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NATIONAL AMBIENT AIR QUALITY OBJECTIVES FOR PARTICULATE MATTER

Executive Summary

PART 1: SCIENCE ASSESSMENT DOCUMENT

A report by the
CEPA/FPAC Working Group
on Air Quality Objectives
and Guidelines

Canada

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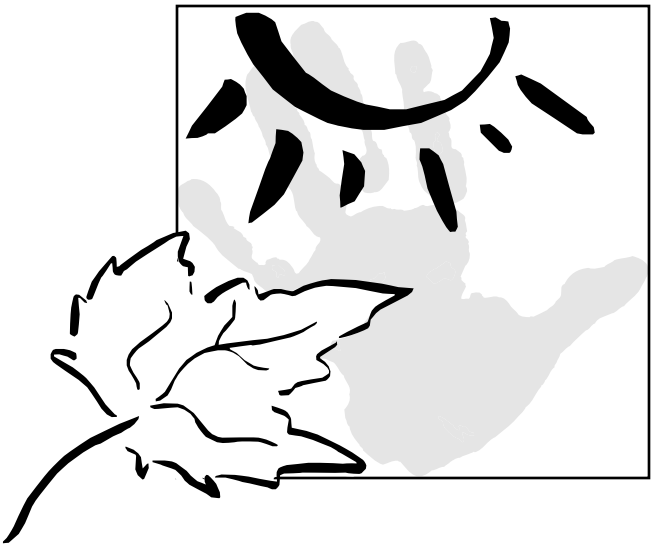

PART 1: SCIENCE ASSESSMENT DOCUMENT

A report by the
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EXECUTIVE SUMMARY

Introduction

The Canadian Environmental Protection Act (CEPA) Federal/Provincial Working Group on Air Quality Objectives and Guidelines (WGAQOG) is directed by the CEPA Federal-Provincial Advisory Committee (FPAC) to develop National Ambient Air Quality Objectives (NAAQOs) for airborne pollutants and to undertake their periodic reassessment. NAAQOs are established to provide a measure of protection to people and the environment from adverse effects due to airborne pollutants.

NAAQOs are developed in two stages, the first of which is the scientific assessment stage, embodied within the Science Assessment Document. The science assessment process concludes with determination of one or more Reference Levels for the pollutant of concern, through appropriate analyses of the scientific data, as described in A Protocol for the Development of National Ambient Air Quality Objectives: Part 1: Science Assessment Document and Derivation of the Reference Level(s). The Reference Level is a level above which an effect on a receptor (human or environmental) has been demonstrated. Reference Levels may be proposed for one or more time periods (e.g. 24 hour, annual) and for one or more receptors. Monitoring technology, economic benefits, and public/stakeholder consultations are introduced in the second phase, which is derivation of the Air Quality Objective (AQO). The AQO may be selected at the Reference Level, or it may be either lower or higher, depending on background levels of the pollutant, uncertainties in the underlying scientific data, and other considerations as listed above. The process by which AQOs are developed is described in A Protocol for the Development of National Ambient Air Quality Objectives: Part 2: Rationale Document and Derivation of Air Quality Objectives (in progress).

Canadian air quality objectives for particulate matter (PM) were first developed in the 1970s and were revised in the 1980s. At that time, the understanding was that episodes of severe pollution could result in adverse health and environmental impacts but that lower concentrations of PM, such as those generally experienced across Canada, were not particularly detrimental. Beginning about 1990, serious doubt was cast on this assumption, because of an avalanche of scientific studies emerging from the U.S., Canada, Britain and Europe. These studies demonstrated adverse effects of PM on cardio-respiratory health, including both increased hospitalizations and increased premature mortality at pollutant levels that were often far below the current air quality objectives. These findings have prompted re-examination of PM air quality objectives not only in Canada, but also in the U.S., Britain, the European Community and the World Health Organization.

The reevaluation of particulate air pollution has also led to a shift in focus from total suspended particulates (TSP), which encompasses a wide size range of particles, to particles equal or less than 10 micrometers in diameter (PM_{10}), and its subfractions ($PM_{2.5}$). This reflects the current belief that it is these smaller particle fractions that are primarily responsible for the observed adverse health and environmental effects.

The Science Assessment Document represents the Canadian reexamination of particulate matter air pollution and its possible consequences on human health and the environment. This document derives Reference Levels for PM_{10} (particles $\leq$10 µm) and $PM_{2.5}$ ($\leq$2.5 µm).

Physical and Chemical Characteristics

Particulate matter refers to all airborne solid and liquid particles, except pure water, that are microscopic in size. Particle size may range from approximately 0.005 µm to 100 µm in diameter, although the suspended portion is generally less than 40 µm. 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 and organic carbon compounds; oxides of silicon, aluminum and iron; trace metals; sulphates; nitrates and ammonia.

PM_{10} refers to particulate matter that is 10 µm or less in diameter. 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. 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 carbons (VOCs) and ammonia (NH_3). Primary particles are found in both the fine and coarse fractions, whereas secondary particles, such as sulphates and nitrates, are found predominantly in the fine fraction. Both primary and secondary PM can result from either natural or (human) anthropogenic sources.

Particle size is considered to be the most important parameter 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 removed from the atmosphere.

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, which result in the physical breakdown of larger particles into smaller ones to yield particles 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.

Other physical characteristics which 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. Particles (such as sulphates and nitrates) remain dry with increasing relative humidity until their deliquescent point is reached (which varies with the chemistry of the particle), at which time a sudden uptake of water occurs with a corresponding increase in particle size. The resultant particles are usually within the size range that are most efficient at scattering light. Therefore, particle growth through deliquescence has a large potential impact on atmospheric visibility.

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 to coarse particles.

As a consequence of their different sources and mechanisms of production, fine and coarse particles have markedly different chemical properties also. 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. 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. Fine particles tend to be acidic in nature. Sulphate has repeatedly been shown to be the most abundant single component of fine particles. 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. Since many of the compounds making up the carbonaceous mass are likely to be toxic, further elucidation of the carbon moiety of particulate matter is clearly required.

Sources

Particulate matter is a ubiquitous pollutant, reflecting the fact that it has both natural and anthropogenic sources. Natural sources of primary PM 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 sources such as wildfires will generate fine

PM. Secondary PM can be formed through reactions involving natural sources of the precursor gases. For example, volatile organic carbons (VOCs) are released from trees and nitrogen oxides are released from soils. Anthropogenic sources also produce both primary and secondary PM, 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, SO_2 and NOx).

Current estimates of the magnitude of actual emission sources in Canada are restricted to primary PM and are thus incomplete. In 1990, an estimated 1.0 Mt of PM_{10} was emitted from primary sources excluding open sources. Primary $PM_{2.5}$ accounted for approximately 75% of this total. The source sector breakdown for primary PM_{10} emissions is as follows: 42% industrial sources, 28% forest fires, 15% non-industrial fuel combustion, 11% transportation and 2% each, incineration and miscellaneous sources. The corresponding breakdown for $PM_{2.5}$ emissions shows equally large (34%) contributions from forest fires and industrial sources, and smaller contributions from non-industrial fuel combustion (16%), transportation (13%), incineration (2%) and miscellaneous sources (1%). Sources considered in each category include the following:

industrial: coal industry, pulp and paper industry, non-ferrous mining and smelting, iron and steel production, wood industry and mining and quarrying;

non-industrial fuel combustion: commercial and residential fuel combustion, residential fuelwood combustion and electric power generation;

transportation: gasoline and diesel vehicles and trucks, propane powered vehicles, railroads, marine craft, aircraft and tire wear;

incineration: wood waste and other types of incineration;

miscellaneous sources: structural fires, pesticides and fertilizer application, cigarette smoking and the marine cargo handling industry.

These emission estimates exclude open sources such as paved and unpaved roads, construction sites and prescribed burning, from which estimates of particulate emissions are particularly difficult to obtain. Although primary PM emissions from these sources have not yet been adequately quantified, these sources are known to release substantial quantities of both fine and coarse PM, although releases are typically episodic in nature. Estimates of the magnitude of secondary particulate sources are required to complement the data on primary emissions; however, these can only be derived using mathematical models which take into account precursor gas emissions and the complex atmospheric chemistry involved in the formation of secondary PM. Such models are still in the development stage. Limited studies in the Lower Fraser Valley of British Columbia have indicated that secondary particulates may account for up to 50% of $PM_{2.5}$ during the summer months. Similarly high contributions may be expected in other urban areas of Canada.

The available data clearly indicate that source contributions vary from province-to-province and by region. In the Yukon and Northwest Territories, British Columbia and Saskatchewan, forest fires and/or prescribed burning are the largest estimated sources of PM_{10}. Industrial sources are a major contributor to provincial PM_{10} emissions in all provinces except Prince Edward Island. In PEI, non-industrial fuel combustion (primarily residential wood combustion) is the major source. The transportation sector is also a large contributor in Ontario and BC, while non-industrial fuel combustion is significant in Alberta, Ontario, Québec 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.

Ambient Monitoring

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 PM 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, once every six days, as in the National Air Pollution Surveillance (NAPS) network. Different sampling periods and frequencies may be used where required, although the fact that the filters are collected manually is a constraint on the operation of these samplers. Indirect measurements are made using parameters other than mass that can then be converted to units of mass concentration based on known relationships between the two parameters.

Historically, the standard manual PM monitor in Canada was the hi-vol sampler, deriving its name from the fact that it draws large volumes of air through the filter upon which the particulate matter is retained. Originally used for the sampling of TSP, a modification of this technology was required to accommodate monitoring of PM_{10}. A specially designed sampling inlet was added that removes particles >10 µm so that particles in the PM_{10} size range are selectively retained on the filter. This type of sampler is therefore called a size selective inlet (SSI) hi-vol sampler.

The dichotomous sampler was the original sampler for inhalable particulates (PM_{10}), dividing particles into two size classes, as its name suggests. The sampler operates at a comparatively low sampling rate relative to the hi-vol samplers. The sample is first drawn through a selective inlet restricted to particles $\leq$10 µm. Subsequently, particles are fractionated into a fine fraction ($\leq$2.5 µm) and a coarse fraction (>2.5–10 µm) which are collected on separate filters for measurement and analysis. Hence dichot samplers enable measurements of PM_{10} (fine plus coarse fractions), $PM_{2.5}$ (fine fraction) and the coarse fraction. The partisol sampler is another low-volume manual sampler used to monitor PM_{10}. It can be fitted with TSP, PM_{10}, or $PM_{2.5}$ inlet heads. Other fine particle samplers include a variety of inertial impactors and cyclone samplers that are fitted with inlets designed to capture particles $\leq$2.5 µm in diameter.

The inlets of these manual PM samplers are designed with specified 50% cut points (D_{50}), which are defined as the particle aerodynamic diameter at which 50% of the particles pass through the inlet and 50% are rejected. The 50% cut points are accurate only at specified flow rates and therefore, the degree to which air flow through the sampler can be controlled is an important design feature of the instrument. Of concern with respect to PM_{10} samplers is that the 50% cut point (at approx. 10 µm) occurs near the maximum of particle mass distributions. Therefore, even slight differences in the cut points of different instruments may result in differences in PM mass measurements. This is less of a concern with $PM_{2.5}$ monitors since the 2.5 µm cut point occurs near a minimum in the particle mass distribution.

The comparability of PM mass measurements made with different samplers is an issue of concern. Differences may arise from cut point biases, differences in maintenance regimes that in turn affect operation of the instrument and other factors. In the national network, both dichot and SSI hi-vol samplers are co-located at five sites: Saint John, Ottawa, Edmonton, Vancouver Rocky Point Park and Vancouver West 10th Ave. Based on samples collected between 1984-1993, mean ratios of [PM_{10}] measured with the dichot samplers to PM_{10} measured with hi-vol samplers ranged from 0.93 at sites in Edmonton and Saint John to 1.17 at the Vancouver West 10th Ave. site. Excellent correlation was found between the samplers (0.84-0.95). Relatively few inter-comparison studies have been conducted to date for $PM_{2.5}$ samplers.

A variety of analytical techniques are available for determining concentrations of inorganic and organic compounds from mass filter specimens of particulate matter. Some of these are non-destructible methodologies that leave the filter intact, enabling further chemical analyses; others are destructive of the filter. While these techniques are routinely used to help assess particle composition at sites across Canada, for the kind of detailed chemical analyses required for source apportionment studies (the attribution of PM at a site to specific sources), specialized particle sampling systems are necessary. One example of such a specialized particle sampler is the IMPROVE sampler, which has been used in intensive studies carried out at few urban and rural sites across Canada to study visual range and PM composition.

Indirect measurements of particulate matter have historically been made using two methodologies: the British Smoke Shade (BSS) sampler and the AISI sampler, which measures Coefficient of Haze (CoH). Both the BSS and the CoH techniques are based on optical properties of particles and are most sensitive to the sooty components of PM that fall in the size range of approximately $\leq$3.5–4.5 µm in diameter. The Beta Attenuation monitor, also known as the beta-gauge monitor, has been used in Europe and Japan for several years. Mass determination is based on the attenuation that a beta-ray particle undergoes as it passes through an exposed filter. The beta attenuation monitor can provide hourly PM concentrations, but is a very expensive instrument and has a number of operational constraints.

In contrast to the techniques described above, development of the Tapered Element Oscillation Microbalance (TEOM) offers the opportunity for much less labour intensive continuous measurement of PM concentrations; The TEOM sampler operates 24 hours a day and is automated. The TEOM operates on the principle that PM accumulations on a filter will result in changes to the oscillation frequency of a specially designed tube attached to the filter. Based upon the direct relationship between PM mass and oscillation frequency, the instrument‘s microprocessor computes the total mass accumulation on the filter, as well as the mass concentrations and mass rate, in real time. TEOM samplers can be fitted with either a PM_{10} or $PM_{2.5}$ sampling inlet, but not both at once.

As with manual samplers, issues of comparability of measurements have arisen. Some data from co-located TEOMS and PM_{10} hi-vols have shown good agreement between the two samplers, with correlation coefficients of 0.977 for 24 hour averages. However, other data seem to indicate that TEOM data are consistently lower than data from manual samplers, a difference that has been linked at least partially to the high sampling temperature used in TEOMS with a corresponding loss through volatilization of some volatile compounds, an issue of particular concern when sampling for $PM_{2.5}$.

Spatial Representativeness

In developing a monitoring network, selection of monitoring sites must be based on siting criteria that reflect the purpose of the data collection exercise and that will minimize undue bias in the resulting measurements. Nonetheless, individual sites are unique with respect to sensor location, surrounding structures, land use patterns, local meteorology etc. all of which will influence ambient pollution levels. As a result, it is recognized that monitoring data from fixed samplers may not portray pollution levels representative of the entire community. Furthermore, fixed monitors do not accurately reflect pollution levels that individuals may be exposed to (see section below on Human Exposure Assessment). To better characterize individual exposures, Personal Exposure Monitors (PEMS), sampling devices worn on the body, have been developed. Due to the nature of their application, however, the design of PEMS must meet a number of challenging criteria: low noise, light weight, portability, rugged design, ease of operation, adequate battery lifetime to meet sampling requirements and comparability with fixed site monitors.

Ambient Levels

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. In addition to the national network, British Columbia, Ontario and Québec operate particulate matter monitors.

PM 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.

PM levels in the atmosphere are a function of both natural and anthropogenic sources. 'Background' PM is generally defined as the distribution of PM concentrations that would be observed in the absence of anthropogenic emissions of PM and precursor emissions of VOC, NOx and SO_2. The actual magnitude of background PM for a given location is difficult to determine because of the influence of long range transport of anthropogenic particles and precursors. The range of expected background concentrations on an annual or long term basis is from 4 $\mu g/m^3$ to 11 $\mu g/m^3$ for PM_{10} and 1–5 $\mu g/m^3$ for $PM_{2.5}$ for remote sites in North America. 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, that can result in short term PM levels comparable to those in polluted urban atmospheres.

Twenty-four hour PM data typically exhibit a strongly skewed distribution dominated by a large number of low values. PM 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 multi-faceted and are related both to emission variability and to variations in geophysical variables such as mixed layer depth, wind speed and humidity levels.

Mean 24 hour PM_{10} concentrations across Canada range from 15–42 $\mu g/m^3$, with most sites in the range of 20-30 $\mu g/m^3$. These levels are substantially above background levels, indicating that anthropogenic activities make a significant contribution to ambient PM_{10} loadings. The highest 24 hour PM_{10} concentrations recorded by the NAPS monitoring network were observed in Québec and Ontario (at sites in Montréal, Windsor, Hamilton and Walpole Island) and at a single site in Calgary, Alberta. However, even within cities, there may be sites that experience comparatively low ambient 24 hour PM_{10} levels, as is the case in Montréal and Calgary. The three rural sites of Kejimkujik, Sutton and Egbert recorded mean 24 hour PM_{10} concentrations of 11, 11 and 17 $\mu g/m^3$ respectively, although observations were only available for 1992-1995 for Kejimkujik and Egbert and for May-September, 1993 for Sutton.

The season of maximum 24 hour 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 spring maximum of both mean and median PM_{10} concentrations and the upper quartile of the distribution, indicating that both average and extreme 24 hour PM_{10} concentrations are typically higher during the months of January, February and March. Sites in Ontario seem to exhibit daily summertime maximum PM_{10} concentration, which may reflect the greater abundance of secondary aerosols in the Windsor-Quebec City Corridor, where precursor concentrations are known to be high.

A hebdomadal cycle of 24 hour 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 concentrations (all sites). This suggests a substantial contribution to PM_{10} concentrations from transportation sources.

Yearly variations in PM_{10} concentrations during the 1984 to 1995 sampling period show an apparent decrease at most sites with a complete data record. The largest percentage 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}] on a national basis averaging 2 percent per year.

Twenty-four hour mean concentrations of $PM_{2.5}$ at the NAPS urban sites ranged from 8.5 to 20.2 µg/m^3. $PM_{2.5}$ concentrations are more spatially homogeneous than PM_{10} 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, Sutton and Egbert recorded mean $PM_{2.5}$ 24 hour concentrations of 7.0, 7.7 and 10.5 µg/m^3 respectively, although again, observations were only available for 1992-1995 for Kejimkujik and Egbert and for May-September, 1993 for Sutton.

The seasonal variability of $PM_{2.5}$ is more pronounced than that of PM_{10}; however, there is no discernible geographic pattern to this variability. The Montréal, Ottawa, Edmonton, Calgary and Vancouver/Victoria sites record higher $PM_{2.5}$ concentrations in the winter months and in particular during January and February. Other Ontario sites record the highest daily concentrations in the summer months with a peak median in August. The Maritime sites show variable seasonal variation in $PM_{2.5}$ concentrations with Saint John and Kejimkujik showing a strong summer maximum and Halifax a winter maximum.

Most urban sites show minimum 24 hour $PM_{2.5}$ concentrations on Sunday and maximum concentrations during the middle of the week. Again, this difference is magnified for roadway sites, where up to a 60% increase in $PM_{2.5}$ midweek relative to Sunday was noted (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 1984-93 showed a statistically significant ($p < 0.001$) decreasing trend in $PM_{2.5}$ on a national basis averaging 3.3% per year. For the Ontario sites, there was no significant change in $PM_{2.5}$ between 1987 and 1993.

In 1994, ten sites (all but two in the Lower Fraser Valley) reported hourly PM_{10} concentrations to the NAPS network using TEOM instruments. A maximum 1 hour PM_{10} concentration of 255 µg/m^3 was measured at the Abbotsford site (in the LFV) and a maximum 1 hour concentration of 204 µg/m at the Edmonton site. Analysis of the diurnal variations in PM_{10} have shown that a substantial increase in PM_{10} levels occur 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 (12-6:00 am.)

Relationships Among TSP, PM_{10} and $PM_{2.5}$ and Inorganic Constituents of PM

Fourteen urban sites in the NAPS dichotomous sampler network operating from 1986 to 1994 had simultaneous measurements of TSP, PM_{10}, $PM_{2.5}$ and sulphate (SO_4^{2-}). This data set is valuable in that it allows exploration of the composition of these different PM fractions at the 14 sites. On average across the 14 sites, PM_{10} accounted for approximately 50% of the 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 found on fine particles, where it comprised on average approximately 17% of the fine PM. However, considerable variation within and among sites exists for these ratios. The relationships between TSP, PM_{10}, $PM_{2.5}$ 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 comprised of very coarse PM).

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 (53% in this case).

These data, collected from 1984-1993 from 19 sites (16 locales), show that 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 ($\leq$2.5µm in diameter). 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 inter-quartile ranges (25th-75th percentiles).

There are relatively strong correlations (r^2) between PM_{10} and $PM_{2.5}$ at each of the19 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 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.

Comparisons of TSP, PM_{10}, $PM_{2.5}$ and sulphate mass distributions at sites across Canada have shown a couple of key trends. 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 expected 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 SO_2 emissions within and upwind of these two areas.

Comparisons of urban and rural sites in close proximity to one another demonstrate, not surprisingly, that urban PM concentrations are greater than rural ones, particularly for coarse PM. This is mirrored by an enrichment in urban areas in the concentration of all inorganic elements and ions assayed for. 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 (Ca, Si, NO_3^-, Fe, Al, Mg, Zn, Ti, Mn, V, Pb, Ni). 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 balance approach. Depending upon site, only about 37 to 61 percent 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, NO_3^- and NH_4^+ dominate the identifiable components of the fine PM mass, consistent with the results of many studies. Due to the increased importance of crustal material, a greater portion of the coarse particle mass (~70%) was explained by the inorganic constituents. This mass balance approach to partitioning PM mass into organic and inorganic fractions should be complemented by more detailed analytical studies of the carbonaceous component.

Human Exposure Assessment

The likelihood of an adverse response to particles is influenced by the degree of exposure, defined as any contact between a pollutant at a specified concentration and the outer (e.g., skin) or inner (e.g., respiratory tract epithelium) surface of the human body. Changes in the degree of exposure are influenced by the duration, magnitude and frequency of exposure. Inhalation is the only PM exposure pathway to considered in this assessment**.**

Ambient concentrations of particles are typically measured over a 24 hour sampling period, as described above. Over a 24 hour period, a person spends their time in many locations or microenvironments. For example, most people spend a great deal of time in indoor environments, at home and at work; some time each day in vehicles; and relatively little time each day outdoors. The proportion of time spent in different environments will vary with age, gender and day of the week. To the extent that microenvironmental PM concentrations are different from outdoor concentrations, population (and individual) exposures to PM will be different from those estimated from ambient monitoring data. The high correlations that have been found between personal exposure and indoor PM concentrations, combined with the amount of time spent indoors, indicate that indoor microenvironments are the most important contributors to PM exposure.

Indoor levels of particles are a function of: indoor sources, outdoor particle levels, the fraction of ambient air penetrating indoors, filtration, air exchange (e.g., older houses tend to be more leaky), particle decay and resuspension rates (e.g. from vacuuming or dusting). The latter source, the so-called “Pigpen” or “personal cloud” effect, helps explains why actual personal exposure is usually greater than indirect estimates combining indoor

and outdoor concentrations and time-activity information. The increase in particle concentration as a result of a person occupying the microenvironment is overlooked.

Several early studies indicated that penetration of ambient air into indoor environments is more effective for fine particles than coarse particles. Some more recent studies have indicated that penetration factors for both fine and coarse particles are close to unity. Nonetheless, current scientific thinking maintains that small particles penetrate indoors more effectively than larger particles. 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 will be found indoors under equilibrium will tend toward 50% or less, particularly in the winter. Once inside, the larger particles tend to settle out more quickly than smaller particles; however, the larger particles are more easily resuspended as a result of indoor activities.

Cigarette smoking has been identified as the major source of indoor particles (particularly fine PM, but also PM_{10}) in smoking households, raising indoor PM concentrations significantly above those in non-smoking households. A $PM_{2.5}$ concentration of 30 $\mu g/m^3$ corresponds to the impact of smoking approximately one pack of cigarettes per day. In non-smoking households, outdoor air is the major source of indoor PM levels. Other indoor sources of particles include such things as wood burning and kerosene stoves and heaters, animal dander, home care and personal care products and various indoor sources of mineral fibres. In general, large home-to-home variations in indoor particle concentrations can be expected. Some studies have reported mean indoor concentrations greater than outdoor levels, while others conclude just the opposite. In many cases, the range of particle concentrations indoors and outdoors is similar. However, in areas where outdoor levels are fairly high, indoor concentrations may well be less, whereas indoor concentrations may greatly exceed outdoor concentrations in areas where outdoor levels are relatively low.

Correlations between ambient PM data obtained from fixed ambient monitors (FAMs) and personal exposure data obtained from PEMs have been explicitly examined in many studies. Most of these reveal poor correlations and show, not surprisingly, that personal exposures are usually greater than (either indoor or) outdoor ambient concentrations. Furthermore, most studies report poor cross-sectional personal-outdoor correlations. If there is a lot of variability in particle concentrations from sources poorly correlated with FAMs, then the percent of variance in personal exposures that can be explained by the FAM data is likely to be small. Sampling error, a non-random sample, very strong indoor sources and personal activities all contribute to a poor correlation between FAMs and PEMs. However, for individuals who are not exposed to 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. Introducing microenvironmental exposures into a time-weighted average exposure estimate will theoretically improve estimates of personal exposure to total PM. Studies have shown this to be the case; however, such indirect personal exposure estimates are still likely to underestimate actual personal exposures.

Personal and population exposure models have been developed that combine ambient measurements of pollutants with information on age-specific time-activity and estimates of microenvironmental pollutant concentrations. A probabilistic PM_{10} exposure model was applied to Canadian data to produce estimates of distributions of 24 hour average personal, indoor, outdoor and in-transit PM_{10} concentrations. Predicted mean 24 hour personal PM_{10} exposure (across all regions and seasons) is 39 $\mu g/m^3$. Median personal PM_{10} exposure is predicted to be 31 $\mu g/m^3$. Comparing these results with exposure predictions based on urban NAPS site data (i.e., ambient PM data) clearly shows that ambient data underestimate average population exposures to total PM (mean and median urban ambient PM_{10} are approximately 28 $\mu g/m^3$ and 24 $\mu g/m^3$ respectively). Canadian estimates of exposure to $PM_{2.5}$ have not yet been predicted through exposure modelling. Given the current information, it is reasonable to conclude that the ambient data alone can represent the lower range in the distribution of total particle exposures. Further studies are required to improve various components of the exposure model, including better characterization of indoor PM sources and penetration rates of ambient particles indoors, specifically in cold climates, since air exchange rates are a function of ambient temperature.

Aesthetic Effects

Increases in the amount of fine particulate and gases in the atmosphere are associated with reductions in the ability of the human eye to see through the atmosphere or identify an object at a distance. Decreased visual range in parks and wilderness areas has become a concern as a result of the potential losses of tourism. The public may also consider visibility to be an indicator of overall air quality. Hence reduced visual range is perceived to indicate poor air quality, and consequently, quality of life in general may be negatively impacted.

Visual range (or conversely, light extinction) is a function of the light scattering and light absorption properties of gas molecules and particles. Fine particles ($PM_{2.5}$), and submicron particles in particular in the size range of

0.3–0.7 µm, are most effective in reducing visibility. Reduced visibility is usually more a function of light scattering than light absorption, except where there are particles (e.g. elemental carbon, otherwise known as "soot") or gases (e.g. NO_2) present in the atmosphere that are particularly effective at absorbing light.

It has been determined that there is a linear relationship between b_{scat}, a measure of light scattering, and fine particle mass, with b_{scat}/fine mass = 3.1 m^2/g. b_{scat} can also be related mathematically to visual range. Thus, visual range may be determined directly from fine particle mass if these values are known, or as a function of PM_{10} mass concentrations, if the ratios of $PM_{2.5}/PM_{10}$ are known. Coarse particles can also disrupt visibility but are typically one-half to one-third as effective as fine particles. In some areas, where coarse particle concentrations are unusually high, they may make significant contributions to reduced visibility.

Particle light extinction is modified by relative humidity. When relative humidity exceeds 70%, light scattering efficiencies begin to increase, as a function of increased water uptake by the particles. As humidity increases to 95% or more, water soluble components of the fine aerosol can swell to seven times their dry radius, dramatically increasing scattering efficiency. The effect of humidity is also very dependent on chemical and microphysical variables. Components of fine particles, such as sulphates (ammonium sulphates), nitrates (ammonium nitrate), organics, elemental carbon and soil dust, will vary in their ability to absorb water. The hygroscopic (water attracting) fraction of the aerosol will affect visibility to a greater extent in regions of Canada with higher relative humidity.

A number of studies have examined the relative contributions of different chemical species to visibility reductions. Typically, sulphates are the largest contributor to visibility reduction. Nitrates are also important to visibility reduction but are less efficient per unit mass than sulphates in light scattering. Even where fine mass has been shown to be dominated by a secondary organic component, sulphate and ammonium nitrate dominated light scattering (e.g. Pacific '93 field study in the Fraser Valley). Particle-phase organic compounds are relatively inefficient contributors to light scattering. Elemental (or black) carbon contributes to light absorption, as previously mentioned, and to a small extent, light scattering.

There is relatively little information on natural visual ranges in Canada. Estimates of visual range from three sites in different regions of Canada relatively unimpacted by anthropogenic PM were obtained using nephelometer measurements of light scattering (b_{scat}), and mathematical relationships between b_{scat} and visual range (b_{scat}/b_{ext} = 0.9; VR = 3.91/b_{ext}) with the following results: Waterton, Alberta – 210-350 km; Egbert, Ontario – 86-120 km; St. Andrews, N.B. – 185-210 km). $PM_{2.5}$ levels were then calculated using the relationship $b_{scat}/PM_{2.5}$ = 3.1 m^2/g (see above) to yield the following estimates of background $PM_{2.5}$: Waterton – 3.2–5.5 µg/m^3; Egbert – 9.7–13.0 µg/m^3; St Andrews – 5.5–6.1 µg/m^3. Southeastern Canada (SE Ontario) clearly has lower visibility and higher background $PM_{2.5}$ levels than do either western or eastern Canada. A 10% increase in $PM_{2.5}$ from background is used to define the level above which effects on visual range are noticeable. Thus in western and eastern Canada, using the upper ranges for background $PM_{2.5}$, impacts on visual range would be observed when $PM_{2.5}$ levels exceed 6–7 µg/m^3 and for southeastern Canada, 14 µg/m^3.

Visual range at urban sites can be estimated from known concentrations of $PM_{2.5}$ and PM_{10} and from the mathematical relationships between $PM_{2.5}$ and visual range (see above). On this basis, visual range at the NAPS urban sites has been estimated to range from 34 km at a site in Montréal to 73 km in St. John, New Brunswick. In general, urban sites in Ontario and Québec have lower estimated visual ranges (approx. 35-50 km) than do urban sites in either eastern or western Canada (approx. 45-70 km.). Likewise, visual range at the NAPS rural sites can be estimated, with the following results: Kejimkujik VR = 136 km; Sutton VR = 132 km; Egbert VR = 93 km. These values are based on average 24 hour $PM_{2.5}$ (or PM_{10}) levels, and thus represent average visibilities. In fact, visual ranges have been shown to vary with the season, as would be expected with changing PM concentrations and humidity levels.

In addition to the regional differences in visibility, there are regional differences in the public's response to changes in air quality. Differences in public perception of unacceptable visibility may be related to the nature of environments and vistas viewed, as the public may be less willing to accept degradation of a wilderness area than an urban environment. In a public perception study in Denver, Co. (U.S.), acceptable visibility as measured by visual range was approximately 50 km. Acceptable visibilities or acceptable changes in visibility in different regions of Canada still need to be determined.

As noted previously, a noticeable change in visibility is expected for a 10% change in fine particulate levels. Therefore, where fine particulate loadings are lower (e.g. rural areas), there will be a larger change in visual range for incremental increases in $PM_{2.5}$ than in (urban) areas with higher particle loadings. Given that mean $PM_{2.5}$ levels at NAPS sites across Canada range from about 10–20 µg/m^3, a 10% change in $PM_{2.5}$ levels (a noticeable change in visibility), corresponds to a 1–2 µg/m^3 increase in $PM_{2.5}$ levels. From observed relationships between

$PM_{2.5}$ and PM_{10} levels at Canadian rural and urban sites, a noticeable change in visibility would be expected to occur for a 1–2 µg/m^3 increase in PM_{10} levels at rural sites and a 2–5 µg/m^3 increase in PM_{10} at urban sites.

Recognizing the paucity and variability of data on natural background $PM_{2.5}$ levels and that it is not possible to define a single background concentration of $PM_{2.5}$ for all of Canada, it is recommended that no Reference Level for $PM_{2.5}$ based on visibility be identified at this time. However, it is emphasized that incremental impacts on visual range can be expected to occur when ambient $PM_{2.5}$ concentrations rise by 10% over existing concentrations. Given that natural visual range estimates for different regions of Canada are in the range of 86 to 350 km (see previous section), and that these visibility measurements give estimated natural levels of $PM_{2.5}$ from 5.5 to 13 µg/m^3 (upper estimates), (with western Canada and eastern Canada having natural levels of $PM_{2.5}$ around 5.5 to 6 µg/m^3, and southeastern Canada having higher $PM_{2.5}$ levels of around 13 µg/m^3), it is conservatively concluded that the level above which effects may be demonstrated on visual range as a function of $PM_{2.5}$ loadings within Canada is 6 to 14 µg/m^3. Any additional particle loading above these levels may be considered to reduce visual range. Since it is recognized that fine particles, in particular those in the 0.3–0.7µm size range, are responsible for reduced visibility, no Reference Level for PM_{10} is identified.

Environmental Impacts

The primary effects of particulate matter on vegetation are reduced growth and productivity due to interference with photosynthesis and phytotoxic impacts as a result of particle composition. The mechanisms of action are through smothering of the leaf; physical blocking of the stomata; bio-chemical interactions; and/or indirect effects through the soil. Particles make contact with vegetation surfaces in three ways: sedimentation, impaction and deposition. The relative efficiency of these methods will depend upon the plant or soil surface, the micro-climate and ambient (temperature and humidity) conditions. Given the limited amount of information available and specifically the lack of quantitative dose-effect information, it is not possible to define a Reference Level for vegetation and particulate matter.

The deposition of particulate matter on materials can reduce their aesthetic appeal as well as increase their physical and chemical degradation. The primary effects of particulate matter on materials are on the rates of corrosion and erosion, and soiling and discolouration. Particles may act as catalysts for the conversion of SO_2 and NOx to sulphuric acid and nitric acid which accelerate the chemical degradation of susceptible material surfaces on which they are deposited. Most information available is on the effects of particle exposure in combination with SO_2. Given the limited amount of information available and specifically the lack of quantitative dose-effect information, it is not possible to define a Reference Level for materials.

Human Health Effects

It has generally been accepted since the 1970s that there is an association between respiratory health and high levels of particulate air pollution. What has not been clear until more recently is that adverse effects also occur at ambient concentrations that are experienced today in North America and Western Europe. At issue still is to what extent the observed effects should be attributed to air pollution as a whole, comprised of multiple air pollutants, or rather to particulate matter specifically, and if so, to which component of particulate matter, either in terms of particle size or chemistry.

A decade and more has now passed since physiologically relevant measures of PM were incorporated into monitoring programs. Many studies have now been published that have used PM_{10} measurements and attempted to associate these levels with various health effects. Additionally, several studies that measure fine particles, as $PM_{2.5}$ or PM_5, British Smoke Shade (BSS), sulphate and /or particle strong acidity (PSA or H^+) have recently become available. The role of chemical speciation of particles has been investigated to only a very limited extent, although several epidemiological studies have used sulphate as the particle metric, and a small number of studies, particle acidity. Very little epidemiological testing of the role of ultrafine particles (< 0.1 µm) has as yet taken place since the current ambient monitoring data do not support such studies.

Epidemiological studies of the effects of PM on human health explore statistical associations between changes in ambient levels of PM and changes in the occurrence of cardiorespiratory health problems in the general population. Five basic health variables have been examined in epidemiological studies: mortality, hospital admissions/emergency department visits, respiratory health (symptoms, medication use, reduced activity days, elementary school absenteeism), pulmonary function, and cancer (the latter in only a few studies, incidental to other endpoints). All of the epidemiological studies are observational in nature; that is, the investigator has no control over the exposure or treatment of the subjects in the study, which differentiates them from the controlled human exposure studies discussed below.

Acute Effects

Daily or short-term variations in particulate matter, as PM_{10}, BSS, $PM_{2.5}$ or sulphate, were significantly associated with increases in all-cause mortality in 43 regressions carried out in 20 cities across North and South America and Europe. Virtually all of these studies demonstrated consistent associations between air pollution and acute mortality. These associations could not be explained by the influence of weather (temperature and humidity were most commonly found to have independent associations with mortality), season, yearly trends, day-to-day variations and variations due to holidays, epidemics, or other non-pollutant factors, since all the analyses looked for some or all of these potential biases and accounted for them in various ways in the analysis. Most of the studies also examined one or more gaseous pollutant in addition to PM.

The magnitude of the risk for PM_{10} was small, varying between 0.4% and 1.7% per 10 $\mu g/m^3$ increase, with an unweighted mean of 0.8% and a weighted mean of 0.5% for concentrations weighted by sample size averaging 25–78 $\mu g/m^3$ except in the two South American studies where concentrations averaged 82 and 115 $\mu g/m^3$. The results were highly consistent under differing PM_{10} exposure conditions. The magnitude of the increase was about the same for BSS as for PM_{10} but even fewer studies included more than one or two co-occurring gaseous pollutants.

Far fewer studies have investigated the relationship between $PM_{2.5}$ concentrations and mortality. In the best-conducted and most reliable study which examined $PM_{2.5}$ (and sulphate), an overall increase in mortality for six U.S. cities of 1.5% per 10 $\mu g/m^3$ increase in $PM_{2.5}$ was observed, with a range of 0.85 – 2.2% per 10 $\mu g/m^3$ for the six cities individually, at average $PM_{2.5}$ concentrations that ranged form 11–30 $\mu g/m^3$. The increase in $PM_{2.5}$ related risk of mortality is thus about twice that for PM_{10}. Although the magnitude of the mortality risk was greater in the six city study for sulphate compared to $PM_{2.5}$ (2.2% vs. 1.5% per 10 $\mu g/m^3$), the strength of the association was greater for $PM_{2.5}$ than sulphate.

While the increases in mortality risks associated with different particle metrics are small, they nonetheless signify substantial numbers of avoidable deaths due to the very large size of populations impacted by air pollution. These increases in relative risk due to air pollution have been observed at particulate matter concentrations well within the range of normal ambient concentrations and substantially below current standards and objectives (the current Canadian 24 hour acceptable TSP objective is approximately equivalent to a PM_{10} level of 60–80 $\mu g/m^3$ and to a $PM_{2.5}$ level of about 30–50 $\mu g/m^3$). Furthermore, there was little evidence in the PM_{10} and $PM_{2.5}$ data that the dose-response curve included a threshold; instead the response was observed to increase monotonically with increasing concentration, in the PM_{10} concentration range below 80–100 $\mu g/m^3$ and average $PM_{2.5}$ concentrations 14.7–21 $\mu g/m^3$. The lack of a threshold down to low concentrations suggests that it will be difficult to identify a level at which no adverse effects would be expected to occur as a result of exposure to particulate matter.

On a population basis, the hypothesis is that what we're seeing is exacerbation of pre-existing disease, or enhanced response of a subpopulation of sensitive individuals. Suggestions that the elderly are a susceptible population, more so than young adults, remains unsolved in the absence of pathology. However, overall the results suggested a surprisingly modest increase in relative risk for the elderly compared to the whole population. This does not support the suggestion that it is only the elderly who are being affected and dying from air pollution, and that their lives are being shortened by air pollution episodes by only a few days or weeks before they would have died anyway.

In all of the analyses that examined one or more air pollutants together in the same statistical model with particulate matter, the association of particulate matter with daily mortality was remarkably robust, despite the problems of disentangling the effects of PM from other air pollutants. This was the case for all four of the normally considered gaseous pollutants, SO_2, NO_2, CO and ozone. Moreover, in most locations, the magnitude of the PM association was greater than any other air pollutant considered, the exception being ozone, in a few cases. The magnitude, robustness and consistency of this association across so many locations with differing air pollutant mixtures indicates that PM is the best indicator of the air pollution effect on mortality, and is considered to give some support to PM of some kind, possibly acting together with other air pollutant(s), as a causal agent.

Particulate matter of some kind has been shown to have significant associations with increased hospitalizations in most of the 26 studies examined. All of the 16 studies that examined PM_{10} and one or more respiratory endpoints requiring hospitalization showed significant associations, varying between 0.45% and 4.7% per 10 $\mu g/m^3$ increase in PM_{10} at mean concentrations varying between 25 and 53 $\mu g/m^3$. Particulate matter was shown to have associations with cardiovascular disease in addition to its associations with respiratory disease, but the magnitudes of the cardiovascular associations were generally smaller than those for respiratory disease. Only three hospitalizations studies, two in Toronto and one in Montréal, directly examined the association between $PM_{2.5}$ and respiratory or cardiac effects, with an increase in respiratory effects observed in all three

studies. At mean $PM_{2.5}$ concentrations 12.2–18.6 µg/m^3, respiratory hospitalization and emergency room visits increased 2.5–9.6% per 10 µg/m^3 $PM_{2.5}$ increase. Five different air pollutants were considered in addition to PM in single, bivariate and multiple analyses in various locations. Particulate matter was the air pollutant with the most consistent and stable association with increases in hospitalizations. Ozone and carbon monoxide were judged to have independent associations as well as PM.

The strongest and most consistent association of particulate matter with respiratory hospitalizations is considered to be with sulphate. A 2.0% – 2.7% increase per 10 µg/m^3 increase in sulphate (co-regressed with ozone) was indicated in southern Ontario in the best conducted study of the series of eight examined. This was calculated to be equivalent to a 1.1% increase per 10 µg/m^3 increase in $PM_{2.5}$, based on site-specific monitoring and conversion factors. The correlations between ozone and sulphate were high (0.5 – 0.8) in all eight studies, which causes difficulties in separating out the effects of one from the other. Overall, there is good evidence for an association between sulphate and respiratory hospitalizations and sulphate is considered to be a good surrogate for fine particles from combustion sources. This does not mean that the sulphate is itself directly toxic however. An association between BSS (a somewhat smaller particle than PM_{10}) and respiratory hospitalizations exists, but is considered to be weak, presumably because this PM metric does not adequately represent secondary PM (much of which is colourless), being an optical measurement of dark coloured particles. Results for acidity (H^+) were inconsistent, with strong associations and high significance in some studies, and none in others.

No evidence for a threshold of effects for respiratory hospitalizations associated with particulate matter or other air pollutants was found at the low (10 to 100 µg/m^3 PM_{10}) concentration ranges examined. Curves appear to increase monotonically, with steep slopes at low concentrations and some suggestion of curvilinear responses (lower slope) at higher concentrations. The effect of age on hospitalizations or emergency department visits was examined in several locations, since historical data from episodes of high air pollution had strongly suggested that it was the elderly, the young and those with pre-existing respiratory and/or cardiovascular conditions who were responding to air pollution. While some studies found that the elderly were at increased risk compared to other age groups in the population, the increases observed in cardiorespiratory hospitalizations were by no means predominantly due to effects on the elderly. Children were also shown to be a high risk group for increased respiratory disease in a few, but not all, studies. Those with preexisting COPD were also identified as a susceptible subgroup.

In addition to its effects on mortality and hospitalizations, increases in particulate matter have been shown to cause small, reversible decrements in lung function in normal asymptomatic children, and in both adults and children who have some form of pre-existing respiratory condition, particularly asthma. These changes were often accompanied, especially in adults, by increases in symptoms such as chronic bronchitis or cough. 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 demonstrated to be associated with high ambient particulate matter, in some cases PM_{10}, and in others, $PM_{2.5}$ or other fine particulate components such as sulphate. Effects on respiratory health (lung function symptoms, absenteeism etc.), although much less serious than hospitalizations, and most certainly than mortality, nonetheless have the potential to impact much more of the population.

Longer Term and Chronic Effects

In contrast to the larger number of studies on daily variations in pollution associated with mortality and morbidity, relatively few studies are available that examine the effects of long term or chronic exposure on health endpoints. Such exposures, varying in duration between one and 16-20 years exposure, were associated with increases in mortality, respiratory disease symptoms, decrements in lung function and, possibly, with increases in lung cancer in both cross-sectional and the more powerful prospective cohort studies. In the first of two cohort mortality studies (six cities), average mortality was increased by 9%, 14% and 35% for each 10 µg/m^3 increase in PM_{10}, $PM_{2.5}$ or sulphate. In the same study, the probablility of survival over a 14 year period was reduced from approximately 88% in the least polluted city to 79% in the most polluted city. Based on the mean pollutant level across the six cities, lifespan was estimated to have been reduced by about two years over the 14 year period, an observation incompatible with suggestions that most or all the observed deaths related to PM are due to harvesting, (the accelerated deaths of persons already ill by a few days or weeks). In a larger study covering 151 cities, mortality increases associated with a 10 µg/m^3 annual increase in fine particle concentrations ($PM_{2.5}$ and sulphate) were lower, at 7% over a period of seven years.

The effects on mortality cannot with certainty be ascribed to a true chronic effect, since they could equally be the result of cumulative effects of daily variations in PM. However, the increases in incidence of chronic bronchitis and decreases in lung function, capacity, growth and development that were shown in cohorts of children

across North America after chronic or lifetime exposure to acidity, sulphate and fine particulate pollution, must be considered to be true chronic effects. There were indications also from a long term (20-25 years) cohort study in older adults that this increased incidence of disease, and probably also the reduced lung capacity that accompanies it, are carried over into adulthood as increased susceptibility to adverse effects of air pollutants. Although the development of lung cancer was also associated with fine particulate air pollution, the associations were weak by comparison with other lifestyle factors such as smoking.

Fine Particles versus Coarse Particles

The attributes that determine the toxicity of particles are poorly understood; however, particle size is known to be a very important determinant of inhalability and eventual deposition within the respiratory tract. In order to be inhalable and to reach the tracheobronchial area of the respiratory tract, particles must be smaller than about 10 µm in diameter (or up to 15 µm with mouth breathing). Particles 2–3 µm and smaller are able to reach the alveoli in the distal parts of the lung, and have been termed respirable (hence the general reference to $PM_{2.5}$ as respirable particles). The chemical composition of particulate matter has also been hypothesized to play an important role in its toxicity and certainly fine PM is far more complex chemically than coarse PM, which derives mainly from crustal material. Particle number, rather than mass, has also been suggested as an important determinant of toxicity, since large numbers of very small particles have a very high surface to volume ratio. They therefore present greater opportunities for surface adsorption of toxic substances such as heavy metals or PAHs, and subsequent deposition in the lungs.

The most valuable health studies for the purpose of attributing effects to a specific particle size and composition are those studies in which several particle metrics were employed, and particularly if these were not too highly correlated with each other. Typically, when PM_{10} and $PM_{2.5}$ concentrations are both measured, it is often not possible to distinguish the effects of one from another, because $PM_{2.5}$ is a part of PM_{10}, and the two are usually highly correlated ($r \geq 0.6$). However, the coarse fraction of PM_{10} (particles of size 2.5–10 µm) is often not highly correlated with $PM_{2.5}$ (particles $\leq$ 2.5 m) because it stems from different sources than the fine fraction. A few recent carefully conducted studies that included large databases have directly compared the coarse fraction of PM_{10} to fine particles.

The results of these and other studies have shown that in almost all cases, in both acute and subchronic mortality studies, fine particles as $PM_{2.5}$, had a stronger and more significant association with mortality than coarse particles, as either the coarse fraction, or PM_{10} and/or TSP. In only two cases was there an association between the coarse fraction and mortality, and even these were of questionable validity. Sulphate, which is part of the fine fraction of PM, appears to have as strong or stronger an association than $PM_{2.5}$ with increased mortality and hospitalizations. In one study in which sulphate and the non-sulphate fraction of $PM_{2.5}$ were directly compared, the non-sulphate portion was equally as, or more toxic than sulphate itself however, suggesting that sulphate would be an inadequate surrogate for all fine particle effects.

Overall, these studies support the hypothesis very well that the fine particle fraction is more important as a predictor of toxicity than the coarse fraction. However, coarse particles have not yet been eliminated from consideration, as there is some indication they may play a role in cardiovascular disease and COPD.

Experimental Data

Carefully controlled, quantitative studies of exposed humans in laboratory settings offer a complementary approach to epidemiological investigations. Advantage is taken of the highly controlled environment to identify responses to individual pollutants or sometimes pollutant mixtures and to characterize exposure response relationships where possible. In addition, such a controlled environment provides the opportunity to examine interactions with other environmental variables, such as exercise, humidity or temperature. Insofar as individuals with acute and chronic respiratory diseases can participate in exposure protocols, potentially susceptible populations may also be studied, although those with more severe preexisting disease and hence those most likely to be affected by air pollutants, are naturally excluded. Clinical studies also have other limitations: for practical and ethical reasons, studies must be limited to small groups, which may not be representative of larger populations; exposure must also be limited to short durations and to concentrations of pollutants that are expected to produce mild and transient responses; and exposures are often limited to a single pollutant, or to a very limited pollutant mix, which never replicates the complex mixture to which populations are actually exposed. Furthermore, transient responses in clinical studies have never been validated as predictors of more chronic and persistent effects.

Controlled human exposures to acidic and inert particles at relatively high levels compared to those generally experienced in the environment have not caused significant alterations in respiratory function in healthy individuals. However, acidity has been shown to slow mucociliary clearance of particles from small airways at concentrations as low as 100 µg/m^3. The clinical studies

identify asthmatics as a susceptible population, but not persons with chronic obstructive pulmonary disease (COPD), or the elderly, at least not for acidic particles. Asthmatics, especially children and adolescents, may experience adverse effects on airway function at concentrations experienced on occasion in ambient air (~35 $\mu g/m^3$ H_2SO_4 for 40 min).

Almost all of the human clinical studies have been based on observations of pulmonary function changes and subjective symptom reports. There are hardly any data published on particle-induced airway inflammatory responses. No data on changes to the cardiovascualr system have been documented. There is reason also to suspect that decrements in pulmonary function may not be a sensitive indicator for particle-induced lung injury. Moreover, based on the assumption that the response of pulmonary function to air pollutants may be a protective mechanism for the lungs from receiving further insults in the deep airways, failure of certain subjects, such as COPD patients, to have pulmonary function responses to particles might render these patients more vunerable to the pulmonary injury. Neither have the human clinical studies used particle generation systems that reflect the complexity of ambient particles.

Based on the extremely limited clinical database available on various species of particles, acidic aerosols produce the most significant bronchoconstriction, while the toxicity of sulfate is related to acidity *per se.* The toxicity of nitrates was not considered, since previous work had shown it not to exert effects on lung function at concentrations below 1000 $\mu g/m^3$ in clinical studies. Inert particles appeared to have no effect on lung function in either healthy or asthmatic volunteers in the few studies available. Very little work has been done on the effect of particle size specifically on airway mucociliary function, although limited studies have shown that fine particles (less than 2.5 µm) are cleared from the lung more slowly than larger particles, and that submicrometre (< 1.0 µm) particles clear very slowly indeed, taking more than one to two years in a few cases, especially in patients with obstructive lung diseases.

Overall, the clinical data do not lend much support to the observations seen in the epidemiology studies, particularly to the observations that high ambient particulate concentrations are associated with mortality within hours or a few days at most. Despite the fact that the ranges of particle concentrations tested usually exceed those experienced by the general population, little evidence for a dose response relationship has been documented in the clinical toxicological literature. Even at high particle concentrations in susceptible subpopulations, acidic aerosols have been found to produce only small decrements in lung function. The data do identify one susceptible subpopulation, asthmatics, who currently comprise five to eight percent of the Canadian population, a percentage that has been rising in the past decade in Canada as well as in other western countries.

The discrepancy between clinical and epidemiological data may be related to a number of factors, many of which are related to the general limitations of clinical studies described earlier. Furthermore, the pulmonary function parameters that are most often used in clinical studies may not be sensitive enough to indicate particle-induced adverse health effects. Particle size and type may also be an issue. In most clinical studies, artificial particles were used, which do not reflect the complexity of real world particles. The sizes of particles often used are above 0.5 µm, which does not include the full range of the particle size distribution found in ambient air. In particular, nanometre-sized ultrafine particles, that have been found in animal studies to induce acute pulmonary inflammation and death at very low concentrations, and are present in ambient air, have not been examined in clinical trials.

Studies on experimental animals (or tissue samples) have many of the same advantages and disadvantages of controlled human studies. A wide range of pollutants and concentrations can be tested under controlled laboratory conditions, and autopsies of study animals can be performed to investigate tissue damage from exposure to pollutants. However, for the most part, experimental studies involve well-defined particle species and do not by any means reflect the full range of complex ambient particle mixtures to which humans are exposed, a problem noted above with respect to Clinical Studies also. There is considerable uncertainty also in extrapolating results from animal inhalation studies and applying these results to humans for the purpose of risk assessment. Therefore, such studies are most appropriately used to explore mechanistic aspects of the toxicity of particles. A summary of the effects of PM from the animal toxicology literature is reviewed below prior to a discussion of mechanistic aspects of the toxicity of PM.

Studies using experimental animals have not provided convincing evidence of particle toxicity at ambient levels. Acute exposures (4-6 hour single exposures) of laboratory animals to a variety of types of particles, almost always at concentrations well above those occurring in the environment, have been shown to cause:

decreases in ventilatory lung function;

changes in mucociliary clearance of particles from the lower respiratory tract (front line of defence in the conducting airways);

increased number of alveolar macrophages and polymorphonuclear leukocytes in the alveoli (primary line of defence of the alveolar region against inhaled particles);

alterations in immunologic responses (particle composition a factor, since particles with known cytotoxic properties, such as metals, affect the immune system to a significantly greater degree);

changes in airway defence mechanisms against microbial infections (appears to be related to particle composition and not strictly a particle effect);

increase or decrease in the ability of macrophages to phagocytize particles (also related to particle composition);

a range of histologic, cellular and biochemical disturbances, including the production of proinflammatory cytokines and other mediators by the lungs alveolar macrophages (may be related to particle size, with greater effects occurring with ultrafine particles);

increased electrocardiographic abnormalities (an indication of cardiovascular disturbance);

increased mortality.

Bronchial hypersensitivity to non-specific stimuli and increased morbidity and mortality from cardio-respiratory symptoms occurs most likely in animals with pre-existing cardio-respiratory diseases.

Subchronic and chronic exposure tests involved repeated exposures for at least half the lifetime of the test species, often on a schedule that mimicked workplace conditions (e.g. 6h/day, 5 days/wk). Particle mass concentrations to which test animals were exposed were very high (> 1 mg/m^3), greatly exceeding levels reported in the ambient environment. Exposure resulted in significant compromises in various lung functions similar to those seen in the acute studies, but including also:

reductions in lung clearance;

induction of histopathologic and cytologic changes (regardless of particle types, mass, concentration, duration of exposure or species examined);

production of chronic alveolitis and fibrosis;

production of lung cancer (a particle and/or chemical effect).

The epidemiological finding of an association between 24 hour ambient particle levels below 100 µg/m^3 and mortality has not been substantiated by animal studies as far as PM_{10} and $PM_{2.5}$ are concerned. With the exception of ultrafine particles ($\leq$ 0.1µm), none of the other particle types and sizes used in animal inhalation studies cause such acute dramatic effects, including high mortality at ambient concentrations. The lowest concentration of $PM_{2.5}$ reported that caused acute death in rats with acute pulmonary inflammation or chronic bronchitis was 250 g/m^3 (3 days, 6 hr/day), using continuous exposure to concentrated ambient particles.

The extrapolation of results from experimental animals to humans is, however, fraught with uncertainty. This uncertainty relates to the dosimetry of the respiratory tract, differences in the sensitivities of specific target cells, differences in cell populations in the individual airway generations of animal species, differences in metabolic activity of lung cells, and differences in the lifespan between laboratory animals and humans. A recent comparative dosimetric analysis conducted by Miller and colleagues has yielded some interesting results, namely that, 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 concentration of PM. This trend is even more pronounced for individuals with compromised lungs (smokers, asthmatics, and patients with chronic obstructive lung disease) compared to normal subjects. Therefore, rats exposed to 1000–1500 µg/m^3 of particles may actually have received a level of particles equivalent to 120–150 µg/m^3 in humans. Given the caution which must be exercised in extrapolating risks from animals to humans, animal studies are best used to help elucidate the mechanism(s) of particle toxicity.

The animal studies clearly show effects on the lungs resulting from the inhalation of particulate matter, effects that can be attributed to a particle effect *per se*, as described above. No firm conclusions can be drawn, however, from the results of the numerous animal toxicology studies to answer the question of which particle type and size is most likely to cause the adverse effects. Particle size does appear to be a very critical character, however, with smaller particles having more pronounced effects, and particle size is believed to be the most important characteristic influencing deposition in the human respiratory system.

The significance of particle size is linked also to particle number and surface area. Ultrafine particles ($\leq$ 0.1 µm), by virtue of their greater numbers (2.4 million particles of 0.02 µm diameter correspond in mass to 1 particle 2.5 µm in diameter), greater surface area and slow clearance from the pulmonary interstitium, may be of particular toxicological importance and may also provide an answer to the puzzle of observed epidemiological effects at low particle mass levels. Ambient monitoring of the ultrafine particle mode of the urban aerosol is very difficult, and therefore, few data are yet available to carry

out epidemiological testing of the role of ultrafine particles in contributing to cardio-respiratory illness and death.

Chemical composition of the particle may also play a role. From the toxicological evidence, 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. The coarse particle mode is less likely to induce acute adverse responses than are either the fine or ultrafine modes, a fact attributed to both size and composition. However, these larger particles may well contribute in some way to effects.

The impact of interactions between different constituents of air pollution has been examined in animal studies to only a limited degree, mostly focusing on particulate and one gas-phase compound only. Such combined exposures have resulted in mixed responses, showing either no effect of the combination or some synergism depending on endpoint, but overall the results are equivocal. However, realistic environmental conditions are far more complex than those utilized in experimental settings. The actual mechanism of particle induced cardiovascular response is not yet clear. Some recent studies have suggested that it may involve the oxidation of low density lipoprotein by reactive oxygen species accompanying particulate pollution. Oxidized low density lipoprotein is known to be very cytotoxic.

Risk Characterization

The data on the health effects of particulate matter have been examined in animal toxicity studies, controlled human exposure studies, and human epidemiological studies. By far the most compelling evidence for adverse health effects of airborne particulate matter at currently experienced levels in the atmosphere has come from the epidemiological studies. The epidemiological data appropriately identify the same susceptible sub-populations that would be expected clinically (the elderly, children and those with pre-existing disease), with responses dependent on the health endpoint. Acute effects, as noted in the epidemiological literature, and ordered according to severity, are as follows:

increases in mortality due to cardio-respiratory diseases;

increases in hospitalizations for cardio-respiratory 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.

This listing of effects does not accommodate the potential numbers of people impacted. If this were considered, then the description of effects would assume a pyramid shape, with relatively few people impacted by the more severe health outcomes (mortality and hospitalizations) while much greater numbers of people may be impacted with greater frequency by the less severe health consequences (respiratory symptoms, absenteeism, reduced lung function). In addition, long term or chronic effects have been found to be associated with exposure to particulate matter, including:

reduced survival;

reduced lung function and capacity in children;

increases in the development of chronic bronchitis and asthma in some adults.

Overall, these findings from the epidemiological literature are not well supported by either the clinical or toxicological literature. Controlled human exposures to acidic and inert particles even at relatively high levels of PM compared to those generally experienced in the environment, have not been associated with significant alterations in respiratory functions in healthy individuals. Asthmatic individuals, especially asthmatic children and adolescents have, however, been identified as a susceptible subpopulation, responding to lower concentrations of acidic particles. The two major reasons for the lack of support by the clinical studies for the findings of the epidemiological studies are the extreme paucity of data on relevant exposures, and the ethical impossibility of investigating effects in precisely those persons most likely to be affected by air pollutants.

Animal toxicology studies have also demonstrated cardiorespiratory effects from acute, subchronic and chronic exposures to PM, but almost always at concentrations well above those occurring in the ambient environment. The toxicological evidence reviewed was able to demonstrate effects on the lung attributable to a particle effect, separate from effects related to the composition of the particle. The interpretation of results from experimental inhalation studies on animals and their significance for human exposures is fraught with uncertainty though. Therefore, the animal studies should be used to contribute primarily to an understanding of the mechanisms which lead to particle effects in humans.

Although the epidemiological studies are observational rather than experimental, they are nonetheless considered more relevant to a characterization of health risks associated with particulate air pollution than either animal toxicology or controlled human studies for several reasons:

they are the most direct way of assessing the adverse health outcomes of real world complex mixtures of pollutants to which people are exposed;

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 for ethical reasons;

population studies based on large administrative databases are able to demonstrate the impacts of pollution on public health, and even to enable some estimate of the costs of such impacts on society;

no extrapolation is necessary when assessing the effects on public health of a particular concentration of air pollutant as measured by the ambient monitoring network, despite our lack of knowledge about the exposures of each individual in the population (we need only know that the correlation is reasonable between the ambient monitor and the personal exposure).

These studies were conducted under a broad range of environmental conditions across many different geographical locales.

On Causality

In evaluating the epidemiological studies as a whole, a number of issues arise, key among them the issue of causality. Epidemiological studies do not themselves provide data on biological mechanisms that would explain the observed associations. Associations found in epidemiological studies between PM and health effects may reflect chance, bias or cause. A weight of evidence approach is used whereby multiple lines of evidence are brought together and duly considered in order to build a case for causality. On the basis of accepted criteria, the weight of evidence from the epidemiological literature that supports a causal link between particulate matter and adverse health effects is summarized as follows:

the **probability** of a relationship between PM and cardio-respiratory health has been ably established;

the **strength of the association** between exposure to PM and health outcomes can be considered relatively strong, since although the magnitude of the estimates of increased risk are generally small, they are remarkably stable among different studies and are often highly statistically significant;

a monotonically increasing (no threshold) **concentration-response curve** was observed from very low ambient levels up to much higher levels with remarkable consistency in many of the studies on acute and chronic mortality and hospitalizations;

the evidence is considered to be strong with respect to the **specificity of the effect** to respiratory and cardiac outcomes; non-respiratory effects are not associated with exposure to particulate pollution;

the **specificity of cause** is considered to be strong enough to conclude that particulate matter *per se*, rather than other pollutants or environmental variables, is associated with adverse health effects;

a logical **temporal relationship** exists, with exposure (e.g. daily peaks in PM), followed by effects (e.g. increased mortality and hospitalizations), although the rapidity with which mortality has been observed following incidents of high exposure remains a puzzle in terms of the mechanism of action of particles;

positive associations between particulate air pollution and cardio-respiratory related mortality and hospitalizations, and respiratory related health effects, have been **consistently** reported in numerous studies conducted under a broad range of environmental conditions in many cities on three continents, by a number of different investigators, providing a strongly **coherent** picture of the nature of particle-induced effects.

One of the most difficult questions has been, and continues to be, the role played by other gaseous pollutants (particularly SO_2, NO_2, CO and O_3) in the toxicity of particulate matter. Many of the available studies could not or did not consider several of these co-occurring gaseous pollutants. In analyses designed to help separate out the effects of one pollutant from another, the association of particulate matter with adverse health outcomes reported in the epidemiology literature was remarkably robust to inclusion (one at a time) of all four of the normally present gaseous air pollutants SO_2, NO_2, CO and ozone). Moreover, the magnitude of this association was often (but not always) greater than any of these other air pollutants individually or combined. The magnitude, robustness, and consistency of this association across so many locations with differing air pollutant mixtures supports the position that particulate matter of some kind is the best indicator for the effects of air pollution on adverse health outcomes. The question of which particle metric is the best indicator of toxicity remains unsettled, but current

evidence suggests that some form of fine particle is the best measure of particle toxicity, although in some locations, and with respect to some endpoints, coarse particles remain important and cannot yet be entirely dismissed.

The second critical outstanding issue with respect to causality relates to the biological plausibility of the effects of particulate matter on human health. When evaluating the effects of low levels of ambient particulates, we need to clearly separate acute adverse effects from chronic effects that reflect long term levels of air pollution. The association of mortality with daily variations in particulate air pollution presents difficulties in establishing a plausible mechanism that could explain these associations, particularly the very short lag period, or in some cases no lag, between the recording of elevated particle concentrations and the occurrence of increased mortality. Several hypotheses have been put forth to explain acute particle related mortality, and although the puzzle is by no means resolved, neither is it beyond explanation. The answer may likely involve exacerbation of preexisting disease conditions and evidence is mounting for a critical role for ultrafine particles on the strength of some recent toxicological evidence that has shown that mortality in rats can be induced after exposure to relatively low concentrations of these tiny particles.

These suggested biological mechanisms still require much more research and confirmation. However, they help close a major gap in our understanding, thus providing some support for the idea of causality. Precise mechanisms of action have yet to be established. It should be noted, however, that biological plausibility is not an absolute requirement for a conclusion of causality. Epidemiological observations have often preceded the biologic knowledge of the day, as evidenced by the example of smoking and lung cancer. A fundamental purpose of epidemiology is to establish a cause with enough certainty that it will be justifiable and highly appropriate to take action to mitigate effects on public health. This point has clearly been reached with respect to particulate matter.

Identification of Reference Levels

The Federal/Provincial Working Group on Air Quality Objectives and Guidelines, as part of its mandate, has identified “Reference Levels”, levels above which effects on human health and the environment can be demonstrated. The scientific information provided within the Science Assessment Document forms the basis upon which Reference Levels are established. Given the limited amount of information available, in particular the lack of quantitative dose-response information related to particle effects *per se*, it was not possible to define Reference Levels for either effects of PM on vegetation or on materials.

Since the effects of particulate matter on visibility are due primarily to the fine particle fraction, defining a Reference Level for PM_{10} is not appropriate. Defining a Reference Level based on $PM_{2.5}$ is extremely difficult for a number of reasons: 1) variability in perceived changes to visual range, 2) the lack of site specific data on fine particle mass in remote areas, and 3) an inability to define a single, natural background concentration for $PM_{2.5}$ across Canada due to regional variations in background $PM_{2.5}$. Therefore, at this time, no Reference Level for $PM_{2.5}$ has been identified to protect visual range.

For particulate matter and human health effects, the Reference Level is derived statistically from several studies and should be interpreted as a level above which there is confidence (statistical significance) in the dose-response relationship and the ability to provide some quantification of adverse endpoints. The Reference Level in this case should **not** therefore be interpreted as a threshold of effects. On the contrary, there is no clear evidence of a threshold level for the positive associations between particulate matter and both daily mortality and hospitalization rates. That is, any increase in ambient PM is associated with a statistical increase in mortality and hospitalizations, and thus, any Reference Level identified is acknowledged to lie within the “effects range”.

Mass concentrations of $PM_{2.5}$ and PM_{10} are recommended as the metrics of choice for PM Reference Levels. According to the weight of evidence presented in the Science Assessment Document, the strength and consistency of the epidemiological evidence for mortality and morbidity effects associated with exposure to both PM_{10} and $PM_{2.5}$ is remarkable, robust, consistent and compelling. $PM_{2.5}$ is most clearly associated with adverse health effects in a number of epidemiological studies, and has been shown to have a more robust association with mortality in most studies than other fine particle metrics (such as sulphate or acidity). $PM_{2.5}$ is also a more general surrogate for fine particle effects in all regions of the country than other measures (given the strong regional differences in sulphate and acidity levels). A Reference level for PM_{10} is also recommended given the consistent associations observed in epidemiological studies with mortality and hospitalizations and due to concerns over its link to particular endpoints such as chronic bronchitis and cardiovascular disease.

The recommended Reference Levels for PM_{10} and $PM_{2.5}$ (24 hour averages), statistically derived on the basis of several key epidemiological studies as detailed in the Science Assessment Document, are:

25 µg/m^3 for PM_{10}

15 µg/m^3 for $PM_{2.5}$

While the mortality and hospitalization endpoints have been emphasized in the derivation of the Reference Levels, because of the superior data on these endpoints, they are really only the tip of the iceberg with respect to PM induced human health effects. Other adverse effects such as bronchitis, reduced lung function, restricted activity, absenteeism and increased costs for medication are evident, and are occurring at ambient concentrations currently experienced within Canada.

As more scientific research is conducted, the Reference values will change, either because of better delineation of the adverse effects at lower concentrations, or because of better statistical analysis of the concentration-response relationship at low ambient concentrations.