Short-term Effects of Coarse Particulate Matter Air Pollution on Population Health

Time Series Studies in Hong Kong

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Outline

- PM pollution
- Health effects of coarse PM
- My studies on coarse PM in HK
- Toxicity implications
- Policy implications
Particulate Matter

- Air quality regulations and research focus on **PM size**
  - **TSP** (Total suspended)
  - **PM\textsubscript{10}** (Respirable suspended): \(\leq\)10\(\mu\text{m}\)
  - **PM\textsubscript{2.5}** (Coarse, PM\textsubscript{10-2.5}): 2.5 - 10\(\mu\text{m}\)
  - **PM\textsubscript{2.5}** (Fine): \(\leq\)2.5\(\mu\text{m}\)
  - **PM\textsubscript{0.1}** (Ultrafine, UFP): \(\leq\)0.1\(\mu\text{m}\)
- PM size is directly linked to their deposition in the airway and potential health effects.

PM – why size matters

Two distinct types of particles:
- **PM\textsubscript{2.5}**: vehicle exhausts and combustion sources. Generally comprised of organic carbon, elemental carbon, sulfate, nitrate, and metals.
- **PM\textsubscript{c}**: crustal elements, re-suspension of road dusts, sea salts, and biogenic components (pollen, spores, molds, bacteria).

These variations in composition, along with differential deposition in the body, suggest that PM\textsubscript{2.5} and PM\textsubscript{c} may differ in their impacts on human health.
Many epidemiologic studies have linked daily counts of mortality/morbidity to PM$_{10}$ and PM$_{2.5}$, but relatively few have investigated the health effects of coarse particles (PM$_c$).

- PM$_c$ were initially considered as potentially less toxic than fine particles (Bell 2004; Wilson and Suh 1997).
- The lack of PM$_c$ measurement data or the concurrent PM$_{10}$ and PM$_{2.5}$ monitoring data.

**Inconsistent findings on PM$_c$**

- Systematic review (Brunekreef and Forsberg 2005) and Meta-analysis (Alder 2014) of studies on associations between PM$_{2.5}$, PM$_c$ and cardio-respiratory hospital admissions
  - PM$_c$ might have adverse effects on the respiratory system;
  - less supportive but could not exclude for a PM$_c$ effect on cardiovascular admissions.

- Some subsequent studies that examined the effects of PM$_{2.5}$ and PM$_c$ simultaneously on cardio-respiratory hospitalizations in European cities (Host 2008, Teger 2008, Halonen 2009, Atkinson 2010, Stafoggia 2013) got similar results.

- Inconsistent results in NMMAPS studies:
  - In 108 US counties: failed to find any significant effects of PM$_c$ on cardio-respiratory admissions (Tang 2006)
  - In 59 US counties: accounting for exposure measurement error, reported a positive association between PM$_c$ and same-day hospital admission for cardiovascular diseases (Chang 2011)
How about HK?

- The Air Quality Objectives for Hong Kong only cover PM$_{10}$, and the Environmental Protection Department (EPD) newly established PM$_{2.5}$ standard in 2014.

- Regulation specifically for PM$_c$ is not in place or under consideration;

- Additional studies to estimate the adverse health effects of PM$_c$ could help support a PM$_c$ standard in the future.

My own studies

- To examine whether PM$_c$ is associated with emergency hospital admissions for cardio-respiratory diseases in Hong Kong

- To examine the differential effects of PM$_c$ on respiratory and cardiovascular diseases.
### Data Sources (2000-2005)

#### Daily air pollution concentrations from EPD
- $PM_c = PM_{1.0} - PM_{2.5}$
- Monitoring data in Tsuen Wan station were used.

#### Daily emergency hospital admissions from HA
- Diseases of respiratory system
  - Chronic obstructive pulmonary diseases (COPD)
  - Asthma
- Diseases of circulatory system
  - Cardiac diseases
  - Ischemic heart diseases (IHD)
  - Cerebrovascular diseases (CBD)

#### Daily mean temperature and relative humidity from HKO

### GAM regression

- Time Series study using generalized additive Poisson regression models.

- Core/Base model:
  
  $log(E(Y_t)) = \alpha + s(t, df=7/\text{year } \times \text{ no. of years}) + s(Temp_{0}, df=6) + s(Temp_{1-3}, df=6) + s(Humidity_{0}, df=3) + \beta_1 \text{DOW} + \beta_2 \text{Holiday} + \beta_3 \text{influenza}$
  
  $= \alpha + \text{COVs}$
Assumption checking

- Use partial autocorrelation function (PACF) to guide the inclusion of the autoregressive terms (Schwartz, 2000);
  - Criteria for an adequate core model: no autocorrelation and no discernible patterns in the residuals
- Use concentration-response relationship curves to justify the assumption of linearity of the PM\textsubscript{c} effect

Co-pollutant control

- Single pollutant models: to examine the effects of PM\textsubscript{c} at different lag structures (lag\textsubscript{0}~lag\textsubscript{5}, lag\textsubscript{01}~lag\textsubscript{03})
- Two-pollutant models: to examine the effects of PM\textsubscript{c}, adjusting for PM\textsubscript{2.5}
- To further adjust for the exposures to gaseous pollutants (NO\textsubscript{2}, SO\textsubscript{2} and O\textsubscript{3})
Sensitivity Analysis

- Excluded days with extremely high (the highest 1%) and low (the lowest 1%) PM$_{10}$ concentrations
- Examined the impact of various $df$ for time trend and weather conditions on PM$_c$ effect estimates
- Restricted the emergency hospital admissions to Tsuen Wan residents

### Data Description

<table>
<thead>
<tr>
<th>Variables</th>
<th>No. of days</th>
<th>Mean</th>
<th>SD</th>
<th>25th</th>
<th>50th</th>
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<tr>
<td>Daily emergency hospital admissions</td>
<td>2192</td>
<td>236.7</td>
<td>55.4</td>
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<td>230</td>
<td>260</td>
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<tr>
<td>Total RES</td>
<td>2192</td>
<td>81.1</td>
<td>20.3</td>
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<td>80</td>
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<td>COPD</td>
<td>2192</td>
<td>19.6</td>
<td>8.0</td>
<td>14</td>
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<td>RES in TW residents</td>
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<td>154.3</td>
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<td>153</td>
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<td>95.1</td>
<td>18.6</td>
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<td>94</td>
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<td>Cardiac</td>
<td>2192</td>
<td>30.1</td>
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<td>IHD</td>
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<table>
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<tr>
<th>Pollution concentration (µg/m$^3$)</th>
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<td>PM$_{10}$</td>
<td>1998</td>
<td>56.1</td>
<td>27.8</td>
<td>34.9</td>
<td>49.2</td>
<td>72.5</td>
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<td>PM$_{2.5}$</td>
<td>1997</td>
<td>39.4</td>
<td>20.7</td>
<td>23.8</td>
<td>34.8</td>
<td>50.1</td>
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<td>PM$_c$</td>
<td>1997</td>
<td>16.6</td>
<td>9.2</td>
<td>10.0</td>
<td>14.5</td>
<td>20.9</td>
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<td>NO$_2$</td>
<td>1995</td>
<td>64.4</td>
<td>22.4</td>
<td>48.4</td>
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<td>SO$_2$</td>
<td>1998</td>
<td>22.9</td>
<td>17.1</td>
<td>11.3</td>
<td>18.3</td>
<td>28.7</td>
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<tr>
<td>O$_3$</td>
<td>1995</td>
<td>31.1</td>
<td>24.3</td>
<td>13.2</td>
<td>24.2</td>
<td>42.8</td>
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<td>Temperature (°C)</td>
<td>2192</td>
<td>23.5</td>
<td>5.0</td>
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<td>27.8</td>
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<td>Relative humidity (%)</td>
<td>2192</td>
<td>78.2</td>
<td>9.7</td>
<td>73</td>
<td>79</td>
<td>85</td>
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</table>
The effects of four-day moving averages (lag 03) of PM concentrations for respiratory hospitalizations and effects of lag 01 for circulatory hospitalizations were estimated in GAMs, adjusting for time trend, weather conditions, day of week, public holidays and influenza outbreaks.

IQR: interquartile range, 10.9 µg/m$^3$ for PM$_{c}$ and 26.3 µg/m$^3$ for PM$_{2.5}$.

Co-pollutant control

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Total Respiratory</th>
<th>COPD</th>
<th>Total Circulatory</th>
<th>Cardiac Diseases</th>
</tr>
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<tr>
<td><strong>Single Pollutant Model</strong></td>
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<tr>
<td>PM$_{c}$</td>
<td>1.94 (1.24, 2.64)</td>
<td>3.37 (2.26, 4.49)</td>
<td>0.83 (0.10, 1.58)</td>
<td>1.49 (0.57, 2.41)</td>
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<tr>
<td>PM$_{2.5}$</td>
<td>2.58 (1.73, 3.44)</td>
<td>4.44 (3.11, 5.80)</td>
<td>1.75 (0.94, 2.57)</td>
<td>2.61 (1.60, 3.64)</td>
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<tr>
<td><strong>Two Pollutants Model</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM$_{c}$</td>
<td><strong>1.05 (0.19, 1.91)</strong></td>
<td><strong>1.78 (0.41, 3.16)</strong></td>
<td>-0.16 (-1.07, 0.76)</td>
<td>0.17 (-0.95, 1.29)</td>
</tr>
<tr>
<td>PM$_{2.5}$</td>
<td>1.81 (0.76, 2.87)</td>
<td>3.13 (1.48, 4.81)</td>
<td>1.86 (0.85, 2.88)</td>
<td><strong>2.50 (1.25, 3.77)</strong></td>
</tr>
</tbody>
</table>

*The effects of four-day moving averages (lag$_{03}$) of PM concentrations for respiratory hospitalizations and effects of lag$_{01}$ for circulatory hospitalizations were estimated in GAMs, adjusting for time trend, weather conditions, day of week, public holidays and influenza outbreaks.

IQR: interquartile range, 10.9 µg/m$^3$ for PM$_{c}$ and 26.3 µg/m$^3$ for PM$_{2.5}$. 

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8
### Sensitivity Analyses

<table>
<thead>
<tr>
<th>Two-Pollutant Models</th>
<th>Restrict analysis for PM&lt;sub&gt;c&lt;/sub&gt; in P1-P9&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Restrict analysis in TW residents&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Additionally adjusted for gaseous pollutants&lt;sup&gt;c&lt;/sup&gt;</th>
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<tbody>
<tr>
<td></td>
<td>NO&lt;sub&gt;x&lt;/sub&gt;</td>
<td>SO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>O&lt;sub&gt;3&lt;/sub&gt;</td>
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<td><strong>Respiratory diseases</strong></td>
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<tr>
<td>PM&lt;sub&gt;c&lt;/sub&gt;</td>
<td>1.32 (0.23, 2.42)</td>
<td>1.78 (0.11, 3.47)</td>
<td>1.07 (0.24, 1.94)</td>
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<tr>
<td>PM&lt;sub&gt;2.5&lt;/sub&gt;</td>
<td>1.70 (0.59, 2.82)</td>
<td>1.72 (-0.26, 3.74)</td>
<td>1.19 (0.05, 2.33)</td>
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<tr>
<td><strong>Circulatory diseases</strong></td>
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<td></td>
</tr>
<tr>
<td>PM&lt;sub&gt;c&lt;/sub&gt;</td>
<td>0.18 (-0.93, 1.30)</td>
<td>-1.40 (-3.14, 0.38)</td>
<td>-0.12 (-1.02, 0.80)</td>
</tr>
<tr>
<td>PM&lt;sub&gt;2.5&lt;/sub&gt;</td>
<td>2.16 (1.02, 3.31)</td>
<td>3.89 (1.95, 5.87)</td>
<td>0.86 (-0.30, 2.03)</td>
</tr>
</tbody>
</table>

* The effects of lag03 of PM concentrations for respiratory diseases and effects of lag01 for circulatory diseases were estimated in GAMs, adjusting for time trend, weather conditions, day of week, public holidays and influenza outbreaks. IQR: interquartile range, 10.9 µg/m<sup>3</sup> for PM<sub>c</sub> and 26.3 µg/m<sup>3</sup> for PM<sub>2.5</sub>.

### Summary of results

- PM<sub>c</sub> pollution was linked to emergency hospital admissions for respiratory diseases but not for cardiovascular diseases in Hong Kong.
- PM<sub>c</sub> effects on respiratory diseases were independent of PM<sub>2.5</sub> and gaseous pollutants.
- The adverse effects of PM<sub>2.5</sub> were confirmed, for both respiratory and cardiovascular diseases.
Discussion – PM$_c$ vs PM$_{2.5}$

- PM$_c$: more likely to deposit/affect the upper and larger airways
  - carry biological materials (bacteria, moulds or pollens), likely to produce adverse health effects in the respiratory system (Almeida 2006)
- PM$_{2.5}$: higher number concentrations, larger surface area and deeper lung deposition
  - reach the small airways and the alveoli of the lung, and not easy to be cleaned;
  - Inhaled ultrafine particles could even translocate rapidly from the lungs into the blood circulation (Nemmar 2002).

Discussion – Strengths

- Largest single city study to date to examine the effects of PM$_c$ after adjustment for PM$_{2.5}$ and gaseous
  - with almost one million of emergency hospital admissions covering over 6 years.
- Used daily data on PM$_{2.5}$ and PM$_c$ concentrations
  - Unlike some studies that used every third or sixth day PM data (Lin 2005; Peng 2008)
  - Was able to estimate the effects of multiday average or cumulative effect
Discussion – Limitations

- PMc were estimated by subtracting PM$_{2.5}$ from PM$_{10}$ measurements:
  - two sources of measurement error;
  - reduce the statistical power of detecting an association.

- Used the monitoring data to represent the population exposure:
  - PMc - less spatially homogeneous, because of shorter travelling distance and suspending time in the air.

Effects of Coarse Particulate Matter on Emergency Hospital Admissions for Respiratory Diseases: A Time-Series Analysis in Hong Kong

Hong Qiu, Ignatius Tak-sum Yu, Linwei Tian, Xiaorong Wang, Lap Ah Tse, Wilson Tam, and Tze Wai Wong
School of Public Health and Primary Care, Chinese University of Hong Kong, Hong Kong Special Administrative Region, China

Backgrounds: Many epidemiological studies have linked daily counts of hospital admissions to particular events (PMc) with an aerodynamic diameter ≤ 10 μm (PM$_{10}$) and ≤ 2.5 μm (PM$_{2.5}$), but relatively few have investigated the relationship of hospital admissions with coarse PM (PMc).

Objective: We conducted this study to estimate the health effects of PMc on emergency hospital admissions for respiratory disease in Hong Kong, after controlling for PM$_{2.5}$ and gaseous pollutants.

Methods: We conducted a time-series analysis of associations between daily emergency hospital admissions for respiratory disease in Hong Kong from January 2000 to December 2005 and daily PM$_{2.5}$ and PMc concentrations. We estimated PM$_{2.5}$ concentrations by subtracting PM$_{2.5}$ from PM$_{10}$ measurements. We used generalised additive models to examine the relationship between PMc, single- and multipollutant lagged exposures, and hospital admissions allowed for time trends, weather conditions, influenza outbreaks, PM$_{2.5}$, and gaseous pollutants (nitrogen dioxide, sulphur dioxide, and ozone).

Materials and Methods

Data on particulate pollutants and meteorology variables. We obtained air pollution data for January 2000 through December 2005 from the Environmental Protection Department. There are a total of 11 general monitoring stations in Hong Kong. All of them monitored PM$_{2.5}$ and gaseous pollutants (nitrogen dioxide, sulphur dioxide, and ozone) during the study period, but only three (Tuen Mun, Tung Ping Chau, and Kowloon) collected simultaneous PM$_{2.5}$ data. The Tuen Mun and Tung Ping Chau stations are located in remote areas of Hong Kong, whereas the Tuen Mun...
Updated Data (2011-2012)

Daily air pollution concentrations from EPD
- PMc = PM10-PM2.5;
- Monitoring data in TEN general stations were used;
- Providing more accurate exposure information than data from one single station.

- Updated daily emergency hospital admissions from HA
  - Pneumonia is an inflammatory condition of lung caused by infections.
  - It may be triggered and exacerbated by coarse particulate matter (PMc) exposure.

- Aim: To estimate the effect of PMc on emergency hospital admissions for pneumonia after controlling for PM_{2.5} and gaseous pollutants.

GAM modeling
- Generalized additive Poisson models;

\[
\log(E(Y)) = \alpha + s(t, df=8/years\times2years) \\
+ s(Temp_0, df=6) + s(Temp_{1-3}, df=6) \\
+ s(Humid_0, df=3) \\
+ \beta_1 DOW + \beta_2 Holiday + \beta_3 influenza
\]

- Single lag effect and distributed lags effect were both estimated;
- Adjusting for PM_{2.5} and gaseous pollutants (NO_{2}, SO_{2} and O_{3});
- Subgroup analyses by gender and age were performed to identify the most susceptible subpopulations.
Single-pollutant models

<table>
<thead>
<tr>
<th>Lag Days</th>
<th>PM_1 lag 0</th>
<th>PM_2 lag 1</th>
<th>PM_3 lag 2</th>
<th>PM_4 lag 3</th>
<th>PM_5 lag 4</th>
<th>PM_6 lag 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>lag_0</td>
<td>1.06 (-0.22, 2.35)</td>
<td>0.68 (-0.07, 1.43)</td>
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<tr>
<td>lag_1</td>
<td>1.57 (0.42, 2.73)</td>
<td>0.72 (0.00, 1.44)</td>
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<tr>
<td>lag_2</td>
<td>1.83 (0.70, 2.97)</td>
<td>0.85 (0.15, 1.56)</td>
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<tr>
<td>lag_3</td>
<td>1.76 (0.65, 2.88)</td>
<td>1.15 (0.46, 1.84)</td>
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<td>lag_4</td>
<td>1.14 (0.03, 2.26)</td>
<td>1.47 (0.80, 2.14)</td>
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<td>lag_5</td>
<td>1.07 (0.03, 2.19)</td>
<td>0.89 (0.22, 1.57)</td>
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<td>lag_6</td>
<td>0.82 (0.27, 1.95)</td>
<td>0.34 (0.03, 1.02)</td>
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<td>dlm03</td>
<td>3.33 (1.54, 5.15)</td>
<td>1.69 (0.68, 2.70)</td>
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<td>dlm46</td>
<td>0.97 (0.65, 2.62)</td>
<td>1.16 (0.20, 2.14)</td>
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Co-pollutant models

<table>
<thead>
<tr>
<th>Lag Days</th>
<th>PM_1+PM_2 lag 0</th>
<th>PM_1+NO_2 lag 1</th>
<th>PM_1+O_3 lag 2</th>
<th>PM_1+SO_2 lag 3</th>
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<tr>
<td>lag_0</td>
<td>0.68 (-0.73, 2.12)</td>
<td>0.99 (-0.30, 2.30)</td>
<td>0.66 (-0.68, 2.03)</td>
<td>1.06 (-2.23, 2.36)</td>
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<td>lag_1</td>
<td>1.32 (0.01, 2.65)</td>
<td>1.52 (0.35, 2.70)</td>
<td>0.65 (-0.61, 1.93)</td>
<td>1.73 (0.57, 2.91)</td>
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<td>lag_2</td>
<td>1.52 (0.20, 2.85)</td>
<td>1.71 (0.56, 2.88)</td>
<td>1.08 (-0.20, 2.37)</td>
<td>1.74 (0.57, 2.91)</td>
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<td>lag_3</td>
<td>1.08 (-0.22, 2.39)</td>
<td>1.41 (0.26, 2.56)</td>
<td>0.92 (-0.35, 2.21)</td>
<td>1.42 (0.27, 2.58)</td>
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<td>dlm03</td>
<td>2.43 (0.41, 4.50)</td>
<td>2.90 (1.06, 4.77)</td>
<td>1.38 (-0.70, 3.51)</td>
<td>3.03 (1.20, 4.89)</td>
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</tbody>
</table>

a: Overall cumulative effects of pollutants lasting for 0–3 days (dlm03) were estimated by unconstrained distributed lag models. b: Two pollutants were included into the model at the same lags. Statistically significant effect estimates were in bold.
### Subgroup Analyses

<table>
<thead>
<tr>
<th></th>
<th>lag0</th>
<th>lag1</th>
<th>lag2</th>
<th>lag3</th>
<th>dlm03 *</th>
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<tr>
<td>Female</td>
<td>1.47 (-0.30, 3.28)</td>
<td>1.06 (-0.53, 2.67)</td>
<td>2.34 (0.79, 3.92)</td>
<td>2.92 (1.40, 4.45)</td>
<td>4.55 (2.07, 7.09)</td>
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<td>Male</td>
<td>0.95 (-0.73, 2.65)</td>
<td>2.38 (0.88, 3.91)</td>
<td>1.89 (0.40, 3.40)</td>
<td>1.40 (-0.07, 2.89)</td>
<td>3.20 (0.86, 5.59)</td>
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<td><strong>Age group</strong></td>
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<tr>
<td>&lt;15</td>
<td>1.26 (-1.96, 4.59)</td>
<td>1.73 (-1.15, 4.70)</td>
<td>3.33 (0.45, 6.29)</td>
<td>3.60 (0.79, 6.49)</td>
<td>5.60 (0.97, 10.44)</td>
</tr>
<tr>
<td>15~64</td>
<td>1.59 (-1.49, 4.78)</td>
<td>0.91 (-1.88, 3.78)</td>
<td>-0.02 (-2.77, 2.81)</td>
<td>1.56 (-1.16, 4.35)</td>
<td>2.56 (-1.69, 6.99)</td>
</tr>
<tr>
<td>65~74</td>
<td>1.90 (-1.61, 5.54)</td>
<td>2.44 (-0.71, 5.69)</td>
<td>3.29 (0.27, 6.39)</td>
<td>5.33 (2.36, 8.39)</td>
<td>7.33 (2.41, 12.49)</td>
</tr>
<tr>
<td>&gt;=75</td>
<td>1.23 (-0.39, 2.88)</td>
<td>2.36 (0.92, 3.82)</td>
<td>2.93 (1.53, 4.35)</td>
<td>2.41 (1.04, 3.80)</td>
<td>4.52 (2.29, 6.80)</td>
</tr>
</tbody>
</table>

*a: Overall cumulative effects of PMc lasting for 0~3 days (dlm03) were estimated by unconstrained distributed lag models. Statistically significant effect estimates were in bold. Differences of the effect estimates of PMc between gender or among age groups were statistically non-significant (p>0.05).*

### Summary of results

- Both PM_{c} and PM_{2.5} significantly associated with pneumonia emergency hospital admissions in Hong Kong.

- The effect estimates of PM_{c} were robust to the adjustment for PM_{2.5}, and gaseous pollutants NO_{2} or SO_{2}, but were attenuated upon adjustment for O_{3}.

- It appears that females, children and elders might be more vulnerable to PM_{c} exposure.
Discussion

- Strength: the average levels across the 10 monitors, more representative of the general population.

- Potential mechanisms:
  - Imbalance of inflammatory mediators; (Morn 1999)
  - Immunosuppressive, undermine the normal pulmonary antimicrobial defense; (Zelikoff 2002)
  - Impair the cellular defense and immune system, increase susceptibility to bacterial pathogens. (Happo 2010)

- PMc may carry biogenic elements (bacteria, etc.)
Toxicity and Policy Implications

- Better understanding the toxicity of different size of PM
- Air quality control policy – not to abandon PM$_{10}$ standards, regulation specifically for PM$_c$ might be considered
- Susceptible populations – reduce relevant exposures

Future Work

- Sources, composition, and health effects of coarse (PM$_c$) particles: a two-year time-series study in Hong Kong
  - better know about PM$_c$ composition, sources
  - better understanding of toxicity
  - more targeted regulations
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