ORIGINAL ARTICLE

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

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ABSTRACT

Background 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. Pneumonia is an inflammatory condition of the 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. Women, children and older people might be more vulnerable to PMc exposure.

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,1–3 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

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.
Respiratory epidemiology

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.\textsuperscript{15} 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 PM\textsubscript{10} and PM\textsubscript{2.5} 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 PM\textsubscript{2.5} on pneumonia. In this study, we conducted a time series analysis to estimate the acute effect of PM\textsubscript{2.5} on emergency hospital admissions for pneumonia in Hong Kong after controlling for PM\textsubscript{2.5} 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 (PM\textsubscript{10}, NO\textsubscript{2}, O\textsubscript{3}, and SO\textsubscript{2}) in 14 monitoring stations dispersed in different districts of Hong Kong since 1990. Hourly concentrations of PM\textsubscript{2.5} 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 PM\textsubscript{10} and PM\textsubscript{2.5}, 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 PM\textsubscript{10} and PM\textsubscript{2.5} and estimated PM\textsubscript{2.5} concentrations by subtracting daily mean PM\textsubscript{2.5} from PM\textsubscript{10} for each station. Daily average concentrations of PM\textsubscript{2.5} 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 NO\textsubscript{2}, SO\textsubscript{2} and 8 h (10:00–18:00) mean concentration of O\textsubscript{3} to represent the citywide pollution exposure.\textsuperscript{16}

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 publicly funded hospitals providing 24 h accident and emergency services and covering 90% of hospital beds in Hong Kong for local residents.\textsuperscript{16} 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.\textsuperscript{17} 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 PM\textsubscript{2.5} concentrations and the emergency pneumonia hospitalisations. We used the smoothing spline, $s(.)$, to filter out seasonal patterns and long-term trends in daily hospitalisations, and the daily mean temperature and relative humidity.\textsuperscript{18} 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.\textsuperscript{18, 19} We used a df of 8/year for the time trend, a df of six for the mean temperature of the current day (Temp\textsubscript{0}) and the previous 3 days’ moving average (Temp\textsubscript{1–3}), and a df of three for the current day relative humidity (Humid\textsubscript{0}). We included the DOW and public holidays (Holiday) in the model as dummy variables.\textsuperscript{20} 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.\textsuperscript{21}

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

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

Here $E(Y)$ means the expected daily counts of emergency hospital admission for pneumonia on day $t$; $s(.)$ 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\textsubscript{0}, lag\textsubscript{1}) to model emergency hospital admissions for pneumonia.\textsuperscript{18}

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 outcome. The linear effects of different fractions of PM\textsubscript{10} were then estimated for the same day and up to 6 days before the outcome (single-lag effect from lag\textsubscript{0} to lag\textsubscript{6}), as previous studies have justified the linear association between the logarithm of particulate matter air pollution and respiratory morbidity.\textsuperscript{14, 17} The overall cumulative effects lasting for 0–3 days and 4–6 days were estimated by unconstrained distributed lag model (dlm03 and dlm46).\textsuperscript{23} Sensitivity analyses were conducted to test the effects of PM\textsubscript{2.5} with longer exposure windows from lag\textsubscript{0} to lag\textsubscript{16}. The acute effects of PM\textsubscript{2.5} on pneumonia were examined in two-pollutant models by further adjustment for the possible confounding effects from PM\textsubscript{2.5} 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.\textsuperscript{22} We tested the statistical significance of differences by gender or age group through calculating $(\beta_1 - \beta_2)/\sqrt{SE_1^2 + SE_2^2}$, where $\beta_1$ and $\beta_2$ are the estimates for the two categories (eg, female and male patients), and $SE_1$ and $SE_2$ are the standard errors of the estimates.
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 increase of PMc, and their respective 95% CIs. All analyses were conducted using the ‘mgcv’ package 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 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 PMc, and PM2.5 were 14.6 and 30.9 μg/m³, with SD of 8.8 and 16.8 μg/m³, respectively. PM2.5 accounted for a substantial part of the mass concentration of PM10 with an average of 67% in Hong Kong. The daily mean concentrations of NO2, SO2 and O3 were 56.1, 12.4 and 49.4 μg/m³, respectively (table 1). Generally, PM10 was strongly correlated with PM2.5 (correlation coefficient, r=0.956) and PMc (r=0.835); PM2.5 and PMc were moderately correlated (r=0.640). The correlation of PMc with gaseous pollutants was low to moderate (r=0.273 with SO2, 0.437 with NO2 and 0.513 with O3) (table 2). 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 PMc 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 PMc 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 PMc became statistically non-significant (table 3). Association with PMc 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 PMc on emergency hospital admissions for pneumonia decreased slightly but remained statistically significant on lag1 and lag2 days, and dlm03 after adjusting for PM2.5 at the same lags. Adjustment for the gaseous pollutants showed that the effect estimates of PMc 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 lag1.

Stratified analyses by gender (table 5) showed that PMc 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 PMc, respectively. At the same time, PMc 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 PMc exposure, the effect estimate differences between genders or among age

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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

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**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>
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<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>0.734</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NO2</td>
<td>0.688</td>
<td>0.513</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>0.496</td>
<td>0.273</td>
<td>0.593</td>
<td>0.312</td>
<td>1.000</td>
<td></td>
</tr>
<tr>
<td>SO2</td>
<td>0.458</td>
<td>0.559</td>
<td>0.513</td>
<td>0.463</td>
<td>1.000</td>
<td></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.

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**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.
PMc exposure misclassification because of the spatial variability of PMc. In the current study, we made use of the daily pairwise monitoring data of PM10 and PM1.5 in 10 general stations dispersed in Hong Kong to correlate the citywide daily average PMc concentrations with the daily counts of pneumonia admissions. The spatial variability of PMc concentrations was one justification for using all the PMc data from the 10 air monitors in the city. But we were not able to assign patients the monitored concentrations based on proximity to hospital or residential address because we did not have access to individual patients’ information on their residential addresses or hospital names. As the average levels across the 10 monitors were more representative of the general population in the area than from a single monitor, we correlated daily counts of pneumonia with the citywide daily average concentrations of PMc. We observed a significant positive association between PMc exposure and emergency hospital admissions for pneumonia. To date, few studies have examined and reported the adverse health effects of coarse particulates, which focused more on the overall respiratory diseases, COPD, asthma, or overall cardiovascular diseases.

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 cytotoxicity and proinflammatory cytokines interleukin-6 and interleukin-8. Particulate matter is likely immunosuppressive and may undermine the normal pulmonary antimicrobial defence mechanisms. Happon and colleagues 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 characteristics, 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.

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 O3. 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 O3 (r=0.513) was higher than that between PMc and NO2 (r=0.437) or PMc and SO2 (r=0.273) in Hong Kong, which might have made the effect estimates of PMc unstable upon adjustment for O3. It is likely PMc and O3 were independent players whereas the larger measurement error of PMc prevented it from remaining statistically significant, along with O3, in the two-pollutant model. Indeed, PMc concentrations estimated by subtracting PM2.5 from PM10 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.

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Table 3. Effects of different fractions of PM10 on emergency hospital admissions for pneumonia by lags in single pollutant models, 2011–2012* (ER% (95% CI) for 10 μg/m3 increment of PM)

<table>
<thead>
<tr>
<th>Lag days</th>
<th>PMc</th>
<th>PM2.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>lag0δ</td>
<td>1.06 (0.22 to 2.35)</td>
<td>0.68 (0.07 to 1.43)</td>
</tr>
<tr>
<td>lag1δ</td>
<td>1.57 (0.22 to 2.73)</td>
<td>0.72 (0.00 to 1.44)</td>
</tr>
<tr>
<td>lag2δ</td>
<td>1.83 (0.70 to 2.97)</td>
<td>0.85 (0.15 to 1.56)</td>
</tr>
<tr>
<td>lag3δ</td>
<td>1.76 (0.65 to 2.88)</td>
<td>1.15 (0.46 to 1.84)</td>
</tr>
<tr>
<td>lag4δ</td>
<td>1.14 (0.03 to 2.26)</td>
<td>1.47 (0.80 to 2.14)</td>
</tr>
<tr>
<td>lag5δ</td>
<td>1.07 (0.03 to 2.19)</td>
<td>0.89 (0.22 to 1.57)</td>
</tr>
<tr>
<td>lag6δ</td>
<td>0.82 (0.27 to 1.93)</td>
<td>0.34 (0.03 to 1.02)</td>
</tr>
<tr>
<td>dlm03†</td>
<td>3.33 (1.54 to 5.15)</td>
<td>1.69 (0.68 to 2.70)</td>
</tr>
<tr>
<td>dlm46†</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.
†Overall cumulative effects of DTR lasting for 0–3 (dlm03) and 4–6 days (dlm46) 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.

Figure 2. Sensitivity analysis to show the effects of PMc and PM2.5 on emergency hospital admissions for pneumonia with longer exposure windows from lag0 to lag43. Effects were estimated as excess risk (95% CI) per 10 μg/m3 increment of PM. PM2.5, particles with an aerodynamic diameter less than 2.5 μm; PMc, coarse particulate matter.
Consistent with previous studies, we found 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.

Acknowledgements The authors thank the Hospital Authority for providing hospital admissions data, the Hong Kong Environmental Protection Department for

### 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>
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<tbody>
<tr>
<td>PMc</td>
<td></td>
<td></td>
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<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>
<tr>
<td>Second pollutant</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<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; PMc, particles with an aerodynamic diameter less than 2.5 μm; PM2.5, 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; PMc, particles with an aerodynamic diameter less than 2.5 μm; PM2.5, coarse particulate matter.
providing air pollution monitoring data, and the Hong Kong Observatory for the temperature and humidity data.

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

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**REFERENCES**