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Respirable Particulate Constituents and Risk of Cause-Specific Mortality in the Hong Kong Population

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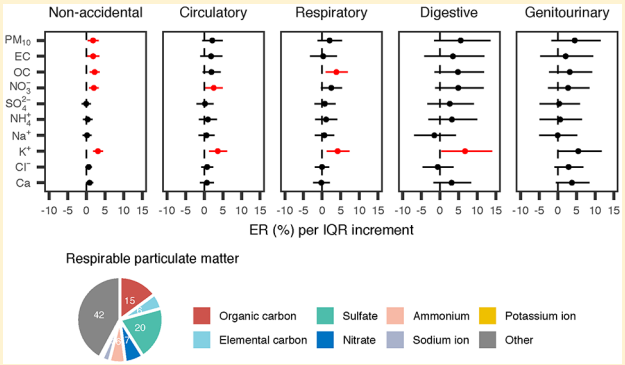
Supporting Information

ABSTRACT: Emerging studies examined the associations of particulate matter constituents with nonaccidental and cardiorespiratory diseases, but few have investigated more specific causes of cardiorespiratory diseases or other system diseases, especially in Asia. We estimated the association between respirable particulate matter (PM_{10}) constituents and a spectrum of deaths using a quasi-Poisson time-series model in Hong Kong. Positive associations were identified between cause-specific deaths and elemental carbon, organic carbon (OC), nitrate, and potassium ion (K^+), but only the associations for OC and K^+ were robust in the two-constituent models adjusting for other constituents. The estimated effects of OC were strongest on mortality from the respiratory system with cumulative percent excess risk (ER%) of 3.82% (95% CI: 0.96%, 6.92%) per interquartile range (6.7 $\mu g/m^3$) increase over 7 days prior to death (lag_{0-7}), especially for pneumonia (ER%: 4.32%; 95% CI: 0.70%, 8.26%). The digestive system was most sensitive to K^+ with cumulative ER% of 6.74% (95% CI: 0.37%, 14.01%) per interquartile range (0.6 $\mu g/m^3$) increase. This study indicates that PM_{10} constituents from biomass burning (OC and K^+) were more toxic than other constituents for deaths in Hong Kong, especially for mortalities from respiratory and digestive systems. These findings should have potential biological and pollution control implications.



INTRODUCTION

It is well documented that particulate matter (PM) is associated with increased risks of adverse health outcomes.[1−4] However, there is a large heterogeneity among risk estimates[5] suggesting that the most harmful aspect of PM to health may not be best quantified by total mass concentration of particles. PM is a complex mixture of constituents (e.g., elemental carbon or sulfate), and these constituents vary spatially and temporally depending on the local source of origin and interactions with local climate and other factors.[6] Thus, more PM constituents studies under various atmospheres are needed to improve our understanding of the adverse health effects of PM.

Information on which PM constituent is most harmful and which disease is more sensitive to certain constituent are crucial to inform local policy making and air pollution control in a more specific and effective way. However, most prior studies that examined the health effects of PM constituents typically only investigated diseases of all causes collectively or all cardiorespiratory diseases. Evidence on health effects of PM constituents on more refined categories of cause-specific cardiorespiratory causes (e.g., heart failure or pneumonia)[7−9] or diseases of other systems (e.g., diseases of the digestive system) is limited. In addition, although a few studies examined the adverse health effects of PM constituents, risk estimates obtained from studies with different population characteristics are not comparable as population characteristics, such as age and sex, could modify the PM-mortality associations.[10,11] To the best of our knowledge, no study has been conducted using the same study population to estimate associations between PM constituents and a spectrum of deaths, which makes identification of more toxic constituents and susceptible diseases difficult.

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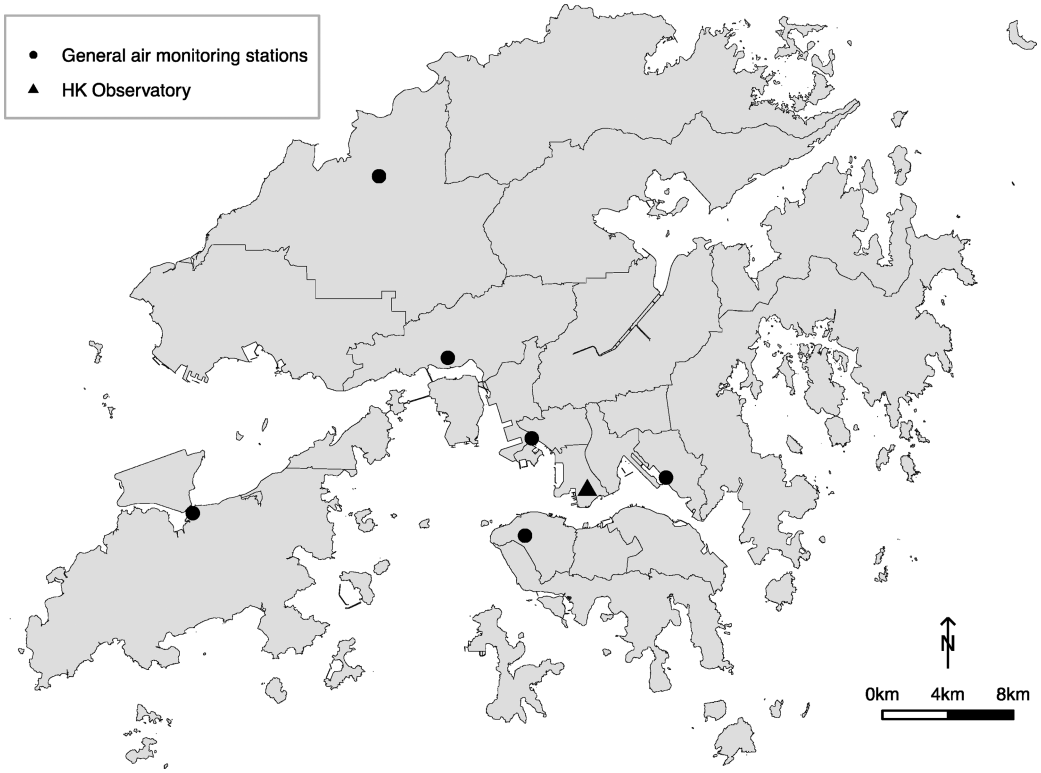

Figure 1. Spatial distribution of air monitoring stations (n = 6) and weather station (n = 1) in Hong Kong, 2001−2010.

Table 1. Summary Statistics for Selected Cause-Specific Mortality in Hong Kong, 2001−2010[a]

ICD-10 codes	disease	total count of study period	daily count			
			mean (SD)	25th	50th	75th
A00−R99	nonaccidental	361 648	99 (16)	88	98	109
I00−I99	diseases of the circulatory system	100 228	27 (7)	23	27	32
I10−I15	hypertensive heart diseases	7827	2 (2)	1	2	3
I20−I25	ischemic heart disease	39 970	11 (4)	8	11	13
I21−I23	myocardial infarction	18 679	5 (2)	3	5	7
I50	heart failure	7097	2 (2)	1	2	3
I60−I69	cerebrovascular diseases	33 980	9 (3)	7	9	11
J00−J99	diseases of the respiratory system	69 703	19 (6)	15	19	23
J12−J18	pneumonia	43 788	12 (5)	8	12	15
J41−J44	chronic obstructive pulmonary disease	18 155	5 (2)	3	5	6
K00−K93	diseases of the digestive system	14 205	4 (2)	2	4	5
N00−N99	diseases of the genitourinary system	18 045	5 (2)	3	5	6

[a]Abbreviations: SD = standard deviation; 25th = 25th percentile; 50th=50th percentile; 75th=75th percentile.

Hong Kong is one of the world's most populous large cities located in the Pearl River Delta area, one of the most polluted areas in China, with air pollution concentrations often exceeding the recommended Air Quality guideline.[12] In addition, Hong Kong has one of the world's longest life expectancy with patterns of chronic diseases different from Western populations.[13] These unique natures of Hong Kong enable us to examine the health effects of PM_{10} constituents on a spectrum of deaths. In the present study, we sought to examine the associations of PM_{10} constituents with a broad spectrum of cause-specific mortality (nonaccidental, systems of cardiovascular, respiratory, digestive, and genitourinary, and cause-specific cardiorespiratory diseases) using 10-year daily time-series data between 2001 and 2010 in Hong Kong.

MATERIALS AND METHODS

PM_{10} Constituents and Meteorological Data. We obtained PM_{10} constituents between 2001 and 2010 from Hong Kong Environmental Protection Department, which collected 24-h PM_{10} filter samples from six general air monitoring stations. PM_{10} samples were first analyzed for mass by gravimetric analysis, and elements of PM_{10} were then determined using inductively coupled plasma atomic emission spectroscopy, elemental carbon/organic carbon using a thermal/optical transmittance method, and ions using ion chromatography.[14] We excluded constituents that individually contributed <1%,[6,15] had been contaminated, had >25% of missing values, or below the analytical detection limit,[16,17] which yielded a total of nine constituents. These constituents included elemental carbon (EC), organic carbon (OC), nitrate (NO_3^-), sulfate (SO_4^{2-}), ammonium ion (NH_4^+), chloride ion

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Table 2. Summary Statistics of Daily Concentrations of Respirable Particulate and Its Constituents and Meteorological Conditions in Hong Kong, 2001−2010[a]

variable	mean (SD)	% of PM_{10} mass	percentile			IQR
			25th	50th	75th	
concentration, $\mu g/m^3$						
PM_{10}	55.0 (30.2)	[b]	30.8	50.1	72.9	42.1
SO_4^{2-}	11.2 (6.9)	20.38	6.0	10.3	14.9	8.8
OC	8.2 (5.2)	14.88	4.3	7.0	10.7	6.4
EC	3.2 (1.5)	5.78	2.1	3.0	4.0	2.0
NO_3^-	3.8 (3.2)	6.91	1.6	2.8	5.1	3.5
NH_4^+	3.3 (2.6)	6.03	1.3	2.9	4.7	3.4
Na^+	1.6 (0.9)	2.83	0.9	1.4	2.0	1.1
Cl^-	0.9 (1.0)	1.57	0.3	0.6	1.1	0.8
Ca	0.7 (0.6)	1.29	0.3	0.6	0.9	0.6
K^+	0.6 (0.5)	1.03	0.2	0.4	0.8	0.6
meteorological conditions						
temperature, °C	23.5 (5.0)	[b]	19.5	24.7	27.7	8.2
relative humidity, %	78.2 (10.2)	[b]	73.4	79.3	85.2	11.8

[a]Abbreviations: IQR = interquartile range; EC = elemental carbon; OC = organic carbon; NO_3^-= nitrate; SO_4^{2-}= sulfate; NH_4^+ = ammonium; Na^+ = sodium ion; K^+ = potassium ion; and Cl^- = chloride ion. [b]Not available.

(Cl^-), sodium ion (Na^+), potassium ion (K^+), and calcium (Ca). The measured constituents were a good proxy for population exposure (Figure 1) and have been used in prior health studies.[16−20] We also obtained daily mean ambient temperature and relative humidity from the Hong Kong Observatory during the same study period.

Mortality Data. We collected daily mortality data from the Hong Kong Census and Statistics Department. Cause-specific mortality was coded according to the International Classification of Diseases, 10th revision (ICD-10): nonaccidental (A00-R99), diseases of the circulatory system [I00−I99; hypertensive heart diseases (I10−I15), ischemic heart disease (I20−I25), myocardial infarction (I21−I23), heart failure (I50), cerebrovascular diseases (I60−I69)], diseases of the respiratory system [J00−J99; pneumonia (J12−J18), chronic obstructive pulmonary disease (J41−J44)], diseases of the digestive system [K00−K93], and diseases of the genitourinary system [N00−N99] (Table 1). Daily cause-specific mortalities were also aggregated by age (<65 versus ≥65 years).

Statistical Analysis. PM_{10} sampling occurred every-sixth-day on average for each monitoring station. The six monitoring stations have distinct sampling schedule, so there may be zero or multiple stations measured PM_{10} constituents for a particular day. Collectively, ∼70% of the study days had at least on station measured constituents. We created a time-series of PM_{10} constituents per day by applying a centring method to remove the station-specific influence on the measurements of each constituent. The centring approach has been previously described.[19,21] Given the risk estimates of PM_{10} constituents being insensitive to missing data or imputation methods to fill those missing values in previous studies of Hong Kong, we imputed missing values on territory-wide mean concentrations of PM_{10} constituents (1123 days) by linear interpolation using the *na.approx* function in the R*zoo* package to obtain the final complete time-series.[17,18] The linear interpolation assumes the estimated missing value lying on the line joining the nearest nonmissing measurement values to the left and right. The time-series plots of the final complete PM_{10} constituents are shown in Figure S1 of the Supporting Information (SI).

Generalized additive quasi-Poisson regression combined with distributed lag model was used to estimate percent excess risks (ER%) of cause-specific deaths associated with an interquartile range (IQR) increase in each PM_{10} constituent. We extended the lag up to 7 days prior to death and modeled the lag function using a priori third degree polynomials.[18] We used natural cubic splines with 8 degrees of freedom per year to filter out the long-term trend and seasonality. To control for temperature effects, we simultaneously included the same day mean ambient temperature and the moving average of previous 1 to 3 days using natural cubic splines with 3 degrees of freedom each in the models.[22] We also controlled for day of the week, public holidays, influenza epidemics, and current day relative humidity using natural cubic splines with 3 degrees of freedom.

We first constructed single-constituent models to estimate the association of cause-specific mortality with each PM_{10} constituent through including the constituent of interest in the models. For constituents demonstrating statistically significant associations over lag_{0-7} days in the single-constituent models, we then used two-constituent models. In the two-constituent model, we adjusted for the other constituents (co-constituent) one at a time when the Pearson correlation between the constituent of interest and the co-constituent was <0.60 to avoid collinearity.[7,20,23] We considered the associations were robust if they were still statistically significant in the two-constituent models.

Sensitivity Analysis. To confirm whether the results were sensitive to imputation values, we reanalysed the time-series data using an alternative imputation method of replacing missing values with nonmissing measurement values from the previous day. To test the robustness of our findings, we varied degrees of freedom of the natural cubic spline of time per year, controlled for PM_{10} mass, and further adjusted for gaseous air pollutants (NO_2 and O_3).

To control for potential type I error rate due to multiple comparisons, we applied the Bonferroni method correcting for the five broad causes of mortality, and a *P*-value <0.01 (0.05/5) was considered as statistically significant.[24] All analyses were conducted in R statistical software (version 3.5.2) with the "mgcv" (version 1.8−26) and "zoo" (version 1.8−4) packages.

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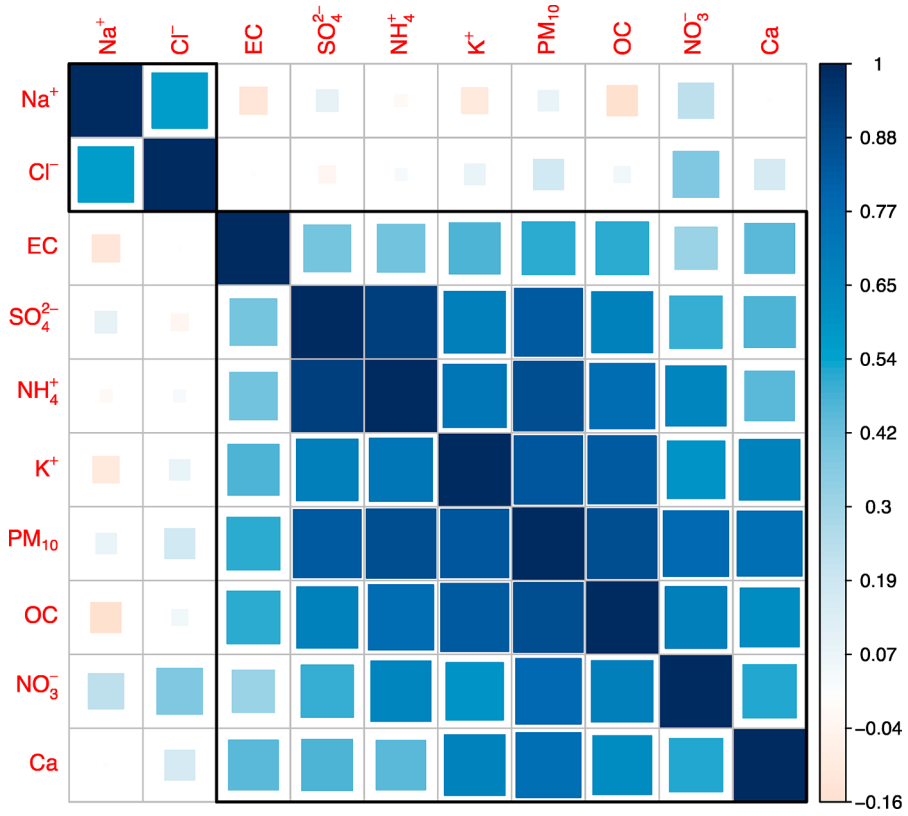

Figure 2. Pearson correlation between respirable particulate constituents in Hong Kong, 2001−2010. Size of the square is proportional to the correlation coefficients.

RESULTS

There were 361 648 nonaccidental deaths occurred during the 10-year study period, of which 100 228 (28%) were died from the circulatory system, 69 703 (19%) from respiratory system, 14 205 (4%) from digestive system, and 18 045 (5%) from the genitourinary system (Table 1). Ischemic heart disease (40%) and cerebrovascular disease (34%) respectively accounted for about one-third of the circulatory deaths. Pneumonia alone accounted for approximately two-thirds of the respiratory deaths (63%).

Table 2 shows the summary statistics of PM_{10} constituents and weather conditions in Hong Kong. Between 2001 and 2010, the daily mean concentration of PM_{10} mass was 55.0 $\mu g/m^3$ (SD: 30.2 $\mu g/m^3$). SO_4^{2-} accounted for the largest fraction of total PM_{10} mass (20.38%), followed by OC (14.88%), NO_3^- (6.91%), NH_4^+ (6.03%), EC (5.78%), Na^+ (2.83%), Cl^- (1.57%), Ca (1.29%), and K^+ (1.03%). These nine constituents in sum consist of ∼61% of the total PM_{10} mass. The daily mean ambient temperature was 23.5 °C and relative humidity was 78.2%.

The Pearson correlations among constituents were generally low to moderate ($r < 0.6$) (Figure 2), except for the correlations of PM_{10} mass with OC, NO_3^-, SO_4^{2-}, NH_4^+, K^+, or Ca, and the correlations between OC and NO_3^-, SO_4^{2-}, NH_4^+, K^+, or Ca, which were >0.6.

The associations between PM_{10} mass and five broad causes of deaths by lag days were generally showed similar pattern with larger risk estimates on later days (2 to 6 days preceding death events) in the single-pollutant models (Figure S2). An IQR (42.1 $\mu g/m^3$) increase in PM_{10} mass over lag_{0-7} days was associated with a 1.63% (95% CI: 0.73%, 2.56%) and 2.08% (95% CI: 0.39%, 3.77%) increase in risk of nonaccidental and diseases of the circulatory system, respectively (Figure 3). OC and K^+ were the two constituents that consistently associated with higher mortality risks of nonaccidental and diseases of the respiratory system. We also found some evidence of significantly positive associations between NO_3^- and mortality of nonaccidental, diseases of the circulatory system, and ischemic heart disease, the association between EC and nonaccidental, and the association between K^+ and diseases of the digestive system. The variance inflation factor for each PM_{10} constituent in the regression model was all <2 indicating low multicollinearity (data not shown). We also found the associations between PM_{10} constituents and cause-specific mortality were more pronounced among elders than younger population (Figures S3 and S4).

In the two-constituent models of adjusting co-constituent for those significant positive associations in the single-constituent models, most associations lost statistical significance, although central effect estimates were still positive (Table 3). Generally, OC and K^+ were the two constituents that were robust to co-constituent adjustment. The associations for OC remained statistically significant for mortalities from nonaccidental (range of central estimates, 2.08%−2.25%), diseases of respiratory system (range of central estimates, 3.82%−5.47%), and pneumonia (range of central estimates, 4.32%−5.99%). The largest effects of OC were found for diseases of respiratory system, especially for pneumonia with an ER% of 4.32% (95% CI: 0.70%, 8.26%) per IQR (6.4 $\mu g/m^3$) increase. The associations for K^+ were robust to co-constituent adjustment for mortalities from nonaccidental (range of central estimates, 2.57%−3.12%), diseases of the circulatory system (range of central estimates,

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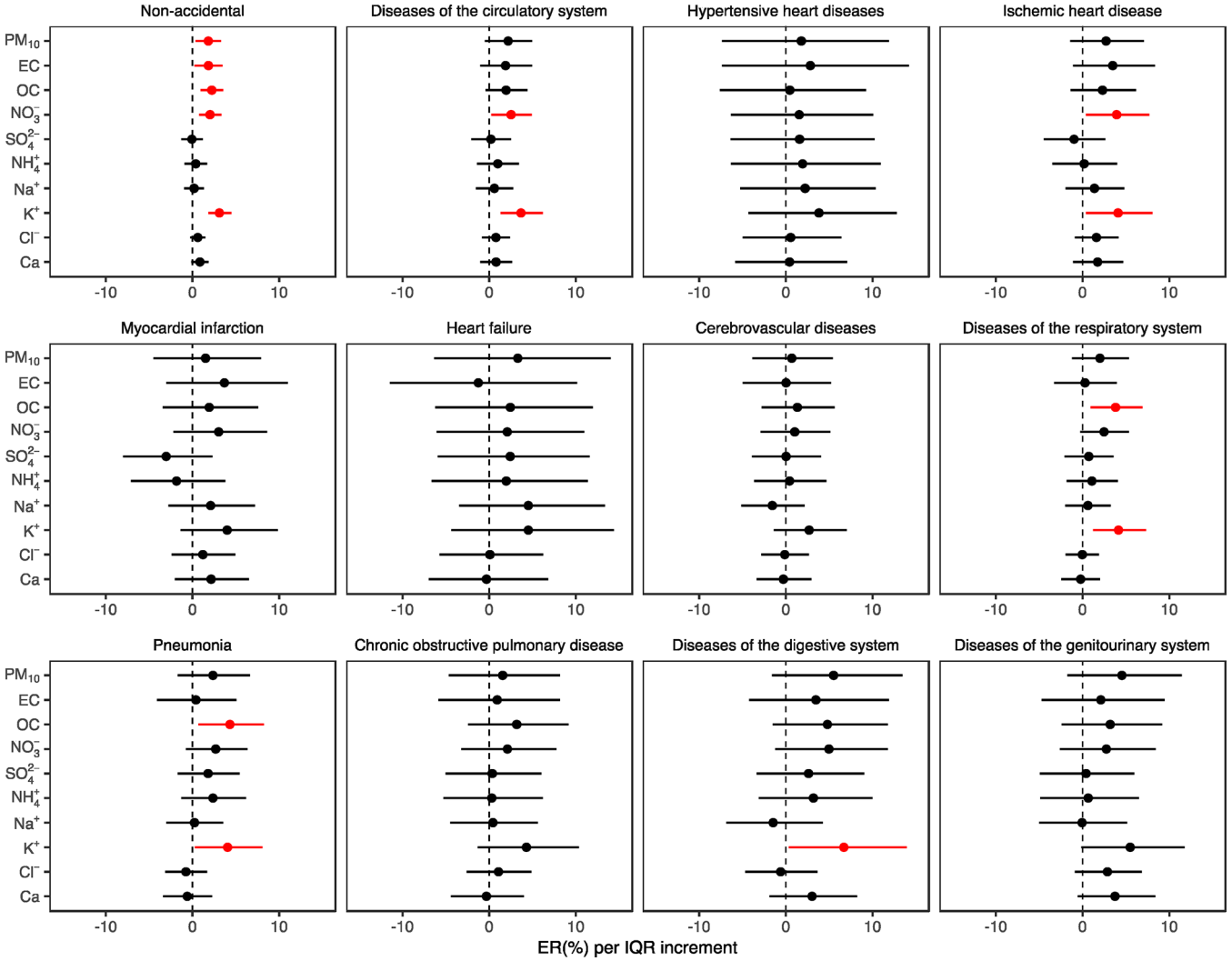

Figure 3. Cumulative excess risk (%) of causes-specific mortality associated with an interquartile range increase in respirable particulate constituents over lag_{0-7} days in Hong Kong, 2001−2010.

Table 3. Cumulative Excess Risk (%) of Cause-Specific Mortality Associated with an Interquartile Range Increase in Constituents over lag_{0-7} Days with Significant Associations in Single-Constituent Models after Adjustment for Co-constituent[a,b,c]

constituents	causes	ER% of single-pollutant [% (95% CI)]	robust to adjustment by	not robust to adjustment by	range of point estimates (%)
EC	nonaccidental	1.82 (0.24, 3.46)	NH_4^+, Cl^-, Na^+, SO_4^{2-}	Ca, NO_3^-, OC, K^+	−0.06 to 2.24
OC	nonaccidental	2.22 (0.95, 3.56)	Cl^-, EC, Na^+		2.08 to 2.25
NO_3^-	nonaccidental	2.01 (0.77, 3.30)	Ca, Cl^-, EC, Na^+, SO_4^{2-}	K^+	0.52 to 2.57
K^+	nonaccidental	3.09 (1.82, 4.48)	Cl^-, EC, NO_3^-, Na^+		2.57 to 3.12
NO_3^-	diseases of the circulatory system	2.54 (0.27, 4.92)	Ca, Cl^-, Na^+, SO_4^{2-}	EC, K^+	0.78 to 3.09
K^+	diseases of the circulatory system	3.62 (1.27, 6.17)	Cl^-, EC, NO_3^-, Na^+		2.97 to 3.75
NO_3^-	ischemic heart disease	3.97 (0.46, 7.76)	SO_4^{2-}, Na^+	Ca, Cl^-, EC, K^+	2.50 to 5.59
K^+	ischemic heart disease	4.05 (0.37, 8.04)	Cl^-, Na^+	EC, NO_3^-	2.40 to 4.20
OC	diseases of the respiratory system	3.82 (0.96, 6.92)	Cl^-, EC, Na^+		3.82 to 5.47
K^+	diseases of the respiratory system	4.17 (1.25, 7.36)	Cl^-, EC, Na^+, NO_3^-		3.63 to 5.27
OC	pneumonia	4.32 (0.70, 8.26)	Cl^-, EC, Na^+		4.32 to 5.99
K^+	pneumonia	4.03 (0.28, 8.08)	Cl^-, EC, Na^+	NO_3^-	3.21 to 5.00
K^+	diseases of the digestive system	6.74 (0.37, 14.01)	Cl^-	EC, Na^+, NO_3^-	5.57 to 7.11

[a]Abbreviations: PM_{10} = respirable particulate matter; ER = excess risk; EC = elemental carbon; OC = organic carbon; NO_3^-= nitrate; K^+ = potassium ion; Cl^- = chloride ion. [b]The selected co-constituents were whose with Pearson correlation coefficient <0.6 in relation to the constituent of interest. [c]Associations were considered robust to co-constituent adjustment if they remained statistically significant, otherwise the associations were not considered robust to co-constituent adjustment.

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2.97%−3.75%) and diseases of respiratory system (range of central estimates, 3.63%−5.27%). We found that the estimated effects of K^+ were strongest on mortality from diseases of the digestive system [ER%: 6.74% (95% CI: 0.37%, 14.01%) per IQR increase (0.6 $\mu g/m^3$) in K^+].

In the sensitivity analysis, we tested whether regression models were sensitive to imputation values by analyzing the time-series data without any imputation or replaced missing values with nonmissing measurement values from the previous day. Although a few associations gained or lost statistical significance compared to the main analysis, our results were generally not sensitive to imputation values (Figures S5). Our findings were also not materially different after varying degrees of freedom of the natural cubic spline of time per year (Figures S6 and S7), adjusting more days of temperature, further controlling for PM_{10} mass or gaseous pollutants (data not shown).

DISCUSSION

To our knowledge, this is the first study to examine associations between PM constituents and a spectrum of mortality in Asian countries. We found PM_{10} mass, EC, OC, NO_3^-, and K^+ were linked with risks of certain cause-specific deaths. OC and K^+ were consistently associated with increased risks of mortalities from nonaccidental and respiratory systems, and the associations were robust to co-constituent adjustment in the two-constituent models. We found mortalities from respiratory system were highly sensitive to OC, and mortalities from digestive system were more susceptible to K^+. These findings should have potential biological and pollution control implication.

Our findings of positive associations between PM_{10} mass, EC, OC, NO_3^-, and K^+ and certain cause-specific deaths were generally consistent with most prior studies.[25,26] Our study is the first to examine PM constituents and mortalities from diseases of the digestive and genitourinary systems, and we found K^+ was statistically associated with increased risk of diseases of the digestive system, and marginally associated with diseases of the genitourinary system. This finding was supported by a few epidemiological studies which found PM was associated with risks of gastrointestinal diseases, chronic kidney disease, and reduced estimated glomerular filtration rate.[27−32] The underlying biological mechanisms to explain the association between PM and digestive and genitourinary diseases remain largely unknown. The increased gut permeability, promotion of the inflammatory response, and alteration of the microbial composition may play a role in the association between PM and digestive diseases.[33,34] Also PM may increase diastolic blood pressure, induce oxidative stress and inflammation, and disturb metabolism (e.g., decreased insulin sensitivity), which can ultimately lead to the development of genitourinary diseases.[35,36]

We found K^+ was consistently associated with increased risk of a spectrum of deaths. These findings were consistent with two recent systematic review and meta-analyses concluding that K^+ was consistently associated with higher risks of nonaccidental and/or cardiovascular deaths.[25,26] These positive associations were mostly observed in areas or regions where concentration of K^+ was relatively high, such as Beijing, China (mean concentration of K^+: 1.08 $\mu g/m^3$),[37] Xi' an, China (1.8 $\mu g/m^3$),[9] South Korea (0.4 $\mu g/m^3$),[38] and five South-European cities (concentrations ranging from 0.18 to 0.41 $\mu g/m^3$),[39] but not in the United States (<0.1 $\mu g/m^3$).[6,7]

K^+ is commonly regarded as a reasonable emission tracer for biomass burning.[14,40] Emerging studies have found that biomass burning for cooking and heating purposes was significantly associated with increased risk of mortality.[41,42] The World Health Organization reported that household air pollution (e.g., wood, crop wastes, coal and dung, charcoal) was linked with deaths from stroke, ischemic heart diseases, chronic obstructive pulmonary disease, and lung cancer, and it claimed about 4 million lives each year.[43] Biomass burning was not common in the developed countries,[41] thus the concentration and contribution of K^+ to the PM mass are usually low.[6] For example, Bell et al. (2007) collected 52 $PM_{2.5}$ component from 187 US counties between 2000 and 2005, and found that K^+ accounted for ~0.5% of $PM_{2.5}$ total mass with mean concentration of 0.07 $\mu g/m^3$ for the yearly average across all 187 counties.[6] In our study, the contribution of K^+ accounted for about 1% of PM_{10} mass with mean concentration of 0.6 $\mu g/m^3$ in Hong Kong, which is much higher than the concentration of K^+ to PM mass in the United States. The positive matrix factorization source apportionment in Hong Kong indicated that sources of coal combustion and biomass burning were nonlocal, which were from Pearl River Delta areas, one of the most polluted areas in China, and sources mainly came from domestic biofuel burning, field burning of rice straw, power plants, and industrial combustion.[14,44]

EC is mainly from vehicle exhaust and OC is a marker for both vehicle exhaust and coal combustion and biomass burning.[14] Most prior studies reported that EC and OC were associated with increased risks of cause-specific deaths.[25,26] For example, one recent meta-analysis reported that EC was associated with higher mortality risk of nonaccidental, cardiovascular, and respiratory diseases, but OC was only associated with nonaccidental and respiratory mortality.[25] Although we found EC was linked with nonaccidental and ischemic heart disease, these associations lost statistically significant when adjusted for co-constituent. We found OC was significantly associated with increased risk of mortalities from nonaccidental, diseases of the respiratory system, pneumonia, and diseases of the digestive system.

Although NO_3^- was significantly associated with mortalities from nonaccidental, circulatory, and ischemic heart disease in the present study, these associations lost statistical significance when adjusted for co-constituents. Evidence on the NO_3^- mortality association is limited and inconsistent. For example, a positive association was found in Xi'an, China,[45] Beijing, China,[37] and six California counties, United States,[46] but not in five South-European cities,[39] and Seoul, Korea.[38] More studies are needed to further understand the health effects of NO_3^-.

One major contribution of this study is that we examined a spectrum of deaths using the same study population, results of this study should be comparable among cause-specific deaths and among constituents. We found OC and K^+ were the two more toxic PM constituents on mortality, and mortalities from respiratory system were highly sensitive to OC, and mortalities from digestive system were more susceptible to K^+ in Hong Kong. Our study adds to the literature that source of biomass burning was associated with increased risks of a spectrum of deaths, which has been rarely studied in Western countries due to the low concentration of K^+ in PM. These findings should have potential biological and pollution control implication.

Our study has some important limitations. First, we used outdoor monitoring stations to represent population exposure,

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which might introduce measurement error. However, this error is shared by other time-series studies and on average bias our results toward the null of no association. Second, constituents from local combustion sources were more likely to have higher spatial heterogeneity, and thus might be subject to more measurement error than those regionally transported.[47] The relative error of measurement associated with each PM constituent might influence their relative strengths of association. Similarly, diagnostic error may also contribute to the difference in the PM constituents-mortality associations. For example, through medical record reviews, a U.S. study found that diagnostic error most commonly occurred in diseases of pneumonia, congestive heart failure, and kidney failure.[48] Finally, besides the broad causes of deaths, we also examined more refined categories of cause-specific mortality. The small sample size for certain causes might have resulted in low statistical power to detect an association.

In summary, we found evidence that PM_{10} constituents from biomass burning (OC and K^+) may be more harmful to the Hong Kong population. Additional studies are needed to confirm or refute these findings. Our findings suggest that the Hong Kong government might need to cooperate with Mainland China to set stricter regulations on PM emissions, especially targeted at the source of biomass burning.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge on the ACS Publications website at DOI: 10.1021/acs.est.9b01635.

Time-series plots of PM_{10} constituents (Figure S1), excess risk (%) for PM_{10} mass by lag day (Figure S2), results of subgroup analysis by age: younger (<65 years) (Figure S3) and elder population (≥65 years) (Figure S4), and results of sensitivity analysis: with missing data replaced by measurement of previous day (Figure S5), using 7 degrees of freedom (Figure S6) or 9 degrees of freedom (Figure S7) per year to control for long-term trend and seasonality (PDF)

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Notes

The authors declare no competing financial interest.

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