Utilizing daily excessive concentration hours to estimate cardiovascular mortality and years of life lost attributable to fine particulate matter in Tehran, Iran

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1. Introduction

The Global Burden of Disease Study (GBD) estimated that cardiovascular diseases (CVDs) caused 17.8 million deaths in 2017, making it the primary mortality risk factor globally (GBD Collaborators, 2018). It was estimated that the incidence and the case fatality rates of major cardiovascular events were higher in low- and middle-income countries than those in high-income countries (Finegold et al., 2013; Yusuf et al., 2014). Specifically, growing epidemiological and clinical evidence indicated potential deleterious effects of environmental factors (e.g., temperature, noise and air pollution) on cardiovascular health (Babich et al., 2005; Brook et al., 2010; Lin et al., 2009; Ran et al., 2018). Ambient fine particulate matter, or particles with aerodynamic diameter less than 2.5 μm (PM$_{2.5}$), is one of the principal air pollutants that has been linked to increased risk of cardiovascular morbidity and mortality (Chen et al., 2017b; Dominici et al., 2006; Pope et al., 2006).

Numerous epidemiological research from the United States and Europe have established the associations between long- and short-term exposure to PM$_{2.5}$ and a variety of acute and chronic cardiovascular consequences such as increased blood pressure, heart rate fluctuations, ischemic heart disease and stroke (Crouse et al., 2012; Kettunen et al., 2007; Pope et al., 2004; Zanobetti and Schwartz, 2005).

In recent years, Iran has suffered a great CVD mortality burden attributable to PM$_{2.5}$ over the past decade due to rapid industrial development, population explosion and desert dust storms in Western Asia (Faridi et al., 2018; Khiani-Badi et al., 2019). However, available evidence from Iran regarding PM$_{2.5}$-CVD mortality relationships is still relatively limited, and age- and gender-specific findings remain inconsistent (Kermani et al., 2018; Varamrani et al., 2018). Besides, most previous studies in Iran investigating PM$_{2.5}$-CVD health relationships used the death counts as the endpoint indicator, which overlooked differences in the life lost from different age groups (Karimi et al., 2019; Khajavi et al., 2019; Mirti et al., 2016). In addition to mortality, years of life lost (YLL) is an important complementary index that has been extensively used to evaluate the disease burden locally, regionally, and globally (Fitzmaurice et al., 2017; Fontaine et al., 2003; Huang et al., 2012).

YLL takes into consideration the premature deaths and the life expectancy by giving different weights to deaths at different ages (Guo et al., 2013; Liang et al., 2018). In comparison with mortality, YLL is a more sensitive and informative indicator in quantifying premature deaths since it partially resolves the disease effects caused by death numbers alone (Zhang et al., 2017; Zhu et al., 2017). Yet, there have been very sparse studies investigating PM$_{2.5}$-YLL relationships in Iran so far, though YLL contributes valuable age-based perspective for public policy making and health resource allocation (Faridi et al., 2018).

Using a time-series design, we applied generalized linear models to assess the attributable effects of PM$_{2.5}$-DECH and PM$_{2.5}\text{mean}$ on CVD mortality and YLL.

Results: For an interquartile range (IQR) rise in PM$_{2.5}$-DECH, total CVD mortality at lag 0–10 days and YLL at lag 0–8 days increased 2.26% (95% confidence interval (CI): 0.85–3.69%) and 23.24 (6.07–40.42) person years, respectively. Corresponding increases were 3.45% (1.44–5.49%) and 35.21 (10.85–59.58) person years for an IQR rise in PM$_{2.5}\text{mean}$. Significant associations between PM$_{2.5}$ pollution (i.e., PM$_{2.5}\text{mean}$ and PM$_{2.5}$-DECH) and cause-specific cardiovascular health (i.e., mortality and YLL) were only identified in stroke. Subgroup analyses showed that male and people aged 0–64 years suffered more from PM$_{2.5}$ pollution. Furthermore, we attributed a greater CVD burden to PM$_{2.5}$-DECH (1.67% for mortality and 2.67% for YLL) than PM$_{2.5}\text{mean}$ (0.63% for mortality and 0.70% for YLL) during the study period.

Conclusions: This study strengthened the evidence for the aggravated CVD mortality burden associated with short-term exposure to PM$_{2.5}$. Our findings also suggested that PM$_{2.5}$-DECH might be a potential alternative indicator of exposure assessment in PM$_{2.5}$-related health investigations.

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emissions, dust storms and natural gas. Residents of Tehran suffered from high concentrations of ambient air pollution especially in cold seasons that schools and government offices were closed to avoid potential health risks (Faridi et al., 2018).

2.2. Data collection

Daily mortality data of Tehran from January 1, 2012, to December 31, 2016, were obtained from the Municipality Tehran (Behesht-e-Zahra organization) (http://www.tehran.ir). According to the International Classification of Diseases (ICD–10), causes of mortality were coded as follows: cardiovascular disease (ICD–10: I00–I99), ischemic heart disease (IHD, I20–I25), stroke (I60–I69) and sudden cardiac death (SCD, I46). Cardiovascular mortality data were further classified by gender (male and female) and age (0–64 years, 65–74 years, 75+ years).

Air pollution data including PM$_{2.5}$, carbon monoxide (CO), ozone (O$_3$), nitrogen dioxide (NO$_2$), and sulfur dioxide (SO$_2$) were collected from Tehran air quality control website (http://airnow.tehran.ir). These items are measured hourly by 22 stations located in different parts of Tehran, and their daily averages were used in subsequent analyses. Daily meteorological data were collected from IRAN Meteorological Organization (IRIMO) (http://www.irimo.ir), including daily average temperature (<C), relative humidity (%), wind speed (m/s), atmospheric pressure (hPa), precipitation (mm) and sunshine hours (h).

2.3. Calculations of YLL and DECH

Life tables for Iran population from 2012 to 2016 were obtained from the World Health Organization’s (WHO) Global Health Observatory (GHO) data, which provided life expectancies at every exact age for both sexes. YLL for each death was calculated by matching age and sex to the life tables, and daily YLL were estimated as the sum for all deaths on the given day (Guo et al., 2013; Zhang et al., 2017). Furthermore, we stratified daily YLL by gender (male, female), age (0–64 years, 65–74 years, 75+ years) and cardiovascular mortality categories (IHD, stroke, SCD). More details are available in our previous publications (Jiao et al., 2019; Zhang et al., 2018).

We adopted DECH as the sum of concentration-hours >35 g/m$^3$ within one day. 35 μg/m$^3$ was chosen as the concentration threshold based on the Clean Air Standards in Iran. For instance, an hour with the concentration of 38 μg/m$^3$ contributed 3 concentration-hours to the DECH values, while hours with concentrations lower than or equal to 35 μg/m$^3$ contributed zero extra concentration-hours to the daily total. DECH is calculated by the formula:

$$\text{DECH} = \sum_{i=0}^{23} \Delta c_i$$

$$\Delta c_i = \begin{cases} c_i - 35, & c_i \geq 35 \\ 0, & c_i < 35 \end{cases}$$

where i is the hour time of observation; $c_i$ is the concentration of PM$_{2.5}$ at the hour of observation; $\Delta c_i$ is the difference between $c_i$ and concentration threshold of 35 μg/m$^3$.

2.4. Statistical analysis

A standard time-series generalized model (GLM) was applied to evaluate the effects of PM$_{2.5}$ on CVD mortality and YLL. Consistent with previous research, daily CVD deaths followed an over-dispersed Poisson distribution, and daily cardiovascular YLL followed a normal distribution (Liang et al., 2018; Lu et al., 2015). Several covariates were adjusted in GLM: (1) a natural cubic smooth function of time with 7 degrees of freedom (df) per year to control for secular trend and seasonality (Chen et al., 2017b); (2) natural cubic smooth (ns) functions with 6 df for the current day’s temperature and moving average of 3 days’ mean temperature (Lin et al., 2017a); (3) ns functions with 3 df for more meteorological factors such as mean relative humidity (RH), wind speed (WS), sunlight hours (SH), precipitation (Pp) and atmospheric pressure (AP) (Chen et al., 2017a); (4) indicator variable for “day of the week (DOW)” (Guo et al., 2011). The GLM model is expressed as:

$$\log(E(\mu_{ij})) = \alpha + \beta_1 x_1 + \text{ns(Time, df} = 7 \times 5) + \text{ns(Temp, df} = 6) + \text{ns(Temp}_{1,3}, \text{df} = 6) + \text{ns(RH, df} = 3) + \text{ns(WS, df} = 3) + \text{ns(AP, df} = 3) + \text{ns(SH, df} = 3) + \text{ns(Pp, df} = 3) + \gamma \text{DOW}_i$$

where $\gamma$ refers to the natural cubic splines; $\beta$ and $\gamma$ are the regression coefficients, and $\chi$ is the intercept.

To estimate potential harvesting and cumulative effects of PM$_{2.5}$mean and PM$_{2.5}$DECH, we first used a single-day lag (current day up to the previous 10 days: lag 0–lag 10). As single-day lag models may underestimate the health impacts of PM$_{2.5}$ (Bell et al., 2004), we further applied moving averages of lag 0–1 to lag 0–10 days to perform analyses. To identify vulnerable sub-populations, subgroup analyses were conducted stratified by gender (male, female), age group (0–64 years, 65–74 years, 75+ years) and cardiovascular mortality categories (IHD, stroke, SCD). We then visually checked the linearity of exposure-response curves between PM$_{2.5}$ pollution (i.e., PM$_{2.5}$mean and PM$_{2.5}$DECH) and CVD health (i.e., mortality and YLL) by applying smoothing functions of the natural cubic spline with 3 df (Smargiassi et al., 2009).

We further quantified the CVD mortality burden (i.e., deaths and YLLs) attributable to PM$_{2.5}$DECH and PM$_{2.5}$mean, respectively (Lin et al., 2016; Lin et al., 2018a). Two indicators, attributable number (AN) and attributable fraction (AF) were applied. First, we calculated attributable number of CVD deaths and YLLs caused by PM$_{2.5}$mean (reference = 35 μg/m$^3$) and PM$_{2.5}$DECH (reference = 0 μg/m$^3$) hours) on each day of the whole series, namely AN, of deaths and YLLs. By summing up AN during the whole series, total attributable deaths and YLLs (AN$_{total}$) can be obtained. Attributable fractions (AF, %) of deaths and YLLs could be provided by the ratios of AN$_{total}$ to the corresponding total number of observed deaths or YLLs.

To test the robustness of our models, we performed several sensitivity analyses by including gaseous pollutants (SO$_2$, NO$_2$, CO and O$_3$) to establish two-pollutant models, and using alternative dfs for smoothing functions of temporal trends (5–9 df/year) and the weather factors (5 and 7 df for temperature and 4 and 5 df for relative humidity).

All analyses were performed using R software (version 3.3.3). The results for PM$_{2.5}$-CVD associations were expressed as the percent increase in daily mortality (%) and absolute changes in YLL (person years) associated with per interquartile range (IQR) increment of PM$_{2.5}$mean and PM$_{2.5}$DECH. The confidence intervals (CIs) were reported at the 95% confidence level. Two-sided statistical tests were conducted, and effects of p < 0.05 were considered statistically significant.
3. Results

3.1. Data description

Table 1 summarizes the statistical characteristics of daily CVD deaths and YLLs. A total of 106,180 CVD deaths were recorded during the study period, of which 48.71% aged 75+ years and 56.45% were male. On each day, approximately 58, 10, 18 and 16 deaths were from CVD, stroke, IHD and SCD. Daily mean YLL was 851.3 person years, more than two-thirds of which were contributed by the youngest group (age 0–64 years). The temporal patterns of daily air pollutants and meteorological factors in Tehran, Iran, 2012–2016 were shown in Fig. 1. Time series plots of daily CVD mortality and YLL in the corresponding period were presented in Fig. S1. The overall PM$_{2.5}$mean and PM$_{2.5}$DECH shared similar periodic fluctuation annually, both of which showed an intuitive seasonal pattern with values higher in winter and lower in summer. We observed an increasing trend for daily SO$_{2}$, while a decreasing trend for NO$_{2}$ during the study period. Summary characteristics of daily air pollutants and meteorological variables were presented in Table S1. Annual average PM$_{2.5}$mean and PM$_{2.5}$DECH were 34.7 µg/m$^3$ and 172.4 µg/m$^3$-hour, respectively (Table S1). Spearman correlation coefficients between air pollutants and meteorological variables were shown in Table S2.

3.2. Associations between air pollution and CVD mortality and YLL

Fig. 2 reveals the exposure-response relationships between PM$_{2.5}$mean, PM$_{2.5}$DECH and CVD mortality, YLL, adjusting for confounders in the time-series analysis. Approximately linear associations between PM$_{2.5}$mean, PM$_{2.5}$DECH and CVD mortality, YLL were observed in the current study. Therefore, we subsequently estimated the effects on mortality and YLL associated with per IQR increase in PM$_{2.5}$mean (IQR = 18.2 µg/m$^3$) and PM$_{2.5}$DECH (IQR = 216.2 µg/m$^3$-hour).

Fig. 3 illustrates the effects of PM$_{2.5}$mean and PM$_{2.5}$DECH on CVD mortality and YLL across different lag days. The lag pattern was generally similar for CVD mortality and YLL. For single lag days, the associations appeared at lag 1 with the most significant effect, and lasted for two or three days with a decreasing trend. For multiple lag days, the effects of PM$_{2.5}$mean and PM$_{2.5}$DECH on CVD mortality gradually increased from lag 0–1 to lag 0–10 days, with PM$_{2.5}$mean and PM$_{2.5}$DECH along lag 0–10 days found to be most strongly associated with CVD mortality. The corresponding effects of PM$_{2.5}$ on CVD YLL showed an upward trend from lag 0–1 to lag 0–8 days, with the highest effect estimates at lag 0–8 days. Thus, we separately selected lag 0–10 days for CVD mortality, and lag 0–8 days for CVD YLL as the exposure periods to report the subsequent results. Specifically, an IQR increase in PM$_{2.5}$mean was associated with 3.45% (95% CI: 1.44–5.48%) increase in CVD mortality at lag 0–10 days, and 35.21% (95% CI: 10.85–59.58) person years increase in YLL at lag 0–8 days. Corresponding increases for an IQR increase in PM$_{2.5}$DECH were 2.26% (95% CI: 0.85–3.69%) and 23.24 (95% CI: 6.07–42.42) person years (Table 2). Estimated changes of CVD mortality and YLL associated with PM$_{2.5}$mean and PM$_{2.5}$DECH at different exposure days were also shown in Table S3.

3.3. Subgroup analyses

Table 2 compares the effects of per IQR increase in PM$_{2.5}$mean and PM$_{2.5}$DECH on CVD mortality (lag 0–10 days) and YLL (lag 0–8 days), stratified by gender, age and the specific cause of CVD mortality. Stratified by gender and cause of CVD mortality, statistically significant associations between PM$_{2.5}$ pollution (i.e., PM$_{2.5}$mean and PM$_{2.5}$DECH) and cardiovascular health (i.e., mortality and YLL) were only identified in male and stroke cases. For example, the ERRs of CVD mortality per IQR increment in PM$_{2.5}$mean and PM$_{2.5}$DECH for stroke was 8.30% (95% CI: 3.65–13.16%) and 5.50% (95% CI: 2.31–8.79%), and for male was 4.68% (95% CI: 1.99–7.45%) and 2.93% (95% CI: 1.04–4.85%). For both CVD mortality and YLL, we observed stronger cardiovascular effects in people aged 0–64 years compared with those aged 75+, while no significant effects of PM$_{2.5}$mean and PM$_{2.5}$DECH on people aged 65–74 years. For example, the ERRs of CVD mortality per IQR increment in PM$_{2.5}$mean and PM$_{2.5}$DECH for people aged 0–64 years was 4.16% (95% CI: 0.62–7.81%) and 2.79% (95% CI: 0.31–5.33%), and for people aged 75+ years was 3.53% (95% CI: 0.74–6.39%) and 2.38% (95% CI: 0.42–4.37%).

3.4. Mortality burden attributable to PM$_{2.5}$mean and PM$_{2.5}$DECH

Table 3 estimates the yearly AN and overall AF for CVD mortality at lag 0–10 days, and YLL at lag 0–8 days associated with PM$_{2.5}$mean and PM$_{2.5}$DECH. Generally, we observed a relatively greater CVD mortality burden attributable to PM$_{2.5}$DECH than PM$_{2.5}$mean. During the study period, approximately 0.63% (95% CI: 0.28–0.98%) CVD deaths (134 cases per year) and corresponding 0.70% (95% CI: 0.23–1.18%) YLLs (2440 person years per year) were attributable to PM$_{2.5}$mean, 1.67% (95% CI: 0.70–2.63%) CVD deaths (355 cases per year) and corresponding 2.67% (95% CI: 0.86–4.48%) YLLs (9280 person years per year) were ascribed to PM$_{2.5}$DECH. People aged 0–64 years contributed more to yearly YLLs (2050 person years) than people aged 75+ (322 person years), suggesting that people dying at younger ages caused greater mortality burden.
3. Sensitivity analyses

Sensitivity analyses showed that the estimated associations of CVD mortality and YLL with exposure to PM$_{2.5}$DECH than PM$_{2.5}$-mean changed a little when adopting two-pollutant models (Tables S4 & S5). Also, PM$_{2.5}$-associated estimates were generally stable in our study regarding gender and age-specific analyses in the two-pollutant models. Furthermore, the results remained robust to additional adjustment of degrees of freedom of temporal trends (5–9/year), mean temperature (4–5) and relative humidity (4–5) (Table S6).

4. Discussion

This is the first study to use PM$_{2.5}$DECH as the exposure metric to evaluate the impacts of PM$_{2.5}$ on cause-specific CVD mortality and YLL in Iran. Evidence gained in the current study showed that rises in both PM$_{2.5}$DECH and PM$_{2.5}$-mean were significantly associated with increased CVD mortality and YLL. For cause-specific CVD mortality, only deaths from stroke were found to be associated with PM$_{2.5}$ pollution. Compared with the most elderly (age 75+ years) and female, younger people (age 0–64 years) and males appeared to be more susceptible. We further obtained a relatively greater CVD mortality burden attributable to PM$_{2.5}$DECH than PM$_{2.5}$-mean. These findings may possess important implications for public agencies of Tehran to prompt the process of clean air action plans and health resource relocation.

While previous epidemiological studies widely reported adverse health effects by using daily mean concentration of air pollutants as the exposure index (Ran et al., 2018a; Zhong et al., 2018), an increasing number of research noticed that significant within-day concentration variations might bias the health-related assessments (Lin et al., 2017b; Lin et al., 2018b). To better capture the health impacts of air pollution, recent studies focused on hourly variations of air pollutants (Bhaskaran et al., 2011; Sullivan et al., 2005), and conceived a series of novel exposure indicators, such as hourly peak concentration, hourly mean concentration and daily excessive concentration hours (Chen et al., 2019; Lin et al., 2017a; Lin et al., 2017b; Lin et al., 2018b). In several prior research, PM$_{2.5}$DECH has been well implemented with advantages (Lin et al., 2017a; Lin et al., 2018b). Compared with PM$_{2.5}$-mean, PM$_{2.5}$DECH is more informative by fully considering the large variations of
Fig. 2. Dose-response curves for PM$_{2.5}$ mean, PM$_{2.5}$ DECH and CVD mortality, YLL in Tehran, Iran, 2012–2016. A natural spline smoother with 3 df was applied. The continuous bold red and blue lines refer to the effect estimates, and the boundaries of the shaded parts refer to the 95% confidence interval. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Fig. 3. Estimated changes with 95% confidence intervals of CVD mortality (%) and YLL (years) associated with per IQR increase in PM$_{2.5}$ mean and PM$_{2.5}$ DECH at different exposure days in Tehran, Iran, 2012–2016.
study calculated PM effects of population exposure to different pollution levels. Our standards, PM

The yearly attributable number and overall attributable fraction of cardiovascular mortality and YLL due to PM\textsubscript{2.5} mean and PM\textsubscript{2.5} DECH.
developing countries such as China, Thailand and Iran, presented evidence regarding short-term PM$_{2.5}$-CVD mortality relationships (Karimi et al., 2019; Pinichka et al., 2017; Shang et al., 2013). The putative biological mechanisms suggested that soluble components of PM$_{2.5}$ could penetrate pulmonary epithelium into the circulation and interact with lung receptors, which might lead to the instability of a vascular plaque or arrhythmias. In addition, less acute cardiac effects from several hours to weeks could be attributable to PM$_{2.5}$ ability to activate hemostatic pathways, impairing vascular function, and accelerating atherosclerosis (Brook et al., 2004). As for specific causes of CVD mortality, we observed significant effects of PM$_{2.5}$ on stroke deaths, which was in line with previous studies investigating the impacts of PM$_{2.5}$ on cause-specific mortality (Kettunen et al., 2007; Shah et al., 2015). A systematic review including 6.2 million stroke events across 28 countries found that with per 10 µg/m$^3$ increase in PM$_{2.5}$, the risk of stroke mortality and hospital admissions increased 0.3%, indicating that PM$_{2.5}$ was a serious environmental risk factor for stroke events (Shah et al., 2015). However, we did not observe statistically significant impacts of PM$_{2.5}$ on IHD and SCD. The results were partly inconsistent with epidemiological research from Tehran evaluating long-term health impacts of PM$_{2.5}$ from 2006 to 2015, which indicated that IHD deaths accounted for most mortality attributable to PM$_{2.5}$ (Faridi et al., 2018). The underlying reasons for the inconsistency remain uncertain, and more studies are warranted to clarify the discrepancy.

Stratified analyses by gender showed that statistically significant effects of all-cause CVD mortality only existed in males. Gender heterogeneity on the fatal CVD impact of PM$_{2.5}$ pollution was also observed in previous research (Clougherty, 2010). While a number of investigations were in agreement with our results (Chen et al., 2018a), numerous prior researchers reported opposite findings that females had a higher risk of CVD mortality due to PM$_{2.5}$ (Franklin et al., 2007; Ostro et al., 2008). This discrepancy might be explained by several potential differences between males and females, such as smoking status, socio-economic status and stress experiences (Chen et al., 2018b; Guo et al., 2013; Seeman et al., 2002). Additionally, men are more likely to have outdoor jobs and may expose to air pollution more frequently. The findings of sex patterns are still unclear and need further investigations.

It is a widely accepted knowledge that elderly population is more susceptible to environmental risk factor, and a large number of epidemiological studies using death counts as endpoint indicator have confirmed this perspective (Chen et al., 2017b; Franklin et al., 2007). However, our results showed relatively smaller effect estimates for people aged 75+ than the younger age group (age 0–64 years) in Tehran, which might be the result of harvesting effects. Additionally, younger people (age 0–64 years) in the current study had higher YLL risk compared with the more elderly (age 75+), which was in line with a retrospective regression analysis from Beijing, the capital city of China (Guo et al., 2013). These findings, contrary to the commonsense view, indicated that giving the same weight to deaths at different ages might underestimate the actual burden of air pollution on younger people. Therefore, as a more informative and comprehensive metric, YLL can serve as an alternative indicator to quantify premature death and evaluate the mortality burden.

In terms of the absolute death counts value and relative mortality proportion, attributable number and fraction can provide more straightforward information for public health policy making compared with relative risk or odds ratio (Wang et al., 2018). Our results showed that Tehran bore a great CVD mortality burden ascribed to ambient PM$_{2.5}$ pollution. In addition, we obtained a relatively larger CVD mortality burden attributable to PM$_{2.5}$DECH (1.67% (95% CI: 0.70–2.63%) CVD deaths) than PM$_{2.5}$mean (0.63% (95% CI: 0.28–0.98%) CVD deaths), indicating that using PM$_{2.5}$mean might underestimate the deleterious health effects of ambient PM$_{2.5}$. Subgroup analyses by age showed that younger people (age 0–64 years) contributed more to YLL than the most elderly (age 75+). Besides, due to a great CVD mortality burden of males and people dying from stroke, public agencies of Tehran should attach much importance to these vulnerable populations.

Some limitations of our research should be considered. First, we used the concentration threshold of Iran’s Clean Air Standards to calculate PM$_{2.5}$DECH rather than referring the threshold estimated by model analyses, which might bias the cardiovascular effect assessments. Second, we used the outdoor 24-hour average concentration of air pollutants, which overlooked the people’s time-activity pattern and indoor-outdoor air quality exchange in different locations (Lin et al., 2018a). Third, as other studies in this field, we used fixed-site monitoring data rather than individual levels, which might lead to some degrees of exposure misclassification and greater heterogeneity of the results (Zeger et al., 2000).

5. Conclusions

This time-series study found that short-term exposure to ambient PM$_{2.5}$ was significantly associated with increased risk of CVD mortality and YLL in Tehran. Significant associations between PM$_{2.5}$ pollution (i.e., PM$_{2.5}$mean and PM$_{2.5}$DECH) and cause-specific cardiovascular health (i.e., mortality and YLL) were only identified in stroke. Subgroup analyses showed that male and younger people were more susceptible to the high concentration of PM$_{2.5}$. Additionally, we observed a larger cardiovascular mortality burden attributable to PM$_{2.5}$DECH than PM$_{2.5}$mean, indicating that DECH might be an alternative exposure metric in health-related assessments. Our findings may provide valuable information for Iran’s public agencies to make a sustainable air pollution control policy and protect the potential vulnerable population.

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Author Contributions

Yunquan Zhang conceived and designed the experiments; Fatemeh Mayyaneh, Mohammad Baaghideh, Alireza Entezari, and Yunquan Zhang collected and cleaned the data; Yunquan Zhang, Dieyi Chen, Qianqian Xiang, Anqi Jiao, and Faxue Zhang performed the data analysis; Dieyi Chen, Yunquan Zhang, and Fatemeh Mayyaneh drafted the manuscript; Hung Chak Ho, Kejia Hu, Gongbo Chen, Qi Zhao, and Shengzhi Sun helped revise the manuscript. All authors read and approved the final manuscript.

Declaration of Competing Interest

The authors declare they have no competing financial interests.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.scitotenv.2019.134909.

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