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BURNCO AGGREGATE PROJECT

Particulate Matter Literature Review

Submitted to:

Mr. Derek Holmes
BURNCO Rock Products Ltd.
1A-2670 Emerson Street
Abbotsford, BC V2T 3S6

REPORT



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Acronyms and Abbreviations

CI	confidence interval
DNA	deoxyribonucleic acid
i.e.	<i>id est</i> (that is)
KS	Kansas
MA	Massachusetts
MO	Missouri
NA	not assessed or not available
NF	no association found between 24-hour PM ₁₀ concentrations and mortality
NO ₂	nitrogen dioxide
PM _{2.5}	particulate matter with a mean aerodynamic diameter of 2.5 microns or smaller
PM ₁₀	particulate matter with a mean aerodynamic diameter of 10 microns or smaller
TN	Tennessee
TSP	total suspended particulate
US	United States
WHO	World Health Organization
WI	Wisconsin

Units of Measure

%	percent
±	plus/minus
µg/m ³	micrograms per cubic metre
µm	micron



1.0 INTRODUCTION

This appendix provides the particulate matter literature review conducted in support of the human health risk assessment being completed as part of the overall Environmental Assessment Certificate Application/Environmental Impact Statement (hereafter referred to as the EA) for the Proposed BURNCO Aggregate Project (the Proposed Project).

2.0 POTENTIAL HEALTH EFFECTS ASSOCIATED WITH PARTICULATE MATTER BASED ON TOXICOLOGY STUDIES

The primary toxicological responses to environmental exposure to airborne particulate matter are respiratory and cardiovascular effects (WHO 2006). A brief summary of the toxicological studies related to health effects associated with exposure to particulate matter is provided below. The World Health Organisation (WHO) states that the risk for various health outcomes increases with exposure and that a threshold below which no adverse effects are expected is not likely to exist (WHO 2006). Given that a threshold has not been identified, WHO (2006) suggest that setting a standard needs to be aimed at achieving the lowest particulate matter concentration possible given the local context and priorities of the region. Further discussion of available information on local background particulate matter concentrations for the region are presented in the Air Quality Section (5.7).

The toxicological mechanism associated with particulate matter is typically inflammation caused by a primary response of alveolar macrophages and pulmonary epithelial cells (Schwarze et al. 2006). The response involves the release of signalling (i.e., cytokines and chemokines) and adherence molecules which mediate a complex interaction between the epithelial cells, the alveolar macrophages and other immune cells such as neutrophils and T-cells (Schwarze et al. 2006). The type of cytokines and chemokines released as a result of exposure determines the resulting health effect. For example, allergic asthma caused by attraction of eosinophils involves different mediators than those that attract neutrophils involved in non-allergic inflammatory disease (Schwarze et al. 2006). The recruited immune cells may release secondary cytokines as well as reactive oxygen species, lipid mediators and toxic proteases that can cause epithelial damage leading to the subsequent release of additional cytokines or chemokines thus increasing or prolonging the inflammatory reaction (Schwarze et al. 2006). The increased inflammatory reaction can ultimately cause chronic inflammation resulting in respiratory illness.

Particulate matter is also believed to be cytotoxic contributing to cell death which can also cause inflammation, leading to the development of acute and chronic lung disease. Particulate matter that is generated from diesel exhaust has been shown to cause lung cancer. The mechanism by which the particulate matter causes cancer is related to direct interaction of the metabolized particle components and products of oxidative stress with deoxyribonucleic acid (DNA) and the subsequent formation of DNA adducts and mutations (Schwarze et al. 2006). The inflammatory process can also contribute to progression of lung cancer but the mechanism(s) by which this occurs are not yet fully understood.



Particulate matter can also cause cardiovascular disease, and several mechanisms for this are possible. One explanation is that the fine particulate matter and substances that are bound to it, such as metals and inflammatory products, enter the blood stream and interact with the heart (Schwarze et al. 2006). In addition, experimental studies have shown that inhalation of particulate matter components or induced substances can alter heart rate, increasing the likelihood of a heart attack (Schwarze et al. 2006). Inflammatory pathways have been identified as being important in various aspects of the development of cardiac disease but the critical pathway has not been identified.

Particle size and substance composition (i.e., metal content) have also been shown to be important contributors of the toxicological mechanism for particulate matter (Stanek 2011). Experimental studies have shown that ultrafine particles are particularly toxic due to a high surface area to mass ratio and surface reactivity. However, other experimental studies have also shown larger particles are equally and often more potent than fine fraction particles, and this may be due to the importance of particle composition (i.e., size effects may not override the potential for a higher concentration of toxic or inflammatory components present in larger particles). The available epidemiological studies indicate that there is greater evidence for particulate matter with a mean aerodynamic diameter of 2.5 microns or smaller ($PM_{2.5}$) and $PM_{2.5-10}$ to cause mortality than particulate matter with a mean aerodynamic diameter of 10 microns or smaller (PM_{10}). However both the coarse and fine fractions seem to contribute to morbidity (Stanek 2011).

Schwarze et al., (2006) conclude that particle size alone is not the critical determinant of particulate matter induced health effects. The concentration of particulate matter component such as metals, soluble organic compounds, and sulphates are also important considerations, but the contribution of these particulate matter components to toxicological effects are generally not well understood. Rohr and Wyzga (2012) examined the data from particulate matter component-based studies and found that most studies had significant findings for specific components of particulate matter but not for particulate matter concentrations. The authors indicate that this demonstrates that particulate matter alone does not drive health responses. Overall the studies demonstrated the need for a greater understanding of the carbon-containing particulate matter components (elemental and organic carbon) because there are data to suggest that these are strongly associated with adverse health outcomes.

A limited number of epidemiological studies indicate an association between metals content and air pollution-related mortality. The available data for particulate matter related morbidity indicate that iron, copper, nickel, vanadium, and zinc are the primary metals of concern (Schwarze, 2006). The Rohr and Wyzga (2012) literature summary suggests that aluminum, silicon, nickel and vanadium are closely associated with adverse health impacts of particulate matter, but it is also possible that other metals could contribute to health effects.

The role of organic compounds in particulate matter induced disease for the general population based on epidemiological studies is not well understood. Information is available from occupational studies for carcinogenic effects associated with organic chemicals in particulate matter. Although experimental studies indicate that soluble organic compounds contribute to the inflammatory response related to exposure of particulate matter from diesel related sources, information is not yet available about the specific organic compounds in particulate matter effects. Information about the relative contribution associated with soluble organic compounds and other components of particulate matter (i.e., metals) is also limited.

Experimental and epidemiological studies are not currently consistent with respect to whether sulphates contribute to particulate matter induced health effects.



Biological agents such as mold and endotoxin are also known to cause allergic reactions. However, epidemiological studies associated with the biological components of particulate matter are quite limited (Schwarze et al. 2006). One study in California indicated that pollen present in particulate matter had an effect on people suffering from asthma. Schwarze et al. (2006) indicate that an experimental study has shown a greater cytokine reaction induced by PM₁₀ relative to PM_{2.5} and that it may be related to the higher endotoxin component of the coarse particulate matter.

Schwarze et al. (2006) indicate that epidemiological studies conducted in areas where crustal particles dominate have caused adverse health effects associated with respiratory morbidity. However, the authors indicate that the majority of the available experimental studies have been conducted with quartz and asbestos and may not be completely representative of coarse particulate matter associated with windblown dust or road abrasion particles. Therefore, although epidemiological studies indicate potential health effects associated with crustal particles, the experimental studies are not yet available to determine the relative importance of the mineral components of particulate matter.

Overall, the mechanisms through which particulate matter influences health, and the role of particle size and composition in causing adverse health outcomes, are still not fully understood (Englert 2004). Current research generally suggests that the composition of particulate matter would be a better predictor of adverse health effects than the mass of particulate matter (Stanek et. al. 2011). Lippmann and Chen (2009a, b) reach similar conclusions in their review of the literature, and state that combustion-driven components of particulate matter that have a high redox potential (e.g., metal and polycyclic aromatic hydrocarbons [PAH] components of particulate matter) are likely to be the primary contributors to adverse health effects.

3.0 EPIDEMIOLOGICAL STUDIES ASSOCIATED WITH PARTICULATE MATTER FROM CRUSTAL DERIVED SOURCES

Air emission sources associated with the Proposed Project will include land clearing, aggregate extraction and processing, conveying materials from the pit to the processing plant, transferring material to a barge, and tug transportation. The activities related to land clearing and conveyance of material from the pit to processing plant that generate fugitive particulate matter emissions from crustal sources are expected to be major contributors to the particulate matter predictions for the Proposed Project. Therefore, a literature review of epidemiological studies was completed to determine if a quantitative relationship can be determined relating exposure to particulate matter from crustal sources and human health effects. Epidemiological studies that evaluated crustal sources were most typically associated with dust storms or other natural events such as volcanic eruptions or geographic locations where there was an absence of industrial activities, thereby leading to the assumption that the particulate matter is generally from crustal rather than combustion sources.

Multiple studies suggest that mortality and morbidity are more closely linked to particulate matter from combustion rather than crustal sources (i.e., dust storms, re-suspended dust from road traffic, agriculture, and mining), unless the particulate is derived from geologic sources and contains high concentrations of metals. Dust events containing high coarse matter particulate as the result of high wind speeds tend to have reduced concentrations of fine particulate and other combustion-related particulate and measurement of dust storm events allows the authors to attribute these events to a crustal source (i.e., non-pollution events). As a result dust storms are often studied from the perspective of the effect of crustal sources on health effects (Schwartz et al. 1999; Staniswalis et al. 2005).



Coarse particulate matter has an effect on mortality, most predominately in arid regions where concentrations of coarse particulate matter are high (Ostro et al. 1999; Staniswalis et al. 2005). Of the available studies on the effects of coarse particulate matter on health, few studies have analyzed coarse and fine particulate matter jointly.

In studies of morbidity, coarse particulate matter generally had a similar effect on asthma and respiratory admissions as fine particulate matter (Anderson 2007). The available data suggests that coarse particles have the potential to cause respiratory and cardiovascular morbidity. The majority of the studies conducted on the effects of coarse particulate matter derived from crustal sources indicate that its effect on mortality and morbidity is less than that of PM10 originating from combustion sources. Stanek et al. (2011) conducted a review of the existing health literature on PM2.5 to determine if there was any consistent association between adverse health outcomes and certain source factors and concluded that the current state of research does not provide enough information to unequivocally relate specific health outcomes to isolated factors or sources.

The literature identified that relates mortality and morbidity associated with crustal sources of PM2.5 and PM10 is summarized below. Acute effects from crustal sources were the only studies identified (i.e., chronic studies related to crustal sources were not identified as these types of studies are often reliant on high winds or dust storms which do not occur continuously).

3.1 PM2.5

3.1.1 Mortality

Information about mortality as the result of exposure to PM2.5 from crustal sources is summarized in Table 9.1-E-1.

Table 9.1-E-1: Summary of Mortality Studies for PM2.5 Originating from Crustal Sources

Table with 3 columns: Location, United States (Various Locations), and United States (Palm Springs and Indio, California). Rows include PM2.5 Concentration during Dust Storm Event, PM2.5 Concentration on Control days, Change in PM2.5 concentration, Increase in Monthly Mortality, All-cause daily increase in mortality, All-cause (3-day lag), All-cause (2-day lag), Cardio-respiratory, and Reference.

Notes:

(a) Confidence intervals (2.5% and 97.5% are 4.2% and 4.7%, respectively).

NA = not assessed or not available; µg/m³ = micrograms per cubic metre; % = percent; PM2.5 = particulate matter with a mean aerodynamic diameter of 2.5 microns or smaller.



Epidemiological studies concerning particulate matter from crustal sources are limited. Laden et al. (2000) found that increased mortality was not associated with fine particulate matter from crustal material. Fine particulate matter from a variety of combustion and vehicular sources in addition to crustal material was categorized by determining the elemental composition of the fraction to identify source related factors. Several factors were identified including a silicon factor in fine particulate matter from crustal material, a lead factor in fine particulate matter from vehicle exhaust, and a selenium factor in particulate matter from coal combustion. Mortality rates were compared to source factor concentrations for six eastern cities in the United States (Watertown, MA, Kingston-Harriman, TN, St. Louis, MO, Portage, WI, and Topeka, KS). The results showed that a 10 microgram per cubic metre ($\mu\text{g}/\text{m}^3$) increase in $\text{PM}_{2.5}$ from mobile (i.e., vehicle) sources accounted for a 3.4 percent (%) increase in daily mortality (95% CI: 1.7 to 5.2%). An equivalent increase in fine particles from coal combustion sources accounted for a 1.1% increase in mortality (95% CI: 0.3 to 2.0%). $\text{PM}_{2.5}$ crustal particles were not associated with daily mortality. The authors concluded that fine particles from mobile and coal combustion, but not crustal sources, are associated with increased mortality.

Fuentes et al. (2006) found a statistically significant positive association between crustal $\text{PM}_{2.5}$ and incidence of mortality from natural causes (i.e., deaths other than from accident, violence or suicide), independent of $\text{PM}_{2.5}$ from other sources at various locations in the United States in June 2000. The percent increase in monthly mortality was 4.5% (95% confidence interval [CI]: 4.2% to 4.7%) per 10 $\mu\text{g}/\text{m}^3$ increase in crustal $\text{PM}_{2.5}$. Other components of the speciated $\text{PM}_{2.5}$ such as sulphate, nitrate and ammonium also showed elevated increases in monthly mortality ranging from 6.6% to 7.5% per 10 $\mu\text{g}/\text{m}^3$ increase in crustal $\text{PM}_{2.5}$. Fuentes et al. (2006) indicated that in the Western United States, crustal particulate matter and nitrate had the greatest impact on mortality relative to the other components of $\text{PM}_{2.5}$.

Ostro et al. (2000) assessed 10 years (1989 to 1998) of daily data on mortality and PM_{10} concentration for two locations (Palm Springs and Indio) in the Coachella Valley, California. In addition, during the final 2.5 years in this same period, daily $\text{PM}_{2.5}$ data were collected to allow for the assessment of size-specific impacts. An association between $\text{PM}_{2.5}$ concentrations and cardiovascular mortality was not found; however, a less robust association between $\text{PM}_{2.5}$ and all-cause mortality was found (Ostro et al. 2000). All-cause mortality was found to be statistically significant only on the fourth day after the dust storm (i.e., four day lag), and the authors suggest that the reduced number of $\text{PM}_{2.5}$ samples in comparison to PM_{10} samples and low $\text{PM}_{2.5}$ concentrations during monitoring (24-hour $\text{PM}_{2.5}$ concentrations ranged from 13 to 17 $\mu\text{g}/\text{m}^3$ in the Coachella Valley) may have impacted statistical power (Ostro et al. 2000). The relative risk was 1.04 (95% CI: 1.00 to 1.08) per 9.0 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$.



3.2 PM₁₀

3.2.1 Mortality

Information about mortality as the result of exposure to PM₁₀ from crustal sources is summarized in Table 9.1-E-2.

A study by Schwartz et al. (1999) examined whether coarse particle concentrations are associated with mortality. Coarse particulate matter (PM₁₀) measurements were collected during 17 dust storm events in Spokane, Washington between 1989 and 1995 and these data were correlated with deaths in the same area. Arid agricultural areas are the primary source of the dust and dust storms occur in the fall after crops have been harvested. Control days were chosen from the same day of the year in a previous year when a dust storm did not occur. The study found that mean 24-hour PM₁₀ concentrations were 263 µg/m³ on exposure days (i.e., dust storm events) compared to concentrations of approximately 42 µg/m³ on control days. The study found that mortality was not elevated on dust storm days in Spokane as compared to control days, and concluded that control of airborne particles should focus on combustion particles not crustal particles if the goal is to reduce human health effects.

Table 9.1-E-2: Summary of Mortality Studies for PM₁₀ Originating from Crustal Sources

Location	Spokane, Washington	Taipei, Taiwan	Coachella Valley, California	Coachella Valley, California	Taipei, Taiwan	El Paso, Texas
PM ₁₀ Concentration during Dust Storm Event (24-hour average) (µg/m ³)	263	101.1	NA	NA	NA	NA
PM ₁₀ Concentration on Control days (24-hour average) (µg/m ³)	42	73.3	NA	NA	NA	NA
Change in PM ₁₀ concentration (24-hour average) (µg/m ³)	221	27.8	10	24.6	64.1	10
Daily Increase in Mortality	NF	NA	1%	3% ^d	NA	1.7% to 2.1%
All-cause (3-day lag)	NF	1.7%	NA	NA	NA	NA
All-cause (2-day lag)	NF	3.4% ^(a) ; 5.3% ^(b)	NA	NA	7.7% ^(c)	NA
Cardio-respiratory	NF	3.7% ^(a)	1.2%	NA	2.6% ^(c)	NA
Reference	Schwartz et al. 1999	Kwon et al. 2002	Ostro et al. 1999	Ostro et al. 2000	Chen et al. 2004	Staniswalis et al. 2005

Notes:

- (a) All age groups combined
- (b) Over 65 years
- (c) Not statistically significant
- (d) increased risk of death from cardiovascular disease

NF = no association found between 24-hour PM₁₀ concentrations and mortality, NA = not assessed or not available; µg/m³ = micrograms per cubic metre; % = percent.



Slaughter et al. (2005) conducted a study to determine whether there is an association between different size fractions of particulate matter and cardiac and respiratory mortality and morbidity. The study examined the association between four fractions of particulate matter (PM₁, PM_{2.5}, PM₁₀ and PM_{2.5-10}) and carbon monoxide and hospital visits/admissions in Spokane, Washington for respiratory and cardiac conditions and mortality between 1995 and 2001. The study did not find any associations with respiratory or cardiac hospital admissions or deaths with any fraction of particulate matter. However, the study did note a greater effect on respiratory health from fine versus coarse particulate matter.

Kwon et al. (2002) studied the effects of wind-blown dust originating from the arid deserts of China and Mongolia on daily mortality for people aged under 65 years and in elderly people (greater than or equal to 65 years) in Seoul, Korea from 1995 to 1998. The dust is composed primarily of crustal sources and ranges between 1.35 micrometers (µm) and 10 µm in size, but may also contain chemicals from combustion sources in Eastern China. The association between dust storm events and daily death counts was assessed using regression analysis which was adjusted for temporal trends and weather variables. The assessment was based on 28 dust storm days observed in Seoul, Korea between 1995 and 1998. The average 24-hour PM₁₀ concentration observed during the dust storm was 101.1 µg/m³ compared to 73.3 µg/m³ on control days (i.e., non-dust storm days). For all-cause mortality (all ages combined) an increase of 1.7% (95% CI: -2.8% to 6.5%) was observed with the 3-day moving average. The risk was highest two days after the event as an increase of 3.4% (95% CI: -0.5% to 7.4%) was calculated by the authors. The results are based on an increase of 27.8 µg/m³ in 24-hour PM₁₀ concentration over that recorded on control days. The analysis of subjects older than 65 years indicated a higher risk 5.3% (95% CI: 0.3% to 10.5%) 2-days after exposure to the dust storm event, which was considered to be significant. For all ages combined, the association with cardio-respiratory mortality was highest on the event day (3.7%; 95% CI: -2.7% to 10.5%) and decreased thereafter. For all other non-accidental causes of mortality, negative and non-statistically significant associations were found with exposure to the dust storm events.

Ostro et al. (1999) examined the role of PM₁₀ in relation to daily mortality in the Coachella Valley, California, where geological particles comprise a significant percentage of the total particulate mass throughout much of the year, especially during wind storms. Analyses were conducted using daily data on mortality from 1989 to 1992 for several pollutants and meteorological variables. Outcome variables included several measures of daily mortality, including all-cause, cardiovascular, and respiratory mortality, and counts of death for those above age 50. The study noted statistically significant associations between PM₁₀ (two- or three-day lags) and each measure of mortality. A 10 µg/m³ change in daily PM₁₀ was associated with an approximately 1% increase in mortality, which is of similar magnitude to particle-associated impacts identified in urban areas.

Ostro et al. (2000) repeated the earlier investigation conducted by Ostro et al. (1999) using 10 years (1989 to 1998) of daily data on mortality and PM₁₀ for two locations (Palm Springs and Indio) in the Coachella Valley, California. Outcome variables included several measures of daily mortality, including all case (minus accidents and homicides), cardiovascular and respiratory mortality. The average 24-hour PM₁₀ concentrations were 29.8 and 47.4 µg/m³ for Palm Springs and Indio, respectively. Ostro et al. (2000) found an association between PM₁₀ and cardiovascular mortality. Ostro et al. (2000) found a statistically significant positive association between PM₁₀ and incidence of death from cardiovascular disease. The relative risk was 1.03 (95% CI: 1.01 to 1.05) per 24.6 µg/m³ increase in PM₁₀.



The authors concluded that although this study was carried out in an area in which PM₁₀ is strongly correlated with the coarse fraction, the magnitudes of the associations are similar to those observed in numerous areas in which variability in particle concentration is due primarily to changes in combustion-related fine particles.

Staniswalis et al. (2005) performed a study in El Paso, Texas to determine if the mortality that occurred during 1992 and 1995 was associated with the temporal variability of PM₁₀ levels within a 24-hour period. In addition, the researchers investigated the association of PM₁₀ with total mortality in relation to wind speed, assuming that at high speeds wind was composed primarily of coarse particulate matter from re-suspended dust whereas at low wind speeds it was mostly fine particulate matter from urban sources. The 24-hour average PM₁₀ concentration ranged from 0.2 and 133.4 µg/m³. In this area, PM_{2.5} is about 25% of the total PM₁₀ concentrations and hourly PM₁₀ concentrations have been noted to peak in the evenings during still-air conditions. Between 1992 and 1995, the daily natural death rate for the area ranged from 1 to 21 deaths/day with an average 8.5 deaths/day.

A principal component analysis showed that 40% of the total variation in daily PM₁₀ concentration was explained by a peak occurring near 8 pm (20:00) and that the daily average only accounts for 28% of this variation. Using the results of the principal component analysis (hourly data), an increase of 2.06% total non-accidental mortality per 10 µg/m³ increase in PM₁₀ concentrations three days after the event (3-day lag) was found. In contrast, a non-significant increase of 1.7% in total mortality for 10 µg/m³ increase in the 24-hour mean PM₁₀ concentration (3-day lag), was found using 24-hour average PM₁₀ levels. This study therefore suggests that using average PM₁₀ over a 24-hour period can result in an underestimation of the health effects.

The mortality risk was derived based on high wind speed and low to mid wind speed conditions, and the differences between those wind conditions were examined. A high wind speed at night (greater than 7.6 metres per second [m/s]) was significantly associated with a 10% lower risk of mortality in the three days following high wind speed event as compared to low and mid wind speed conditions. This suggests that crustal particles may have a weaker negative impact on health outcomes as compared with ultrafine particle exposure. Coarse particles in El Paso are believed to contain deposited metals from historic mining and smelting, and the mineral content of the coarse particulate matter may affect the mortality results presented here, when compared to crustal particulate matter from other locations.

Pope et al. (1999) examined the weak association between particulate matter (PM₁₀) concentrations and mortality in Salt Lake City, Utah; however, a reasonably strong association was found in a neighbouring community (Provo, Utah). The study found that Salt Lake City is subject to significantly more episodes of dust storms than Provo. Exclusion of data (24-hour PM₁₀ measurements) that were associated with dust storm events and the use of particulate matter measurements from multiple monitors resulted in a revised association that was similar to that for Provo, Utah. The study concluded that particulate matter from combustion sources was more closely associated with increased mortality than wind-blown particulate matter, which is high in coarse crustal material.



Chen et al. (2004) also studied the effects of Asian dust storm events from 1995 to 2000 on daily mortality in Taipei, Taiwan. The mean number of deaths due to non-accidental causes was 27, while the mean numbers of deaths due to cardiovascular and respiratory causes were respectively 7.31 and 2.8. Increases of 4.92% and 2.59% per $64.1 \mu\text{g}/\text{m}^3$ increase in PM_{10} were observed for all-cause mortality and cardiovascular mortality, respectively, two days after the dust event, which had caused an increase of $68.14 \mu\text{g}/\text{m}^3$ in PM_{10} . The highest effect was found for respiratory mortality with an increase of 7.66% per $64.1 \mu\text{g}/\text{m}^3$ increase in PM_{10} one day after the event. However, all estimates were non-statistically significant.

In Finland, PM_{10} particles originate from re-suspended coarse road dust and the spreading of sand on streets in the spring. Penttinen et al. (2004) examined the association between air particulate concentrations in air in the greater Helsinki area for all-cause, respiratory and cardiovascular mortality between 1988 and 1996. A measure of the blackness of total suspended particulates (TSP) was used as a surrogate for fine and combustion-derived particles to evaluate the impact of fine particulate, $\text{PM}_{2.5}$, on mortality. The TSP blackness was also highly correlated with carbon monoxide indicating that combustion-derived particles were a major contributor to this measure. The study identified positive but non-significant relationships between PM_{10} and total and cardiovascular mortality for all age groups. Median 24-hour average concentrations for TSP, PM_{10} and $\text{PM}_{2.5}$ were found to be 57, 28 and $15 \mu\text{g}/\text{m}^3$, respectively while maximum concentrations ranged from 234, 122 and $55 \mu\text{g}/\text{m}^3$, respectively. Positive and significant associations were identified for PM_{10} and respiratory mortality. Increases of respiratory mortality of 3.94% (95% CI: 0.01% to 7.87%) on the same day of measurement, 3.96% (95% CI: 0.11% to 7.81%) on 1-day after measurement (1-day lag) and 2.13% (95% CI: 0.03% to 4.22%) and four days after measurement (4-day lag) were noted. Results were not consistent for the association with TSP concentrations and TSP blackness with mortality. Overall, this study provided little evidence of a role for coarse particulate matter from re-suspended road dust in increased mortality; results suggested that combustion-derived particles are more strongly associated with mortality than crustal-derived particles.

3.2.2 Morbidity

Information about morbidity health outcomes as the result of exposure to PM_{10} from crustal sources is summarized in Table 9.1-E-3.

Several authors conducted studies to assess the possible effects of windblown dust storms originating in the deserts of Mongolia and China (Asian Dust Storm) on hospital admissions for various health conditions for residents in Taipei, Taiwan, during the period from 1996 to 2001. Mean concentrations of 24-hour PM_{10} during dust storms was $111.68 \pm 38.32 \mu\text{g}/\text{m}^3$ compared to the mean concentration during comparison days (usually the same day of the week, but a week before and a week after a dust storm) of $55.43 \pm 24.66 \mu\text{g}/\text{m}^3$. The study authors found that there may not have been enough statistical power to detect associations resulting from inadequate sample size of hospital admissions for the various health endpoints on dust storm event days. The studies are summarized below in greater detail.

Chen and Yang (2005) conducted a study to assess the possible effects of Asian Dust Storm on hospital cardiovascular disease admissions of residents in Taipei, Taiwan, during the period from 1996 to 2001. A 3.65% increase in the risk of cardiovascular disease admissions during the Asian Dust Storm events (1 day following the day of the Asian Dust Storm) was observed; however, this increase was not statistically significant.



Andersen et al. (2007) found a statistically significant positive association between crustal PM₁₀ and the incidence of hospital admission for cardiovascular disease, independent of PM₁₀ from all other sources in Copenhagen. The relative risk for cardiovascular disease was 1.051 (95% CI: 1.018 to 1.084) per 1.8 µg/m³ increase in crustal PM₁₀. Yang et al. (2005a) conducted a study to assess the possible associations of Asian Dust Storm on the hospital asthma admissions of residents in Taipei, Taiwan, during the period from 1996 to 2001. The association between dust storms and asthma admission was prominent two days after the Asian Dust Storm event. The estimated relative risk was 1.08 (95% CI: 0.97 to 8.76); however, this increase was not statistically significant.

Yang et al. (2005b) designed a study to assess the possible associations of Asian Dust Storm on the hospital stroke admissions of residents in Taipei, Taiwan, during the period from 1996 to 2001. The study results indicated a statistically significant association between Asian Dust Storm events and daily primary intracerebral hemorrhagic stroke admissions three days after the event (relative risk of 1.15; CI: 1.01 to 10.10). Yang et al. (2005b) also found a positive but not significant association between Asian Dust Storm events and ischemic stroke admissions three days following the dust storms, which was due primarily to PM₁₀.

Yang et al. (2009) conducted a study to assess the possible associations of Asian Dust Storm on the hospital admissions for congestive heart failure for residents in Taipei, Taiwan, during the period from 1996 to 2001. The association between dust storms and congestive heart failure admission was prominent one day after the Asian Dust Storm event. The estimated relative risk was 1.11 (95% CI: 0.99 to 1.25); however, this increase was not statistically significant.

Chang et al. (2006) conducted a study to assess the possible effects of windblown dust storms originating in the deserts of Mongolia and China on the daily clinical visits for allergic rhinitis of residents in Taipei, Taiwan, during the period of 1997 to 2001. The study found that the mean concentration of PM₁₀ during dust storms was 110.37 ± 37.86 µg/m³ compared to the mean concentration during comparison days (usually the same day of the week, but a week before and a week after a dust storm) of 61.73 ± 30.22 µg/m³. A 19% increase in the risk of clinical visits for allergic rhinitis during the dust storm events (two days following the Asian Dust Storm) was observed; however, this increase was not statistically significant.

A study by Yang (2006) assessed the possible effects of exposure to windblown dust storms originating in the deserts of Mongolia and China on the clinical visits for conjunctivitis in residents of Taipei, Taiwan during the period from 1997 to 2001. The study found that the mean concentration of PM₁₀ during dust storms was 110.37 ± 37.86 µg/m³ compared to the mean concentration during comparison days (usually the same day of the week, but a week before and a week after a dust storm) of 61.73 ± 30.22 µg/m³. An 11% increase in the risk of clinical visits for conjunctivitis during the dust storm events (4 days following the day of the storm event) was observed; however, this increase was not statistically significant. There may not have been enough statistical power to detect associations resulting from inadequate sample size of conjunctivitis visits on dust storm event days.

Gordian et al. (1996) examined the effects of average 24-hour PM₁₀ concentrations and carbon monoxide and temperature on the number of daily outpatient visits for respiratory disease (asthma, bronchitis and upper respiratory tract illnesses) in Anchorage, Alaska between 1992 and 1994. Particulate matter less than 10 µm (PM₁₀) in the Anchorage area is composed primarily of material from crustal sources (unpaved roads, road sanding) and volcanic ash due to the lack of industrial sources of pollution.



A volcanic eruption occurred during the study (Mount Spurr on August 18, 1992) and the 24-hour average PM₁₀ concentration was 565 µg/m³ on the day after the eruption. The composition of the volcanic ash was analyzed by electron microscopy and it was determined that the majority of the particle mass (greater than 80%) was composed of particles between 2.5 and 10 µm containing primarily silica and silica-aluminum mixture. The assessment of the volcanic ash composition was consistent with a previous investigation that showed the TSP in the Anchorage area was composed primarily of material from crustal sources. The mean 24-hour PM₁₀ concentration measured during the study was 45.54 µg/m³ and the maximum was the 565 µg/m³ as the result of a volcanic eruption. Vehicular emissions are a source of benzene and carbon monoxide which have been attributed to incomplete combustion of Alaskan gasoline which is high in benzene content. Carbon monoxide was only measured in the winter in this study. Based on the available information, PM₁₀ and carbon monoxide concentrations were not correlated.

The study found statistically significant positive associations between PM₁₀ and incidence of outpatient medical visits due to respiratory illnesses, independent of the outdoor temperature. For asthma, the predicted percent change was 4.2% per 10 µg/m³ increase in PM₁₀. For bronchitis, the predicted percent change was 2.3% per 10 µg/m³ increase in PM₁₀. For upper respiratory tract infections, the predicted percent change was 2.7% per 10 µg/m³ increase in PM₁₀. Temperature is a marker for season, which could influence PM₁₀ levels and respiratory illness. The association of PM₁₀ and increased incidences of outpatient visits was higher and only statistically significant during the period of time following the volcanic eruption. Winter carbon monoxide concentrations were found to be correlated with bronchitis and upper respiratory illnesses but not asthma.

Another study from Washington State (Hefflin et al. 1991) found a small increase in hospital admissions for respiratory illness following dust storms where maximum concentrations exceeded 1,000 µg/m³ over a 24-hour period. Hefflin et al. (1991) found positive associations between PM₁₀ and incidence of hospital emergency room visits for bronchitis (increase was 3.5% per 100 µg/m³), and for sinusitis (increase was 4.5% per 100 µg/m³ PM₁₀).

A study conducted after the eruption of Mount St. Helens looked at potential health effects to children attending a camp in the vicinity of the eruption who were exposed to elevated concentrations of dust (10,000 µg/m³). Although the dust levels measured during the camp were above the particulate matter standard at that time (260 µg/m³), the authors did not find either a within-day or between-day effect from the dust on lung function in the children (Buist et al. 1983).

The data derived from epidemiological studies between exposure to crustal particulate matter and morbidity health effects are presented in Table 9.1-E-3.



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Table 9.1-E-3: Summary of Morbidity Studies from PM10 Originating from Crustal Sources

Location	Taipei, Taiwan						Anchorage, Alaska	Copenhagen, Denmark
PM ₁₀ Concentration during Dust Storm Event (24-hour average) (µg/m ³)	111.68	111.68	111.68	111.68	111.68	110.37	NA	NA
PM ₁₀ Concentration on Control days (24-hour average) (µg/m ³)	55.43	55.43	55.43	55.43	55.43	61.73	NA	NA
Change in PM ₁₀ concentration (24-hour average) (µg/m ³)	38.32	38.32	38.32	38.32	38.32	48.64	10	25
Daily Increase in Morbidity								
Cardiovascular Disease Hospital Admissions	3.65% (1-day lag)	NA	NA	NA	NA	NA	NA	5.1%
Asthma Hospital Admissions	NA	8% ^(a) (2-day lag)	NA	NA	NA	NA	4.2%	-8.8% ^(a,c)
Stroke Hospital Admissions	NA	NA	NA	15% (3-day lag)	NA	NA	NA	NA
Congestive Heart Failure Hospital Admissions	NA	NA	NA	NA	11% (1-day lag) ^a	NA	NA	NA
Allergic Rhinitis Hospital/Clinic Visits	NA	NA	19% (2-day lag) ^a	NA	NA	NA	NA	NA
Clinic Visits for Conjunctivitis	NA	NA	NA	NA	NA	11% (4-day lag) ^a	NA	NA
Upper Respiratory Hospital Admissions	NA	NA	NA	NA	NA	NA	2.7% ^(b)	0.7% ^{(a),(d)}
Reference	Chen and Yang (2005)	Yang et al. (2005a)	Chang et al. (2006)	Yang et al. (2005b)	Yang et al. (2009)	Yang (2006)	Gordian et al. (1996)	Andersen et al. (2007)

Notes:

- (a) Not statistically significant.
 - (b) Correlated with carbon monoxide concentrations from vehicular traffic.
 - (c) School-aged children (5 to 18 years).
 - (d) Respiratory disease in the elderly (≥65 years).
- NA = not assessed or not available; µg/m³ = micrograms per cubic metre; % = percent.



4.0 CLOSURE

We trust this information is sufficient for your needs at this time. Should you have any questions or concerns, please do not hesitate to contact the undersigned at 604-296-4200.

GOLDER ASSOCIATES LTD.

Victoria Hart, M.Sc.
Environmental Scientist

Audrey Wagenaar, M.Sc., DABT, PChem.
Associate, Senior Environmental Scientist

VH/AW/asd

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5.0 REFERENCES

- Andersen ZJ, Wahlin P, Raaschou-Nielsen O, Scheike, TS Loft. 2007. Ambient Particle Source Apportionment and Daily Hospital Admissions Among Children and Elderly in Copenhagen. *J Expo Sci Env Epi* 17: 625-636.
- Bourcier T, Viboud C, Cohen JC, Thomas F, Bury T, Cadiot L, Mestre O, Flahault, A, Borderie V, Laroche L. 2003. Effects of Air Pollution and Climatic Conditions on the Frequency of Ophthalmological Emergency Examinations. *Brit J Ophthalmology* 87: 809-811.
- Buist AS, Johnson LR, Vollmer WM, Sexton GJ, Kanarek, PH. 1983. Acute Effects of Volcanic Ash from Mount St. Helens on Lung Function in Children. *Am Rev Respir Dis* 127: 714-719.
- Chang C, Lee I, Tsai S, Yang C. 2006. Correlation of Asian Storm Events with Daily Clinic Visits for Allergic Rhinitis in Taipei, Taiwan. *J Toxicol Env Heal A* 69: 229-235.
- Chen YS, Sheen PC, Chen ER, Liu YK, Wu TN, Yang CY. 2004. Effects of Asian Dust Storm Events on Daily Mortality in Taipei, Taiwan. *Environ Res* 95: 151-155.
- Chen Y, Yang C. 2005. Effects of Asian Dust Storm Events on the Daily Hospital Admission for Cardiovascular Disease in Taipei, Taiwan. *J Toxicol Env Heal A* 68: 1457-1464.
- Englert 2004. Fine Particles and Human Health-a review of epidemiological studies. *Toxicology Letters* 149 (1): 235-242.
- Fuentes M, Song HR, Ghosh SK, Holland DM, Davis JM. 2006. Spatial association between speciated fine particles and mortality. *Biometrics* 62: 855-63.
- Gordian ME, Ozkaynak H, Xue J, Morris S, Spengler JD. 1996. Particulate Air Pollution and Respiratory Disease in Anchorage, Alaska. *Environ Health Persp* 104: 290-297.
- Gupta SK, Gupta SC, Agarwal R, Sushma S, Agrawal SS, Rohit S. 2007. A Multicentric Case-control Study on the Impact of Air Pollution on Eyes in a Metropolitan City of India. *Indian J Occup Environ Med* 11: 37-40.
- Hefflin BJ, Jalaludin B, McClure E. 1991. Surveillance for Dust Storms and Respiratory Diseases in Washington State. *Arch Environ Health* 49: 170-174.
- Kwon HJ, Cho SH, Chun Y, Lagarde F, Pershagen G. 2002. Effects of the Asian Dust Events on Daily Mortality in Seoul, Korea. *Environ Res Section A* 90: 1-5.
- Laden F, Neas LM, Dockery DW, & Schwartz J. 2000. Association of Fine Particulate Matter from Different Sources with Daily Mortality in Six U.S. Cities. *Environ Health Persp* 108: 941-947.
- Lippmann M, Chen LC. 2009a. Health Effects of Concentrated Ambient Air Particulate Matter (CAPs) and its Components. *Crit Rev Toxicol*. 39: 865-913.
- Lippmann M, Chen LC. 2009b. Effects of metals within ambient air particulate matter (PM) on human health. *Inhal Toxicol*. 21:1-31.



PARTICULATE MATTER LITERATURE REVIEW

- Ostro BD, Hurley S, Lipsett M. 1999. Air Pollution and Daily Mortality in the Coachella Valley, California: A Study of PM₁₀ Dominated by Coarse Particles. *Environ Res, Section A* 81: 231-238.
- Ostro BD, Broadwin R, Lipsett, MJ. 2000. Coarse and Fine Particles and Daily Mortality in the Coachella Valley California: A Follow-up Study. *J Expo Anal Env Epid* 10: 412-419.
- Penttinen P, Tiittanen P, Pekkanen J. 2004. Mortality and Air Pollution in Metropolitan Helsinki, 1988-1996. *Scandinavian Expo Anal Env Epid* 30: 19-27.
- Pope CA., Hill RW, Villegas GM. 1999. Particulate Air Pollution and Daily Mortality on Utah's Wasatch Front. *Environ Health Persp* 107: 567-573.
- Rohr AC and RE Wyzga. 2012. Attributing health effects to individual particulate matter constituents. *Atmos Environ* 62: 130-152.
- Schwartz J, Norris G, Larson T, Sheppard L, Claiborne C, Koenig J. 1999. Episodes of High Coarse Particle Concentrations Are Not Associated with Increased Mortality. *Environ Health Persp* 107: 339-342.
- Schwarze PE, Ovrevik J, Lag M, Refsnes M, Nafstad P, Hetland RB, Dybing E. 2006. Particulate matter properties and health effects: consistency of epidemiological and toxicological studies. *Hum Exp Toxicol* 25: 559-579.
- Slaughter JC, Kim E, Sheppard L, Sullivan, JH, Larson T, Claiborn, C. 2005. Association between Particulate Matter and Emergency Room Visits, Hospital Admissions, and Mortality in Spokane, Washington. *J Expo Anal Env Epid* 15(15): 153-159.
- Stanek LW, Sacks JD, Dutton SJ, Dubois JJB. 2011. Attributing health effects to apportioned components and sources of particulate matter: An evaluation of collective results. *Atmos Environ* 45(32): 5655-5663.
- Staniswalis JG, Parks NJ, Bader JO, Maldonado YM. 2005. Temporal Analysis of Airborne Particulate Matter Reveals a Dose-rate Effect on Mortality in El Paso: Indications of Differential Toxicity for Different Particle Mixtures. *J Air Waste Manage* 55 (7): 893-902.
- WHO (World Health Organization). 2006. Air Quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Geneva, Switzerland.
- Yang C, Tsai S, Chang C, Ho S. 2005a. Effects of Asian Dust Storm Events on Daily Admissions for Asthma in Taipei, Taiwan. *Inhal Toxicol* 17: 817-821.
- Yang C, Chen Y, Chiu H, Goggins W. 2005b. Effects of Asian Dust Storm Events on Daily Stroke Admissions in Taipei, Taiwan. *Environ Res* 99: 79-84.
- Yang C. 2006. Effects of Asian Dust Storm Events on Daily Clinical Visits for Conjunctivitis in Taipei, Taiwan. *J Toxicol Env Heal A* 69: 1673-1680.
- Yang C, Cheng M, Chen C. 2009. Effects of the Asian Dust Storm Events on Hospital Admissions for Congestive Heart Failure in Taipei, Taiwan. *J Toxicol Env Heal A* 72: 324-328.

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Africa	+ 27 11 254 4800
Asia	+ 86 21 6258 5522
Australasia	+ 61 3 8862 3500
Europe	+ 44 1628 851851
North America	+ 1 800 275 3281
South America	+ 56 2 2616 2000

solutions@golder.com
www.golder.com

Golder Associates Ltd.
Suite 200 - 2920 Virtual Way
Vancouver, BC, V5M 0C4
Canada
T: +1 (604) 296 4200

