December (EPA-600/8-82-029a-c).

U.S. EPA (United States Environmental Protection Agency). 1989. An acid aerosol issue paper, health effects and aerometrics. Office of Health and Environmental Assessment, Washington, D.C. (EPA Report No. 600/8-88-005F).

U.S. EPA (United States Environmental Protection Agency). 1995. Exposure factors handbook. Office of Research and Development, Washington, D.C. (EPA Review Draft No. EPA/600/P-95/002A).

U.S. EPA (United States Environmental Protection Agency). 1996. Air quality criteria for particulate matter. Vols. 1–3. Office of Research and Development, Washington, D.C. (EPA Report No. EPA/001a-c).

Utell, M.J., P.E. Morrow and R.W. Hyde. 1983a. Latent development of airway hyperreactivity in human subjects after sulfuric acid aerosol exposure. J. Aerosol Sci. 14: 202–205.

Utell, M.J., P.E. Morrow, D.M. Speers, J. Darling and R.W. Hyde. 1983b. Airway responses to sulfate and sulfuric acid aerosols in asthmatics: an exposure–response relationship. Am. Rev. Respir. Dis. 128: 440–450.

Utell, M.J., P.E. Morrow and R.W. Hyde. 1984. Airway reactivity to sulfate and sulfuric acid aerosols in normal and asthmatic subjects. J. Air Pollut. Control Assoc. 34: 931–935.

Utell, M.J., J.A. Mariglio, P.E. Morrow, F.R. Gibb and D.M. Speers. 1989. Effect of inhaled acid aerosols on respiratory function: the role of endogenous ammonia. J. Aerosol Med. 2: 141–147.

van Houdt, J.J. 1990. Mutagenic activity of airborne particles in indoor and outdoor environments. Atmos. Environ. 24B: 207–220.

Verhoeff, A.P., G. Hoek, J. Schwartz and J.H. van Wijnen. 1996. Air pollution and daily mortality in Amsterdam. Epidemiology 7: 225–230.

Wallace, J.M. and P.V. Hobbs. 1977. Atmospheric science: an introductory survey. Academic Press, Orlando, Florida. 467 pp.

Wallace, L. 1996. Indoor particles, a review. J. Air Waste Manage. Assoc. 46: 98–126.

Wallace, L., H. Özkaynak, J.D. Spengler, E.D. Pellizzari and P. Jenkins. 1993. Indoor, outdoor, and personal air exposures to particles, elements, and nicotine for 178 residents of Riverside, California. *In*: J.J. Jaakkola, R. Ilmarinen and O. Sappanen (eds.), Indoor Air '93. Proceedings of the 6th International Conference on Indoor Air Quality and Climate, July 1993, Helsinki, Finland. Helsinki University of Technology, Espoo, Finland. pp. 445–450.

Walters, S., R.K. Griffiths and J.G. Ayres. 1994. Temporal association between hospital admissions for asthma in Birmingham and ambient levels of sulphur dioxide and smoke. Thorax 49: 133–140.

Warheit, D.B., W.C. Seidel, M.C. Carakostas and M.A. Hartsky. 1990. Attenuation of perfluoropolymer fume pulmonary toxicity: effect of filters, combustion method and aerosol age. Exp. Mol. Pathol. 52: 309–329.

Warheit, D.B., M.C. Carakostas, M.A. Hartsky and J.F. Hansen. 1991. Development of a short-term inhalation bioassay to assess pulmonary toxicity of inhaled particles: comparisons of pulmonary responses to carbonyl iron and silica. Toxicol. Appl. Pharmacol. 107: 350–368.

WGAQOG (Working Group on Air Quality Objectives and Guidelines). 1998. National Ambient Air Quality Objectives for Particulate Matter. 1: Science Assessment Document. A report prepared by the Federal–Provincial Working Group on Air Quality Objectives and Guidelines for the *Canadian Environmental Protection Act* Federal–Provincial Advisory Committee.

WGAQOG (Working Group on Air Quality Objectives and Guidelines). 1999. National Ambient Air Quality Objectives for Particulate Matter. Addendum to the Science Assessment Document. A report prepared by the Federal-Provincial Working Group on Air Quality Objectives and Guidelines for the *Canadian Environment Protection Act* Federal-Provincial Advisory Committee. December 1997, revised April 1999.

Wiessner, J.H., N.S. Mandel, P.G. Sohnle and G.S. Mandel. 1989. Effect of particle size on quartz-induced hemolysis and on lung inflammation and fibrosis. Exp. Lung Res. 15: 801–812.

Wiessner, J.H., N.S. Mandel, P.G. Sohnle, A. Hasgawa and G.S. Mandel. 1990. The effect of chemical modification of quartz surfaces on particulate-induced pulmonary inflammation and fibrosis in the mouse. Am. Rev. Respir. Dis. 141: 111–116.

Wiester, M.J., R. Iltis and W. Moore. 1980. Altered function and histology in guinea pigs after inhalation of diesel exhaust. Environ. Res. 22: 285–297.

Yocom, J.E. 1982. Indoor–outdoor air quality relationships. J. Air Pollut. Control Assoc. 32: 500–520.

Yokoyama, T., L. Vaca, R.D. Rossen, W. Durante, P. Hazarika and D.L. Mann. 1993. Cellular basis for the negative inotropic effects of tumor necrosis factor in the adult mammalian heart. J. Clin. Invest. 92: 2303–2312.

Zelikoff, J.T. and R.B. Schlesinger. 1992. Modulation of pulmonary immune defense mechanisms by sulfuric acid: effects on macrophage-derived tumor necrosis factor and superoxide. Toxicology 76: 271–281.

Zmirou, D., T. Barumandzadeh, F. Balducci, P. Ritter, G. Laham and J.-P. Ghilardi. 1996. Short term effects of air pollution on mortality in the city of Lyon, France, 1985–90. J. Epidemiol. Commun. Health 50 (Suppl. 1): S30–S35.