PARTICULATE MATTER LESS THAN OR EQUAL TO 10 MICRONS (PM-10)

Comments on the CEPA PSL2 Draft Assessment Report on Particulate Matter Less than or Equal to 10 Microns (PM-10) were provided by:

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4. Ontario Forest Industries Association, Toronto, Ontario
5. Ontario Power Generation, Toronto, Ontario
6. Environmental Bureau, Policy Branch, Agriculture and Agri-Food Canada, Ottawa, Ontario
7. Tilbury Cement Limited, Delta, British Columbia
8. Canadian Vehicle Manufacturers Association, Toronto, Ontario
9. Natural Resources Canada, Ottawa, Ontario
10. Canadian Federation of Agriculture, Ottawa, Ontario
11. The Canadian Chamber of Commerce, Ottawa, Ontario

Comments and responses are summarized below by Environment Canada and Health Canada.

Comment	Response
The 60 day public comment period is too short.	The decision on the period of review (60 days) for this substance was a policy decision based on the 60-day review period specified in CEPA 1999 (Bill C-32) and that this is standard government practice for other similar types of consultations.
How will PM resulting from farming operations and forest fires be accounted for?	Addressing PM sources through risk management initiatives will be complex. Emphasis will be placed on areas of the country where ambient levels are highest. PM emitted from farming practices can be addressed to some extent through actions aimed at reducing resuspension of crustal material during tilling and other activities in the field as well as through emissions from off-road engines. The contribution of natural sources of PM to ambient levels is a known phenomenon that will be taken into account in developing and implementing actions to reduce the ambient levels of PM and

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	its precursors.
There is no clear definition of PM that meets the legal definition of a substance under CEPA.	**[1]** Under CEPA 1999, "substance" is defined as "any distinguishable kind of organic or inorganic matter, whether animate or inanimate, and includes: any matter that is capable of being dispersed in the environment or of being transformed in the environment into matter that is capable of being so dispersed or that is capable of causing such transformations in the environment; any element or free radical; any combination of elements of a particular molecular identity that occurs in nature or as a result of a chemical reaction, and; complex combinations of different molecules that originate in nature or are the result of chemical reactions but that could not practicably be formed by simply combining individual constituents, as well as: any mixture that is a combination of substances and does not itself produce a substance that is different from the substances that were combined; any manufactured item that is formed into a specific physical shape or design during manufacture and has, for its final use, a function or functions dependent in whole or in part on its shape or design, and; any animate matter that is or any complex mixtures of different molecules that are, contained in effluents, emissions or wastes that result from any work, undertaking or activity ". We therefore believe that PM10 meets the definition of "substance" in CEPA 1999.
The size of a particle should not be the only criteria used; the nature of the particulate matter should be considered instead.	Particle size is believed to be the most important characteristic influencing the deposition of the particles in the respiratory system. This in turn affects the toxicity. Smaller particles can deposit into pulmonary region where there is not much mucociliary movement to expel particles. Thus smaller particles are more readily retained in the lungs and are more likely than larger particles to exert toxicity. It is recognized that the components of ambient PM10 may vary in

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	different locations. However, studies worldwide have consistently demonstrated that PM10 and PM2.5 are associated with mortality and morbidity, in cities with various PM sources, and different PM components. The health effects manifested are in a PM concentration-dependent fashion. Thus, overall information supports the position that ambient PM10 and PM2.5 represent the components of PM which are toxic to human health.
Particulate matter, classified only by particle size, should not have been added to the PSL2, and therefore a means other than the PSL2 should be used to deal with respirable particular matter.	Since PM_{10} is considered to be a "substance" as defined in CEPA 1999 and was added to the Second Priority Substance List (PSL2), it was both necessary and acceptable to assess PM_{10} under the PSL2 program. The rationale for assessing PM10 provided by the Ministers' Expert Advisory Panel on PSL2 was as follows: "Exposure to respirable particulate matter in the Canadian environment is widespread. Sources include vehicle exhaust, construction, industrial air pollution and the bulk shipping of minerals. Small particles, irrespective of their origins, are trapped in the lungs. Effects associated with ambient exposure to respirable particulate matter include respiratory and pulmonary health dysfunction, which can lead to school absenteeism and increased hospital admissions. As assessment is needed to evaluate health risks".
There are knowledge gaps and uncertainties that are being addressed, which will deliver results in 3 to 5 years.	**[2]** The assessment on "toxic" is based on the current knowledge. Further information will provide evidence on the mechanism(s) of the health effects. However, the knowledge on mechanism is not a prerequisite for establishing a causal relationship. This has been proven by many examples in the history of medicine. For example, when Percival Pott discovered a link between the chimney sweep profession and scrotal cancer in 18th century, there was absolutely no knowledge of the mechanism, until

Comment	Response
	some 200 years later when polycyclic aromatic hydrocarbons in tar were found to be the cause of this cancer. Another good example is that when John Snow discovered the cholera epidemic of 1854 in England was associated with certain drinking water supplies, Louis Pasteur and Robert Koch hadn't developed bacteriology. It was discovered much later that the bacterium *Vibrio comma* causes cholera. These demonstrate that what is biologically plausible depends upon the biological knowledge of the day, and we should not wait without action until the biological cause is absolutely certain. In terms of PM, overall information supports the position that ambient PM10 and PM2.5 represent the components of PM which are toxic to human health.
The presence of particles in all kinds of consumer products makes implementation of risk management measures problematic.	The large number of sources emitting PM-10 make implementation of risk management measures challenging. However, because of the seriousness of potential health effects, investigation of options to reduce exposure to PM-10 is considered to be a high priority. The specific sources to be addressed under risk management will be decided in an open transparent multi-stakeholder process, in which a range of scientific, socio-economic and technical issues will be considered.
Other regulatory alternatives exist, especially the Canada-Wide-Standard (CWS) for PM.	Initiatives taken under CEPA 1999 to reduce exposure to PM will complement and be integrated with those related to the Canada-Wide Standard process.
The assessment inappropriately generalizes that all forms of PM10 have similar and definite health effects and that therefore all PM10 is toxic.	PM10 and PM2.5 are associated with mortality and morbidity, in cities with various PM sources, and different PM components, and with various combinations of gaseous pollutants. The health effects manifested are in a PM concentration-dependent fashion. Thus, overall information supports the position that ambient PM10 and PM2.5 represent the components of PM which

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	are toxic to human health.
It is inappropriate to duplicate the efforts of the CWS process while having clearly committed (through the Harmonization Agreement) to make best efforts to eliminate such duplication.	The Harmonization Accord and its subagreement provide a framework for jurisdictions to work together to develop and achieve common environmental targets. Each jurisdiction remains responsible for taking action using their respective tools and approaches. In the case of the federal government, the Canadian Environmental Protection Act, 1999 (CEPA,1999) is the most important tool available for reducing the ambient levels of PM. Due to the large number of activities leading to the release of PM and its precursors, it is envisaged that some actions will be led by the federal government, using the authority of CEPA 1999, while others will be implemented by provinces and territories using the most appropriate tools available to them.
Further study is required to address key scientific uncertainties prior to determining [a toxic declaration] [as per the 1998 National Research Council report].	See Response **[2]**.
Predicted health impacts may be significantly overestimated based on univariate analysis.	It is true that because of the intercorrelation between PM and the co-existing gaseous pollutants, a single-pollutant model may overestimate the effects of PM. However, studies (n=10) using multi-variate models have demonstrated that PM10 retained its association with acute mortality in analyses that adjusted for other pollutants, although the relative risks of PM10 were slightly reduced. For studies on hospitalizations and ER visits (n=18), and consistent with mortality observations, gaseous pollutants in multi-variate models reduced but did not abolish the significance of the risk attributable to PM. It should be noted that multi-variate models tend to underestimate PM effects due to the intercorrelation among co-pollutants.
Four causality criteria are weak in demonstrating an association between PM exposure and	1) Strength of association: As indicated in the Assessment Report, although the relative risks of

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mortality/morbidity.	PM are not as high as the risk of infectious diseases, the risks of PM are statistically significant. This indicates that there is little probability that the risks detected in the studies are due to pure chance. Moreover, given the fact that the whole population is exposed to PM indiscriminately, PM may in fact have adverse impact on a large portion of the population. 2) Specificity: The conclusion of a good specificity concerning PM health effects was drawn based on the observations that many studies investigated the associations of PM with non-cardio-respiratory as well as cardio-respiratory hospitalizations, and found that the associations exist only with cardio-respiratory diseases. For mortality, as discussed in the Report, the risk of respiratory death in some studies was not as high as the risk of total mortality, which can be explained by the fact that in these cities the mortality rates due to respiratory diseases were too low to give a meaningful result. 3) Temporality: This conclusion was drawn based on the observation that none of the studies showed an effect with a "reverse lag" time, or effects happening several days prior to the episode. For mortality, the lag time between a PM episode and an increase in mortality was very short (24 hr). This short lag time might be due to the susceptibility of certain subpopulations who already had pre-existing cardio-respiratory diseases and were particularly vulnerable to environmental changes. In the Report, we did note a possibility of "an acute coronary artery spasm and a subsequent massive myocardial infarction or a malignant arrhythmia" taking place during a PM episode. Indeed, several very recent studies have demonstrated that metals consistently present in ambient PM can cause acute cardiac arrhythmia and death in animals, especially in those with pulmonary hypertension. 4) Biological plausibility: Biological plausibility is

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	an important element, but not a must, for concluding a causal relationship. There have been many examples in the history of medicine to prove this. For example, when John Snow discovered that the cholera epidemic of 1854 in England was associated with certain drinking water supplies, Louis Pasteur and Robert Koch hadn't developed bacteriology. It was discovered much later that the bacterium *Vibrio comma* causes cholera. Another good example is that when Percival Pott discovered a link between chimney sweep profession with scrotal cancer in 18th century, there was absolutely no knowledge about the mechanism, until 20th century when PAHs in tar were found to cause this cancer. These demonstrate that what is biologically plausible depends upon the biological knowledge of the day, and we should not wait without action until the biological mechanism is absolutely certain. In terms of PM, overall information supports the position that ambient PM10 and PM2.5 represent the components of PM which are toxic to human health.
Regarding the emissions inventories, distinguish between "agriculture tilling" and "wind erosion" and clarify the proportion of PM attributed to each these sources.	In the national inventories it is not yet possible to differentiate between dust generated as a result of agricultural operations and wind generated dust.
Involvement of agricultural expertise	Any management initiatives related to farming will engage appropriate stakeholders.
Explain management options such as Canada-Wide Standards (CWS) and Strategic Options Process (SOP).	CWS (under the framework of the Canada-Wide Accord on Environmental Harmonization) provide a framework for jurisdictions to establish common goals and targets. SOP is an approach developed by the federal government to facilitate discussion with interested parties. Where there is an SOP underway, it will be used as the mechanism to engage stakeholders. It is conceivable that other SOPs could be developed in the future if appropriate.

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The assessment lacks balanced presentation of available scientific information.	The Assessment Report is based on the Science Assessment Document (SAD) produced in support of the National Ambient Air Quality Objectives (NAAQO), which presented all the relevant health effect studies, be they statistically significant or non-significant. The document compared the merits and weaknesses in each study and reached a conclusion. Concerning the publications the stakeholder attached, papers by Gamble (1996), Lipfert & Wyzge (1997), McClellan & Miller (1997), and Moolgavkar & Luebeck (1996) were not included in the SAD because they are review papers rather than original research papers. The paper by McCunney (1997) was not published in a peer-reviewed journal and is not an original research paper, and was appropriately not included in SAD. Other papers were published in late 1997 to 1999, which are beyond the cutoff time point for the assessment. However, in the only original research paper (Burnett et al. 1999) published after the cutoff point, the results show that PM is significantly associated with increased respiratory infection and cardiac diseases after controlling for gaseous co-pollutants.
The assessment overly relies on modeled estimates.	The conclusion of the Assessment Report is largely based on epidemiological evidence. Epidemiology is a scientific discipline, which has been well established and extensively used throughout the history of medicine in research on infectious diseases, cancers, cardiovascular diseases, occupational health, etc., to investigate causal relationships, often before a biological mechanism is found. Epidemiological studies consistently demonstrated significantly positive associations between PM and health effects worldwide, in a concentration-dependent fashion, with a logical temporal relationship. Epidemiological models were used in these studies in order to control for the confounding effects of seasonal cycles, epidemics, weather

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	and co-pollutants, etc. It should be noted that these models (which stringently control for confounders) may result in an underestimate of PM effects, because of the intercorrelations between environmental factors.
The assessment depends upon epidemiologically insignificant associations to create appearance of causality; lack of biological plausibility.	In terms of experimental studies to obtain biological plausibility, see Response **[2].**
The assessment does not persuasively argue in favour of toxic declaration.	The Section "**3.1 CEPA 1999 64c: Human health**" has been revised.
The definition of PM as toxic is based upon size.	As discussed in Section 2.5, TOXICOKINETICS, the sizes of the PM determine the deposition of the particles in the respiratory system to exert their toxicity. Smaller particles can deposit into pulmonary region where there is not much mucociliary movement to expel particles. We recognize that the components of ambient PM10 may vary in different locations. However, studies worldwide have consistently demonstrated that PM10 and PM2.5 are associated with mortality and morbidity, in cities with various PM sources, and different PM components. The health effects manifested are in a PM concentration-dependent fashion. Thus, overall information supports the position that ambient PM10 and PM2.5 represent the components of PM which are toxic to human health.
There are current scientific uncertainties and ongoing research.	See response **[2].**
Consider the U.S. actions regarding PM NAAQS and California's actions on diesel PM.	The decision to remand the new PM Standards in the United States is an internal US issue that has no bearing on the responsibilities and authority of the Ministers of Health and Environment to take action to protect Canadians from the harmful impacts of air pollution. The mandate of the Expert Panel established by the Royal Society of Canada is focused on the tools and methodologies used to estimate the cost and benefits associated with taking action to

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	implement the recommendations of the Expert Panel and to review the Standards in 3 to 5 years. It is the view of the federal government that the uncertainties and data gaps identified are not sufficient to postpone taking action to protect the health of Canadians from air pollution caused by PM and its precursors. The overlap between the PM2.5 and PM10 standards is recognized and no PM-10 standards were proposed to the Ministers.
There are deficiencies in the report regarding, biological plausibility, exposure, and treatment of confounders.	**[3]** Regarding **biological plausibility**, see Response [2]. Regarding **exposure**, we acknowledged in the Report that there is a degree of uncertainty when using fixed ambient monitors (FAM). However, studies on personal exposure to PM have demonstrated that personal exposure data correlate well with data from FAMs (Section 3.1.1), indicating that FAM data can serve as adequate surrogate for population exposure. Regarding treatment of **confounders**, all recent time-series studies have employed models such as linear filters, Fourier series or logically smooth regression techniques, to control for confounding of seasonal cycles, epidemics and weather. For gaseous co-pollutants, studies (n=10) using multi-variate models have demonstrated that PM10 retained its association with acute mortality in analyses that adjusted for other pollutants, although the relative risks for PM10 were slightly reduced. For studies on hospitalizations and ER visits using multi-variate models (n=18), and consistent with mortality observations, gaseous pollutants reduced but did not eliminate the significant risk attributable to PM. It should be noted that multi-variate models tend to underestimate PM effects due to the intercorrelation among co-pollutants.
Lack of stakeholder involvement (specifically agriculture), particularly regarding emission sources; clarification of process from this point	Stakeholders will be engaged in the development of risk management initiatives aimed at addressing PM. The federal government will

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on and involvement of stakeholders in risk management discussions.	continue to engage stakeholders through the various consultation processes - through SOPs or other processes it develops. Farming activities contribute to both direct release of PM through tilling practices as well as through equipment (diesel and gasoline engine emissions). Any initiatives aimed at addressing farming practices will take place in the context of appropriate agricultural stakeholder engagement.
The level of confidence in the scientific evidence needs to be improved (ie: clinical studies, weak epidemiological associations, lack of individual exposure data, plausible biological mechanisms and co-occurring pollutants).	For comments on individual exposure data and co-pollutants, see Response **[3].** For biological plausibility, see Response **[2].** Clinical studies provide evidence for biological plausibility and for susceptible populations. As discussed in the Assessment Report, clinical studies have shown that asthmatic children are more responsive to acidic PM at concentrations close to ambient levels. There had been very limited clinical studies, by the time we finished the Assessment Report, focusing on the mechanisms of PM effects. However, given the examples we presented in Response **[2],** what is biologically plausible depends upon the biological knowledge of the day, and we should not wait without action until the biological plausibility has been completely elucidated. For PM, overall information supports that ambient PM10 and PM2.5 are good surrogates for whatever components of PM are toxic to human health. Regarding **epidemiological associations**, while the relative risks for PM are not as dramatic as some infectious diseases, they are statistically significant, even after adjusting for confounding factors. Moreover, results from epidemiological studies worldwide have shown consistent and coherent results, with a concentration-dependent fashion, and a logical temporal relationship. All these elements considered, we believe there is a causal relationship between PM and human health.

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The report does not address the specific toxicity of the different chemical species of PM.	The assessment focuses on ambient PM10 whose components may vary in different locations. However, studies worldwide have consistently demonstrated that PM10 and PM2.5 are associated with mortality and morbidity, in cities with various PM sources, and different PM components, and with various combinations of gaseous pollutants. The health effects manifested are in a PM concentration-dependent fashion. Thus, overall information supports the position that ambient PM10 and PM2.5 represent the components of PM which are toxic to human health.
Conclusions with regard to Paragraphs 11a and b are not clear. Can it be concluded that PM10 is not toxic under Paragraphs 11a and b of CEPA?	In the last paragraph on page 3 of the report, it is clearly stated that only effects under Paragraph 11c (Section 64c under CEPA,1999) are addressed. No conclusions about "toxic" under Paragraphs 11a and b can be drawn based upon the information presented. Formal assessments of risk under Paragraphs 11a and b were not conducted since information was limited and data available suggested that key concerns for PM related to human health. In practice, it has not been considered necessary to reach formal conclusions under all parts of Section 11 (now Section 64 CEPA, 1999), if the conclusion under at least one of them is "toxic". This approach is reasonable in this case, since regardless of what might have been concluded under Paragraphs 11a or b, it is expected that actions to reduce exposure will ultimately be driven by human health concerns.
Incomplete review regarding source attribution, particularly from biogenic VOC emissions.	An intensive review of source attribution was not within the scope of this assessment. For additional information, the reader is referred to the WGAQOG PM SAD (1999) and the Addendum (2000). These are available at: www.hc-sc.gc.ca/ehp/ehd/catalogue/bch.htm
There is no linkage between ambient trends and corresponding health outcomes, with an	The ambient PM trends discussed by the stakeholder show long term variations of ambient

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evaluation of those aspects of PM responsible for the health outcomes.	PM concentrations. Time-series mortality and hospitalization studies focus on acute effects of PM, i.e., the effects of daily PM fluctuation such as an episode. These studies demonstrate significant changes of acute mortality and hospitalizations with an increase in PM concentrations. Studies on long term effects examine the relationship between PM and health effects after adjusting for confounders and co-variates, and conclude that PM concentrations are significantly associated with health effects. Because there are so many factors contributing to changes of mortality and hospitalizations including social-economic and environmental factors etc., a direct comparison between the trends of PM2.5 concentrations and health outcomes without teasing out other factors will not give meaningful results. That is why in epidemiological studies stringent measures have been applied to control for confounders.
There are contradicted claims on page 18 and page 40 concerning additive effect of PM and ozone.	The issues discussed on pages 18 and 40 are quite different. On page 40, the conclusion is drawn that given the entire database of evidence, the most robust conclusion is that PM measures provide the best explanation of the health effects seen in the several hundred studies reviewed. The earlier discussion related to the combined effects of ozone and sulphuric acid observed by one researcher. While acid has been discussed as a culprit in some air pollution studies, it should not be equated with particulate matter.
The definition of PM as a substance should not be based upon size.	In terms of the definition of a "substance", see Response **[1].** Regarding PM size, as discussed in Section 2.5, TOXICOKINETICS, the sizes of the PM determine the deposition of the particles in the respiratory system. Smaller particles can deposit into pulmonary region where there is not much mucociliary movement to expel particles. Thus smaller particles are

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	more readily retained in the lungs and more likely than larger particles to exert toxicity.
The drawing a conclusion of toxic is not appropriate.	The most convincing component of the health science database for PM is the consistent associations between ambient levels of this pollutant and adverse health effects. This association has been found to be consistent and robust in studies on six continents, in a wide variety of cities, in a concentration-dependent manner. The associations between PM and health effects still exist after adjusting for co-pollutants. This variety of situations indicates that despite variations in the local pollution mix, the local sources of PM, and the specific characteristics of population and climate, PM as a size fraction is an appropriate characteristic. Thus, overall information supports the position that ambient PM10 and PM2.5 are representative of the toxic components of PM.
Report does not provide enough solid scientific information necessary to support future risk management activities.	The PSL assessment report is aimed at determining whether or not a given substance is toxic as defined under CEPA, 1999. The development and implementation of management initiatives will require further analyses that will be discussed in a multi-stakeholder environment.
The treatment of uncertainties is inadequate (fixed monitor data, biological plausibility, covariates, a study by Burnett et al. 1998).	Regarding gaps on actual human exposure, we acknowledged in the Report that there is a degree of uncertainty when using fixed ambient monitors (FAM). However, studies on personal exposure to PM have demonstrated that personal exposure data correlate well with data from FAMs (Section 3.1.1), indicating that FAM data can serve as adequate surrogate for population exposure. Regarding biological plausibility, see Response **[2].** Regarding confounders and covariates, see Response **[3].** In the study (Burnett et al., 1998) cited by the stakeholder, the authors focused on the gaseous pollutants in the multi-variate models. The very limited PM risk estimates presented in this paper

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	were obtained using models different from those for gaseous pollutants. The authors acknowledged that due to the limitation of PM exposure data, they could not directly analyze PM risks in conjunction with gaseous pollutants. Although the authors concluded that the gaseous pollutants examined in this study may contribute to greater risk than do PM2.5 or sulfate, they did not eliminate the notion of PM risk.
A revision of the description linking PM to lung cancer is needed.	Assessment on lung cancer has been revised.
There are multiple sources, multiple characteristics, multiple challenges with PM.	Regarding multiple sources and multiple characteristics of PM, studies worldwide have consistently demonstrated that PM10 and PM2.5 are associated with mortality and morbidity, in cities with various PM sources, and different PM components, and with various combinations of gaseous pollutants. The health effects manifested are in a PM concentration-dependent fashion, and with a logical temporal relationship. Overall information supports the position that ambient PM10 and PM2.5 adequately represent the components of PM which are toxic to human health.
Are Canadian-based control measures the answer to public health improvements?	Regarding Canadian-based control measures and cross-boundary transport of PM, we recognize the need to provide better estimates of daily and annual long-range transport of PM from US before deciding what local control measures should be in effect. However, the control policy for PM is out of the realm of the Assessment Report.
How much reliance should be placed on fixed ambient monitors?	In terms of using exposure data from fixed ambient monitors (FAMs), see Response **[3].**
Is the claim that no "threshold" dose exists justified?	Regarding "non-threshold" for PM effects and mechanism(s), see Response **[2].** The "non-threshold" effects have been observed in many epidemiological studies. We recognize that there

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	has been a wealth of toxicological data emerging since our cutoff time point, to investigate the mechanisms of PM toxicity. Effects observed in experimental animals include acute cardiac arrhythmia, inflammation, and acute death in animals with pre-existing pulmonary hypertension, when exposed to ROFA or concentrated ambient PM or transition metals often present in ambient PM. Transition metals have been specifically pointed out to be one of the putative culprits for these effects. However, given the complexity regarding the choice of the biomarkers for effects, the timing of the effects and the species differences in physiology and biochemistry, there has not been convincing evidence published so far, including those cited by CANTOX, to conclude whether the PM or the components of PM are "threshold toxicants" or "non-threshold toxicants". Therefore, we shall base our assessment on the knowledge of the day. It should be noted that in the Assessment Report we presented the studies reporting PM effects with a threshold as well. Should any new data emerge with significant implications, they will be incorporated into the document.
Could human behaviour patterns explain the association between PM and health endpoints?	Human behavior patterns: In most of the time-series epidemiological studies (for acute effects) reviewed in the Assessment Report, researchers have realized the impact of several cyclic patterns such as the day-of-week patterns of both health endpoints (and PM pollution) on the true associations between PM and health endpoints. In order to control for the confounding effect of these patterns, various filtering methods such as linear filters, Fourier series, or locally smooth regression techniques, have been used extensively in time-series studies. To demonstrate a simple example, suppose on a certain day (say a workday) the PM10 level is 100 mg/m^3 and the average PM concentrations in the 31-day period around this day is 80

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	mg/m^3. Further consider a day (say a weekend) in which the average PM value is 40 mg/m^3 and is surrounded by a 31-day average PM level of 20 mg/m^3. In this example, a 31-day span of the smoother is used. The filtered data represent differences between the observed data and an average of data on the specified day and 15 days in the past and 15 days in the future. Likewise, the health endpoint can be filtered using a similar method. Thus, the filtered values on both days are 20 mg/m^3, and yet the absolute value of PM is quite different. These filtering functions remove any low frequency cyclic patterns and non-cyclic trends in both the event and PM data that might obscure the true association between daily variations in both time series.
Are the statistical analyses and techniques embraced by the PSL assessment report justified?	Regarding the statistics used in epidemiological studies: 1) Dose-response curve: A monotonic response does not contradict curvilinear response, as the term "monotonic" means that the trend of a response does not change to an oppose direction at any point, not like a letter U, even when the dose-response curve reaches a plateau. E.g., a line in the enclosed Figure 1 is considered both monotonic and curvilinear. As reviewed in the Assessment Report, except one study, all the other time-series studies have shown a monotonic increase in health effects in response to the increase of PM, be the relationship linear or curvilinear. These studies expressed data at 95% confidence interval ($P<0.05$), and did not try to extrapolate the response at low concentrations if the data did not show a statistical significance. Statistical uncertainty: For exposure uncertainty, see Response **[3].** For statistical methods, time-series analyses have been a well-established method and have been used extensively for acute effects in various fields. Smooth techniques, parametric and non-parametric modeling etc., as

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	discussed above, are used to remove the impact of cyclic variations of data. For control of cyclic data variations, see the response above. For control of co-pollutants, see Response **[3].** For distributional properties of the data, Poisson distribution has been used most frequently due to the low mortality and morbidity rates. However, studies have shown that in practice data are not sensitive to the choice of distribution, Poisson or Gaussian (Kinney et al., 1995; Lipfert, 1994).
PM and Lung cancer: Is the association justified?	Regarding lung cancer, see above Response.
What should be the next steps?	Regarding the next step, the Assessment Report has pointed out the uncertainties, which indicate the directions of further research. Meanwhile, Canada Wide Standards will be under review periodically in order to incorporate new information and to be revised when necessary.