ORIGINAL ARTICLE

Coarse particulate matter associated with increased risk of emergency hospital admissions for pneumonia in Hong Kong

Hong Qiu,1 Lin Wei Tian,1,2 Vivian C Pun,1 Kin-fai Ho,1,2 Tze Wai Wong,1 Ignatius T S Yu1

ABSTRACT
Background Epidemiological research on the effects of coarse particles (PMc, particulate matter between 2.5 μm and 10 μm in aerodynamic diameter) on respiratory morbidity is sparse and inconclusive. Pneumonia is an inflammatory condition of lung caused by infections, which may be triggered and exacerbated by PMc exposure. Aim To estimate the effect of PMc on emergency hospital admissions for pneumonia after controlling for PM2.5 and gaseous pollutants. Method PMc concentrations were estimated by subtracting PM2.5 from PM10 measurements in each of the 10 air monitoring stations from January 2011 to December 2012 in Hong Kong and then citywide daily average concentrations of PMc were computed from the 10 stations. Generalised additive Poisson models were used to examine the relationship between PMc and daily emergency hospital admissions for pneumonia, adjusting for PM2.5 and gaseous pollutants (NO2, SO2 and O3). Subgroup analyses by gender and age were also performed to identify the most susceptible subpopulations. Results PMc and PM2.5 were significantly associated with emergency pneumonia hospitalisations. Every 10 μg/m3 increment of PMc in the past 4 days (lag0–lag3) was associated with a 3.33% (95% CI 1.54% to 5.15%) increase in emergency hospitalisations for pneumonia. The effect estimates of PMc were robust to the adjustment of PM2.5, NO2 or SO2, but attenuated on the inclusion of O3 in the model. Conclusions Short-term PMc exposure is associated with emergency hospitalisations for pneumonia in Hong Kong. Air quality regulation specifically for PMc might be considered.

INTRODUCTION
Although the effects of fine particulate matter pollution (PM2.5, particles with an aerodynamic diameter less than 2.5 μm) associated with respiratory diseases have been well documented,1 2 epidemiological research on the effects of coarse particles (PMc, particulate matter between 2.5 and 10 μm in aerodynamic diameter) on respiratory morbidity is sparse and inconclusive.2 3 Examining the association between PMc and health outcomes may be more difficult because coarse particles show greater spatial heterogeneity due to their larger size and shorter suspending period in the atmosphere.1 3

Key messages
What is the key question?
▸ Pneumonia is an inflammatory condition of the lung caused by infections; can it be triggered and exacerbated by coarse particulate matter (PMc) exposure?

What is the bottom line?
▸ We found an association between PMc exposure and emergency hospital admissions for pneumonia and the effect estimates of PMc were robust to the adjustment of PM2.5, NO2 or SO2, but were attenuated on the inclusion of O3 in the model.

Why read on?
▸ The reliable daily pairwise monitoring data of PM10 and PM2.5 in 10 general stations throughout Hong Kong give more accurate exposure information than data from one single station, and provide an opportunity to assess the relationship between PMc and pneumonia emergency hospitalisations.

Pneumonia is an inflammatory condition of a lobe or the whole lung caused by bacterial, viral and fungal infections. In Hong Kong, pneumonia was the second leading cause of death in 2012. An increasing trend was observed in the number of deaths and death rate since 2002. The number of deaths were 6960, accounting for 15.9% of all registered deaths in 2012.6 Inadequate nutrition, exposure to tobacco smoke, air pollution, and not receiving immunisation may predispose people to lower respiratory tract infection.7 Indoor and outdoor air pollution have been identified as important risk factors for pneumonia.8–12 However, only a few studies have examined the association between coarse particulate matter and pneumonia13 and the results have been inconsistent. In a previous study, we used data from a single monitoring station and found the association between PMc and emergency hospitalisations for overall respiratory diseases and COPD, but failed to detect the effects of PMc on the other endpoints of respiratory diseases such as asthma, etc.14 which was probably due to the spatial heterogeneity of PMc distribution or smaller statistical power.
Toxicological evidence supports the possibility that short-term coarse particle exposure may independently impact respiratory health by inducing inflammation that may incite or exacerbate disease. Pneumonia is an inflammatory condition of the lung, which may also be triggered and exacerbated by coarse particles. Hong Kong Environmental Protection Department (EPD) has begun to monitor the hourly concentrations of PM10 and PM2.5 in each of the 14 monitoring stations dispersed in the whole territory of Hong Kong since January 2011. The accurate PM concentration data provide an opportunity to assess the effects of PM2.5 on pneumonia. In this study, we conducted a time series analysis to estimate the acute effect of PM2.5 on emergency hospital admissions for pneumonia in Hong Kong after controlling for PM2.5 and gaseous pollutants. Subgroup analyses by gender and by age groups were also performed to identify the most susceptible subpopulations.

**MATERIALS AND METHODS**

**Data collection**

Hong Kong EPD has begun to monitor the hourly concentrations of four criteria air pollutants (PM10, NO2, O3, and SO2) in 14 monitoring stations dispersed in different districts of Hong Kong since 1990. Hourly concentrations of PM2.5 have been monitored in three general stations and one roadside station since 1998 and in all the 14 stations since 2011. In this study, we collected the pairwise data of PM10 and PM2.5, and gaseous pollutants in each monitoring station from January 2011 to December 2012. Three roadside stations and one station on a remote island were excluded, leaving 10 general stations to compute the citywide daily mean concentrations to represent the background air pollution level. We calculated 24 h mean concentrations of PM10 and PM2.5 and estimated PM2.5 concentrations by subtracting daily mean PM2.5 from PM10 for each station. Daily average concentrations of PM2.5 across the 10 general stations were used to represent the general population’s daily exposure. We also applied similar approaches to calculate 24 h mean concentrations of NO2, SO2 and 8 h (10:00–18:00) mean concentration of O3 to represent the citywide pollution exposure.

The daily count of emergency hospital admissions for pneumonia (International Classification of Diseases, ninth revision (ICD-9): 480–486) as the principal diagnosis was obtained from the Hospital Authority Corporate Data Warehouse. Hospital Authority is the statutory body running all public hospitals in Hong Kong. The records of admission were taken from the publicely funded hospitals providing 24 h accident and emergency services and covering 90% of hospital beds in Hong Kong for local residents. For the current study period of 2011–2012, the Hospital Authority provided us with daily counts of emergency hospital admissions aggregated over age, gender, date of admission, and principal diagnosis on discharge. We abstracted the overall daily pneumonia emergency admissions, admissions by gender and by age groups (age <15, 15–64, 65–74, ≥75 years old) as the health outcomes, respectively. Daily admissions for influenza (ICD-9: 487) were used to identify influenza epidemics, which were then treated as a potential confounder in the data analysis. Ethics approval and consent from individual subjects were not required by our institute as we used only aggregated data but not any individualised data in this study.

The meteorological information including the daily mean temperature and relative humidity were collected from the Hong Kong Observatory.

### Statistical modelling

In this time series study, generalised additive Poisson regression models were used to fit the relationship between the citywide daily PM2.5 concentrations and the emergency pneumonia hospitalisations. We used the smoothing spline, $s(t)$, to filter out seasonal patterns and long-term trends in daily hospitalisations, and the daily mean temperature and relative humidity. We also adjusted for the day of the week (DOW) and dichotomous variables such as public holidays and influenza epidemics.

We followed previous studies to select a priori model specifications and the degree of freedom (df) for the time trend and other meteorological variables. We used a df of 8/year for the time trend, a df of six for the mean temperature of the current day (Temp0) and the previous 3 days’ moving average (Temp1–3), and a df of three for the current day relative humidity (Humid0). We included the DOW and public holidays (Holiday) in the model as dummy variables. To adjust for the potential confounding effect of an influenza epidemic on emergency hospital admissions, we entered a dummy variable for the weeks with a number of influenza hospital admissions exceeding the 75th centile of the same year into the core model.

Briefly, we set up a core model to remove the long-term trend, seasonal variations, and adjust for time-varying confounders as follows:

$$
\log(E(Y)) = \alpha + s(t, \text{df} = 8/\text{year} \times 2 \text{ years}) + s(\text{Temp}_0, \text{df} = 6) + s(\text{Temp}_{1–3}, \text{df} = 6) + s(\text{Humid}_0, \text{df} = 3) + \beta_1 \text{DOW} + \beta_2 \text{Holiday} + \beta_3 \text{influenza}
$$

Here $E(Y)$ means the expected daily counts of emergency hospital admission for pneumonia on day $t$; $s(\cdot)$ is the smoothing spline function for nonlinear variables. We examined the residuals of the core model to check whether there were discernable patterns and autocorrelation by means of residual plot and partial autocorrelation function (PACF) plot. The PACF of residuals of the core model (1) was larger than 0.1 for the first two lags, resulting in the addition of two autoregressive terms ($lag_0, lag_1$) to model emergency hospital admissions for pneumonia.

No discernible patterns and no autocorrelation in the residuals are the criteria for an adequate core model set up which is intended to remove all potential confounders in the daily variations of health outcomes. The linear effects of different fractions of PM10 were then estimated for the same day and up to 6 days before the outcome (single-lag effect from lag0 to lag6), as previous studies have justified the linear association between the logarithm of particulate matter air pollution and respiratory morbidity. The overall cumulative effects lasting for 0–3 days and 4–6 days were estimated by unconstrained distributed lag model (dlm03 and dlm46). Sensitivity analyses were conducted to test the effects of PM2.5 with longer exposure windows from lag0 to lag11. The acute effects of PM2.5 on pneumonia were examined in two-pollutant models by further adjustment for the possible confounding effects from PM2.5 and gaseous pollutants.

To identify the most susceptible subpopulation, effect differences by gender and age group were also examined by using the subgroups of pneumonia hospitalisations as the health outcomes. We tested the statistical significance of differences by gender or age group through calculating $(\beta_1 - \beta_2)/\sqrt{\text{SE}_1^2 + \text{SE}_2^2}$, where $\beta_1$ and $\beta_2$ are the estimates for the two categories (eg, female and male patients), and SE1 and SE2.
SE₂ are their respective SEs.  

An absolute value larger than 1.96 indicates a statistically significant difference at the α=0.05 level.

The results were expressed in terms of the percentage increases (Excess Risk (%)) in emergency pneumonia hospital admissions for 10 μg/m³ increment of PMc, and their respective 95% CIs. All analyses were conducted using the ‘mgcv’ package²⁴ in the statistical environment R 3.0.3 (R Development Core Team, 2014: http://www.r-project.org).

RESULTS

Descriptive statistics

We recorded a total of 75 863 emergency hospital admissions for pneumonia in the study population from 1 January 2011 to 31 December 2012, accounting for 38.2% of the total respiratory diseases. The mean daily number of emergency hospital admissions for pneumonia was 104, among which 46.5% were female patients and 53.5% were male patients. The mean daily number of emergency hospital admissions for pneumonia was 104, among which 46.5% were female patients and 53.5% were male patients. The mean daily number of admissions in the different age groups were 12, 15, 11 and 66 for age <15, 15–64, 65–74 and ≥75 years, respectively (table 1).

The citywide daily mean concentrations of PM₂.⁵ and PM₂.₅ were 14.6 and 30.9 μg/m³, respectively (table 1). Generally, PM₁₀ was more than 56.1, 12.4 and 49.4 μg/m³, respectively (table 1). Generally, PM₁₀ was more than 56.1, 12.4 and 49.4 μg/m³, respectively (table 1). Generally, PM₁₀ was more than 56.1, 12.4 and 49.4 μg/m³, respectively (table 1). Generally, PM₁₀ was more than 56.1, 12.4 and 49.4 μg/m³, respectively (table 1). Generally, PM₁₀ was more than 56.1, 12.4 and 49.4 μg/m³, respectively (table 1). Generally, PM₁₀ was more than 56.1, 12.4 and 49.4 μg/m³, respectively (table 1). Generally, PM₁₀ was more than 56.1, 12.4 and 49.4 μg/m³, respectively (table 1). Generally, PM₁₀ was more than 56.1, 12.4 and 49.4 μg/m³, respectively (table 1). Generally, PM₁₀ was more than 56.1, 12.4 and 49.4 μg/m³, respectively (table 1). Generally, PM₁₀ was more than 56.1, 12.4 and 49.4 μg/m³, respectively (table 1).

The daily mean concentrations of NO₂, SO₂ and O₃ were 56.1, 12.4 and 49.4 μg/m³, respectively (table 1). The time series graph showed the daily variations of emergency hospital admissions for pneumonia and air pollution concentrations during the study period (figure 1).

Regression results

Table 3 summarised the effects of the two fractions of PM₁₀ on emergency hospital admissions for pneumonia examined in single pollutant models. We found PM₂.⁵ and PM₂.₅ to be significantly associated with pneumonia emergency hospital admissions on lag₁ to lag₄ days. The 0–3-day cumulative effect (dlm03) of PM₂.⁵ and PM₂.₅ per 10 μg/m³ increment was respectively associated with a 3.33% (95% CI 1.54% to 5.15%) and 1.69% (95% CI 0.68% to 2.70%) increase in emergency hospitalisations for pneumonia. A delayed effect of PM₂.₅ was also found with a 4–6-day cumulative effect (dlm46) of 1.16% (95% CI 0.20% to 2.14%), while the association with PM₂.⁵ became statistically non-significant (table 3). Association with PM₂.⁵ and PM₂.₅ became statistically non-significant on lag₆ day and approached null on longer lag days (figure 2).

In the two-pollutant models, the effects of PM₂.⁵ on emergency hospital admissions for pneumonia decreased slightly but remained statistically significant on lag₁ and lag₄ days, and dlm03 after adjusting for PM₂.₅ at the same lags. Adjustment for the gaseous pollutants showed that the effect estimates of PM₂.⁵ were affected by the inclusion of O₃, but not NO₂ or SO₂ in the model (table 4). O₃ had independent associations with pneumonia on lag₁, lag₄, and dlm03, while PM₂.₅, NO₂ and SO₂ only had independent effects on lag₆.

Stratified analyses by gender (table 5) showed that PM₂.⁵ exposure exhibited slightly larger effects for female patients than for male patients, with the cumulative effect estimates (dlm03) of 4.55% (95% CI 2.07% to 7.09%) and 3.20% (95% CI 0.86% to 5.59%) increase in pneumonia hospitalisations per 10 μg/m³ increment of PM₂.⁵, respectively. At the same time, PM₂.⁵ exposure exhibited a relatively larger effect on older people aged 65 years and older, and on children younger than 15 years old (table 5). Although it appears that female patients, children, and older people might be more vulnerable to the daily PM₂.⁵ exposure, the effect estimate differences between genders or among age groups were not statistically significant.
groups did not reach statistical significance, possibly due to the reduced study power in subgroup analyses.

DISCUSSION
This study is one of the few that have investigated the association between particulate matter pollution and pneumonia hospitalisations. We found PMc and PM2.5 were significantly associated with pneumonia emergency hospital admissions in Hong Kong. The effect estimates of PMc were robust to the adjustment of PM2.5, and gaseous pollutants NO2 or SO2, but were attenuated upon adjustment of O3. It appears that female patients, children and older people might be more vulnerable to PMc exposure.

One of our previous studies detected significant positive associations of PMc and PM2.5 with emergency hospitalisations for overall respiratory diseases and COPD, but not for other specific causes.14 One single site monitoring data was used in that study to estimate the population exposure, which may have resulted in overestimation or underestimation of the true exposure levels.

Table 2  Pearson correlation coefficients between particle concentration, gaseous pollutants and weather conditions*

<table>
<thead>
<tr>
<th>Pollutants</th>
<th>PM10</th>
<th>PM2.5</th>
<th>PMc</th>
<th>NO2</th>
<th>O3</th>
<th>SO2</th>
<th>Temperature</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM10</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM2.5</td>
<td>0.956</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PMc</td>
<td>0.835</td>
<td>0.640</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NO2</td>
<td>0.688</td>
<td>0.734</td>
<td>0.437</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O3</td>
<td>0.600</td>
<td>0.559</td>
<td>0.513</td>
<td>0.463</td>
<td>1.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SO2</td>
<td>0.458</td>
<td>0.496</td>
<td>0.273</td>
<td>0.593</td>
<td>0.312</td>
<td>1.000</td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>−0.400</td>
<td>−0.413</td>
<td>−0.292</td>
<td>−0.282</td>
<td>0.144</td>
<td>0.067</td>
<td>1.000</td>
</tr>
<tr>
<td>Relative humidity</td>
<td>−0.528</td>
<td>−0.472</td>
<td>−0.498</td>
<td>−0.311</td>
<td>−0.530</td>
<td>−0.443</td>
<td>0.172</td>
</tr>
</tbody>
</table>

*All correlation coefficients except that between SO2 and temperature are statistically significant (p<0.05). PM2.5, particles with an aerodynamic diameter less than 2.5 μm; PM10, particles with an aerodynamic diameter less than 10 μm; PMc, coarse particulate matter.

Figure 1  Time series graph to show the daily variation of emergency hospital admissions for pneumonia and concentrations of air pollutants. PM2.5, particles with an aerodynamic diameter less than 2.5 μm; PMc, coarse particulate matter.
Toxicological studies proposed that the acute lung injury and an imbalance of inflammatory mediators might be causative mechanisms for the short-term association of PMc with pneumonia development. Exposure of human monocytes to particle extracts for 6 h at 37°C induced significant cytoxicity and proinflammatory cytokines interleukin-6 and interleukin-8.\textsuperscript{13} Particulate matter is likely immunsuppressive and may undermine the normal pulmonary antimicrobial defence mechanisms.\textsuperscript{30} Hoppo and colleagues\textsuperscript{31} instilled particulate samples intratracheally to healthy mice either once or repeatedly on days 1, 3, and 6 of the study week; they found repeated intratracheal instillation of fine and coarse particulate samples evoked enhanced pulmonary inflammation and cytotoxicity. The particulate matter induced oxidative stress and inflammation which may impair the cellular defence and immune system and increase susceptibility to bacterial pathogens. Besides the toxicological mechanisms related to its physical and chemical properties, PMc originated from the soil and abrasive mechanical processes may also carry biological materials such as bacteria, moulds or pollens, and are therefore likely to produce additional adverse health effects in the respiratory system.\textsuperscript{32}

Our current time-series study findings on the short-term association between PMc pollution and pneumonia emergency hospitalisations were consistent with the toxicological findings of the acute adverse effects of PMc.

The associations of PMc with pneumonia hospitalisations were generally robust to the adjustment of all co-pollutants, except for \textit{O}_3. These results may reflect the actual difference in toxicity of the corresponding pollutants themselves, but it is impossible to differentiate such factors in multi-pollutant models. The correlation coefficient between PMc and \textit{O}_3 (\textit{r}=0.513) was higher than that between PMc and NO\textsubscript{2} (\textit{r}=0.437) or PM\textsubscript{2.5} and SO\textsubscript{2} (\textit{r}=0.273) in Hong Kong, which might have made the effect estimates of PMc unstable upon adjustment for \textit{O}_3. It is likely PMc and \textit{O}_3 were independent players whereas the larger measurement error of PMc prevented it from remaining statistically significant, along with \textit{O}_3, in the two-pollutant model.\textsuperscript{33} Indeed, PMc concentrations estimated by subtracting PM\textsubscript{2.5} from PM\textsubscript{10} measurements were subject to double measurement error whereas directly measured ozone was subject to fewer measurement errors.

Female patients, children and elders might be more vulnerable to daily PMc exposure. Children generally breathe more rapidly than adults; they may have more exposure to air pollutants per kilogram of body weight. Older people may have a weaker immune system and higher frequency of chronic respiratory and heart diseases and thus be more vulnerable to air pollution. Female patients had substantially lower smoking prevalence compared with male patients, while the non-smokers may be more sensitive to air pollution exposure.\textsuperscript{22}

![Figure 2](http://thorax.bmj.com) Sensitivity analysis to show the effects of PM\textsubscript{c} and PM\textsubscript{2.5} on emergency hospital admissions for pneumonia with longer exposure windows from lag\textsubscript{0} to lag\textsubscript{13}. Effects were estimated as excess risk (95% CI) per 10 μg/m\textsuperscript{3} increment of PM. PM\textsubscript{2.5}, particles with an aerodynamic diameter less than 2.5 μm; PM\textsubscript{c}, coarse particulate matter.

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**Table 3** Effects of different fractions of PM\textsubscript{10} on emergency hospital admissions for pneumonia by lags in single pollutant models, 2011–2012.\textsuperscript{\*} (ER\% (95% CI) for 10 μg/m\textsuperscript{3} increment of PM)

<table>
<thead>
<tr>
<th>Lag days</th>
<th>PM\textsubscript{c}</th>
<th>PM\textsubscript{2.5}</th>
</tr>
</thead>
<tbody>
<tr>
<td>lag\textsubscript{0}</td>
<td>1.06 (–0.22 to 2.35)</td>
<td>0.68 (–0.07 to 1.43)</td>
</tr>
<tr>
<td>lag\textsubscript{1}</td>
<td>1.57 (0.42 to 2.73)</td>
<td>0.72 (0.00 to 1.44)</td>
</tr>
<tr>
<td>lag\textsubscript{2}</td>
<td>1.83 (0.70 to 2.97)</td>
<td>0.85 (0.15 to 1.56)</td>
</tr>
<tr>
<td>lag\textsubscript{3}</td>
<td>1.76 (0.65 to 2.88)</td>
<td>1.15 (0.46 to 1.84)</td>
</tr>
<tr>
<td>lag\textsubscript{4}</td>
<td>1.14 (0.03 to 2.26)</td>
<td>1.47 (0.80 to 2.14)</td>
</tr>
<tr>
<td>lag\textsubscript{5}</td>
<td>1.07 (–0.03 to 2.19)</td>
<td>0.89 (0.22 to 1.57)</td>
</tr>
<tr>
<td>lag\textsubscript{6}</td>
<td>0.82 (–0.27 to 1.93)</td>
<td>0.34 (–0.33 to 1.02)</td>
</tr>
<tr>
<td>dim\textsubscript{03} \textdagger</td>
<td>3.33 (1.54 to 5.15)</td>
<td>1.69 (0.68 to 2.70)</td>
</tr>
<tr>
<td>dim\textsubscript{46} \textdagger</td>
<td>0.97 (–0.65 to 2.62)</td>
<td>1.16 (0.20 to 2.14)</td>
</tr>
</tbody>
</table>

\*Generalised additive Poisson models were used, adjusting for long-term trend, seasonality, weather factors, calendar effect and influenza epidemics.

\dag Overall cumulative effects of DTR lasting for 0–3 (dim\textsubscript{03}) and 4–6 days (dim\textsubscript{46}) were estimated by unconstrained distributed lag models. Statistically significant effect estimates are in bold.

ER, excess risk; PM\textsubscript{2.5}, particles with an aerodynamic diameter less than 2.5 μm; PM\textsubscript{c}, coarse particulate matter.
Table 4 Effects of PMc on emergency hospital admissions for pneumonia by lags in two-pollutant models, 2011–2012 (ER% (95% CI) for 10 μg/m³ increment of PMc)

<table>
<thead>
<tr>
<th>Lag days</th>
<th>PMc+PM2.5*</th>
<th>PMc+NO₂*</th>
<th>PMc+O₃*</th>
<th>PMc+SO₂*</th>
</tr>
</thead>
<tbody>
<tr>
<td>PMc</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lag0</td>
<td>0.68 (−0.73 to 2.12)</td>
<td>0.99 (−0.30 to 2.30)</td>
<td>0.66 (−0.68 to 2.03)</td>
<td>1.06 (−0.23 to 2.36)</td>
</tr>
<tr>
<td>lag1</td>
<td>1.32 (0.01 to 2.65)</td>
<td>1.52 (0.35 to 2.70)</td>
<td>0.65 (−0.61 to 1.93)</td>
<td>1.73 (0.57 to 2.91)</td>
</tr>
<tr>
<td>lag2</td>
<td>1.52 (0.20 to 2.85)</td>
<td>1.71 (0.56 to 2.88)</td>
<td>1.08 (−0.20 to 2.37)</td>
<td>1.74 (0.57 to 2.91)</td>
</tr>
<tr>
<td>lag3</td>
<td>1.08 (−0.22 to 2.39)</td>
<td>1.41 (0.26 to 2.56)</td>
<td>0.92 (−0.35 to 2.21)</td>
<td>1.42 (0.27 to 2.58)</td>
</tr>
<tr>
<td>dlm03†</td>
<td>2.43 (0.41 to 4.50)</td>
<td>2.90 (1.06 to 4.77)</td>
<td>1.38 (−0.70 to 3.51)</td>
<td>3.03 (1.20 to 4.89)</td>
</tr>
</tbody>
</table>

Second pollutant

<table>
<thead>
<tr>
<th>Lag days</th>
<th>PMc+PM2.5*</th>
<th>PMc+NO₂*</th>
<th>PMc+O₃*</th>
<th>PMc+SO₂*</th>
</tr>
</thead>
<tbody>
<tr>
<td>lag0</td>
<td>0.51 (−0.32 to 1.34)</td>
<td>0.21 (−0.46 to 0.88)</td>
<td>0.39 (−0.03 to 0.81)</td>
<td>−0.03 (−1.94 to 1.91)</td>
</tr>
<tr>
<td>lag1</td>
<td>0.32 (−0.50 to 1.14)</td>
<td>0.15 (−0.49 to 0.79)</td>
<td>0.64 (0.26 to 1.02)</td>
<td>−1.38 (−3.09 to 0.37)</td>
</tr>
<tr>
<td>lag2</td>
<td>0.38 (−0.43 to 1.19)</td>
<td>0.30 (−0.33 to 0.92)</td>
<td>0.47 (0.09 to 0.85)</td>
<td>0.59 (−1.19 to 2.41)</td>
</tr>
<tr>
<td>lag3</td>
<td>0.80 (0.00 to 1.61)</td>
<td>0.77 (0.16 to 1.39)</td>
<td>0.50 (0.13 to 0.88)</td>
<td>1.92 (0.17 to 3.70)</td>
</tr>
<tr>
<td>dlm03†</td>
<td>1.00 (−0.13 to 2.14)</td>
<td>0.70 (−0.21 to 1.62)</td>
<td>0.96 (0.36 to 1.57)</td>
<td>1.13 (−1.64 to 3.97)</td>
</tr>
</tbody>
</table>

*Two pollutants were included in the model at the same lags. Statistically significant effect estimates are in bold.
†Overall cumulative effects of DTR lasting for 0–3 days (dlm03) were estimated by unconstrained distributed lag models. ER, excess risk; PM2.5, particles with an aerodynamic diameter less than 2.5 μm; PMc, coarse particulate matter.

Table 5 Effects of PMc on emergency hospital admissions for pneumonia by gender and age groups, 2011–2012 (ER% (95% CI) for 10 μg/m³ increment of PMc)

<table>
<thead>
<tr>
<th>Gender</th>
<th>lag0</th>
<th>lag1</th>
<th>lag2</th>
<th>lag3</th>
<th>dlm03†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>1.47 (−0.30 to 3.28)</td>
<td>1.06 (−0.53 to 2.67)</td>
<td>2.34 (0.79 to 3.92)</td>
<td>2.92 (1.40 to 4.45)</td>
<td>4.55 (2.07 to 7.09)</td>
</tr>
<tr>
<td>Male</td>
<td>0.95 (−0.73 to 2.65)</td>
<td>2.38 (0.88 to 3.91)</td>
<td>1.89 (0.40 to 3.40)</td>
<td>1.40 (−0.07 to 2.89)</td>
<td>3.20 (0.86 to 5.59)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Age group</th>
<th>lag0</th>
<th>lag1</th>
<th>lag2</th>
<th>lag3</th>
<th>dlm03†</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;15</td>
<td>1.26 (−1.96 to 4.59)</td>
<td>1.73 (−1.15 to 4.70)</td>
<td>3.33 (0.45 to 6.29)</td>
<td>3.60 (0.79 to 6.49)</td>
<td>5.60 (0.97 to 10.44)</td>
</tr>
<tr>
<td>15–64</td>
<td>1.59 (−1.49 to 4.78)</td>
<td>0.91 (−1.88 to 3.78)</td>
<td>−0.02 (−2.77 to 2.81)</td>
<td>1.56 (−1.16 to 4.35)</td>
<td>2.56 (−1.69 to 6.99)</td>
</tr>
<tr>
<td>65–74</td>
<td>1.90 (−1.61 to 5.54)</td>
<td>2.44 (−0.71 to 5.69)</td>
<td>3.29 (0.27 to 6.39)</td>
<td>5.33 (2.36 to 8.39)</td>
<td>7.33 (2.41 to 12.49)</td>
</tr>
<tr>
<td>≥75</td>
<td>1.23 (−0.39 to 2.88)</td>
<td>2.36 (0.92 to 3.82)</td>
<td>2.93 (1.53 to 4.35)</td>
<td>2.41 (1.04 to 3.80)</td>
<td>4.52 (2.29 to 6.80)</td>
</tr>
</tbody>
</table>

*Overall cumulative effects of pollutants lasting for 0–3 days (dlm03) were estimated by unconstrained distributed lag models. Statistically significant effect estimates are in bold.
ER, excess risk; PM2.5, particles with an aerodynamic diameter less than 2.5 μm; PMc, coarse particulate matter.

Consistent with previous studies, our study suggested that special attention can be paid to the vulnerable populations such as female patients, children and older people in terms of PMc exposure.

This study had some limitations. We estimated PMc concentrations by subtracting PM2.5 from PM10 measurements so that PMc concentrations were affected by double measurement errors, which may dilute/underestimate the true associations. As in all other monitor-based time series studies, indoor air pollution and personal exposure data were not available, so outdoor monitoring data were used to represent the population exposure to ambient particles. Although a simulation study suggested that for PM2.5, ambient concentrations available from local monitoring stations might be adequate surrogates for the corresponding total personal exposures, the relationship between personal exposure and ambient concentrations of PMc is much less certain. Another limitation was that we could not identify the readmissions for patients with pneumonia according to the available data. It is likely that some patients, especially children and older people, were admitted to hospital more than once during the study period. Such repeated admissions could lead to a temporal dependence reflected by autocorrelation in the time series of hospitalisation counts.

This study also had a few strengths. Although we used only 2 years of data for analysis in the current study due to the constraints of PMc data availability in multiple air monitoring stations, our daily PMc concentration time series were contiguous in the whole study period, which may facilitate the standard computation procedures and prevent the loss of study power. This was different from some earlier studies that used every third or sixth day PMc data. We used the emergency hospital admissions for pneumonia as the health outcome. These unscheduled pneumonia hospitalisations were more likely to be community acquired and might reflect the acute effects of ambient PMc air pollution. We used air monitoring data averaged across 10 general stations dispersed in Hong Kong, which were more representative of the general population exposure than from one single monitoring station.

In conclusion, we found that PMc could play an important role in emergency hospitalisations for pneumonia in Hong Kong. The effects of PMc were robust to the adjustment for PM2.5, and gaseous pollutants NO₂ or SO₂, but not O₃. Air quality regulation specifically for PMc might be considered.

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Contributors HQ, LWT and VCP designed the study, analysed the data and drafted the manuscript; K-FH and TW carried out data collection and interpreted the results; ITSY supervised the conduction of the study.

Competing interests The author HQ was supported by the Postdoctoral Fellowship Scheme 2013–2014 of the Faculty of Medicine, the Chinese University of Hong Kong.

Ethics approval Ethics approval was not required because only aggregated but not any individualised data were used in this study.

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