The relationships between short-term exposure to particulate matter and mortality in Korea: impact of particulate matter exposure metrics for sub-daily exposures

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Abstract
Most studies of short-term particulate matter (PM) exposure use 24 h averages. However, other pollutants have stronger effects in shorter timeframes, which has influenced policy (e.g., ozone 8 h maximum). The selection of appropriate exposure timeframes is important for effective regulation. The US EPA identified health effects for sub-daily PM exposures as a critical research need. Unlike most areas, Seoul, Korea has hourly measurements of PM$_{10}$, although not PM$_{2.5}$. We investigated PM$_{10}$ and mortality (total, cardiovascular, respiratory) in Seoul (1999–2009) considering sub-daily exposures: 24 h, daytime (7 am–8 pm), morning (7–10 am), nighttime (8 pm–7 am), and 1 h daily maximum. We applied Poisson generalized linear modeling adjusting for temporal trends and meteorology. All PM$_{10}$ metrics were significantly associated with total mortality. Compared to other exposure timeframes, morning exposure had the most certain effect on total mortality (based on statistical significance). Increases of 10 µg m$^{-3}$ in 24 h, daytime, morning, nighttime, and 1 h maximum PM$_{10}$ were associated with 0.15% (95% confidence interval 0.02–0.28%), 0.14% (0.01–0.27%), 0.10% (0.03–0.18%), 0.12% (0.03–0.22%), and 0.10% (0.00–0.21%) increases in total mortality, respectively. PM$_{10}$ was significantly associated with cardiovascular mortality for 24 h, morning, and nighttime exposures. We did not identify significant associations with respiratory mortality. The results support use of a 24 h averaging time as an appropriate metric for health studies and regulation, particularly for PM$_{10}$ and mortality.

Keywords: exposure timeframes, sub-daily averaging times, mortality, PM$_{10}$, time-series

1. Introduction
Particulate matter (PM) has been linked to numerous adverse health effects including increased risk of hospital admissions and mortality [1–3]. Epidemiological studies of the health effects of PM have reported significant associations in...
many parts of the world [4–6]. The majority of the studies examining short-term exposure have used the daily (i.e., 24 h) average as the exposure metric, as this measure is most commonly used in the majority of government monitoring networks.

Selecting and investigating the appropriate exposure timeframes for PM is important for effective regulatory control and risk management. The exposure metric or averaging time is one of the basic elements of a regulatory standard for air pollution, with the others being indicator, level, and form [7]. Regulations and guidelines aimed at protecting public health from airborne particles are typically based on the 24 h metric. Examples are the US Environmental Protection Agency’s (EPA’s) National Ambient Air Quality Standards [8], the World Health Organization’s Air Quality Guidelines [9], and Korea’s Air Quality Standards [10]. The choice of a 24 h average metric is founded on the state of scientific evidence, which indicates health impacts for this exposure metric; however, in comparison few population-based studies have investigated shorter exposure timeframes.

In a 2005 review of research on the health impacts of particles, the US EPA determined that ‘the study of shorter-term averaging times, on the order of one or more hours, is an important research priority’ [7]. The US EPA noted ‘There is a growing body of studies that provide additional evidence of effects associated with exposure periods shorter than 24 h (e.g., one to several hours)…’ [US EPA] staff concludes that this information remains too limited to serve as a basis for establishing a shorter-than-24 h fine particle primary standard at this time’ [7]. Several years later in the most recent review, published in 2009, US EPA’s synthesis of research on PM and health called for analysis on ‘What new evidence is available on effects occurring from exposures at sub-daily averaging times?’ and noted a critical question was ‘To what extent is key scientific evidence becoming available to improve our understanding of the health effects associated with various time periods of PM exposures, including not only short-term (daily or multiday) and chronic (months to years) exposures, but also peak PM exposures (<24 h)?’ However, based on the current studies, the US EPA concluded that ‘There were insufficient data on peak (i.e., <24 h) exposures for any PM size fraction with health effects to make causality determinations for this exposure category’ [11, 12]. This indicates that studies on particles’ health impacts at sub-daily exposures for all PM size fractions are still needed. Further, such results could add to our understanding of biological mechanism.

Findings from studies on the health impacts of PM at sub-daily exposures might suggest that different exposure metrics could be more suitable for policies, or they may indicate that a 24 h exposure is appropriate. This issue has played an important role in policy for other air pollutants that have been shown to have stronger effects at timeframes shorter than the 24 h period, such as the daily 8 h maximum for ozone [13]. In fact, the US EPA’s health-based regulations for ozone have evolved in response to scientific evidence on sub-daily exposure metrics, starting with the daily 1 h maximum in 1971 and moving to a daily 8 h maximum in 1997.

Although few population-based studies of PM at sub-daily exposures exist, some studies do provide additional evidence of effects associated with exposure periods shorter than 24 h. Associations were observed between ambient PM concentrations averaged over shorter time intervals (e.g., one to several hours) and cardiovascular or respiratory effects [14–20]. These studies reported statistically significant associations between shorter-term PM10 (particulate matter ≤10 µm in aerodynamic diameter) or PM2.5 (particulate matter ≤2.5 µm in aerodynamic diameter) concentrations and cardiovascular health outcomes such as myocardial infarction and heart rate variability and respiratory outcomes. A study in the US reported that effect estimates for myocardial infarction with PM2.5 levels 2 and 24 h before onset are quite similar in magnitude and both are statistically significant [18]. Another US study found higher associations with asthma symptoms for daily 1 h and 8 h maximum PM10 exposures, as compared with the standard metric of the 24 h mean [15]. On the other hand, a study in Los Angeles reported higher effect estimates for 24 h PM10 concentrations and respiratory symptoms than for a metric of the 1 h maximum [21]. These studies did not use the data from standard, government monitoring networks.

The lack of population-based studies using sub-daily exposures largely relates to data limitations as daily data are often more readily available. Many regions of the world, such as the US, have routine measurements for daily levels of PM, but do not have monitoring networks providing information at a higher resolution such as hourly data. For example, some of the earlier studies in the United States used 24 hr PM data every third day [22]. The Ministry of Environment, Republic of Korea, does collect hourly PM10 data allowing a unique opportunity to study PM exposure metrics. In this study, we investigated associations of PM10 with daily mortality in Seoul, Korea for the period 1999–2009, using several short-term exposure metrics of PM10: 24 h average, daytime, morning, nighttime, and 1 h maximum. To the best of our knowledge, this is the first study to compare the mortality impacts of PM10 using sub-daily exposure metrics in Korea or elsewhere.

2. Methods

2.1. Health, pollution, and weather data

Hourly PM10 concentrations in Seoul were obtained for 27 monitoring stations operated by the Ministry of Environment, Republic of Korea. We used 5 short-term exposure metrics of PM10: (1) the traditional 24 h exposure (i.e., daily exposure), (2) daytime exposure (7 am–8 pm), (3) morning exposure (7 am–10 am), (4) nighttime exposure (8 pm–7 am), and (5) a peak exposure of the 1 h daily maximum.

The National Meteorological Administration, Republic of Korea, provided hourly measurements of ambient temperature and relative humidity, and 3 h measurements of barometric pressure for Seoul during the study period. We converted weather data into 24 h values (i.e., daily) by
averaging the 24 hourly values for temperature and relative humidity or averaging the eight 3 h values for barometric pressure for each day.

We obtained daily mortality data for Seoul between 1 January 1999 and 31 December 2009 from the National Statistical Office, Republic of Korea. We classified mortality data into all causes of death except external causes (International Classification of Diseases, ICD-10; World Health Organization 2007, A00-R99), cardiovascular causes (ICD-10, I00-I99), and respiratory causes (ICD-10, J00-J99). Analyses were stratified by cause of death.

2.2. Statistical analysis

We assessed how various PM metrics are associated with risk of mortality by using time-series modeling, an approach that has been applied widely to examine the health effects of short-term exposure to many pollutants, including PM. This method estimates how variation in exposure, in this case PM, is associated with changes in health outcomes such as mortality, and allows controlling for time-variant confounding factors such as weather and seasonal trends. A review of time-series methodology in relation to the study of PM and health is provided elsewhere [23]. Epidemiological findings from time-series studies have provided strong evidence on the associations between short-term PM exposure and health outcomes and results have played a crucial role in the setting of regulatory standards [24, 25]. Also, the results of time-series studies have been central to analyses aimed at estimating the overall health impacts or economic damages attributable to air pollution exposure [26–28].

To estimate the relationship between daily mortality and several exposure metrics of PM$_{10}$, we applied an over-dispersed Poisson generalized linear model with natural cubic splines for time and meteorology. In this time-series model, the daily number of deaths $Y_t$ is assumed to have an over-dispersed Poisson distribution with expected value $E(Y_t)$.

$$\ln(E(Y_t)) = \beta_0^t + \beta_j^t X_j^t + d^t \text{DOW}_t + \text{ns}(t) + \text{ns}(\text{temperature})_t + \text{ns}(\text{humidity})_t + \text{ns}(\text{pressure})_t$$

(1)

where $E(Y_t)$ = expected number of deaths on day $t$; $\beta_0^t$ = model intercept for exposure metric $j$ (e.g., 24 h PM$_{10}$ exposure); $d^t$ = vector of regression coefficients for day of the week for model of exposure metric $j$; DOH$_t$ = categorical variable for day of the week; $\text{ns}(\text{time})_t$ = natural cubic spline of a variable representing time to adjust for long-term trends and seasonality, with 7 degrees of freedom (df) per year; $\text{ns}(\text{temperature})_t$ = natural cubic spline of temperature on day $t$, with 3 df; $\text{ns}(\text{humidity})_t$ = natural cubic spline of humidity on day $t$, with 4 df, and $\text{ns}(\text{pressure})_t$ = natural cubic spline of pressure on day $t$, with 4 df. The variable $X_j^t$ represents the level of exposure metric $j$ on day $t$. The variable $\beta_j^t$ denotes the relationship between exposure metric $j$ and mortality risk. Each exposure metric was modeled separately.

We examined the effect of each exposure metric of PM$_{10}$ with single day lags of the same day (lag0) and previous days (lag1, lag2 and lag3) and cumulative lags (lag01, lag02 and lag03). All analyses were conducted using R 2.10.1 (R Foundation for Statistical Computing, Vienna, Austria). Results were expressed as the percentage change in mortality per 10 µg m$^{-3}$ increase of each metric. Because this increment is not equivalent across the various exposure metrics, results were also presented based on an interquartile range (IQR) increment.

3. Results

Health impacts for various short-term exposure metrics may differ if the pollutant exhibits daily fluctuations. We examined the pattern of PM$_{10}$ concentrations by time of day and time of year. Figure 1 shows daily patterns of hourly averages of PM$_{10}$ concentrations for the study period and by season. PM$_{10}$ concentrations displayed a peak in the morning (10:00 am) and a secondary peak in the evening (10:00 pm) for the whole period. Levels started to rise around 7:00 am or 6:00 pm when the heavy traffic usually occurs and increased for several hours. The morning peak is present for all seasons except summer. In the autumn, the concentration of the evening peak is higher than that of morning peak, whereas in winter the morning peak is higher.

Supplementary figure 1 (available at stacks.iop.org/ERL/8/014015/mmedia) shows monthly average PM$_{10}$ concentrations by different exposure metrics across all years. Monthly average concentrations show similar patterns among different exposure metrics. PM$_{10}$ levels generally decreased in summer months, and higher concentrations are found in winter or spring months, when Asian dust events frequently occurred. The months with the highest values vary by year; however, most years exhibit peaks in spring (e.g., March) and the lowest concentrations in summer (e.g., August) (results not shown).

Table 1 provides descriptive statistics of mortality data and PM$_{10}$ concentrations by different exposure metrics for the study period. The average PM$_{10}$ concentrations by different exposure metrics showed similar patterns, ranging from 63.2 to 64.5 µg m$^{-3}$, except the 1 h maximum, which exhibits more variation, reaching 1784 µg m$^{-3}$ and averaging 94.1 µg m$^{-3}$. Descriptive statistics by season are presented in supplementary table 1 (available at stacks.iop.org/ERL/8/014015/mmedia). PM$_{10}$ concentrations by different exposure metrics generally showed similar seasonal patterns, with lower concentrations in summer and autumn and higher concentrations in spring and winter.
Table 1. Summary statistics of mortality and PM\textsubscript{10} concentration by different exposure metrics in Seoul, Korea, 1999–2009.

|                | Mean | SD  | Min  | 25%  | 50%  | 75%  | Max  | IQR  |
|----------------|------|-----|------|------|------|------|------|------|
| Mortality (observations/day) | 92.9 | 11.7| 55.0 | 85.0 | 93.0 | 100.0| 148.0| 15.0 |
| PM\textsubscript{10} (µg m\textsuperscript{-3}) | 24 h exposure | 63.5 | 44.6 | 9.0  | 38.9 | 55.7 | 77.5 | 1016.4 | 38.6 |
|                | Daytime exposure (7 am–8 pm) | 63.8 | 46.3 | 8.3  | 37.4 | 55.3 | 78.5 | 1025.2 | 41.1 |
|                | Morning exposure (7 am–10 am) | 64.5 | 49.5 | 7.2  | 35.6 | 55.5 | 81.9 | 1227.3 | 46.4 |
|                | Nighttime exposure (8 pm–7 am) | 63.2 | 45.6 | 7.8  | 38.7 | 55.4 | 78.0 | 1238.7 | 39.3 |
|                | 1 h maximum | 94.1 | 80.3 | 11.5 | 56.5 | 80.3 | 112.6 | 1784.0 | 56.1 |

Table 2. Correlation coefficients among different PM\textsubscript{10} exposure metrics, 1999–2009.

|                | Daytime (7 am–8 pm) | Morning (7 am–10 am) | Nighttime (8 pm–7 am) | 1 h maximum |
|----------------|---------------------|----------------------|-----------------------|-------------|
| 24 h exposure  | 0.97                | 0.92                 | 0.96                  | 0.92        |
| Daytime (7 am–8 pm) | 0.90                | 0.88                 | 0.88                  | 0.87        |
| Morning (7 am–10 am) | 0.88                | 0.88                 | 0.88                  | 0.90        |
| Nighttime (8 pm–7 am) | 0.88                | 0.88                 | 0.88                  | 0.90        |

Table 3. Effects of different PM\textsubscript{10} exposure metrics and lags on total mortality, for a 10 µg m\textsuperscript{-3} increase in PM\textsubscript{10} (95% confidence intervals). (Note: significant findings are shaded. For each exposure, the most certain effect is shown in bold font. The effect estimates shown in this table are for a 10 µg m\textsuperscript{-3} increase for all exposure metrics; however, a 10 µg m\textsuperscript{-3} increase is not equivalent across all exposure metrics. For example, a 10 µg m\textsuperscript{-3} increase in the 24 h exposure corresponds to approximately a 15 µg m\textsuperscript{-3} increase in the daily 1 h maximum.)

|                | 24 h exposure | Daytime (7 am–8 pm) | Morning (7 am–10 am) | Nighttime (8 pm–7 am) | 1 h maximum |
|----------------|---------------|---------------------|----------------------|-----------------------|-------------|
| Lag0 | 0.09 (0.00, 0.17) | 0.07 (–0.01, 0.16) | **0.10 (0.03, 0.18)** | 0.09 (0.01, 0.17) | 0.06 (–0.01, 0.12) |
| Lag1 | 0.07 (–0.01, 0.16) | 0.05 (–0.03, 0.13) | 0.06 (–0.02, 0.13) | 0.09 (0.01, 0.17) | 0.05 (–0.02, 0.11) |
| Lag2 | 0.05 (–0.03, 0.14) | 0.05 (–0.03, 0.13) | 0.05 (–0.03, 0.12) | 0.05 (–0.03, 0.13) | 0.04 (–0.03, 0.11) |
| Lag3 | 0.05 (–0.04, 0.13) | 0.05 (–0.03, 0.13) | 0.04 (–0.03, 0.12) | 0.04 (–0.04, 0.12) | 0.04 (–0.03, 0.10) |
| Lag01 | 0.11 (0.01, 0.21) | 0.09 (–0.01, 0.19) | 0.12 (0.03, 0.21) | **0.12 (0.03, 0.22)** | 0.07 (–0.01, 0.15) |
| Lag02 | 0.13 (0.02, 0.24) | 0.12 (0.00, 0.23) | 0.14 (0.03, 0.25) | 0.14 (0.03, 0.24) | 0.09 (–0.01, 0.18) |
| Lag03 | **0.15 (0.02, 0.28)** | **0.14 (0.01, 0.27)** | 0.16 (0.04, 0.28) | 0.15 (0.03, 0.27) | **0.10 (0.00, 0.21)** |

Table 2 provides the correlation coefficients among different PM\textsubscript{10} short-term exposure metrics. The correlations between different exposure metrics were high, ranging from 0.80 to 0.97. The highest correlation was for 24 h exposure and daytime exposure (0.97) and correlation between morning exposure and 1 h maximum was the lowest, although still high (0.80). Correlations among different PM\textsubscript{10} exposure metrics by season are provided in supplementary table 2 (available at stacks.iop.org/ERL/8/014015/mmedia). Correlations between 24 h exposure and daytime exposure were the highest in all seasons (range 0.97–0.99). The lowest correlation was between morning exposure and the 1 h maximum in fall at 0.78.

Table 3 shows the percentage change in risk of total mortality per 10 µg m\textsuperscript{-3} increase in PM\textsubscript{10} for each exposure metric by lag and supplementary table 3 (available at stacks.iop.org/ERL/8/014015/mmedia) shows results based on an increment of an IQR. Note that a 10 µg m\textsuperscript{-3} increase is not equivalent across all exposure metrics. For example, a 10 µg m\textsuperscript{-3} increase in the 24 h exposure corresponds to approximately a 15 µg m\textsuperscript{-3} increase in the daily 1 h maximum. In table 3, statistically significant findings are shaded. For each exposure metric, the lag with the most certain effect estimates (largest t-statistic) is shown in bold font. These lags were the 4-day cumulative lags (L03) for 24 h exposure, daytime exposure, and 1 h maximum; same day (L0) for morning exposure; and 2 day cumulative lags (L01) for nighttime exposure. Associations between mortality risk and PM\textsubscript{10} were observed for all exposure metrics. For example, a 10 µg m\textsuperscript{-3} increase in 24 h exposure at L03 was associated with a 0.15% (0.02, 0.28%), 0.14% (0.01, 0.27%) increase in total mortality, respectively. For all PM\textsubscript{10} exposure metrics, associations were higher when multiday lags were considered rather than single day lag, based on central estimates. Although statistically significant associations with risk of mortality were found for all PM\textsubscript{10} exposure metrics, the most certain effects were for the morning exposure ($p = 0.007$) and nighttime exposure ($p = 0.012$), and least for the 1 h max ($p = 0.049$). To compare effects across the exposure metrics, we also provided the percentage change in risk of mortality per IQR increase in PM\textsubscript{10} for each exposure metric by lag in supplementary table 3 (available at stacks.iop.org/ERL/8/014015/mmedia). We found similar results with table 3.

Tables 4 and 5 show the percentage change in risk of cardiovascular and respiratory mortality per 10 µg m\textsuperscript{-3} increase in PM\textsubscript{10} for each exposure metric by lag, respectively. Supplementary tables 4 and 5 (available at...
Table 4. Effects of different PM$_{10}$ exposure metrics and lags on cardiovascular mortality, for a 10 $\mu$g m$^{-3}$ increase in PM$_{10}$ (95% confidence intervals). (Note: significant findings are shaded. For each exposure, the most certain effect is shown in bold font. The effect estimates shown in this table are for a 10 $\mu$g m$^{-3}$ increase for all exposure metrics; however, a 10 $\mu$g m$^{-3}$ increase is not equivalent across all exposure metrics. For example, a 10 $\mu$g m$^{-3}$ increase in the 24 h exposure corresponds to approximately a 15 $\mu$g m$^{-3}$ increase in the daily 1 h maximum.)

| Lag  | 24 h exposure | Daytime (7 am–8 pm) | Morning (7 am–10 am) | Nighttime (8 pm–7 am) | 1 h maximum |
|------|---------------|---------------------|----------------------|-----------------------|-------------|
| Lag0 | 0.15 (−0.00, 0.31) | 0.12 (−0.03, 0.27) | **0.18 (0.04, 0.32)** | 0.16 (0.02, 0.31) | 0.07 (−0.05, 0.20) |
| Lag1 | 0.16 (0.01, 0.31) | 0.11 (−0.04, 0.26) | 0.11 (−0.03, 0.25) | 0.20 (0.05, 0.34) | **0.11 (−0.01, 0.24)** |
| Lag2 | 0.04 (−0.12, 0.19) | 0.05 (−0.10, 0.20) | 0.03 (−0.11, 0.17) | 0.02 (−0.13, 0.17) | 0.03 (−0.09, 0.16) |
| Lag3 | −0.02 (−0.17, 0.13) | 0.01 (−0.14, 0.16) | −0.05 (−0.19, 0.09) | −0.05 (−0.20, 0.10) | −0.02 (−0.15, 0.10) |
| Lag01 | **0.22 (0.03, 0.40)** | **0.17 (−0.01, 0.36)** | 0.21 (0.04, 0.38) | **0.25 (0.07, 0.42)** | 0.13 (−0.02, 0.27) |
| Lag02 | 0.21 (0.00, 0.42) | 0.18 (−0.03, 0.40) | 0.21 (0.01, 0.41) | 0.23 (0.03, 0.43) | 0.13 (−0.04, 0.31) |
| Lag03 | 0.19 (−0.05, 0.43) | 0.18 (−0.06, 0.42) | 0.17 (−0.06, 0.39) | 0.18 (−0.04, 0.41) | 0.12 (−0.08, 0.31) |

Table 5. Effects of different PM$_{10}$ exposure metrics and lags on respiratory mortality, for a 10 $\mu$g m$^{-3}$ increase in PM$_{10}$ (95% confidence intervals). (Note: for each exposure, the most certain effect is shown in bold font. The effect estimates shown in this table are for a 10 $\mu$g m$^{-3}$ increase for all exposure metrics; however, a 10 $\mu$g m$^{-3}$ increase is not equivalent across all exposure metrics. For example, a 10 $\mu$g m$^{-3}$ increase in the 24 h exposure corresponds to approximately a 15 $\mu$g m$^{-3}$ increase in the daily 1 h maximum.)

| Lag  | 24 h exposure | Daytime (7 am–8 pm) | Morning (7 am–10 am) | Nighttime (8 pm–7 am) | 1 h maximum |
|------|---------------|---------------------|----------------------|-----------------------|-------------|
| Lag0 | 0.04 (−0.29, 0.37) | 0.04 (−0.28, 0.35) | 0.09 (−0.20, 0.38) | 0.04 (−0.27, 0.36) | −0.04 (−0.30, 0.23) |
| Lag1 | 0.06 (−0.26, 0.39) | 0.02 (−0.29, 0.34) | −0.03 (−0.32, 0.26) | 0.10 (−0.21, 0.41) | **0.10 (−0.15, 0.36)** |
| Lag2 | **0.16 (−0.15, 0.48)** | **0.15 (−0.15, 0.45)** | 0.15 (−0.14, 0.43) | **0.16 (−0.14, 0.46)** | 0.08 (−0.17, 0.34) |
| Lag3 | 0.14 (−0.17, 0.45) | 0.14 (−0.16, 0.45) | 0.20 (−0.08, 0.48) | 0.12 (−0.18, 0.42) | 0.09 (−0.16, 0.34) |
| Lag01 | 0.07 (−0.31, 0.46) | 0.04 (−0.34, 0.43) | 0.05 (−0.31, 0.40) | 0.10 (−0.26, 0.46) | 0.05 (−0.26, 0.35) |
| Lag02 | 0.17 (−0.27, 0.61) | 0.14 (−0.30, 0.59) | 0.15 (−0.27, 0.56) | 0.19 (−0.23, 0.60) | 0.09 (−0.26, 0.44) |
| Lag03 | 0.24 (−0.24, 0.73) | 0.23 (−0.26, 0.73) | 0.27 (−0.19, 0.74) | 0.25 (−0.22, 0.71) | 0.14 (−0.25, 0.54) |

stacks.iop.org/ERL/8/014015/mmedia) show results based on an increment of an IQR. Statistically significant associations between cardiovascular mortality risk and PM$_{10}$ were observed for 24 h, morning, and nighttime exposure metrics. We did not find a significant association between respiratory mortality and PM$_{10}$ for any exposure metrics.

Because the daily pattern of PM$_{10}$ differs somewhat by season (figure 1), we performed sensitivity analysis to estimate the effects of different PM$_{10}$ exposure metrics of lag 0–3 days on mortality by season (supplementary table 6 available at stacks.iop.org/ERL/8/014015/mmedia). All central estimates were positive except for nighttime exposure in summer and all exposure metrics in winter. The effect estimates in autumn were higher than other seasons, except for the 1 h max, which has the highest effect in spring, although confidence intervals for all seasons overlapped for all exposure metrics.

4. Discussion

In this study, we investigated associations of PM$_{10}$ with daily mortality in Seoul, Korea using several sub-daily exposure metrics. We found that PM$_{10}$ was associated with daily total mortality for all exposure metrics (24 h exposure, daytime exposure, morning exposure, nighttime exposure, and 1 h maximum) and with cardiovascular mortality for 24 h, morning, and nighttime exposure metrics, with little difference in the magnitude of risk, but some difference in the lag for the most certain result. We did not find evidence that any sub-daily exposure metrics are more associated with mortality than the 24 h metric. To the best of our knowledge, this is the first study to report particle effects on daily mortality from shorter time intervals (e.g., one to several hours) of PM$_{10}$ compared with the standard 24 h metric. Although this study provides information on sub-daily PM$_{10}$ exposure metrics for mortality, many research questions remain.

The PM exposure metric most associated with health may vary by health outcome due to different physiological responses. Possible biological mechanisms of particle effects on mortality include alveolar inflammation causing acute changes in blood coagulability, impairment of lung defenses, and physiological disturbances of gas transfer [29, 30]. Previous studies suggest that shorter-term PM may be more informative in explaining findings of adverse particle effects on acute health response than the 24 h average. Based on biological plausibility, cardiovascular disease presents an acute response to trigger agents [31]. On the other hand, respiratory disease (e.g., pneumonia, chronic disease) has a more delayed effect because of slower progression [32]. Gold et al [16] observed associations with sub-daily averaging periods of 4 h for PM$_{2.5}$ on reduced heart rate variability. On the other hand, another US study found no differences in associations with forced expiratory volume in one second (FEV$_1$) by averaging time (1, 8 and 24 h average) for personal PM in the 0.1–10 $\mu$m range [33]. Our study investigated
total non-accidental, cardiovascular, and respiratory mortality, but future work could explore whether the most appropriate short-term exposure metric differs by cause of mortality or other cause-specific health endpoints.

Our findings on PM exposure metrics are not generalizable to other pollutants. Darrow et al. [20] examined the relationships between air pollutants (PM2.5, CO, NO2, and O3) and respiratory emergency department visits using various temporal metrics that included the daily 1 h maximum, 24 h, nighttime average. They reported that different averaging times had little effect on effect estimates for PM, however, results for other pollutants were highly sensitive to the exposure metric used. Further study is needed to examine associations between health outcomes and sub-daily exposure of other pollutants. Further, additional work is warranted on other size fractions of PM, such as PM2.5, and specific chemical components and sources of particles. The chemical structure of particles varies by location and season [34]. For example, recent work found that the chemical composition of PM2.5 in Seoul is more similar to that of the western US than the eastern US [35].

Previous short-term exposure studies provide no evidence of a clear threshold level in relationship between PM and mortality, in terms of 24 h average concentrations. Several findings indicate that linear models without a threshold are appropriate for assessing the effect of particulate air pollution on mortality [36, 37]. However, future work could examine the existence and level of thresholds that may exist and aim to reduce the uncertainty in effect estimates in relation to different exposure metrics.

The availability of hourly data allowed us to estimate exposure metrics shorter than a 24 h average, providing a distinct advantage over many other commonly used datasets. However, the health data of the number of mortalities are daily. A more sophisticated analysis of sub-daily exposure would have temporally aligned data for both exposure and health outcomes. Whereas our research benefited from the availability of sub-daily pollution data, sub-daily health data are also needed (e.g., hour of death rather than day of death). Thus, future research is still needed to investigate effects of various short-term exposure metrics with datasets that allow incorporation of the onset of the health outcome (i.e., time of death rather than day of death).

We performed sensitivity analyses with respect to season and lag structure although other uncertainties still exist that cannot be quantified or evaluated by this research. These include the impact of sub-daily exposure metrics on other health outcomes, such as hospital admissions, or cause-specific mortality. The importance of sub-daily exposure metrics may differ by health outcome. We used a PM size distribution of PM10 as PM2.5 measurements were not available for this region; however, future studies could investigate these research questions for other size fractions of particles, such as PM2.5. The size fraction of particles is relevant to human health, yet particulate matter is a complex mixture of multiple chemical components from various sources [35]. These issues are also relevant to sub-daily exposure metrics as different formulations of particles may have different health impacts and therefore relevant exposure timeframes. Finally, the biological mechanisms through which various types of particles affect health in different exposure timeframes are not fully understood.

Our findings that PM10 is associated with mortality are consistent with other studies conducted with various populations. For example, a study in Delhi reported that daily PM10 was associated with increased the rate of non-accidental mortality [6]. Qian et al. [38] examining the association of daily mortality with ambient air pollution in China, observed that a 10 µg m−3 increase in daily PM10 was associated with a 0.43% (95% CI, 0.24–0.62%) increase in mortality due to all natural causes. A study in Italy observed that a 10 µg m−3 increase in daily PM10 was associated with a 0.75% (95% CI, 0.42–1.09%) increase in mortality due to all natural causes among subjects age 65 years and older [39].

Many PM studies have been unable to study effects of multiple days of exposure due to the lack of daily data. We observed that for all PM10 exposure metrics, associations were higher when multiday lags were used rather than a single day lag, confirming the evidence reported in previous studies. Some studies have demonstrated that multiday averages of pollution are found to be better predictors of daily mortality than a single day’s exposure [40, 41]. A study by Zeka et al. [42] in the US suggested that analyses using a one day lag underestimated the effects of PM10 for all cause, heart, and respiratory mortality.

We did not find a clear seasonal pattern of daily mortality effects for any PM10 exposure metric. Most central estimates were higher in autumn, however, confidence intervals of effects for all seasons overlapped for all exposure metrics. Previous findings of seasonal variation of daily mortality effects of air pollution are inconsistent. Qian et al. [43] examined seasonal patterns of associations between daily mortality and PM10 in China. They found that the strongest effects occurred in winter for all natural mortality, with a 0.69% increase (95% CI: 0.44–0.94%) for winter, 0.34% (95% CI: 0.00–0.69%) for spring, 0.45% (95% CI: −0.13–1.04%) for summer, and −0.21% (95% CI: −0.54–0.12%) for fall for a 10 µg m−3 increase in daily PM10. Peng et al. [44] in the US found that a 10 µg m−3 increase in daily PM10 was associated with a 0.15% (95% PI: −0.08–0.39%), 0.14% (−0.14–0.42%), 0.36% (0.11–0.61%), and 0.14% (−0.06–0.34%) increase in mortality for winter, spring, summer, and autumn, respectively. Another study in Korea, examining seasonal effects of PM10 on non-accidental mortality reported that the association increased during the summer compared to other times of the year [45]. Seasonal differences in the short-term effects of PM10 may be related with several factors such as differences of particulate matter compositions or variations in behavioral and individual activity patterns (e.g., time spent outdoors, ventilation, air conditioning) [44].

5. Conclusions
This study contributes to the literature on the evidence of daily mortality with different short-term exposure metrics.
of PM$_{10}$. As the US EPA has noted, there is a paucity of scientific information on health impacts from sub-daily PM exposures [11, 12]. In conclusion, we identified similar associations between different sub-daily exposure metrics of PM$_{10}$, and did not find evidence that one metric is preferable to another for mortality associations. These findings indicate that the use of a 24 h exposure metric for PM$_{10}$ is appropriate for mortality studies, and support the use of a 24 h averaging time for regulatory standards, although they do not exclude the possibility that other exposure metrics are better suited to other health endpoints.

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References

[1] Franklin M, Zeka A and Schwartz J 2007 Association between PM$_{2.5}$ and all-cause and specific-cause mortality in 27 US communities J. Expo. Sci. Environ. Epidemiol. 17 279–87
[2] Pope C A III and Dockery D W 2006 Health effects of fine particulate air pollution: lines that connect J. Air Waste Manag. Assoc. 56 709–42
[3] Schwartz J et al 1996 Methodological issues in studies of air pollution and daily counts of deaths or hospital admissions J. Epidemiol. Community Health 50 (Suppl. 1) S3–11
[4] Guaita R, Fichiule M, Máté T, Linares C and Díaz J 2011 Short-term impact of particulate matter (PM$_{2.5}$) on respiratory mortality in Madrid Int. J. Environ. Health Res. 21 260–74
[5] Ito K, Mathes R, Ross Z, Nadas A, Thurston G and Matte T 2011 Fine particulate matter constituents associated with cardiovascular hospitalizations and mortality in New York City Environ. Health Perspect. 119 467–73
[6] Rajaratnam U, Sehgal M, Nairy S, Patnayak R C, Chhabra S K, Kilani, Ragavan K V and HEI Health Review Committee 2011 Time-series study on air pollution and mortality in Delhi Res. Rep. Health Eff. Inst. 157 47–74
[7] US EPA 2005 Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information (Research Triangle Park, NC: US Environmental Protection Agency, Office of Air Quality Planning and Standards)
[8] US EPA 2006 National ambient air quality standards for particulate matter 40 CFR Part 50 Federal Register 71 61144–233
[9] World Health Organization 2006 WHO Air Quality Guidelines for Particulate Matter, Ozone, Nitrogen Dioxide and Sulfur Dioxide: Global Update 2005 (Copenhagen: WHO)
[10] MOE (Ministry of Environment, Korea) 2011 Annual Report of Air Quality in Korea 2010 (available at http://library.me.go.kr/search/DetailViewPopup.ax?cid=5506169, accessed 14 December 2011)
[11] US EPA 2009 Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter EPA 452-R-08-004 (Research Triangle Park, NC: US Environmental Protection Agency)
[12] US EPA 2009 Integrated Science Assessment for Particulate Matter EPA/600/R-08/139F (Research Triangle Park, NC: US Environmental Protection Agency)
[13] Gent J F, Triche E W, Holford T R, Belanger K, Bracken M B, Beckett W S and Leaderer B P 2003 Association of low-level ozone and fine particles with respiratory symptoms in children with asthma J. Am. Med. Assoc. 290 1859–67
[14] Burgan O, Smargiassi A, Perron S and Kosatsky T 2010 Cardiovascular effects of sub-daily levels of ambient fine particles: a systematic review Environ. Health 9 26–41
[15] Deltino R J, Zeiger R S, Seltzer J M and Street D H 1998 Symptoms in pediatric asthmatics and air pollution: differences in effects by symptom severity, anti-inflammatory medication use and particulate averaging time Environ. Health Perspect. 106 751–61
[16] Gold D R, Litonjua A, Schwartz J, Lovett E, Larson A, Nearing B, Allen G, Verrier M, Cherry R and Verrier R 2000 Ambient pollution and heart rate variability Circulation 101 1267–73
[17] Magari S R, Schwartz J, Williams P L, Hauser R, Smith T J and Christiani D C 2002 The association between personal measurements of environmental exposure to particulates and heart rate variability Epidemiology 13 305–10
[18] Peters A, Dockery D W, Muller J E and Mittleman M A 2001 Increased particulate air pollution and the triggering of myocardial infarction Circulation 103 2810–5
[19] Bhaskaran K, Hajat S, Armstrong B, Haines A, Herrett E, Wilkinson P and Smeeth L 2011 The effects of hourly differences in air pollution on the risk of myocardial infarction: case crossover analysis of the MINAP database BMJ 343 d5531
[20] Darrow L A, Klein M, Sarnat J A, Mulholland J A, Strickland J M, Sarnat S E, Russell A G and Tolbert P E 2011 The use of alternative pollutant metrics in time-series studies of ambient air pollution and respiratory emergency department visits J. Expo. Sci. Environ. Epidemiol. 21 10–9
[21] Ostro B, Lipsett M, Mann J, Braxton-Owens H and White M 2001 Air pollution and exacerbation of asthma in African–American children in Los Angeles Epidemiology 12 200–8
[22] Ostro B, Broadwin R, Green S, Feng W Y and Lipsett M 2006 Fine particulate air pollution and mortality in nine California counties: results from CALFINE Environ. Health Perspect. 114 29–33
[23] Bell M L, Samet J M and Dominici F 2004 Time-series studies of particulate matter Ann. Rev. Public Health 25 247–80
[24] Curtis L, Rea W, Smith-Willis P, Fenyes E and Pan Y 2006 Adverse health effects of outdoor air pollutants Environ. Int. 32 815–30
[25] Pope C A III, Brook R D, Burnett R T and Dockery W D 2011 How is cardiovascular disease mortality risk affected by duration and intensity of fine particulate matter exposure? An integration of the epidemiologic evidence Air Qual. Atmos. Health 4 5–14
[26] Künzli N et al 2000 Public health impact of outdoor and traffic-related air pollution: a European assessment Lancet 356 795–801
[27] Muller N Z and Mendelsohn R 2007 Measuring the damages of air pollution in the United States J. Environ. Econ. Manage. 54 1–14
[28] Vlachokostas C, Achillas C, Moussiopoulos N, Kalogeropoulos K, Sigalas G, Kalognomou E A and Banias G 2012 Health effects and social costs of particulate air pollution: a case study for Thessaloniki, Greece Air Qual. Atmos. Health 5 325–34
[29] Katsouyannni K et al 1997 Short term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: results from time series data from the APHEA project BMJ 314 1658–63
[30] Seaton A, MacNee W, Donaldson K and Godden D 1995 Particulate air pollution and acute health effects Lancet 345 176–8
[31] Peters A et al 2000 Air pollution and incidences of cardiac arrhythmia Epidemiology 11 11–7
[32] Zanobetti A et al 2003 The temporal pattern of respiratory and heart disease mortality in response to air pollution Environ. Health Perspect. 111 1188–93
[33] Delfino R J, Quintana P J E, Floro J, Gastañaga V M, Samimi B S, Kleinman M T, Liu L J S, Bufalino C, Wu C F and McLaren C E 2004 Association of FEV₁ in asthmatic children with personal and microenvironmental exposure to airborne particulate matter Environ. Health Perspect. 112 932–41
[34] Bell M L, Dominici F, Ebisu K, Zeger S L and Samet J M 2007 Spatial and temporal variation in PM₂.₅ chemical composition in the United States for health effects studies Environ. Health Perspect. 115 989–95
[35] Son J Y, Lee J T, Kim K H, Jung K and Bell M L 2012 Characterization of fine particulate matter and associations between particulate chemical constituents and mortality in Seoul, Korea Environ. Health Perspect. 120 872–8
[36] Daniels M J, Dominici F, Samet J M and Zeger S L 2000 Estimating particulate matter–mortality dose–response curves and threshold levels: an analysis of daily time-series for the 20 largest US cities Am. J. Epidemiol. 152 397–406
[37] Hong Y C, Leem J H, Ha E H and Christiansi D C 1999 PM₁₀ exposure, gaseous pollutants, and daily mortality in Inchon, South Korea Environ. Health Perspect. 107 873–8
[38] Qian Z et al 2010 Part 2. Association of daily mortality with ambient air pollution, and effect modification by extremely high temperature in Wuhan, China Res. Rep. Health Eff. Inst. 154 91–217
[39] Forastiere F et al 2008 Particulate matter and daily mortality: a case-crossover analysis of individual effect modifiers Epidemiology 19 571–80
[40] Kelsall J E, Samet J M, Zeger S L and Xu J 1997 Air pollution mortality in Philadelphia: 1974–1988 Am. J. Epidemiol. 146 750–62
[41] Schwartz J and Dockery D W 1992 Increased mortality in Philadelphia associated with daily air pollution concentrations Am. Rev. Respir. Dis. 145 600–4
[42] Zeka A, Zanobetti A and Schwartz J 2005 Short term effects of particulate matter on cause specific mortality: effects of lags and modification by city characteristics Occup. Environ. Med. 62 718–25
[43] Qian Z et al 2010 Seasonal pattern of the acute mortality effects of air pollution J. Air Waste Manag. Assoc. 60 481–8
[44] Peng R D, Dominici F, Pastor-Barriuso R, Zeger S L and Samet J M 2005 Seasonal analyses of air pollution and mortality in 100 US cities Am. J. Epidemiol. 161 585–94
[45] Yi O, Hong Y C and Kim H 2010 Seasonal effect of PM₁₀ concentrations on mortality and morbidity in Seoul, Korea: a temperature-matched case–crossover analysis Environ. Res. 110 89–95