Short-term Effects of Fine and Coarse Particles on Deaths in Hong Kong Elderly Population: an Analysis of Mortality Displacement

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Abbreviations:

DLM, distributed lag model; GAM, generalized additive model; ICD-10, international statistical classification of diseases, 10th revision; IQR, inter-quartile range; PM_{10}, particulate matter with an aerodynamic diameter less than 10 μm; $PM_{2.5}$, fine particles with an aerodynamic diameter less than 2.5 μm; PMc, coarse particles with an aerodynamic diameter between 2.5 and 10 μm.

Abstract

Background: While numerous studies worldwide have evaluated the short-term associations of fine and coarse particulate matter (PM) air pollution with mortality and morbidity, these studies may be susceptible to short-term harvesting effect. We aimed to investigate the short-term association between mortality and PM with aerodynamic diameter less than 2.5 μm ($PM_{2.5}$) and those between 2.5 and 10 μm (PMc) within a month prior to death, and assess the mortality displacement by $PM_{2.5}$ and PMc among elderly population in Hong Kong.

Methods: We obtained air pollution data from January 2011 to December 2015 from Environmental Protection Department, and daily cause-specific mortality data from Census and Statistical Department of Hong Kong. We performed generalized additive distributed lag model to examine the acute, delayed and long-lasting effects of $PM_{2.5}$ and PMc within one month on mortality.

Results: We observed a statistically significant association of $PM_{2.5}$ and PMc exposure over lags 0-6 days with all natural mortality and cardio-respiratory mortality. The overall cumulative effect of $PM_{2.5}$ over 0-30 lag days was 3.44% (95% CI: 0.30-6.67%) increase in all natural mortality and 6.90% (95% CI: 0.58-13.61%) increase of circulatory mortality, which suggested the absence of mortality displacement by $PM_{2.5}$. On the other hand, no significant cumulative association with mortality was found for PMc over 0-30 lag exposure window, and thus mortality displacement by PMc cannot be ruled out. Findings remained robust in various sensitivity analyses.

Conclusions: We found adverse effect of both $PM_{2.5}$ and PMc exposure within one week prior to death. While there was no evidence of mortality displacement in the association of $PM_{2.5}$ exposure over one month prior with all natural and circulatory mortality, mortality displacement by PMc cannot be ruled out. $PM_{2.5}$ may contribute more to the longer term effect of particulate matter than PMc.

Key Words: Coarse Particulate Matter; Fine Particulate Matter; Generalized Additive Distributed Lag Model; Mortality Displacement; Time Series Study

Capsule: This time series study with analysis of mortality displacement demonstrated the acute effects of both fine and coarse PM on mortality within one week and no evidence of mortality displacement by $PM_{2.5}$ within one month prior to death.

Introduction

The associations of fine and coarse particulate matter (PM) air pollution with mortality and morbidity have been widely discussed (Adar et al., 2014; Brunekreef and Forsberg, 2005), with consistent evidence of an acute health effect of fine particles with aerodynamic diameter ≤ 2.5 µm ($PM_{2.5}$) (Englert, 2004), and supporting evidence of an effect of coarse particles with aerodynamic diameter between 2.5-10 µm (PMc), especially with respiratory diseases (Adar et al., 2014; Qiu et al., 2012). While majority of the epidemiological studies on the health effects of $PM_{2.5}$ and PMc focused on short exposure period, i.e., from the day of disease onset/death to previous few days (Meister et al., 2012; Qiu et al., 2012; Samoli et al., 2013; Zanobetti and Schwartz, 2009), these studies may be susceptible to potential short-term harvesting effect (Schwartz, 2000a). Harvesting effect, also known as mortality displacement, of air pollution-related deaths is a phenomenon where air pollution principally affects frail population by advancing their deaths by a number of days or weeks because of their already poor health conditions, and the initial increase in mortality rate is then followed by a period with a lower-than-expected mortality rate (Schwartz, 2001; Zeger et al., 1999). The presence of harvesting effect could limit the public health significance of air pollution.

A combination of *generalized additive model* and *distributed lag model* has been suggested to model the relationship between lagged exposure of multiple days (e.g., lag 0 to 30 days), and subsequently to quantify the mortality displacement or harvesting effect effectively in air pollution epidemiological studies (Schwartz, 2000a; Zanobetti et al., 2000). However, there remains limited research on the harvesting effect and distributed lag effects of PM. In 2000, Schwartz examined various distributed lag of $PM_{2.5}$ on mortality using seasonal-trend decomposition algorithm, and found evidence of harvesting effects on different time scales (Schwartz, 2000b). Costa et al. showed evidence of mortality displacement within 30 days for nonaccidental and circulatory deaths using distributed lag model in elderly residents of São Paulo (Costa et al., 2017). To our knowledge, no studies have assessed the harvesting effect and distributed lag effects of $PM_{2.5}$ and PMc in the same setting.

Hong Kong is an ideal place to study the health effects of short-term exposure to $PM_{2.5}$ and PMc, because of its readily available measures of hourly PM_{10} and $PM_{2.5}$ concentrations monitored simultaneously in every monitoring station dispersed across the territory since January 2011. Our previous studies have demonstrated acute effects of both $PM_{2.5}$ and PMc within one week on cardio-pulmonary diseases in Hong Kong (Qiu et al., 2014, 2013, 2012). The current study built upon this observation by quantifying mortality displacement and examining the short-term association between cause-specific mortality and $PM_{2.5}$ and PMc exposure in a month prior to death, using the package 'dlnm' developed within the statistical environment R (Gasparrini et al., 2010). Given that the aging population in Hong Kong is increasing [from 12% in 2006 to 16% in 2016 (http://www.censtatd.gov.hk)], and because they are most vulnerable to air pollution, we studied the mortality displacement in Hong Kong elderly population aged 65 or above.

Materials and Methods

Data collection

We obtained pairwise hourly measures of $PM_{2.5}$ and PM_{10} concentrations collected between January 1, 2011 to December 31, 2015 at 14 general air quality monitoring stations maintained by the Hong Kong Environmental Protection Department (EPD) (HKEPD, 2016). Missing data accounted for only 3.1% and 10.7% of PM_{10} and $PM_{2.5}$ measurements, respectively, and thus data were not imputed. Hourly PMc concentrations were calculated by subtracting $PM_{2.5}$ from PM_{10} measurements for each station. We then computed daily 24-hr mean concentrations of $PM_{2.5}$ and PMc if at least 18 of 24 hourly measurements were available for each monitor. Air pollution measurements from one general station on a remote island and three roadside stations were excluded, and the final analysis included daily measurements of $PM_{2.5}$ and PMc from 10 general monitoring stations that represent the citywide background air pollution level and general population's daily exposure (Qiu et al., 2014). Meteorological data of daily mean temperature and relative humidity were collected from Hong Kong Observatory for the same study period.

Mortality data among elderly Hong Kong residents aged 65 or above between 2011 and 2015 were obtained from Hong Kong Census and Statistical Department. The causes of death were identified according to the WHO *International Statistical Classification of Diseases, 10th Revision* (ICD-10). Daily counts of mortality from all natural causes (ICD-10: A00-R99), circulatory diseases (ICD-10: I00-I99) and respiratory diseases (ICD-10: J00-J99) were computed and subsequently linked to air pollution and meteorological data. Ethics approval and consent from individual subjects were not required, as no individualized data but aggregated data were used in this study.

Statistical modelling

We used time series Poisson model to examine the association between PM in different size fractions and daily mortality. Generalized additive regression model (GAM) integrated with distributed lag model (DLM) were used to investigate the potential mortality displacement in the association (Zanobetti et al., 2000). We used smoothing spline function, *s(.)*, to filter out long-term trend and seasonality in time series of daily mortality as well as daily mean temperature and relative humidity (Peng et al., 2006; Schwartz et al., 1996). Based on previous studies we followed a priori model specifications and the degree of freedom (*df*) for the time trend and other time-varying covariates, in order to reduce the problems coming from multiple testing and model selection strategies (Peng et al., 2006; Qiu et al., 2012). Time trend with a *df* of 7 per year, temperature of the current day ($Temp_0$) and the mean of previous 3 days ($Temp_{1-3}$) with a *df* of 6, and relative humidity of the current day ($Humid_0$) with a *df* of 3 were used.The day of the week (*DOW*) and public holidays (*Holiday*) were included as dummy variables to the model (Qiu et al., 2012).

Briefly, a core model was set up to remove the long-term trend, seasonality, with adjustment for time varying covariates as follows:

$$log[E(Y)] = a + s(t, df=7/year \times 5years) + s(Temp_0, df=6) + s(Temp_{1-3}, df=6) + s(Humid_0, df=3) + \beta_{1,i} DOW + \beta_2 Holiday \quad (1)$$

Here *E[Y)* is the expected daily counts of mortality on day t; *s(.)* is the smoothing spline function for nonlinear covariates. We observed no discernible patterns and autocorrelation in the model residuals assessed by residual plot and partial autocorrelation function (PACF) for all three mortality outcomes (Costa et al., 2017). The standardized deviance residuals shown in the Q-Q plot were also normal (Figure 1), which suggested that all unmeasured time-varying confounding in the daily variations of mortality series had been controlled for. Once the model was correctly specified, the terms of $PM_{2.5}$ or PMc were included into the model to estimate their single-pollutant association with daily cause-specific mortality.

Distributed lag model (DLM), which was integrated in the GAM as *cross-basis* function to account for the potential distributed and lagged effect of pollution on mortality flexibly, was used to estimate the short-term health effects associated with $PM_{2.5}$ or PMc exposure in the 30-days prior to mortality (Gasparrini et al., 2010). In the primary analysis, second-degree (quadratic) polynomials was used to constrain the smooth shape of the effects of distributed lags ≤ 30 days for daily mortality (Costa et al., 2017). We calculated the cumulative effect of $PM_{2.5}$ or PMc distributed over 0-30 lag days to estimate the overall effect lasting for one month, as well as the cumulative effects of $PM_{2.5}$ or PMc over the different lag periods: 0-6 days, 7-13 days, and 14-30 days, representing the acute, delayed and long-lasting effects, respectively (Qiu et al., 2016). Single-lag effects of $PM_{2.5}$ and PMc over 30 days exposure on daily mortality were shown, as well as the cumulative effects of $PM_{2.5}$ and PMc for the investigation of mortality displacement (Costa et al., 2017; Gasparrini, 2011). As described in detail by Schwartz and Zanobetti, the cumulative effects of PM on daily mortality should reduce to zero, and the confidence intervals for the cumulative risks should include zero in the presence of mortality displacement (Schwartz, 2000a; Zanobetti et al., 2000).

We conducted sensitivity analysis by repeating the regression analysis using an unconstrained DLM, which has been shown to give unbiased estimate of the cumulative effect even though it is more noisy than their constrained counterpart to provide information about the shape of the associations along the lags (Zanobetti et al., 2003, 2002). We also constructed a cubic polynomial DLM to test the robustness of our effect estimates against different degrees of polynomial. Moreover, exposure windows of 0-20 and 0-40 lag days were examined, respectively, to compare with lag period over 0-30 days in the main analysis. Two-pollutant model with $PM_{2.5}$ and PMc over 0-30 lag days included simultaneously in the model was constructed to assess the independent effect of each pollutant on mortality.

The effect estimates were presented as the percentage changes of daily deaths for an interquartile range (IQR) increase of $PM_{2.5}$ or PMc, and their corresponding 95% confidence intervals (CI). All analyses were conducted in the statistical environment R 3.3.3 while loading 'mgcv' and 'dlnm' packages (R Development Core Team, 2017: http://www.r-project.org).

Results

Between January 1, 2011 and December 31, 2015, a total of 168,541 all natural deaths in Hong Kong elderly population (age > 65 years) were reported; among which 42,264 deaths were attributed to circulatory diseases and 44,810 deaths from respiratory diseases. On average, there were 92, 23 and 24 deaths from all natural causes, circulatory diseases and respiratory diseases, respectively, per day. The daily 24-hour mean concentrations were 29.1 $\mu g/m^3$ for $PM_{2.5}$ and 14.6 $\mu g/m^3$ for PMc, while the corresponding IQRs were 24.9 and 10.0 $\mu g/m^3$, respectively. Pearson correlation coefficient between $PM_{2.5}$ and PMc was 0.71. The daily mean temperature was 23.5^{o}C and the relative humidity was 78.3% (Table 1).

Table 2 shows the cumulative effects of $PM_{2.5}$ and PMc on daily mortality over the different lag periods in single-pollutant models. We observed a statistically significant association of $PM_{2.5}$ exposure over lags 0-6 days with all natural mortality (3.23%, 95% CI: 1.85-4.63%), circulatory mortality (4.81%, 95% CI: 2.06-7.63%), as well as respiratory mortality (3.74%, 95% CI: 0.97-6.57%). The associations between $PM_{2.5}$ and for all natural and circulatory mortality persisted for $PM_{2.5}$ exposure over 7-13 days prior to death, though the magnitude of the effects attenuated. No significant associations with respiratory mortality were observed for $PM_{2.5}$ exposure after lag 6 days, and no associations with all natural and circulatory mortality after lag 14 days (i.e., lag 14-30 days). The overall cumulative effect of $PM_{2.5}$ over 0-30 lag days was 3.44% (95% CI: 0.30-6.67%) increase in all natural mortality, and 6.90% (95% CI: 0.58-13.61%) increase in circulatory mortality. Since the effect estimates were not zero and the confidence intervals didn't include zero, no mortality displacement for all natural and circulatory deaths by $PM_{2.5}$ was observed over 0-30 lag exposure window.

Similar to $PM_{2.5}$, increment in PMc exposure over 0-6 lag days was also significantly associated with increase of 2.90% (95% CI: 1.68-4.14%), 2.77% (0.34-5.25%) and 4.19% (1.76-6.69%) for all natural deaths, circulatory deaths and respiratory deaths, respectively. However, the effects of PMc attenuated and rendered insignificant for exposure over 7-13 days prior to death, suggesting a less lasting effect of PMc on mortality than that of $PM_{2.5}$. Since the confidence intervals of the cumulative effects of PMc over 0-30 lag days included zero, mortality displacement by PMc within one month cannot be ruled out (Table 2). Figures 2 and 3 showing single-lag effects and cumulative effects of $PM_{2.5}$ and PMc, respectively, also provided consistent findings of acute effects of both $PM_{2.5}$ and PMc on mortality within one week, and the absence of harvesting effects in the association between $PM_{2.5}$ and all natural mortality and circulatory mortality over 30-days lag window.

Findings from sensitivity analyses using unconstrained DLM or cubic polynomial DLM, or longer exposure windows were similar to those in the primary analysis (Table 3). Two-pollutant models with $PM_{2.5}$ and PMc adjusted simultaneously showed that the association of $PM_{2.5}$ with mortality persisted whereas that of PMc rendered insignificant upon controlling for $PM_{2.5}$ (Table 3).

Discussion

This is the first study that investigated the distributed lag effects of $PM_{2.5}$ and PMc exposure in 30-days prior to death on cause-specific mortality, and assessed whether mortality displacement existed in the associations. We observed both $PM_{2.5}$ and PMc to be associated with significant increase in all natural, cardio-respiratory mortality in lag 0-6 days, which are consistent with findings from existing literature of an adverse association between short-term exposure of $PM_{2.5}$ and PMc and mortality (Lee et al., 2015; López-Villarrubia et al., 2012; Malig and Ostro, 2009; Meister et al., 2012; Perez et al., 2009; Yorifuji et al., 2016; Zanobetti and Schwartz, 2009). However, only $PM_{2.5}$ was found to be significantly linked to all natural and circulatory mortality in lag 0-30 days, suggesting the absence of mortality displacement by $PM_{2.5}$. Yet, mortality displacement by $PM_{2.5}$ in the $PM_{2.5}$-respiratory association or by PMc cannot be ruled out.

Common plausible mechanisms for the association between PM and cardio-respiratory health include the oxidative stress and inflammation pathways. Fine particles could trigger redox-sensitive pathways via catalytic generation of reactive oxygen species, leading to inflammation and cell death (Lodovici and Bigagli, 2011). Studies with healthy volunteers demonstrated that coarse concentrated PM exposure may induce pulmonary inflammation, decrease blood tissue plasminogen activator (Graff et al., 2009), influence systemic biomarkers (Graff et al., 2009; Liu et al., 2015), as well as trigger the hemodynamic alternations through elevating systolic blood pressure and heart rate (Morishita et al., 2015). High level of PMc was associated with reduction in heart rate variability among older subjects (Graff et al., 2009; Lipsett et al., 2006). An animal study also suggested that the chemical compositions of $PM_{2.5}$ and PMc may be responsible for inflammation and lung tissue damage (Happo et al., 2010).

Harvesting effect or mortality displacement principally exists when the mortality rate of susceptible population decreases following an increase in air pollution-associated mortality rate, resulting in an inverse effect of air pollution in longer exposure lags (Schwartz, 2000a; Zanobetti et al., 2000). We extended our exposure window to 0-30 lag days, and observed the statistically significant cumulative effects of $PM_{2.5}$ on all natural and circulatory mortality, which suggests the absence of mortality displacement over this window. Meanwhile, we cannot rule out possible harvesting effect of $PM_{2.5}$ on respiratory mortality, as well as harvesting effect of PMc on all natural and cardiorespiratory deaths within one month. A limited number of studies have investigated potential short-term mortality displacement in the association of air pollution and mortality, but these studies only assessed the harvesting for PM_{10} (Costa et al., 2017; Goodman et al., 2004; Schwartz, 2001; Zanobetti et al., 2003, 2002), $PM_{2.5}$ or black smoke (Goodman et al., 2004; Schwartz, 2000b). Our findings are similar to those from Schwartz, 2000, and further contributed to the existing literature by showing potential evidence of mortality displacement by PMc. While direct comparison is not possible, our results may be different from those in a recent study conducted in Brazil, in which Costa et al. provided evidence of mortality displacement for PM_{10} within 30 days for all natural and circulatory deaths but not for respiratory and cancer deaths (Costa et al., 2017). The different population characteristics, weather conditions and ambient pollution profiles, together with different disease spectrum in San Paulo and Hong Kong may contribute to the diverse results. Differential harvesting effects of fine and coarse particles examined in other places of the world are required to better understand sources of heterogeneity.

Findings from our study also inferred that $PM_{2.5}$ may contribute more in the longer-term effect of PM_{10} exposure than the PMc. Consistent with our study, the Nurses' Health Study also reported evidence of a stronger chronic $PM_{2.5}$ effect on risk of all-cause and cardiovascular mortality than that of PMc (Puett et al., 2009). Furthermore, two-pollutant models adjusting for $PM_{2.5}$ and PMc simultaneously also supported less independent effect of coarse fraction. Compared with PMc, $PM_{2.5}$ has higher number concentration, greater surface area, and better lung deposition (Dockery, 2009). $PM_{2.5}$ has a greater probability of penetrating deeply into the small airways and the alveoli of the lung (Dominici et al., 2006), whereas PMc is more likely to affect the upper and larger airways (Host et al., 2008). Since particles in the alveolar region are removed at a slower rate than those in the conducting airways, this may explain the greater toxic effect of $PM_{2.5}$ (Englert, 2004).

Our study had several strengths. We are the first study to assess mortality displacement by $PM_{2.5}$ and PMc in the same setting, as previous research focused only on PM_{10} or black smoke probably due to data scarcity. Hong Kong is an ideal Asian city for studying the health effect of air pollution, with its greater variability in air pollution than that in most of western countries. Also, the availability of high quality of hourly PM_{10} and $PM_{2.5}$ measurements from a descent network of air monitoring stations across the entire Hong Kong territory provided more representative assessment for population exposure than those used data monitored from only one fixed-site station (Kan et al., 2007) or every sixth day sampling window (Peng et al., 2008).

Some limitations of the study should be considered. Firstly, we calculated PMc concentrations by subtracting $PM_{2.5}$ from PM_{10} measurements, thus the PMc estimation may be susceptible to double measurement errors. Pollutant with large measurement error may lose statistical significance in co-pollutant model, which may explain the null findings of PMc upon adjusting for $PM_{2.5}$. Secondly, exposure misclassification cannot be eliminated in our time series study design. While ambient $PM_{2.5}$ concentrations measured from local monitoring stations might be adequate surrogate for the total personal exposures (Schwartz et al., 2007), the representativeness of ambient PMc concentrations is less certain. Moreover, we did not estimate season-specific effects as stratification could lead to more missing data in the 30-day PM exposure assessment, thereby increasing the variability and bias of the effect estimates. However, seasonal analysis using a shorter exposure lag (i.e., lag0-6 days) for $PM_{2.5}$ and PMc showed stronger association with mortality for the cold season as compared to those in the warm season (data not shown); this finding is generally consistent with previous literature (Meister et al., 2012; Zanobetti and Schwartz, 2009). Finally, residual confounding remains possible as we did not adjust for potential confounding from gaseous pollutants in consideration of collinearity, unstable estimates, and to avoid the problems of model misconvergence.

Conclusions

In conclusion, we found adverse effect of both $PM_{2.5}$ and PMc exposure within one week prior to death. While there was no evidence of mortality displacement in the association between all natural and circulatory mortality and $PM_{2.5}$ exposure over one month prior to death, mortality displacement by PMc cannot be ruled out. $PM_{2.5}$ may contribute more to the longer term effect of PM air pollution and impose stronger public health impact than PMc.

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Table-1. Descriptive Statistics of Daily Mortality Counts in the Elders, Air Pollution Concentrations and Weather Conditions in Hong Kong, 2011-2015 (1826 days)

	Mean	SD	Min	25th	50th	75th	Max
Daily Mortality Counts							
All Natural Deaths	92.3	15.8	48.0	81.2	90.0	101.1	150.0
Circulatory Deaths	23.1	6.5	8.0	19.0	22.0	27.0	52.0
Respiratory Deaths	24.5	7.1	8.0	19.0	24.0	29.0	53.0
Pollution concentration							
PM_{10} (μg/m^3)	43.7	23.8	7.6	24.0	38.9	57.8	157.4
$PM_{2.5}$ (μg/m^3)	29.1	17.3	4.9	14.6	25.9	39.5	115.7
PMc (μg/m^3)	14.6	8.2	2.4	8.7	12.6	18.7	108.9
Weather conditions							
Temperature (^{o}C)	23.5	5.3	8.4	19.0	24.8	28.2	32.4
Relative humidity (%)	78.3	10.3	29.0	74.0	79.0	85.0	99.0

Abbreviations: SD-standard deviation; Min-minimum; Max-maximum; PM_{10}, particulate matter with aerodynamic diameter less than 10 μm; $PM_{2.5}$, particulate matter with aerodynamic diameter less than 2.5 μm; PMc, particulate matter with aerodynamic diameter between 2.5 and 10 μm; NO_2, nitrogen dioxide; SO_2, sulfur dioxide; O_3, ozone.

Table-2 Cumulative effects (% change with 95% CIs) in Daily Mortality in the Elderly Associated with an IQR Increase of PM over Different Lag Periods in Hong Kong, 2011-2015*

	All Natural Death	Circulatory Death	Respiratory Death
$PM_{2.5}$			
Lags 0-6	3.23 (1.85, 4.63)	4.81 (2.06, 7.63)	3.74 (0.97, 6.57)
Lags 7-13	0.87 (0.04, 1.70)	1.79 (0.15, 3.46)	1.04 (-0.61, 2.71)
Lags 14-30	-0.67 (-2.65, 1.36)	0.19 (-3.71, 4.26)	0.04 (-3.94, 4.18)
Lags 0-30	3.44 (0.30, 6.67)	6.90 (0.58, 13.61)	4.85 (-1.44, 11.54)
PMc			
Lags 0-6	2.90 (1.68, 4.14)	2.77 (0.34, 5.25)	4.19 (1.76, 6.69)
Lags 7-13	0.44 (-0.27, 1.15)	1.10 (-0.30, 2.52)	-0.18 (-1.57, 1.23)
Lags 14-30	-1.84 (-3.54, -0.11)	0.14 (-3.25, 3.66)	-2.32 (-5.64, 1.12)
Lags 0-30	1.45 (-1.30, 4.29)	4.05 (-1.48, 9.89)	1.59 (-3.81, 7.30)

*: Interquartile ranges (IQRs) for $PM_{2.5}$, and PMc are 24.9, and 10.0 μg/m^3, respectively. Effects are estimated from Poisson generalized additive distributed lag model, constrained with a second-degree (quadratic) polynomial, while adjusting for time trend and seasonality, weather conditionals, day of week and public holidays. The highest 0.5% and the lowest 0.5% extreme values of PMc have been excluded from the analysis. Statistically significant effect estimates are in bold.

Table-3 Sensitivity analyses showing the cumulative effects (% change with 95% CIs) in Daily Mortality in the Elderly per IQR Increase of PM in Hong Kong, 2011-2015*

	All Natural Death	Circulatory Death	Respiratory Death
Cubic polynomial DLM over 0-30 lag days			
$PM_{2.5}$	3.43 (0.30, 6.67)	6.88 (0.56, 13.60)	4.85 (-1.44, 11.54)
PMc	1.42 (-1.34, 4.26)	3.97 (-1.56, 9.81)	1.62 (-3.78, 7.33)
Unconstrained DLM over 0-30 lag days			
$PM_{2.5}$	3.31 (0.11, 6.62)	6.44 (-0.02, 13.31)	4.37 (-2.06, 11.23)
PMc	1.20 (-1.65, 4.13)	2.73 (-2.92, 8.71)	1.73 (-3.88, 7.67)
Quadratic polynomial DLM over 0-20 lag days			
$PM_{2.5}$	3.14 (0.72, 5.62)	6.52 (1.65, 11.62)	4.01 (-0.83, 9.09)
PMc	1.62 (-0.5, 3.78)	3.63 (-0.58, 8.03)	0.19 (-3.91, 4.46)
Quadratic polynomial DLM over 0-40 lag days			
$PM_{2.5}$	2.61 (-1.70, 7.12)	8.93 (0.05, 18.61)	-1.26 (-9.45, 7.66)
PMc	2.29 (-1.46, 6.19)	7.08 (-0.57, 15.33)	1.91 (-5.43, 9.83)
Quadratic polynomial DLM over 0-30 lag days in two-pollutant model			
$PM_{2.5}$	5.60 (0.69, 10.75)	8.09 (-1.59, 18.72)	9.36 (-0.52, 20.23)
PMc	-2.29 (-6.39, 1.98)	-1.30 (-9.26, 7.35)	-4.59 (-12.29, 3.80)

*: Interquartile ranges (IQRs) for $PM_{2.5}$ and PMc are 24.9 and 10.0 $\mu g/m^3$, respectively. Effects are estimated from Poisson generalized additive distributed lag model, while adjusting for time trend and seasonality, weather conditionals, day of week and public holidays. The highest 0.5% and the lowest 0.5% extreme values of PMc have been excluded from the analysis. Statistically significant effect estimates are in bold.

Figure legends:

Figure-1 Diagnostic plots of the residuals of the core model for A: All Natural Deaths; B: Circulatory Deaths; C: Respiratory Deaths

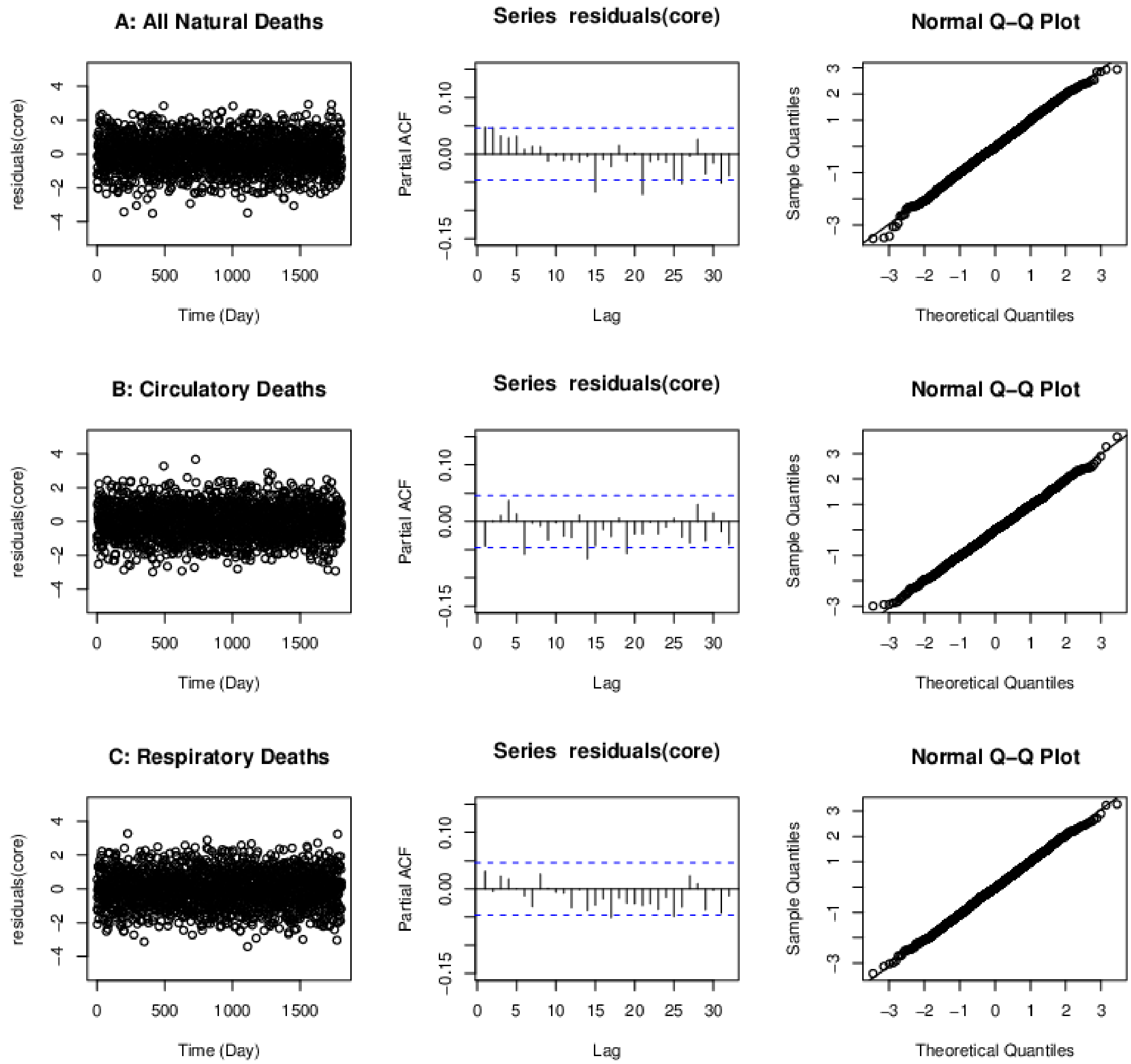

Figure-2 Single-lag effects of $PM_{2.5}$ and PMc per IQR increase over 0-30 lag days on daily mortality of Hong Kong Elderly population, 2011-2015

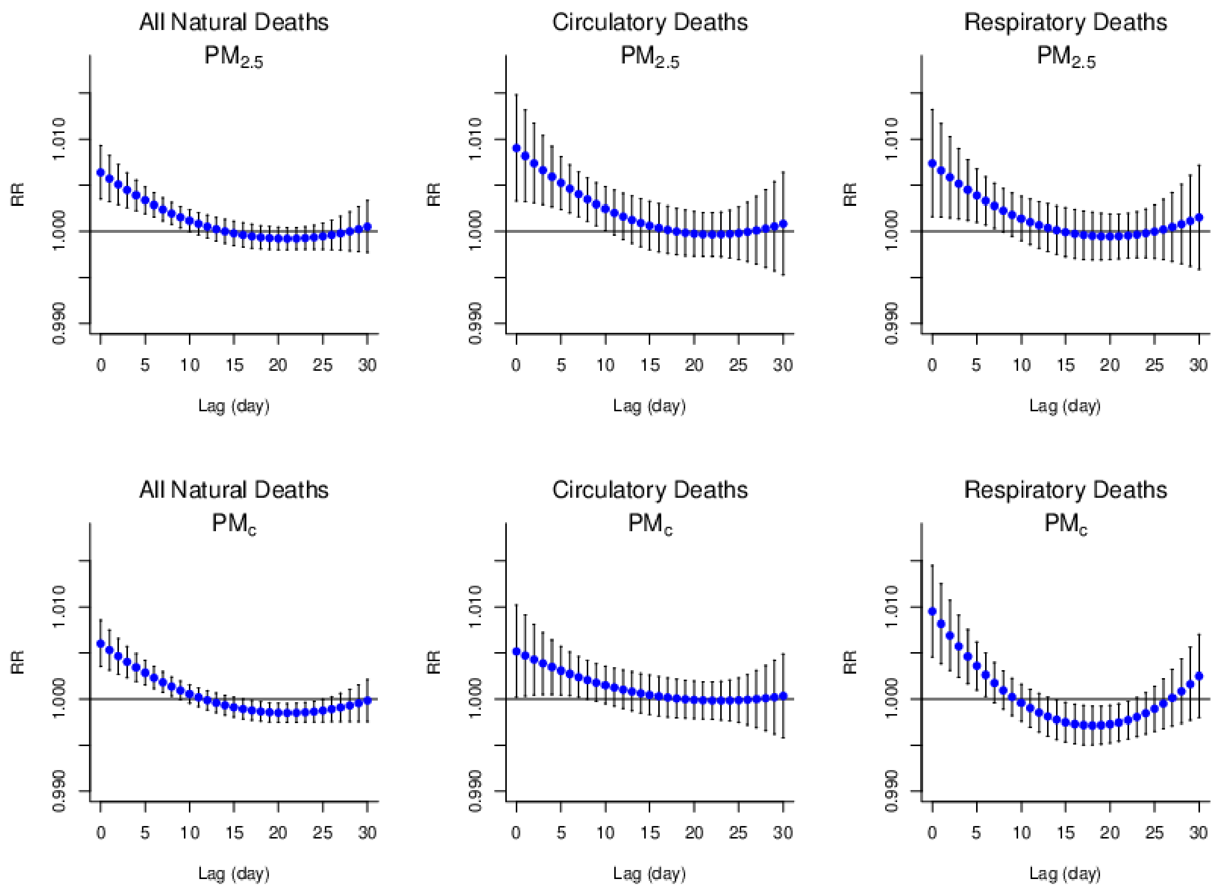

Figure-3 Cumulative effects of $PM_{2.5}$ and PMc per IQR increase over 0-30 lag days on daily mortality of Hong Kong Elderly population, 2011-2015