Short-term effects of ambient $PM_{2.5}$ and $PM_{2.5\text{-}10}$ on mortality in major cities of Korea

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Abstract

While many epidemiological studies have examined the health effects of different size of ambient particulate matter (PM), the findings have been mixed. PM is a heterogeneous mixture and its chemical components differ by size with more combustion related materials in fine mode and more crustal materials in coarse mode. This study estimates the risk of mortality associated with $PM_{2.5}$ (particulate matter less than 2.5 μm in aerodynamic diameter) and $PM_{2.5\text{-}10}$ (particulate matter less than 10 μm and greater than 2.5 μm in aerodynamic diameter) exposure. Long-term measurements of $PM_{2.5}$ and $PM_{2.5\text{-}10}$ were compared with all-cause, cardiovascular, and respiratory mortality observed from January 2006 to December 2012 in three large cities in Korea (i.e. Seoul, Busan, and Incheon). A time-series analysis based on a quasi-Poisson distribution was used to evaluate the associations of $PM_{2.5}$ and $PM_{2.5\text{-}10}$ with mortality. A 10 μg m^{-3} increase in $PM_{2.5}$ (lag01) was associated with an increase of 1.18% (95% CI: 0.64, 1.72), 0.34% (95% CI: 0.03, 0.64), and 0.43% (90% CI: 0.02, 0.95) in all-cause mortality in Busan, Seoul, and Incheon, respectively, during the study period. An increase in respiratory mortality of 0.52% (95% CI: 0.09, 0.96) and 2.25% (95% CI: 0.38, 4.15) were associated with a 10 μg m^{-3} increase in $PM_{2.5}$

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(lag01) in Seoul and Busan, respectively. Overall, the strongest associations were observed in Busan as well as elderly population. Statistically significant associations between ambient $PM_{2.5}$ and $PM_{2.5\text{-}10}$ and mortality were observed in this study. Exposure to fine particles, which mostly originate from combustion and mobile emissions, showed stronger effects on human health than coarse particles, which mostly originate from natural sources such as soil and mechanical processes.

Keywords: Coarse particles; Fine particles; Health effects; Time-series analysis.

INTRODUCTION

Many epidemiological studies have identified the associations between ambient fine (less than 2.5 µm in aerodynamic diameter particulate matter: $PM_{2.5}$) or coarse (less than 10 µm and greater than 2.5 µm in aerodynamic diameter particulate matter: $PM_{2.5\text{-}10}$) particles and health (Katsouyanni *et al.*, 1997; Pope III and Dockery, 2006; Samoli *et al.*, 2013; Apte *et al.*, 2015). The findings show that exposure to ambient particles (PM) is significantly related to increased mortality and morbidity outcomes, but the results vary in different regions (depending on geological and meteorological factors, population structure, and cultural factors), as well as the sizes, chemical components, and sources of PM. For instance, Samoli *et al*. (2013) studied the associations between $PM_{2.5}$ or $PM_{2.5\text{-}10}$ and mortality in 10 European Mediterranean metropolitan areas and reported that these particles were significantly associated with all-cause, respiratory, and cardiovascular mortality. Significant health risks associated with PM exposure were also observed in communities in the United States and East Asian cities (Franklin *et al*., 2007; Lee *et al*., 2015). However, findings vary according to regions due to different composition and sources of PM, geological and meteorological factors, population structure, and cultural factors including lifestyle of the community in each of study areas (Lee *et al.*, 2000).

As interest in the health effects associated with PM exposure have increased, the United States Environmental Protection Agency (U.S. EPA) announced a revision of the ambient air quality standards of $PM_{2.5}$ concentration for 24-hour from 65 μg m^{-3} to 35 μg m^{-3}, and annually from 15 μg m^{-3} to 12 μg m^{-3} to offer increased protection against the negative health effects related to short-term and long-term $PM_{2.5}$ exposure (U.S. EPA, 2008). The air quality standard for $PM_{2.5}$ in Korea was established in 2015 and was set at 50 μg m^{-3} for 24-hour and 25 μg m^{-3} per year (Bae, 2014). While many health issues related with severe air pollution, mainly due to increases in the urban population, have been reported in Korea, the $PM_{2.5}$ standard in Korea is still weaker than the standards set by the United States and the World Health Organization (WHO).

According to a report by the WHO, approximately 3.7 million people including a considerable number of Asian people, died due to $PM_{2.5}$ exposure in 2012. Highly dangerous air pollution levels have been observed in Asian regions including Korea (Kan *et al.*, 2007; Huang *et al.*, 2012; Wang *et al.*, 2017). In particular, recent economic growth in China has affected air pollution levels in Korea (Chen *et al.*, 2013). Extreme PM events (i.e., yellow sand events, smog dust events, and the mixed smog and Asian dust events relevant to long-range transport of air pollutants from China) are also serious threats to ambient air conditions in Korea (Kim *et al.*, 2012; Kim *et al.*, 2015). Approximately 92% of Korea's population lives in urban areas, which was only 16% of the total area of Korea in the present. Local air pollution caused by rapid urbanization and long-range transported air pollutants are exacerbating adverse health outcomes in Korea.

Despite the awareness of the serious health threat of PM in urban areas, not enough research has studied the sizes, chemical constituents, and sources of PM and the associated health effects, using detailed measurement PM data in Korea. More research of the health effects of PM exposure is needed to develop effective air quality management system in Korea.

To address the research gaps in the health effects of PM in Korea, we examined the health risks of all-cause, cardiovascular, and respiratory mortality associated with short-term $PM_{2.5}$ and $PM_{2.5-10}$ exposure in three major cities in Korea (i.e. Seoul, Busan, and Incheon).

METHODS

Mortality and air pollutants

Three cities in Korea (i.e., Seoul, Busan, and Incheon) were selected for this study, as shown in Fig. 1. These cities were chosen because of the available mortality and air pollutant data, including $PM_{2.5}$ and $PM_{2.5-10}$, continuously observed from 2006 to 2012. Also, Seoul, Busan, and Incheon are the most populous cities in Korea. Table 1 shows the population sizes of major cities in Korea from 2000 to 2010 in five-year intervals.

Daily mortality data from January 1, 2006 to December 31, 2012 were obtained from the Korean Statistical Information Service (http://kosis.kr). A death was included only when it was for a resident of the three cities. The data were classified using the International Classification of Disease (ICD) as all-cause (non-accidental and specific diseases, ICD-10, A00-R99), respiratory (ICD-10, J00-J99), and cardiovascular (ICD-10, I00-I99) mortality. The data were also classified by age (all ages and greater than 65 years of age).

Our study used data from 35, 19, and 15 national air quality monitoring sites in Seoul, Busan, and Incheon, respectively. The hourly measured air pollutants (i.e., PM_{10}, $PM_{2.5}$, CO, SO_2, NO_2, and O_3) from January 1, 2006 to December 31, 2012 were acquired from the national air quality monitoring sites operated by the Research Institute of Public Health and Environment of Seoul (http://health.seoul.go.kr), Busan (http://www.busan.go.kr/ihe), and Incheon (http://air.incheon.go.kr/airinch/inch.html). The hourly measurements of PM_{10}, $PM_{2.5}$, CO, SO_2, and NO_2 at the multiple monitoring sites of each city were averaged for each day. Eight-hour averages (10:00 AM - 18:00 PM) of O_3 across the monitoring sites of each city for each day were used and the $PM_{2.5-10}$ was calculated as the difference between the daily average PM_{10} and $PM_{2.5}$ levels at a co-located site. We calculated daily concentrations of each of air pollutants in each city as previously described in Yi *et al.* (2010). In briefly, the every hour value from all of the monitoring stations were averaged by time in each city, and then the 24-hour values were averaged as the daily mean values for each of air pollutants, except for O_3 for which 8-hour values were averaged. We considered those daily means as the representative of daily exposure to PM concentrations in each city. Only three days of total of 2,557 days in Busan had a missing value of only PM concentrations and the three days were omitted. Peak values of PM concentrations may influence the short-term effects in a time-series analysis. We tested our main analysis that used all of data points of PM concentrations by excluding days with the highest 0.5% of PM concentrations. There were no difference of risk estimates between two data sets in each city and thus, we considered all the data of PM concentrations in this study. Daily meteorological data for each city, including temperature, relative humidity, and barometric pressure, were obtained from the Korea Meteorological Administration (KMA, http://www.kma.go.kr).

Statistical Analysis

We conducted a time-series analysis to estimate the adverse health effects of $PM_{2.5}$ and $PM_{2.5\text{-}10}$ exposure on mortality in the three cities. A generalized additive model (GAM) based on the assumption of a quasi-Poisson distribution using natural splines (ns) was used for the analysis. We controlled for mean temperature, relative humidity, and barometric pressure. The day of the week and holidays were included as dummy variables. The model equation was

$$\mathrm{Log}[\mathrm{E}(Y_t)] = \alpha + \beta \times PM_{t:t-1} + \mathrm{s}(time_t, \mathrm{df} = 7/\mathrm{year}) + \mathrm{s}(temperature_t, df = 6) + s(temperature_{t:t-1}, df = 6) + s(humidity_t, df = 6) + s(pressure_t, df = 3) + DOW + holidays$$

where E(Yt) is the number of expected deaths on day t, α is the intercept of each city, and β is the log-relative risk corresponding to a unit increase of $\mathrm{PM}_{t:t-1}$ that represents the 2-day moving average of $\mathrm{PM}_{2.5}$ and $\mathrm{PM}_{2.5\text{-}10}$ concentrations on day t and day t-1. The variable s is the natural spline smoothing function to control seasonality, time trend, and the non-linear relationship with *a priori* degrees of freedom (df), which was based on Lee *et al.* (2015) and references therein (Peng *et al.*, 2006; Qiu *et al.*, 2012). We applied calendar time with 7 df, temperature on day t and day t-1 with 6 df, and meteorological variables with 3 df for each city. DOW is the variable for day of the week.

Since the concentration of PM could affect not only the mortality on the same day of exposure, but also the mortality on a few days after exposure, we considered lag effects in this study. Previous studies have found that the associations between mortality and PM were generally larger with lagged exposures than a single-day exposure (Braga *et al.*, 2001; Zanobetti *et al.*, 2002; Franklin *et al.*, 2007; Dai *et al.*, 2014). Therefore, we used 2-day moving averages in the study (lag01, cumulative exposures of the same day of exposure and the day after exposure). We considered the effect of different single-day exposures from lag0 to lag7, as well as eight-day moving average of current to previous seven day's concentrations (lag07) and the cumulative effects from lag0 to lag7 using the dlnm package proposed by Gasparrini *et al.* (2010) for sensitivity analyses. Quasi-Poisson model has been frequently used in count data given the over- or under-dispersion dataset but it may produce inconsistent outcomes in some cases. Therefore, we considered negative binomial models to check the robustness of our main analysis. Finally, we used a two-pollutant model to examine the effects of relationships among the pollutants on the risk estimates of the single pollutant models. The coefficients obtained from the single lag and two-pollutant analysis were compared with the lag01 results.

The statistical significance of differences between the effect estimates between cities was calculated by the 95% confidence intervals as follows;

$$(\mathrm{Q1} - \mathrm{Q2}) \pm 1.96\sqrt{(SE1)^2 + (SE2)^2}$$

where Q1 and Q2 were the effect estimates for each city, and SE1 and SE2 were their corresponding standard error (Lin *et al.*, 2016).

We used SAS (Statistical Analysis System version 9.4, the SAS Institute) to arrange the data and the R program (version 3.2.1, The R Foundation) for time series analysis. The risk effect estimates were presented as the percentage of excess risk in daily mortality associated with a 10 μg/m^3 increase in each size of PM concentrations. All statistical tests were two sided, and alpha level of 0.05 was considered statistically significance.

RESULTS

Descriptive statistics of the data from 2006 to 2012 for each city are summarized in Table 2. We examined 431,743 all-cause deaths, 29,757 respiratory deaths, 113,212 cardiovascular deaths, and the air pollutants data including $PM_{2.5}$, $PM_{2.5-10}$, SO_2, NO_2, CO, and O_3 for 2,557 days for the three cities. During the study period, the averages of daily all-cause deaths were 95, 47, and 26 in Seoul, Busan, and Incheon, respectively, for all ages, and 67, 34, and 18 in Seoul, Busan, and Incheon, respectively, for the elderly. For respiratory deaths, there were 6 (Seoul), 4 (Busan), and 2 (Incheon) for all ages and 5 (Seoul), 3 (Busan), and 2 (Incheon) for the elderly. For cardiovascular deaths, there were 23 (Seoul), 14 (Busan), and 7 (Incheon) for all ages and 18 (Seoul), 11 (Busan), and 6 (Incheon) for the elderly.

The averages of daily concentrations of $PM_{2.5}$ were 26.0 $\mu g\ m^{-3}$, 27.0 $\mu g\ m^{-3}$, and 32.1 $\mu g\ m^{-3}$, in Seoul, Busan, and Incheon, respectively. The average $PM_{2.5-10}$ concentrations in Seoul, Busan, and Incheon were 27.5 $\mu g\ m^{-3}$, 23.8 $\mu g\ m^{-3}$, and 26.9 $\mu g\ m^{-3}$, respectively. We also summarized the mean concentration of other gaseous air pollutants (SO_2, NO_2, CO, and O_3), as well as the daily averages for temperature, humidity, and air pressure (Table 2).

Table 3 shows the excess mortality for a 10 $\mu g\ m^{-3}$ increase of $PM_{2.5}$ and $PM_{2.5-10}$ at lag01 for each cause of death across the three cities. In all ages, $PM_{2.5}$ was associated with 0.34% (90% CI: 0.03, 0.64), 1.18% (95% CI: 0.64, 1.72), and 0.43% (90% CI: 0.02, 0.95) increases in all-cause mortality in Seoul, Busan, and Incheon, respectively. Respiratory mortality had the highest relative risk for Busan's elderly (2.43%; 95% CI: 0.51, 4.38). For $PM_{2.5\text{-}10}$, respiratory mortality increased 0.72% (90% CI: 0.05, 1.40) in Seoul for all ages and cardiovascular mortality increased by 0.56% (90% CI: 0.06, 1.07) in Busan for the elderly. No significant association was observed in Incheon for $PM_{2.5\text{-}10}$. There were stronger associations of $PM_{2.5}$ with mortality in Busan than the other two cities. The elderly were more vulnerable to $PM_{2.5}$ and $PM_{2.5\text{-}10}$ exposure than all ages in all three cities. $PM_{2.5}$ was associated with higher risks of respiratory mortality than other causes of death. Overall, $PM_{2.5}$ was more significantly associated with various types of mortality than $PM_{2.5\text{-}10}$.

Table 4 shows the results of the single lag (lag0-lag3) effects of $PM_{2.5}$ and $PM_{2.5\text{-}10}$ exposure. Statistically significant associations were observed, but the coefficients did not increase with longer lags. The highest estimated relative risks with a 10 μg m^{-3} increase of $PM_{2.5}$ were associated with respiratory mortality at lag0 (1.77%; 95% CI: 0.55, 3.01) in Seoul, respiratory mortality at lag1 (1.92%; 95% CI: 0.27, 3.60) in Busan, and all-cause mortality at lag1 (0.48%; 95% CI: 0.02, 0.93) in Incheon for all ages. In the elderly population, the estimated associations were generally greater than those of the all ages category, similar to the lag01 results. We also found that $PM_{2.5–10}$ had a lower estimated risk effect on mortality than $PM_{2.5}$. The risk estimates of the single lags from lag0 to lag7, as well as the moving average lag07 are presented in supplemental materials (see Figs. S1-S6). The main findings at lag01 are compared with the results from lag07 and the cumulative effects from lag0 – 7 in Tables S1-S3. Overall, the excess risks of mortality associated with each 10 μg/m^3 increase of lag01 PM concentrations were attenuated when considering the cumulative effects of PM concentrations from lag0 – 7 as well as lag07 PM concentrations.

We also performed two-pollutant analysis to examine the confounding effects among air pollutants as shown in Table 5. Most of the estimated results, after adjusting for the second pollutant, showed similar or smaller associations, but there were a few cases of higher coefficients in the model with a 10 μg m^{-3} increase of $PM_{2.5}$ (lag01) (e.g., adjusted O_3 with cardiovascular mortality in Seoul, $PM_{2.5-10}$ and SO_2 for all-cause mortality as well as $PM_{2.5-10}$ and O_3 for respiratory mortality in Busan for all ages. $PM_{2.5-10}$ had showed mostly negative associations with mortality after adjusting for other air pollutants in the three cities.

DISCUSSION

In this study, we considered 570 thousand deaths across three metropolitan areas in Korea and found that $PM_{2.5}$ and $PM_{2.5-10}$ were significantly associated with increases in daily mortality (i.e., all-cause, respiratory, and cardiovascular mortality). The $PM_{2.5}$ concentration in Incheon was higher than in Busan and Seoul due to emissions from the industrial complex around Incheon. Seoul and Incheon's $PM_{2.5-10}$ levels were higher than Busan's due to heavy traffic volume in these metropolitan areas. Several previous studies have shown that $PM_{2.5-10}$ is largely comprised of re-suspended road dust (Manoli *et al.*, 2002; Masri *et al.*, 2015).

We found that there were significant, adverse health effects of PM, and the effect of PM on respiratory mortality was much larger than for other causes of deaths. These results are analogous to the findings of Franklin *et al.* (2007) and van Eeden *et al.* (2005), who reported that respiratory mortality is related to inflammatory reactions in alveolar cells caused by $PM_{2.5}$. In addition, the elderly (over 65 years) had greater increases in mortality for a unit increase in PM than the all ages group in the three cities, which aligns with other studies (Goldberg *et al.*, 2001; Franklin *et al.*, 2007; Samoli *et al.*, 2013; Lee *et al.*, 2015).

The greatest effects were observed in Busan, representing regional differences in PM health effects (Franklin *et al.*, 2007). These regional differences may be due to a number of factors, such as unrepresentative sampling, different components of PM from different sources, geographical and meteorological differences, and different exposure patterns for each city (Lee *et al.*, 2000; Chen *et al.*, 2012; Ueda *et al.*, 2016). We cannot confidently assert that the monitoring sites used in this study comprehensively represented the air pollution of each city. Also, several studies have investigated the risks of $PM_{2.5}$ exposure with different chemical components and showed different effects from different PM chemicals (Laden *et al.*, 2000; Lee *et al.,* 2000; Heo *et al.*, 2014). Various PM emission sources, weather conditions, and the geography of the study region can affect PM composition. Seoul, Incheon, and Busan are three biggest cities in Korea. In particular, Incheon and Busan are two biggest seaport cities in Korea. Previous studies have shown that there were different sources contributing to $PM_{2.5}$ mass concentrations in each city (Heo *et al.*, 2009; Choi *et al.*, 2013; Jeong *et al.*, 2017). Industrial activity, biomass burning, and motor vehicle sources were the major sources of $PM_{2.5}$ mass concentrations in Seoul and Incheon, whereas ship emissions were highly contributing to $PM_{2.5}$ mass concentrations in Busan. The different source contributions to PM concentrations between each city may lead to different risk effects between each city. In addition, water soluble metals from ship emissions, such as nickel and vanadium, are major chemicals that are highly associated with increases in reactive oxygen species (ROS) production in human body when exposed to ambient PM, subsequently triggering a case of events associated with inflammation and potential apoptosis (cell death) (Heo *et al.*, 2015). Thus, our findings that Busan has the highest risk effects of exposure to PM concentrations may be further explained by the toxicological evidence. Also, residents' adaptive behavior in more polluted areas can also affect exposure-response relationships relevant to ambient PM.

We found that the effects of $PM_{2.5\text{-}10}$ on mortality were lower than those of $PM_{2.5}$ or showed no significant association with mortality. Many studies have found higher adverse health effects of $PM_{2.5}$ than $PM_{2.5\text{-}10}$ (Kan *et al.*, 2007; Chen *et al.*, 2011; Samoli *et al.*, 2013; Lee *et al.*, 2015).

This result may be due to different components and sizes of the two categories of PM. $PM_{2.5}$ is a mixture of organic and inorganic compounds including organic carbon, elemental carbon, sulfate, nitrate, and biological particles, and $PM_{2.5\text{-}10}$ is mainly composed of crustal materials, suspended dusts, and primary organic materials (Kan *et al.*, 2007; Heo *et al.*, 2014). Also, $PM_{2.5}$ penetrates deeper into alveoli cells and results in toxic reactions (Ueda *et al.*, 2016).

Since ambient PM affects both the mortality of the current exposure day and the mortality of a few days after exposure, the lag effect has been considered in most PM exposure epidemiological studies. We observed a significant effect of $PM_{2.5}$ on cardiovascular mortality in Busan four days after exposure (lag4), but there was no significant effect on the exposure day (lag0) and three days after exposure (lag3).

In the two-pollutant models, $PM_{2.5}$ showed slightly increased effects after adjusting the single pollutant models for a second pollutants in a few cases; there were no significant effects in more cases of $PM_{2.5\text{-}10}$. These results are consistent with a previous study (Samoli *et al.*, 2013). However, Lee *et al.* (2015) estimated significant increased effects of $PM_{2.5\text{-}10}$ when the associations of $PM_{2.5\text{-}10}$ with respiratory and cardiovascular-related deaths were adjusted with O_3. The different findings between the current study and previous studies are likely due to different study regions and study periods.

To the best of our knowledge, this is the first study on the health effects of exposure to different sizes of PM using relatively long-term field measurements. However, this study had some limitations. First, we could not reflect individual exposure to ambient PM. We used air pollutant data derived from the National Ambient Monitoring Sites for each city; a few of the central monitoring sites may have had exposure misclassification. Secondly, there were likely measurement errors in the observed air pollutant data. National Ambient Monitoring Sites in Korea are controlled by the Korea Environment Corporation or local governments, and there may be different quality assurance and quality control protocols. Also, some cities use different instruments to measure air pollutants; for example, Incheon has five TEOM (Tapered Element Oscillating Microbalance) monitors and 10 beta attenuation monitors. Standardized control and quality assurance and quality control systems are needed to reduce regional differences. Moreover, increasing monitoring sites and appropriate considerations for choosing the locations of new sampling sites are needed to gather representative data. We calculated $PM_{2.5\text{-}10}$ by subtracting $PM_{2.5}$ from PM_{10}, which were not measured data, and this may have led to systemic errors (Son *et al.*, 2012). Finally, we did not consider regional characteristics of each city, such as geographical and meteorological conditions, cultural background, and sociodemographic features to definitively identify the differences in the adverse health effects of ambient PM among the cities. Further investigations with consideration of the factors affecting exposure to PM and the resulting health risk are required.

CONCLUSIONS

In summary, statistically significant associations of fine and coarse particles with mortality in three major metropolitan areas of Korea were observed in this study. Exposure to fine particles, which mostly originate from combustion and mobile emissions, showed stronger effects on human health than coarse particles, which mostly originate from natural sources such as soil and mechanical processes. This study indicates that air quality management must be strengthened, and further studies with more detailed data are needed in Korea.

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Table 1. Population sizes of three cities.

	2000		2005		2010	
	Population	%	Population	%	Population	%
Korea	46,136,101	100	47,278,951	100	48,580,293	100
City						
Seoul	9,895,217	21.4	9,820,171	20.8	9,794,304	20.2
Busan	3,662,884	7.9	3,523,582	7.5	3,414,950	7.0
Incheon	2,475,139	5.4	2,531,280	5.4	2,662,509	5.5

Table 2. Summary of statistics for the number of deaths, air pollutants, and meteorological variables in three cities.

Mortality Counts	Seoul					Busan					Incheon				
	Min	25th	Mean	75th	Max	Min	25th	Mean	75th	Max	Min	25th	Mean	75th	Max
Number of death															
All ages															
All cause	56	87	95.2	103	145	24	42	47.4	52	80	11	22	26.2	30	47
Respiratory	0	4	6.1	8	21	0	2	3.6	5	13	0	1	2.0	3	10
Cardiovascular	8	19	22.8	26	42	2	11	14.1	17	32	0	5	7.3	9	20
Ages ≥ 65															
All cause	35	60	67.3	74	108	12	29	33.5	38	62	4	15	18.1	21	37
Respiratory	0	3	5.4	7	18	0	2	3.2	4	13	0	1	1.7	3	9
Cardiovascular	4	15	18.0	21	35	0	8	11.1	13	27	0	4	5.7	7	16
Air pollutants															
$PM_{2.5}$ (μg m^{-3})	3.3	15.7	26.0	32.1	190.6	4.8	17.2	27.0	33.3	104.6	5.3	19.8	32.1	39.4	258.7
$PM_{2.5\text{-}10}$ (μg m^{-3})	2.8	15.0	27.5	32.9	673.5	1.1	14.7	23.8	27.3	769.5	4.7	15.9	26.9	31.1	402.0
SO_2 (ppb)	2.5	3.9	5.6	6.6	21.2	0.5	4.2	5.8	6.9	23.0	3.1	5.7	7.7	9.2	24.3
NO_2 (ppb)	9.7	27.8	37.7	46.6	92.4	2.0	15.3	21.2	25.9	52.1	6.5	21.3	30.8	38.4	101.8
CO (ppm)	0.2	0.5	0.6	0.7	1.8	0.1	0.3	0.4	0.5	1.0	0.2	0.5	0.6	0.7	1.8
O_3 (ppb)	2.0	10.5	18.2	24.7	60.5	1.5	19.4	26.1	32.4	63.4	2.6	14.1	21.9	28.8	65.7
Meteorology															
Temperature (℃)	-14.6	3.9	12.7	22.1	31.8	-7.2	8.1	14.9	21.6	30.1	-14.6	3.9	12.7	22.1	31.8
Humidity (%)	19.4	49.1	60.3	71.3	96.3	11.6	46.8	61.3	76.0	97.1	19.4	49.1	60.3	71.3	96.3
Air pressure (hPa)	993.3	1009.6	1016.0	1022.5	1038.1	994.1	1010.1	1015.5	1021.0	1034.6	993.3	1009.6	1016.0	1022.5	1038.1

Table 3. Excess risks of mortality associated with a 10 μg m^{-3} increase of PM$_{2.5}$ and PM$_{2.5\text{-}10}$ at lag01 in three cities.

Air pollutant	Ages	Mortality	City		
			Seoul	Busan	Incheon
PM$_{2.5}$	All ages	All cause	0.34 (0.03 to 0.64)[a]	1.18 (0.64 to 1.72)	0.43 (0.02 to 0.95)[a]
		Respiratory	2.08 (0.74 to 3.44)	2.25 (0.38 to 4.15)	0.82 (-0.99 to 2.69)
		Cardiovascular	0.90 (0.19 to 1.62)	0.56 (-0.40 to 1.53)	0.19 (-0.75 to 1.14)
	Elderly (≥65)	All cause	0.52 (0.09 to 0.96)	1.26 (0.62 to 1.91)	0.66 (0.03 to 1.29)
		Respiratory	2.24 (0.83 to 3.68)	2.43 (0.51 to 4.38)	0.35 (-1.59 to 2.33)
		Cardiovascular	0.61 (-0.19 to 1.41)	0.56 (-0.52 to 1.65)	0.14 (-0.92 to 1.21)
PM$_{2.5\text{-}10}$	All ages	All cause	0.20 (0.02 to 0.38)[a]	-0.00 (-0.32 to 0.32)	0.13 (-0.31 to 0.57)
		Respiratory	0.72 (0.05 to 1.40)[a]	0.47 (-0.66 to 1.60)	0.69 (-0.85 to 2.26)
		Cardiovascular	0.44 (0.03 to 0.85)	0.40 (-0.14 to 0.95)	0.03 (-0.78 to 0.85)
	Elderly (≥65)	All cause	0.38 (0.12 to 0.64)	0.11 (-0.27 to 0.49)	0.07 (-0.46 to 0.62)
		Respiratory	0.70 (-0.15 to 1.56)	0.43 (-0.77 to 1.65)	-0.18 (-1.87 to 1.55)
		Cardiovascular	0.53 (0.07 to 0.99)	0.56 (0.06 to 1.07)[a]	0.07 (-0.84 to 0.99)

[a] 90% Confidence Interval

Table 4. Excess risks of mortality associated with a 10 μg m^{-3} increase of PM$_{2.5}$ and PM$_{2.5\text{-}10}$ at different single lag in three cities.

Air pollutant	Ages	Mortality	Lag	City		
				Seoul	Busan	Incheon
PM$_{2.5}$	All ages	All cause	0	0.33 (0.01 to 0.66)	0.92 (0.44 to 1.40)	0.24 (-0.23 to 0.71)
			1	0.20 (-0.12 to 0.52)	0.92 (0.44 to 1.39)	0.48 (0.02 to 0.93)
			2	0.02 (-0.29 to 0.33)	0.44 (-0.01 to 0.90)	0.35 (-0.11 to 0.81)
			3	-0.14 (-0.45 to 0.16)	0.41 (-0.04 to 0.85)	-0.16 (-0.62 to 0.30)
		Respiratory	0	1.77 (0.55 to 3.01)	1.59 (-0.09 to 3.29)	0.18 (-1.51 to 1.89)
			1	1.52 (0.33 to 2.72)	1.92 (0.27 to 3.60)	1.13 (-0.49 to 2.78)
			2	1.16 (-0.01 to 2.34)	1.40 (-0.21 to 3.03)	0.91 (-0.73 to 2.57)
			3	0.60 (-0.55 to 1.76)	0.47 (-1.10 to 2.07)	-0.49 (-2.13 to 1.19)
		Cardiovascular	0	0.76 (0.12 to 1.41)	0.60 (-0.25 to 1.46)	-0.05 (-0.92 to 0.83)
			1	0.66 (0.03 to 1.29)	0.30 (-0.54 to 1.15)	0.35 (-0.49 to 1.19)
			2	-0.01 (-0.63 to 0.61)	-0.26 (-1.07 to 0.56)	0.45 (-0.39 to 1.31)
			3	-0.13 (-0.73 to 0.47)	0.25 (-0.54 to 1.05)	-0.06 (-0.91 to 0.79)
	Elderly (≥65)	All cause	0	0.56 (0.17 to 0.96)	0.92 (0.35 to 1.50)	0.43 (-0.15 to 1.01)
			1	0.27 (-0.12 to 0.66)	1.04 (0.48 to 1.61)	0.65 (0.09 to 1.21)
			2	-0.12 (-0.50 to 0.26)	0.62 (0.07 to 1.16)	0.62 (0.05 to 1.18)
			3	-0.27 (-0.64 to 0.10)	0.46 (-0.07 to 1.00)	0.01 (-0.55 to 0.57)
		Respiratory	0	2.22 (0.93 to 3.52)	1.89 (0.17 to 3.65)	-0.43 (-2.23 to 1.41)
			1	1.37 (0.12 to 2.64)	2.01 (0.30 to 3.75)	0.96 (-0.77 to 2.72)
			2	0.93 (-0.31 to 2.19)	1.31 (-0.39 to 3.03)	1.10 (-0.64 to 2.87)
			3	0.26 (-0.96 to 1.50)	0.17 (-1.50 to 1.87)	-0.22 (-1.97 to 1.57)
		Cardiovascular	0	0.65 (-0.07 to 1.37)	0.59 (-0.37 to 1.56)	-0.01 (-0.99 to 0.98)
			1	0.30 (-0.40 to 1.01)	0.29 (-0.66 to 1.25)	0.23 (-0.71 to 1.19)
			2	-0.16 (-0.85 to 0.53)	-0.22 (-1.13 to 0.70)	0.38 (-0.58 to 1.34)
			3	-0.12 (-0.79 to 0.56)	0.54 (-0.34 to 1.44)	0.13 (-0.82 to 1.10)
PM$_{2.5\text{-}10}$	All ages	All cause	0	0.16 (-0.01 to 0.34)	0.08 (-0.17 to 0.34)	0.13 (-0.25 to 0.50)
			1	0.11 (-0.07 to 0.29)	-0.08 (-0.34 to 0.17)	0.06 (-0.31 to 0.43)
			2	0.06 (-0.11 to 0.24)	-0.16 (-0.42 to 0.10)	0.12 (-0.25 to 0.49)
			3	0.12 (-0.06 to 0.29)	0.09 (-0.16 to 0.33)	0.18 (-0.18 to 0.55)
		Respiratory	0	0.36 (-0.31 to 1.03)	0.21 (-0.73 to 1.16)	0.21 (-1.12 to 1.57)

		1	0.62 (-0.03 to 1.27)	0.37 (-0.52 to 1.26)	0.77 (-0.52 to 2.07)
		2	0.91 (0.30 to 1.53)	0.24 (-0.66 to 1.14)	-0.42 (-1.80 to 0.97)
		3	0.50 (-0.15 to 1.15)	-0.87 (-1.94 to 0.21)	-0.26 (-1.63 to 1.13)
	Cardiovascular	0	0.32 (-0.02 to 0.66)	0.52 (0.11 to 0.94)	0.11 (-0.57 to 0.80)
		1	0.28 (-0.06 to 0.62)	-0.04 (-0.48 to 0.41)	-0.07 (-0.76 to 0.62)
		2	-0.02 (-0.36 to 0.33)	-0.18 (-0.63 to 0.28)	0.19 (-0.49 to 0.88)
		3	0.05 (-0.30 to 0.39)	-0.14 (-0.60 to 0.32)	-0.13 (-0.83 to 0.57)
Elderly (≥65)	All cause	0	0.32 (0.11 to 0.53)	0.18 (-0.12 to 0.49)	0.08 (-0.38 to 0.55)
		1	0.19 (-0.02 to 0.41)	-0.04 (-0.35 to 0.26)	0.02 (-0.44 to 0.48)
		2	0.05 (-0.17 to 0.27)	-0.18 (-0.49 to 0.13)	0.24 (-0.22 to 0.69)
		3	0.08 (-0.14 to 0.29)	-0.06 (-0.36 to 0.25)	0.23 (-0.22 to 0.69)
	Respiratory	0	0.42 (-0.29 to 1.14)	0.30 (-0.68 to 1.28)	-0.54 (-2.03 to 0.97)
		1	0.53 (-0.17 to 1.24)	0.27 (-0.69 to 1.24)	0.26 (-1.16 to 1.71)
		2	0.81 (0.15 to 1.48)	0.28 (-0.67 to 1.24)	-0.74 (-2.24 to 0.78)
		3	0.32 (-0.38 to 1.04)	-1.20 (-2.39 to 0.00)	-0.11 (-1.56 to 1.36)
	Cardiovascular	0	0.45 (0.08 to 0.82)	0.71 (0.26 to 1.16)	0.13 (-0.65 to 0.91)
		1	0.27 (-0.11 to 0.65)	-0.04 (-0.54 to 0.47)	-0.03 (-0.81 to 0.75)
		2	-0.09 (-0.48 to 0.31)	-0.25 (-0.77 to 0.27)	0.04 (-0.73 to 0.82)
		3	-0.03 (-0.42 to 0.36)	-0.21 (-0.73 to 0.32)	-0.14 (-0.93 to 0.65)

Table 5. Excess risks of mortality associated with a 10 μg m^{-3} increase of $PM_{2.5}$ and $PM_{2.5-10}$ in three cities using two-pollutant model.

Air pollutant	Ages	Mortality	Second pollutant	City		
				Seoul	Busan	Incheon
$PM_{2.5}$	All ages	All cause	None	0.34 (-0.02 to 0.70)	1.18 (0.64 to 1.72)	0.44 (-0.07 to 0.95)
			SO_2	0.15 (-0.29 to 0.59)	1.26 (0.61 to 1.91)	0.34 (-0.29 to 0.98)
			NO_2	0.37 (-0.05 to 0.79)	1.13 (0.50 to 1.77)	0.31 (-0.29 to 0.92)
			CO	0.43 (-0.03 to 0.90)	1.00 (0.28 to 1.73)	0.50 (-0.17 to 1.17)
			O_3	0.31 (-0.06 to 0.67)	1.18 (0.64 to 1.72)	0.45 (-0.05 to 0.97)
		Respiratory	None	2.08 (0.74 to 3.44)	2.25 (0.38 to 4.15)	0.82 (-0.99 to 2.69)
			SO_2	1.14 (-0.51 to 2.82)	1.61 (-0.63 to 3.90)	-0.01 (-2.31 to 2.35)
			NO_2	1.99 (0.38 to 3.62)	1.85 (-0.35 to 4.10)	0.12 (-2.06 to 2.35)
			CO	1.56 (-0.22 to 3.36)	2.32 (-0.21 to 4.91)	0.29 (-2.15 to 2.80)
			O_3	2.06 (0.72 to 3.43)	2.26 (0.39 to 4.16)	0.84 (-1.00 to 2.71)
		Cardiovascular	None	0.90 (0.19 to 1.62)	0.56 (-0.40 to 1.53)	0.19 (-0.75 to 1.14)
			SO_2	0.71 (-0.16 to 1.59)	0.82 (-0.33 to 1.98)	-0.03 (-1.22 to 1.17)
			NO_2	0.87 (0.05 to 1.70)	0.73 (-0.39 to 1.86)	-0.10 (-1.22 to 1.04)
			CO	0.90 (0.00 to 1.82)	0.18 (-1.10 to 1.48)	0.43 (-0.83 to 1.71)
			O_3	0.92 (0.20 to 1.65)	0.58 (-0.38 to 1.54)	0.25 (-0.70 to 1.21)
	Elderly (≥65)	All cause	None	0.52 (0.09 to 0.96)	1.26 (0.62 to 1.91)	0.66 (0.03 to 1.29)
			SO_2	0.24 (-0.29 to 0.78)	1.39 (0.62 to 2.16)	0.42 (-0.37 to 1.22)
			NO_2	0.46 (-0.05 to 0.98)	1.26 (0.51 to 2.02)	0.40 (-0.34 to 1.14)
			CO	0.50 (-0.07 to 1.07)	1.04 (0.18 to 1.91)	0.60 (-0.22 to 1.43)
			O_3	0.53 (0.09 to 0.97)	1.24 (0.60 to 1.89)	0.66 (0.03 to 1.30)
		Respiratory	None	2.24 (0.83 to 3.68)	2.43 (0.51 to 4.38)	0.35 (-1.59 to 2.33)
			SO_2	0.86 (-0.89 to 2.64)	1.22 (-1.13 to 3.64)	-0.18 (-2.64 to 2.34)
			NO_2	1.89 (0.18 to 3.62)	1.82 (-0.50 to 4.20)	-0.35 (-2.68 to 2.04)
			CO	1.40 (-0.47 to 3.31)	2.22 (-0.43 to 4.94)	-0.02 (-2.64 to 2.66)
			O_3	2.30 (0.88 to 3.73)	2.42 (0.51 to 4.38)	0.40 (-1.55 to 2.40)
		Cardiovascular	None	0.61 (-0.19 to 1.41)	0.56 (-0.52 to 1.65)	0.14 (-0.92 to 1.21)
			SO_2	0.39 (-0.58 to 1.38)	0.85 (-0.45 to 2.16)	0.02 (-1.31 to 1.38)
			NO_2	0.53 (-0.39 to 1.46)	0.69 (-0.57 to 1.97)	-0.31 (-1.57 to 0.97)
			CO	0.52 (-0.49 to 1.55)	-0.02 (-1.46 to 1.44)	0.43 (-0.96 to 1.85)

			O_3	0.62 (-0.19 to 1.43)	0.56 (-0.51 to 1.65)	0.21 (-0.86 to 1.28)
$PM_{2.5-10}$	All ages	All cause	None	0.20 (-0.01 to 0.41)	-0.00 (-0.32 to 0.32)	0.13 (-0.31 to 0.57)
			SO_2	0.15 (-0.07 to 0.37)	-0.02 (-0.34 to 0.31)	0.05 (-0.40 to 0.51)
			NO_2	0.20 (-0.02 to 0.42)	-0.02 (-0.34 to 0.30)	0.05 (-0.41 to 0.51)
			CO	0.18 (-0.04 to 0.41)	-0.05 (-0.38 to 0.27)	0.08 (-0.38 to 0.54)
			O_3	0.20 (-0.02 to 0.41)	0.00 (-0.32 to 0.32)	0.13 (-0.31 to 0.57)
		Respiratory	None	0.72 (-0.08 to 1.53)	0.47 (-0.66 to 1.60)	0.69 (-0.85 to 2.26)
			SO_2	0.48 (-0.35 to 1.32)	0.40 (-0.76 to 1.58)	0.45 (-1.16 to 2.08)
			NO_2	0.63 (-0.19 to 1.46)	0.40 (-0.76 to 1.57)	0.49 (-1.11 to 2.12)
			CO	0.50 (-0.34 to 1.35)	0.39 (-0.77 to 1.56)	0.52 (-1.09 to 2.16)
			O_3	0.73 (-0.07 to 1.54)	0.45 (-0.70 to 1.61)	0.69 (-0.85 to 2.26)
		Cardiovascular	None	0.44 (0.03 to 0.85)	0.40 (-0.14 to 0.95)	0.03 (-0.78 to 0.85)
			SO_2	0.37 (-0.05 to 0.79)	0.40 (-0.15 to 0.94)	-0.05 (-0.89 to 0.80)
			NO_2	0.42 (0.00 to 0.84)	0.40 (-0.15 to 0.94)	-0.08 (-0.92 to 0.77)
			CO	0.38 (-0.04 to 0.81)	0.37 (-0.18 to 0.92)	0.05 (-0.79 to 0.91)
			O_3	0.44 (0.03 to 0.85)	0.40 (-0.14 to 0.94)	0.04 (-0.77 to 0.85)
	Elderly (≥65)	All cause	None	0.38 (0.12 to 0.64)	0.11 (-0.27 to 0.49)	0.07 (-0.46 to 0.62)
			SO_2	0.32 (0.05 to 0.58)	0.10 (-0.28 to 0.48)	-0.05 (-0.61 to 0.52)
			NO_2	0.35 (0.09 to 0.61)	0.10 (-0.29 to 0.48)	-0.03 (-0.59 to 0.53)
			CO	0.35 (0.09 to 0.62)	0.06 (-0.33 to 0.45)	-0.01 (-0.58 to 0.56)
			O_3	0.38 (0.12 to 0.64)	0.11 (-0.27 to 0.49)	0.07 (-0.47 to 0.62)
		Respiratory	None	0.70 (-0.15 to 1.56)	0.43 (-0.77 to 1.65)	-0.18 (-1.87 to 1.55)
			SO_2	0.36 (-0.54 to 1.27)	0.32 (-0.91 to 1.57)	-0.37 (-2.14 to 1.43)
			NO_2	0.56 (-0.32 to 1.44)	0.28 (-0.95 to 1.53)	-0.40 (-2.17 to 1.41)
			CO	0.40 (-0.51 to 1.31)	0.32 (-0.93 to 1.59)	-0.34 (-2.12 to 1.48)
			O_3	0.70 (-0.15 to 1.56)	0.43 (-0.77 to 1.66)	-0.18 (-1.87 to 1.55)
		Cardiovascular	None	0.53 (0.07 to 0.99)	0.56 (-0.04 to 1.17)	0.07 (-0.84 to 0.99)
			SO_2	0.48 (0.01 to 0.95)	0.58 (-0.02 to 1.19)	0.02 (-0.92 to 0.98)
			NO_2	0.50 (0.04 to 0.97)	0.56 (-0.05 to 1.17)	-0.08 (-1.02 to 0.88)
			CO	0.50 (0.02 to 0.97)	0.52 (-0.08 to 1.14)	0.12 (-0.83 to 1.07)
			O_3	0.53 (0.07 to 0.99)	0.56 (-0.04 to 1.17)	0.08 (-0.83 to 0.99)

Figure Captions

Fig. 1. Location of study area in Korea.

Fig. 1.