

1 **Using daily excessive concentration hours to explore the short-term mortality effects of**  
2 **ambient PM<sub>2.5</sub> in Hong Kong**

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19 **ABSTRACT**

20 We developed a novel indicator, daily excessive concentration hours (DECH), to explore the  
21 acute mortality impacts of ambient particulate matter pollution (PM<sub>2.5</sub>) in Hong Kong. The  
22 DECH of PM<sub>2.5</sub> was calculated as daily concentration-hours >25 µg/m<sup>3</sup>. We applied a  
23 generalized additive models to quantify the association between DECH and mortality with  
24 adjustment for potential confounders. The results showed that the DECH was significantly  
25 associated with mortality. The excess mortality risk for an interquartile range (565  
26 µg/m<sup>3</sup>\*hours) increase in DECH of PM<sub>2.5</sub> was 1.65% (95% CI: 1.05%, 2.26%) for all natural  
27 mortality at lag 02 day, 2.01% (95% CI: 0.82%, 3.21%) for cardiovascular mortality at lag 03  
28 days, and 1.41% (95% CI: 0.34%, 2.49%) for respiratory mortality at lag 2 day. The  
29 associations remained consistent after adjustment for gaseous air pollutants (daily mean  
30 concentration of SO<sub>2</sub>, NO<sub>2</sub> and O<sub>3</sub>) and in alternative model specifications. When compared  
31 to the mortality burden of daily mean PM<sub>2.5</sub>, DECH was found to be a relatively conservative  
32 indicator. This study adds to the evidence by showing that daily excessive concentration  
33 hours of PM<sub>2.5</sub> might be new predictor of mortality in Hong Kong.

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35 **Keywords:** Excessive concentration hours; PM<sub>2.5</sub>; Mortality; mortality burden; Hong Kong

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37 **Capsule:** Excessive concentration hours of PM<sub>2.5</sub>, as one new indicator, is significantly  
38 associated with increased mortality in Hong Kong.

39

40 **1. Introduction**

41 Associated with China's rapid social and economic development, ambient air pollution  
42 has become the fourth most significant contributor to disability adjusted life years lost  
43 (DALYs) in China, behind dietary factors, high blood pressure and tobacco smoking (Yang et  
44 al., 2013). Serious air pollution in China, specifically in urban areas like Hong Kong, has  
45 increased interest in assessing the health impacts of air pollution (Chen et al., 2013).

46 The previous studies mainly employed daily mean concentration to represent the  
47 exposure (Pope et al., 2006; Schwartz et al., 2017). This approach ignores the enormous  
48 variations among different hours within one day (Moreno et al., 2009). A few studies have  
49 proposed other indicators, such as daily peak concentration (Delfino et al., 2002; Lin et al.,  
50 2016a; Madsen et al., 2012). It is believed that the peak concentration may play a more  
51 important role in overwhelming certain body defense mechanisms, and may better capture the  
52 adverse effects of outdoor air pollution exposures (Delfino et al., 2002). Both approaches  
53 have their own advantages, but neither approach can adequately represent the complex  
54 exposure patterns of ambient air pollution, particularly the different exposure intensities and  
55 durations among different days even with the same daily mean concentration.

56 In this study, we developed a novel indicator, daily excessive concentration hours  
57 (DECH), to quantify the acute mortality effects of daily PM<sub>2.5</sub> in Hong Kong, China.

58

59 **2. Methods**

60

61 *2.1 Air pollution*

62 The hourly monitored concentrations of air pollution were retrieved from Hong Kong  
63 Environmental Protection Department between January 1, 1998 and December 31, 2011 (Qiu  
64 et al., 2012). A total of 14 stations were operated to monitor the daily concentrations of  
65 particulate matter of 10 microns in diameter or smaller (PM<sub>10</sub>), nitrogen dioxide (NO<sub>2</sub>), sulfur  
66 dioxide (SO<sub>2</sub>), and ozone (O<sub>3</sub>). Hourly concentrations of PM<sub>2.5</sub> were only monitored in four  
67 stations (Central (CL), Tung Chung (TC), Tap Mun (TM), and Tsuen Wan (TW)) (Figure 1).  
68 The air pollution data from these four stations were used in this study. Hong Kong covers an  
69 area of 1100 km<sup>2</sup>, however, most of the population live in the central areas of Hong Kong,  
70 which are nearby the four air monitoring stations.

71 We developed a new indicator, DECH of PM<sub>2.5</sub>, which was calculated as the daily total  
72 concentration-hours >25 µg/m<sup>3</sup>. The concentration threshold of 25 µg/m<sup>3</sup> was chosen  
73 according to the guideline of the World Health Organization (WHO) (World Health  
74 Organization, 2006). When calculating DECH, any overages of the threshold contributed  
75 concentration-hours to the daily total while any concentrations below the threshold did not.  
76 For instance, one hour with an hourly concentration of 26.5 µg/m<sup>3</sup> would contributed 1.5  
77 concentration-hours to the daily total; while hours with the concentrations equal to or lower  
78 than 25 µg/m<sup>3</sup> did not contribute to the daily total.

79 The daily mean concentrations of ambient NO<sub>2</sub>, SO<sub>2</sub> and O<sub>3</sub> were also calculated using  
80 the data from these four stations. Daily mean temperature (°C) and relative humidity (%),  
81 were also retrieved from the Weather station of Hong Kong.

82

83 *2.2 Mortality*

84 Daily count of mortality was retrieved from the Hong Kong Census and Statistics  
85 Department over the period of 1998-2011. The causes of death were recorded in accordance  
86 with the International Classification of Diseases (ICD), the 9<sup>th</sup> revision was applied for  
87 1998-2000 and the 10<sup>th</sup> revision was for 2001-2011. We constructed the daily time series for  
88 mortalities for all diseases (ICD-9: 001-799 or ICD-10: A00-R99), cardiovascular diseases  
89 (ICD-9: 390-459 or ICD-10: I00-I99) and respiratory diseases (ICD-9: 460-519 or ICD-10:  
90 J00-J99). Daily count of hospitalizations for influenza was also utilized to represent the  
91 influenza outbreaks.

92

### 93 *2.3 Statistical analysis*

94 We employed a generalized additive model (GAM) with a quasi-Poisson to assess the  
95 association between DECH of PM<sub>2.5</sub> and mortality. A penalized spline function was used to  
96 adjust the seasonal pattern and long-term trend in daily mortality and the non-linear mortality  
97 effects of the weather. The influenza outbreaks was coded as a dummy variable based on  
98 whether the weeks with the hospitalization number more than the 75<sup>th</sup> percentile of a year  
99 (Qiu et al., 2012). We also coded both day of the week and public holidays as dummy  
100 variables in the model.

101 In line with previous air pollution time series studies, we conducted the model  
102 specification and selected the degrees of freedom (df) for smoothing functions (Peng et al.,  
103 2008). For example, we applied a df of 6 per year for temporal trends, a df of 6 for the mean  
104 temperature of the same day (Temp<sub>0</sub>) and average temperature of the previous three days  
105 (Temp<sub>1-3</sub>), and a df of 3 the relative humidity (Humidity<sub>0</sub>) of the same day.

106 First we examined the concentration-response relationships graphically using a  
107 smoothing function. The curves showed an approximately linear relationship, we thus  
108 estimated the linear effects with across different lag days. We also investigated the mortality  
109 effects using moving averages for the same day (lag<sub>0</sub>), previous one day (lag<sub>01</sub>), two days  
110 (lag<sub>02</sub>), and three days (lag<sub>03</sub>). We conducted both single-pollutant and two-pollutant models  
111 to examine the associations. In the single-pollutant model, DECH of PM<sub>2.5</sub> was included  
112 alone in the model; while in the two-pollutant models, the daily mean contraptions of other  
113 air pollutants were included separately in the model, such as SO<sub>2</sub>, NO<sub>2</sub> and O<sub>3</sub>. However,  
114 when two pollutants had a high correlation coefficient (>0.80), they were not be included in  
115 the same model simultaneously to avoid the possible multicollinearity (Li et al., 2016b).

116 To compare the mortality effect of the new indicator with that of daily mean  
117 concentration, we also examined the short-term mortality effects of daily mean concentration  
118 of PM<sub>2.5</sub> using the same method as described above.

119

#### 120 *2.4 Mortality burden attributable to daily mean PM<sub>2.5</sub> and DECH*

121 To examine the advantage of the new indicator, we compared the mortality burdens  
122 attributable to daily DECH of PM<sub>2.5</sub> and daily mean PM<sub>2.5</sub> (Li et al., 2016a). Population  
123 attributable fraction (PAF) and attributable mortality (AM) were applied (Zhu et al., 2013).

124 The formulas were shown below:

$$125 \text{ AM} = \text{baseline mortality} * [\exp(\beta * \Delta\text{PC})]$$

$$126 \text{ PAF} = \text{AM} / \text{overall mortality}$$

127 where baseline mortality refers to daily death count at days with reference PM<sub>2.5</sub>

128 concentration (or zero DECH);  $\Delta PC$  represents the differences in concentrations between the  
129 measured and reference concentrations of  $PM_{2.5}$  (or DECH higher than zero  $\mu g/m^3 \cdot hour$ );  
130 reference is zero  $\mu g/m^3 \cdot hour$  for DECH and  $25 \mu g/m^3$  for daily mean  $PM_{2.5}$ , and  $\beta$  represents  
131 a coefficient. The difference between the mortality burden of DECH and mean  $PM_{2.5}$  was  
132 tested by calculating the 95% confidence interval as:  $(b_1 - b_2) \pm 1.96 * \sqrt{(SE_1)^2 + (SE_2)^2}$ ,  
133 where  $b_1$  and  $b_2$  were the effect estimates for each variable, and  $SE_1$  and  $SE_2$  were the  
134 standard errors (Lin et al., 2016b).

135

### 136 2.5 Sensitivity analysis

137 The robustness of the key findings was assessed using different degrees of freedom in the  
138 smoothing functions. We used “mgcv” package in R to conduct the time series analysis.

139

## 140 3. Results

141 We obtained 496,042 deaths from all-natural causes in the study population. Among them,  
142 there were 138,067 cardiovascular disease deaths and 95,857 respiratory disease deaths. On  
143 average, there were 97 all-natural deaths per day, 27 cardiovascular deaths, and 19 respiratory  
144 death (Table 1).

145 There were 44 days without valid monitoring data at CL station, 456 days at TC station,  
146 90 days at TM station, and 190 days at TW station, corresponding to 0.9%, 8.9%, 1.8%, and  
147 3.7% of the overall study period, respectively. For all the four stations, there was only one  
148 day without valid  $PM_{2.5}$  concentrations. The average DECH of  $PM_{2.5}$  was  $370 \mu g/m^3 \cdot hours$ ,  
149 the  $PM_{2.5}$  daily mean was  $38 \mu g/m^3$ . The daily mean concentrations of  $NO_2$ ,  $SO_2$  and  $O_3$  were

150 56, 18, and 45  $\mu\text{g}/\text{m}^3$ , respectively. The daily mean relative humidity and temperature were  
151 78%. and were 24 °C.

152 According to the Pearson correlation (Table 2), there was a high correlation of DECH of  
153  $\text{PM}_{2.5}$  with daily mean  $\text{PM}_{2.5}$  concentration ( $r=0.99$ ), and moderate correlation with  $\text{NO}_2$   
154 ( $r=0.79$ ) and  $\text{SO}_2$  ( $r = 0.53$ ). Low to moderate correlations existed between other air  
155 pollutants and weather variables.

156 We observed an approximately linear dose-response relationship between DECH of  
157  $\text{PM}_{2.5}$  and mortality (Figure 2). Figure 3 shows the linear associations between DECH of  
158  $\text{PM}_{2.5}$  and mortality across different lag days in models without adjustment for other air  
159 pollutants. We found significantly positive associations between DECH of  $\text{PM}_{2.5}$  and  
160 all-cause mortality and cardiovascular mortality across all the lag days. For example, an  
161 interquartile range (IQR) ( $565 \mu\text{g}/\text{m}^3 \cdot \text{hours}$ ) increase in  $\text{lag}_{02}$  DECH of  $\text{PM}_{2.5}$  corresponded  
162 to a 1.65% (95% CI: 1.05%, 2.26%) increase in all-cause mortality; an IQR increase in  $\text{lag}_{03}$   
163 DECH of  $\text{PM}_{2.5}$  was associated with a 2.01% (95% CI: 0.82%, 3.21%) increase in  
164 cardiovascular mortality. While the association between DECH of  $\text{PM}_{2.5}$  and respiratory  
165 mortality was only found to be statistically significant at lag 2 day, the corresponding excess  
166 risk was 1.41% (95% CI: 0.34%, 2.49%). Table 3 reports the effect estimates from  
167 two-pollutant models and the results were comparable and statistically significant.

168 Figure s1 illustrated the short-term mortality effects of daily mean concentrations of  
169  $\text{PM}_{2.5}$ . The analysis found that daily mean  $\text{PM}_{2.5}$  was also significantly associated with  
170 all-cause mortality, cardiovascular and respiratory mortality. For example, each IQR (27  
171  $\mu\text{g}/\text{m}^3$ ) increase in daily mean  $\text{PM}_{2.5}$  at  $\text{lag}_{03}$  corresponded to 2.13% (95% CI: 1.45%, 2.82%)

172 increase in all-cause mortality, 2.77% (95% CI: 1.50%, 4.05%) increase in cardiovascular  
173 mortality, and 2.07% (95% CI: 0.49%, 3.67%) increase in respiratory mortality.

174 Table 4 illustrates the estimated AM and PAF associated with both DECH and daily  
175 mean concentration of PM<sub>2.5</sub>. We obtained a relatively greater mortality burden attributable to  
176 daily mean PM<sub>2.5</sub> than DECH of PM<sub>2.5</sub>, though the differences were not statistically  
177 significant. Based on daily mean concentration of PM<sub>2.5</sub>, we estimated that about 1.14% (95%  
178 CI: 0.77%, 1.51%) of all-cause mortality were attributable to higher daily PM<sub>2.5</sub>  
179 concentrations above 25 µg/m<sup>3</sup>, corresponding to 5635 (95% CI: 3821, 7466) attributable  
180 deaths; and about 1.04% (95% CI: 0.62%, 1.45%) of the mortality (5142, 95% CI: 3088,  
181 7216) could be attributed to daily DECH in PM<sub>2.5</sub> in the study population.

182 The additional analyses with varying DF for the smoothing functions produced consistent  
183 results (Table 3). All these sensitivity analyses indicated that the observed associations  
184 between DECH of PM<sub>2.5</sub> and all mortality categories were robust.

185

#### 186 **4. Discussion**

187 This study developed DECH as a new exposure indicator to measure the short-term  
188 mortality impacts of ambient PM<sub>2.5</sub>, which, to our knowledge, was the first time to do so.  
189 Using 14 years of data with about half million deaths, our analysis suggested that DECH may  
190 serve as an important health predictor of air pollutants.

191 We observed a high correlation between the DECH of PM<sub>2.5</sub> and PM<sub>2.5</sub> daily mean  
192 concentrations, and it was hard to exclude the possible confounding effects of daily mean  
193 concentration of PM<sub>2.5</sub>. Though some may argue that the observed effects of DECH of PM<sub>2.5</sub>

194 may possibly serve as one proxy of daily mean concentration of  $PM_{2.5}$ , our purpose was not  
195 to clarify that the mortality effects of daily excessive concentration hours of  $PM_{2.5}$  were  
196 independent of daily mean  $PM_{2.5}$ . Instead, the findings of this study may suggest that the  
197 previous reported adverse health impacts of daily mean concentration of  $PM_{2.5}$  might have  
198 been driven by DECH of  $PM_{2.5}$ . These hourly excessive concentrations should be considered  
199 in environmental policy-making to reduce the ambient  $PM_{2.5}$  concentrations and in  
200 epidemiological health impact analysis (Lin et al., 2016a).

201 Furthermore, besides providing similar information to the daily mean  $PM_{2.5}$ , it was likely  
202 that the DECH of  $PM_{2.5}$  represented additional independent exposure information, making it  
203 possible to serve as a new exposure indicator. In addition, the collinearity issue may exist  
204 when both  $PM_{2.5}$  mean and  $PM_{2.5}$  DECH were included in the same model. This could result  
205 in effect estimates in two directions, either larger or smaller, especially when several other  
206 important covariates were included in the same model.

207 We did an additional comparison of the mortality burden between these two variables,  
208 and observed a relatively conservative effect estimate of daily excessive concentration hours  
209 of  $PM_{2.5}$ . For example, about 1.04% and 1.14% of all-cause mortality was estimated to be  
210 attributable to DECH of  $PM_{2.5}$  and daily mean  $PM_{2.5}$ , respectively. This finding may suggest  
211 that, compared with daily DECH, daily mean  $PM_{2.5}$  might have over-estimated the mortality  
212 burden of  $PM_{2.5}$  in the study population.

213 The results of our analysis indicated that days with a greater frequency and severity of  
214 higher hourly air pollution level tended to lead to higher mortality risks than those with more  
215 moderate hourly air pollution distributions in Hong Kong. The results were in agreement with

216 previous studies that have used daily mean concentration to examine air pollution-mortality  
217 associations (Guo et al., 2013; Lin et al., 2016c; Qiu et al., 2013). For example, a study from  
218 Shanghai reported that PM<sub>2.5</sub> was significantly associated with increased risk of mortality,  
219 with a corresponding excess risk for mortality of 0.36% (95% CI: 0.11%, 0.61%) for each 10  
220 µg/m<sup>3</sup> increase in the daily mean concentration of PM<sub>2.5</sub> (Kan et al., 2007). One recent Hong  
221 Kong study demonstrated that an IQR increase in the daily mean concentration of PM<sub>2.5</sub>  
222 corresponded to a 1.86% (95% CI: 0.85%, 2.88%) increase in cardiovascular morbidity (Qiu  
223 et al., 2013). Our recent analysis found the excess risk of cardiovascular mortality was 6.11%  
224 (95% CI: 1.76%, 10.64%) for a 31.5 µg/m<sup>3</sup> increase in the daily mean concentration of PM<sub>2.5</sub>  
225 in Guangzhou, China (Lin et al., 2016c).

226 The strongest mortality effects of DECH of PM<sub>2.5</sub> were observed at lag 2 or 3 days'  
227 exposure and a 3-day moving average (lag<sub>02</sub>). Given that the time course of human body  
228 response and subsequent mortality risk can be on the order of hours to days for late-stage  
229 reactions (Bhaskaran et al., 2011; Yorifuji et al., 2014), this result corresponded with our  
230 expectations that the mortality was acutely related to airborne particulate pollution within 3  
231 days of exposures. We further observed slightly stronger associations with a three-day  
232 moving average than with single lag days, suggesting cumulative effects to some degree,  
233 which was consistent with previous studies (Pun et al., 2014).

234 A few biological mechanisms supported the observed acute mortality effects of DECH of  
235 PM<sub>2.5</sub>. For instance, it was possible that people may inhale higher dose of the fine particles on  
236 days with higher DECH of PM<sub>2.5</sub>, as during the hours with higher concentrations of PM<sub>2.5</sub>, as  
237 one may inhale air with more dense particles. Another possibility might be that the higher

238 PM<sub>2.5</sub> exposures may adversely affect the capacity of adapting to the extremely high pollution  
239 concentrations during the days with higher DECH of PM<sub>2.5</sub>. Such adaptation capacity may be  
240 adversely affected by existing cardiopulmonary conditions. One of our recent studies found  
241 that daily peak concentration of PM<sub>2.5</sub> was significantly associated with increased mortality  
242 risk among Chinese population (Lin et al., 2017), thus exposure to high concentrations above  
243 the threshold (25 µg/m<sup>3</sup>) may share similar pathways.

244 This study possessed a few advantages. Results of this study supported the necessity of  
245 considering relatively higher hourly concentrations of air pollution in both environmental  
246 management and air pollution epidemiology studies. Furthermore, harmful effects of air  
247 pollution have been observed below the standards/guidelines set by various countries and  
248 World Health Organization (WHO) (Chan et al., 2005; Moreno et al., 2009), taking the  
249 findings of our study into consideration, it was possible that the effects of high air pollution  
250 levels of some hours within one day might have been omitted. More specifically, though the  
251 daily mean concentration attained the standards in a given day, some hours may have higher  
252 concentrations than the standards, which might have been the underlying reasons for the  
253 adverse health effects.

254 A few limitations should also be considered. Being an ecological study, we could not  
255 establish the causal relationship. The exposure assessment based on the average of a few  
256 stations might have caused non-differential exposure misclassifications, leading to an  
257 under-estimation of the association. Furthermore, due to the limited observations with the  
258 PM<sub>2.5</sub> concentrations lower than 25 µg/m<sup>3</sup> in the study area, making it difficult to examine the  
259 health effects of the air pollution lower than the threshold and limit the ability to control for

260 them in the model.

261

## 262 **5. Conclusions**

263 In summary, this study adds to the evidence by showing that daily excessive

264 concentration hours of PM<sub>2.5</sub> may work as a new predictor of mortality.

265

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270

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346

347 **Table 1**

348 Basic characteristics of the daily mortality, weather variables and air pollution in Hong Kong

Variable	Observation days	Mean±SD	Percentile				
			Min	P <sub>25</sub>	P <sub>50</sub>	P <sub>75</sub>	Max
<b>Daily mortality</b>							
All natural	5113	97±17	51	85	95	108	180
CVD	5113	27±7	8	22	26	31	68
Respiratory	5113	19±6	4	14	18	22	58
<b>Air pollution (µg/m<sup>3</sup>)</b>							
PM <sub>2.5</sub> DECH	5112	370±418	1.0	25	235	590	3398
PM <sub>2.5</sub>	5112	38±20	5.8	22	34	49	170
SO <sub>2</sub>	5112	18±11	0.1	11	15	23	120
NO <sub>2</sub>	5112	56±21	7.7	40	54	68	150
Ozone	5112	45±24	1.1	26	42	61	140
<b>Weather factors</b>							
Temperature (°C)	5113	24±5.0	8.2	20	25	28	32
Relative humidity	5113	78±10	28	73	79	85	98

349 Abbreviation: SD, standard deviation; P<sub>x</sub>, xth percentile; Min, minimum; Max, maximum;

350 CVD, cardiovascular diseases; DECH, daily excessive concentration hours.

351

352 **Table 2**

353 Correlation coefficients between daily DECH of PM<sub>2.5</sub>, weather factors and air pollution in

354 Hong Kong

Pollutants	PM <sub>2.5</sub> DECH	PM <sub>2.5</sub> mean	SO <sub>2</sub>	NO <sub>2</sub>	O <sub>3</sub>	Temperature
PM <sub>2.5</sub> mean	0.99**					
SO <sub>2</sub>	0.53**	0.52**				
NO <sub>2</sub>	0.79**	0.78**	0.56**			
O <sub>3</sub>	0.55**	0.56**	-0.02	0.43		
Temperature	-0.46**	-0.46**	-0.13**	-0.50**	-0.19**	
Humidity	-0.44**	-0.45**	-0.34**	-0.40**	-0.45**	0.13**

355 Abbreviation: DECH, daily excessive concentration hours. \*\*p< 0.01, \* P< 0.05.

356

357 **Table 3**

358 ER in mortality for an IQR increase in daily excessive concentration hours (DECH) of PM<sub>2.5</sub>

359 of lag 02 day in different models.

Model	All natural mortality	CVD mortality	Respiratory mortality
Model-single*	1.65 (1.05, 2.26)	2.01 (0.82, 3.21)	1.41 (0.34, 2.49)
<b>Two-pollutant model</b>			
<del>With PM<sub>2.5</sub>-mean</del>	<del>1.48 (0.55, 2.42)</del>	<del>1.84 (0.19, 3.50)</del>	<del>2.13 (0.06, 4.25)</del>
With SO <sub>2</sub>	1.74 (1.11, 2.38)	2.09 (0.86, 3.33)	1.42 (0.35, 2.50)
With NO <sub>2</sub>	1.46 (0.78, 2.14)	1.81 (0.51, 3.13)	1.32 (0.24, 2.41)
With O <sub>3</sub>	1.62 (0.96, 2.27)	1.80 (0.53, 3.09)	1.59 (0.49, 2.71)
<b>Degree of freedom of temporal trend adjustment</b>			
df=5/year	1.51 (0.91, 2.11)	1.95 (0.75, 3.16)	1.59 (0.52, 2.68)

df=7/year	1.54 (0.93, 2.15)	2.24 (1.05, 3.45)	1.17 (0.09, 2.25)
df=8/year	1.41 (0.81, 2.02)	1.86 (0.66, 3.08)	1.18 (0.10, 2.27)

360 \* Results obtained from single-pollutant models.

361 Abbreviations: ER, excess risk; IQR, interquartile range; CVD, cardiovascular.

362

363 **Table 4**

364 The attributable fraction and attributable all natural mortality due to daily excessive  
365 concentration hours and daily mean PM<sub>2.5</sub> in Hong Kong during 1998-2011.

	PM <sub>2.5</sub> DECHs	Daily mean PM <sub>2.5</sub>	P value
ER * (%)	1.65 (1.05, 2.26)	2.13 (1.45, 2.82)	
Attributable fraction (%)	1.04 (0.62, 1.45)	1.14 (0.77, 1.51)	>0.05
Attributable mortality	5142 (3088, 7216)	5635 (3821, 7466)	>0.05

366 The reference PM<sub>2.5</sub> concentrations were the WHO's Ambient Air Quality guidelines (25  
367 µg/m<sup>3</sup>). \* ER is the excess risk of mortality for per IQR increase in daily excessive  
368 concentration hours of PM<sub>2.5</sub> (565 µg/m<sup>3</sup>\*hours) and in daily mean PM<sub>2.5</sub> (27 µg/m<sup>3</sup>).

369

370

371 **Figure legends:**

372

373 **Figure 1.** Geographical distribution of air pollution monitoring stations in Hong Kong.

374

375 **Figure 2.** Exposure-response curves for daily excess concentration hours (DECH) of PM<sub>2.5</sub>

376 and mortality in Hong Kong. A natural spline smoother with 3 df was applied.

377

378 **Figure 3.** Excess risk of mortality for per IQR increase in daily excess concentration hours

379 (DECH) of PM<sub>2.5</sub> (565 µg/m<sup>3</sup>\*hours) at different lag days in single-pollutant models.

380