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 (PM_c, particulate matter between 2.5 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 PM_c exposure.

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

Method PM_c concentrations were estimated by subtracting $PM_{2.5}$ from PM_{10} 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 PM_c were computed from the 10 stations. Generalised additive Poisson models were used to examine the relationship between PM_c and daily emergency hospital admissions for pneumonia, adjusting for $PM_{2.5}$ and gaseous pollutants (NO_2, SO_2 and O_3). Subgroup analyses by gender and age were also performed to identify the most susceptible subpopulations.

Results PM_c and $PM_{2.5}$ were significantly associated with emergency pneumonia hospitalisations. Every 10 μg/m^3 increment of PM_c in the past 4 days (lag_0–lag_3) was associated with a 3.33% (95% CI 1.54% to 5.15%) increase in emergency hospitalisations for pneumonia. The effect estimates of PM_c were robust to the adjustment of $PM_{2.5}$, NO_2 or SO_2, but attenuated on the inclusion of O_3 in the model. Women, children and older people might be more vulnerable to PM_c exposure.

Conclusions Short-term PM_c exposure is associated with emergency hospitalisations for pneumonia in Hong Kong. Air quality regulation specifically for PM_c might be considered.

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 (PM_c) exposure?

What is the bottom line?

- We found an association between PM_c exposure and emergency hospital admissions for pneumonia and the effect estimates of PM_c were robust to the adjustment of $PM_{2.5}$, NO_2 or SO_2, but were attenuated on the inclusion of O_3 in the model.

Why read on?

- The reliable daily pairwise monitoring data of PM_{10} and $PM_{2.5}$ 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 PM_c and pneumonia emergency hospitalisations.

INTRODUCTION

Although the effects of fine particulate matter pollution ($PM_{2.5}$, particles with an aerodynamic diameter less than 2.5 μm) associated with respiratory diseases have been well documented,[1] epidemiological research on the effects of coarse particles (PM_c, particulate matter between 2.5 and 10 μm in aerodynamic diameter) on respiratory morbidity is sparse and inconclusive.[2 3] Examining the association between PM_c 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.[4 5]

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 pneumonia[5 13] and the results have been inconsistent. In a previous study, we used data from a single monitoring station and found the association between PM_c and emergency hospitalisations for overall respiratory diseases and COPD, but failed to detect the effects of PM_c on the other endpoints of respiratory diseases such as asthma, etc,[14] which was probably due to the spatial heterogeneity of PM_c 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.[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_{10} and $PM_{2.5}$ 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_c on pneumonia. In this study, we conducted a time series analysis to estimate the acute effect of PM_c on emergency hospital admissions for pneumonia in Hong Kong after controlling for $PM_{2.5}$ 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_{10}, NO_2, O_3, and SO_2) in 14 monitoring stations dispersed in different districts of Hong Kong since 1990. Hourly concentrations of $PM_{2.5}$ 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_{10} and $PM_{2.5}$, 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_{10} and $PM_{2.5}$ and estimated PM_c concentrations by subtracting daily mean $PM_{2.5}$ from PM_{10} for each station. Daily average concentrations of PM_c 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_2, SO_2 and 8 h (10:00–18:00) mean concentration of O_3 to represent the citywide pollution exposure.[14]

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.[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.[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_c 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.[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.[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 ($Temp_0$) and the previous 3 days' moving average ($Temp_{1-3}$), and a df of three for the current day relative humidity ($Humid_0$). We included the DOW and public holidays (Holiday) in the model as dummy variables.[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.[17]

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

$$\begin{aligned}\log(\mathrm{E(Y)}) = \alpha &+ \mathrm{s(t,\ df} = 8/\mathrm{year} \times 2\ \mathrm{years)} \\ &+ \mathrm{s(Temp_0,\ df} = 6) + \mathrm{s(Temp_{1-3},\ df} = 6) \\ &+ \mathrm{s(Humid_0,\ df} = 3) \\ &+ \beta_1 \mathrm{DOW} + \beta_2 \mathrm{Holiday} + \beta_3 \mathrm{influenza}\end{aligned} \quad (1)$$

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_1, lag_2) to model emergency hospital admissions for pneumonia.[14] 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_{10} were then estimated for the same day and up to 6 days before the outcome (single-lag effect from lag_0 to lag_6), as previous studies have justified the linear association between the logarithm of particulate matter air pollution and respiratory morbidity.[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).[21] Sensitivity analyses were conducted to test the effects of PM_c with longer exposure windows from lag_0 to lag_{13}. The acute effects of PM_c on pneumonia were examined in two-pollutant models by further adjustment for the possible confounding effects from $PM_{2.5}$ 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.[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 β_1 and β_2 are the estimates for the two categories (eg, female and male patients), and SE_1 and

Table 1 Summary statistics of daily emergency hospital admissions for pneumonia, air pollution concentrations and weather conditions in Hong Kong 2011–2012

Variables	No of days	Mean	SD	Centiles Min	25th	50th	75th	Max
Daily emergency hospital admissions								
Total pneumonia	731	103.8	23.8	52	86	99	118.5	184
Female patients	731	48.3	12.7	20	39	46	56	87
Male patients	731	55.5	13.4	25	46	53	63	105
Age <15	731	11.7	5.1	2	8	11	14.5	32
Age 15–64	731	14.7	5.3	3	11	14	17	40
Age 65–74	731	11.1	3.9	1	8	11	14	24
Age ≥75	731	66.3	16.1	34	54	64	77	119
Pollution concentration (μg/m^3)								
PM_{10}	731	45.44	23.24	7.56	25.82	42.95	61.40	157.35
$PM_{2.5}$	731	30.88	16.79	4.92	16.70	28.38	42.80	85.81
PM_c	731	14.64	8.78	1.86	8.38	12.77	18.68	108.91
NO_2	731	56.05	17.74	12.99	43.53	54.21	66.44	136.22
O_3	731	49.42	32.40	6.12	24.73	41.71	68.52	189.76
SO_2	731	12.42	6.01	3.27	8.00	11.15	15.27	45.32
Meteorology measures								
Temperature (°C)	731	23.2	5.5	8.7	18.7	24.8	28.2	31.8
Relative humidity (%)	731	78.4	9.8	39.0	74.0	79.0	85.0	99.0

Max., maximum; Min., minimum; $PM_{2.5}$, particles with an aerodynamic diameter less than 2.5 μm; PM_{10}, particles with an aerodynamic diameter less than 10 μm; PM_c, coarse particulate matter.

SE_2 are their respective SEs.[22 23] 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/m^3 increment of PM_c, and their respective 95% CIs. All analyses were conducted using the 'mgcv' package[24] 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 PM_c, and $PM_{2.5}$ were 14.6 and 30.9 μg/m^3, with SD of 8.8 and 16.8 μg/m^3, respectively. $PM_{2.5}$ accounted for a substantial part of the mass concentration of PM_{10} with an average of 67% in Hong Kong. The daily mean concentrations of NO_2, SO_2 and O_3 were 56.1, 12.4 and 49.4 μg/m^3, respectively (table 1). Generally, PM_{10} was strongly correlated with $PM_{2.5}$ (correlation coefficient, r=0.956) and PM_c (r=0.835); $PM_{2.5}$ and PM_c were moderately correlated (r=0.640). The correlation of PM_c with gaseous pollutants was low to moderate (r=0.273 with SO_2, 0.437 with NO_2 and 0.513 with O_3) (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 PM_{10} on emergency hospital admissions for pneumonia examined in single pollutant models. We found PM_c and $PM_{2.5}$ to be significantly associated with pneumonia emergency hospital admissions on lag_1 to lag_4 days. The 0–3-day cumulative effect (dlm03) of PM_c and $PM_{2.5}$ per 10 μg/m^3 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 $PM_{2.5}$ 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 PM_c became statistically non-significant (table 3). Association with PM_c and $PM_{2.5}$ became statistically non-significant on lag_6 day and approached null on longer lag days (figure 2).

In the two-pollutant models, the effects of PM_c on emergency hospital admissions for pneumonia decreased slightly but remained statistically significant on lag_1 and lag_2 days, and dlm03 after adjusting for $PM_{2.5}$ at the same lags. Adjustment for the gaseous pollutants showed that the effect estimates of PM_c were affected by the inclusion of O_3, but not NO_2 or SO_2 in the model (table 4). O_3 had independent associations with pneumonia on lag_1–lag_3 and dlm03, while $PM_{2.5}$, NO_2 and SO_2 only had independent effects on lag_3.

Stratified analyses by gender (table 5) showed that PM_c 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/m^3 increment of PM_c, respectively. At the same time, PM_c 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 PM_c exposure, the effect estimate differences between genders or among age

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

Pollutants	PM_{10}	$PM_{2.5}$	PM_c	NO_2	O_3	SO_2	Temperature
PM_{10}	1.000						
$PM_{2.5}$	0.956	1.000					
PM_c	0.835	0.640	1.000				
NO_2	0.688	0.734	0.437	1.000			
O_3	0.600	0.559	0.513	0.463	1.000		
SO_2	0.458	0.496	0.273	0.593	0.312	1.000	
Temperature	−0.400	−0.413	−0.292	−0.282	0.144	0.067	1.000
Relative humidity	−0.528	−0.472	−0.498	−0.311	−0.530	−0.443	0.172

*All correlation coefficients except that between SO_2 and temperature are statistically significant ($p<0.05$).
$PM_{2.5}$, particles with an aerodynamic diameter less than 2.5 μm; PM_{10}, particles with an aerodynamic diameter less than 10 μm; PM_c, coarse particulate matter.

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 PM_c and $PM_{2.5}$ were significantly associated with pneumonia emergency hospital admissions in Hong Kong. The effect estimates of PM_c were robust to the adjustment of $PM_{2.5}$, and gaseous pollutants NO_2 or SO_2, but were attenuated upon adjustment of O_3. It appears that female patients, children and older people might be more vulnerable to PM_c exposure.

One of our previous studies detected significant positive associations of PM_c and $PM_{2.5}$ 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

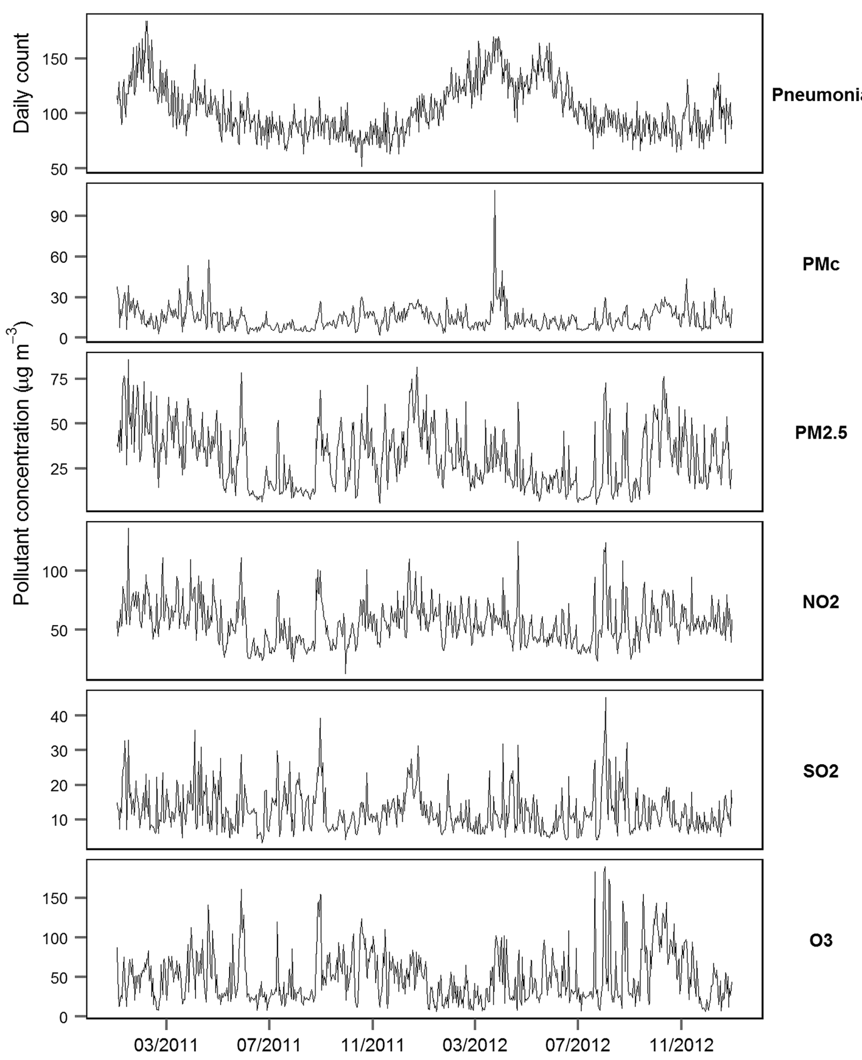

Figure 1 Time series graph to show the daily variation of emergency hospital admissions for pneumonia and concentrations of air pollutants. $PM_{2.5}$, particles with an aerodynamic diameter less than 2.5 μm; PM_c, coarse particulate matter.

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

Lag days	PM_c	$PM_{2.5}$
Lag_0	1.06 (−0.22 to 2.35)	0.68 (−0.07 to 1.43)
lag_1	**1.57 (0.42 to 2.73)**	**0.72 (0.00 to 1.44)**
lag_2	**1.83 (0.70 to 2.97)**	**0.85 (0.15 to 1.56)**
lag_3	**1.76 (0.65 to 2.88)**	**1.15 (0.46 to 1.84)**
lag_4	**1.14 (0.03 to 2.26)**	**1.47 (0.80 to 2.14)**
lag_5	1.07 (−0.03 to 2.19)	**0.89 (0.22 to 1.57)**
lag_6	0.82 (−0.27 to 1.93)	0.34 (−0.33 to 1.02)
dlm03†	**3.33 (1.54 to 5.15)**	**1.69 (0.68 to 2.70)**
dlm46†	0.97 (−0.65 to 2.62)	**1.16 (0.20 to 2.14)**

*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; $PM_{2.5}$, particles with an aerodynamic diameter less than 2.5 μm; PM_c, coarse particulate matter.

PM_c exposure misclassification because of the spatial variability of PM_c.[25] In the current study, we made use of the daily pairwise monitoring data of PM_{10} and $PM_{2.5}$ in 10 general stations dispersed in Hong Kong to correlate the citywide daily average PM_c concentrations with the daily counts of pneumonia admissions. The spatial variability of PM_c concentrations was one justification for using all the PM_c 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 PM_c. We observed a significant positive association between PM_c exposure and emergency hospital admissions for pneumonia. To date, few studies have examined and reported the adverse health effects of coarse particles, which focused more on the overall respiratory diseases, COPD, asthma, or overall cardiovascular diseases.[3 5 13 14 26–28] Only one study conducted in Toronto, Canada reported the significant association between coarse particles and hospitalisation for respiratory infections, including pneumonia in children.[13] Another Taiwan study found an acute increase in pneumonia hospitalisations on Asian dust storm event days,[29] in which air pollution was predominated by high concentrations of coarse particles.

Toxicological studies proposed that the acute lung injury and an imbalance of inflammatory mediators might be causative mechanisms for the short-term association of PM_c 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.[15] Particulate matter is likely immuosuppressive and may undermine the normal pulmonary antimicrobial defence mechanism.[30] Happo and colleagues[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 characteristics, PM_c 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.[32] Our current time-series study findings on the short-term association between PM_c pollution and pneumonia emergency hospitalisations were consistent with the toxicological findings of the acute adverse effects of PM_c.

The associations of PM_c with pneumonia hospitalisations were generally robust to the adjustment of all co-pollutants, except for 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 PM_c and O_3 (r=0.513) was higher than that between PM_c and NO_2 (r=0.437) or PM_c and SO_2 (r=0.273) in Hong Kong, which might have made the effect estimates of PM_c unstable upon adjustment for O_3. It is likely PM_c and O_3 were independent players whereas the larger measurement error of PM_c prevented it from remaining statistically significant, along with O_3, in the two-pollutant model.[33] Indeed, PM_c concentrations estimated by subtracting $PM_{2.5}$ from PM_{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 PM_c 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 nonsmokers may be more sensitive to air pollution exposure.[22]

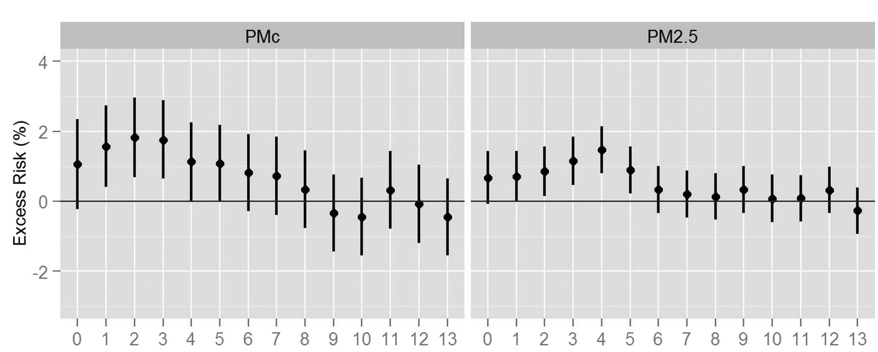

Figure 2 Sensitivity analysis to show the effects of PM_c and $PM_{2.5}$ on emergency hospital admissions for pneumonia with longer exposure windows from lag_0 to lag_{13}. Effects were estimated as excess risk (95% CI) per 10 μg/m^3 increment of PM. $PM_{2.5}$, particles with an aerodynamic diameter less than 2.5 μm; PM_c, coarse particulate matter.

Table 4 Effects of PM_c on emergency hospital admissions for pneumonia by lags in two-pollutant models, 2011–2012 (ER% (95% CI) for 10 μg/m^3 increment of PM_c)

Lag days	PM_c+$PM_{2.5}$*	PM_c+NO_2*	PM_c+O_3*	PM_c+SO_2*
PM_c				
lag_0	0.68 (−0.73 to 2.12)	0.99 (−0.30 to 2.30)	0.66 (−0.68 to 2.03)	1.06 (−0.23 to 2.36)
lag_1	**1.32 (0.01 to 2.65)**	**1.52 (0.35 to 2.70)**	0.65 (−0.61 to 1.93)	**1.73 (0.57 to 2.91)**
lag_2	**1.52 (0.20 to 2.85)**	**1.71 (0.56 to 2.88)**	1.08 (−0.20 to 2.37)	**1.74 (0.57 to 2.91)**
lag_3	1.08 (−0.22 to 2.39)	1.41 (0.26 to 2.56)	0.92 (−0.35 to 2.21)	**1.42 (0.27 to 2.58)**
dlm03†	**2.43 (0.41 to 4.50)**	**2.90 (1.06 to 4.77)**	1.38 (−0.70 to 3.51)	**3.03 (1.20 to 4.89)**
Second pollutant				
lag_0	0.51 (−0.32 to 1.34)	0.21 (−0.46 to 0.88)	0.39 (−0.03 to 0.81)	−0.03 (−1.94 to 1.91)
lag_1	0.32 (−0.50 to 1.14)	0.15 (−0.49 to 0.79)	**0.64 (0.26 to 1.02)**	−1.38 (−3.09 to 0.37)
lag_2	0.38 (−0.43 to 1.19)	0.30 (−0.33 to 0.92)	**0.47 (0.09 to 0.85)**	0.59 (−1.19 to 2.41)
lag_3	**0.80 (0.00 to 1.61)**	**0.77 (0.16 to 1.39)**	**0.50 (0.13 to 0.88)**	**1.92 (0.17 to 3.70)**
dlm03†	1.00 (−0.13 to 2.14)	0.70 (−0.21 to 1.62)	**0.96 (0.36 to 1.57)**	1.13 (−1.64 to 3.97)

*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; $PM_{2.5}$, particles with an aerodynamic diameter less than 2.5 μm; PM_c, coarse particulate matter.

Consistent with previous studies,[13 22 34] our study suggested that special attention can be paid to the vulnerable populations such as female patients, children and older people in terms of PM_c exposure.

This study had some limitations. We estimated PM_c concentrations by subtracting $PM_{2.5}$ from PM_{10} measurements so that PM_c 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 $PM_{2.5}$, ambient concentrations available from local monitoring stations might be adequate surrogates for the corresponding total personal exposures,[35] the relationship between personal exposure and ambient concentrations of PM_c 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.[34]

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 PM_c data availability in multiple air monitoring stations, our daily PM_c 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 PM_c data.[3 13] 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 PM_c 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 PM_c could play an important role in emergency hospitalisations for pneumonia in Hong Kong. The effects of PM_c were robust to the adjustment for $PM_{2.5}$, and gaseous pollutants NO_2 or SO_2, but not O_3. Air quality regulation specifically for PM_c might be considered.

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Table 5 Effects of PM_c on emergency hospital admissions for pneumonia by gender and age groups, 2011–2012 (ER% (95% CI) for 10 μg/m^3 increment of PM_c)

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

*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.
Differences of the effect estimates of PM_c between genders or among age groups were statistically non-significant (p>0.05).
ER, excess risk; $PM_{2.5}$, particles with an aerodynamic diameter less than 2.5 μm; PM_c, 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 TWW carried out data collection and interpreted the results; ITSY supervised the conduction of the study.

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

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