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Does temperature enhance acute mortality effects of ambient particle pollution in Tianjin City, China☆,☆☆

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ABSTRACT

Only a few epidemiological studies have explored whether there were interactive effects between temperature and particulate matter <10 μm in aerodynamic diameter (PM_{10}) on mortality, especially in Asian countries. The present study used time-series analysis to explore the modification effects of temperature on the association between PM_{10} and the cause-specific mortality for cardiovascular, respiratory, cardiopulmonary, stroke and ischemic heart diseases (IHD), as well as non-accidental mortality in Tianjin between 2007 and 2009. Results showed that the PM_{10} effects were stronger on high temperature level days than that on low temperature level days. The interactions between PM_{10} and temperature were statistically significant on cardiovascular, cardiopulmonary, and IHD mortalities. The effect estimates per 10-μg/m^3 increase in PM_{10} concentrations at the moving average of lags 0 and 1 day in high temperature level were 0.62% (95% confidence interval (CI): 0.27, 0.97) for non-accidental, 0.92% (0.47, 1.36) for cardiovascular, 0.74% (−0.33, 1.82) for respiratory, 0.89% (0.47, 1.32) for cardiopulmonary, 0.65% (0.00, 1.31) for stroke and 1.20% (0.63, 1.78) for IHD mortalities. In addition, the PM_{10} effects on high temperature level days were stronger on older (≥65 years) compared with younger subjects (<65 years). This suggests that the modifying effects of the temperature should be considered when analyzing health impacts of ambient PM_{10}.

1. Introduction

Numerous epidemiologic studies have provided sufficiently convincing evidence that non-accidental mortality, including cardiopulmonary mortality, is associated with exposures to ambient particulate matter (Kan and Chen, 2003; Ostro et al., 2000; Pope et al., 2002; Samet et al., 2000). These reports are consistent in many countries and concern the overall population, e.g., in the United States (Basu and Ostro, 2008), Italy (Rossi et al., 1999), Japan (Omori et al., 2003), Thailand (Vichit-Vadakan et al., 2008), China (Qian et al., 2007), and Korea (Lee and Schwartz, 1999; Yi et al., 2009).

Another environmental factor may also impact mortality; a strong association between high temperature and mortality has also been found in some studies (Baccini et al., 2008; Barnett, 2007; Conti et al., 2005; Kan et al., 2007; Tong et al., 2010). The effects are more pronounced in the elderly, women and those with chronic disease, such as psychiatric disease, depression, cardiovascular disease, diabetes, and chronic obstructive pulmonary disease (COPD) (Medina-Ramón and Schwartz1, 2007; Schwartz, 2005; Stafoggia et al., 2006).

In current studies on health impacts of air pollution and temperature, the temperature was generally adjusted for analyzing health effects of ambient particles efficiently, otherwise, air pollutants were also adjusted when studying the effects of temperature on mortality. The modifying effect of the temperature on the air pollution–mortality relationship has been scarcely reported in recent years. A few papers tried to explore the interaction between air pollution and temperature (Qian et al., 2008; Ren and Tong, 2006; Roberts, 2004). Each identified a significantly enhanced mortality effect for increasing values of both particulate matter and temperature. However, in a recent multicity case-crossover analysis in Italy performed with data from 1997 to 2004, Stafoggia et al. found that the interaction between PM_{10} and temperature within three temperature strata resulted in positive but, in most cases, nonstatistically significant coefficients (Stafoggia et al., 2008). Therefore, the interaction between the temperature and the population remains to be unclear.

In present study, we aimed to evaluate the potential modifying effect of the temperature when analyzing the health effects of PM_{10} pollution in Tianjin City of China.

☆ Temperature modification on ambient particle hazard effects attracted more attention. There is less studies in this field. Current studies in China are all about southern cities, such as Wuhan, Shanghai. Tianjin is a big northern city of China. So this paper provides good evidence in northern Chinese cities.

☆☆ There is no conflict of interest or ethic problems.

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2. Materials and methods

2.1. Study area and population

Tianjin City is a metropolis in northern China, located at 38°34 – 40°15 north latitude, 116°43 –118°04 east longitude, the population is approximately 12 million people. Our study area includes the eleven urban and suburban districts (3340 km^2). Its ports, some distance away, are located on the Bohai Gulf in the Pacific Ocean.

Tianjin features a four season, monsoon-influenced climate, typical of East Asia, with cold, windy, very dry winters, and hot, humid summers, due to the monsoon. Tianjin is also an important industrial city in northern China. The major sources of air pollution in the city are coal burning and wind-blown dust, as well as the vehicle exhaust from trucks and a rapidly growing number of automobile on the streets.

2.2. Data sources

The data set used in this study consisted of daily data of meteorological conditions, air quality, and death records of population mortality in the eleven urban and suburban districts of Tianjin City from 1 January 2007 to 31 December 2009, covered around 6 millions local residents. Mortality data were provided by the center for public health surveillance and information service of China Centers for Disease Control and Prevention (CDC). Beginning in 2004, the China CDC electronically archives all death certificates all around the country. All data were validated each year by China CDC. The health outcomes we employed are classified by International Classification of Diseases 10th version (ICD-10), including non-accidental (A00-R99), cardiovascular (I00-99), respiratory (J00-99), cardiopulmonary (I00-99, J00-99), stroke (I60-69), and IHD mortalities (I20-25). Influenza (J09-11) was excluded from respiratory diseases.

The air pollution data, including 24 h average values of ambient PM_{10}, NO_2, and SO_2, were obtained from the Tianjin Environmental Monitoring Center. Daily mass concentrations for each pollutant were averaged from the available monitoring results of 12 fixed-site substations (Hedong, Hexi, Nankai, Heping, Hongqiao, Hebei, Dagang, Tanggu, Dongli, and Beichen districts) respectively under National Quality Control located in the urban and suburban areas of Tianjin. Meteorological data were provided by the National Meteorological Information Center (CMA) of China, including daily mean temperature and relative humidity.

2.3. Statistical methods and data analysis

2.3.1. Spearman correlation

Spearman correlation analyses were conducted to evaluate the bivariate associations between the weather factors and the air pollutants during the period of study.

Poisson generalized additive model (GAM) approach was used to model the natural logarithm of the expected daily death counts as a function of the predictor variables (Hasite and Tibshirani, 1990). It is generally assumed that the daily number of population mortality belongs to a kind of small probability event and had a Poisson distribution. The potential nonlinear effects of some confounding factors on the dependent variable could be modeled with nonparametric smoothing functions in GAM model, such as seasonal variation and weather conditions. We used days of calendar time with a cubic smoothing function to adjust for the confounder from the seasonality and short-term fluctuation using day of the week as a factor. Other potential confounders were also adjusted for, such as relative humidity and public holidays.

Before exploring the effect modification of air temperature on PM10–mortality relationship, we used first an independent model to explore the patterns of the relationship between the temperature and the mortality. The independent model is described below:

$$\begin{aligned}\mathrm{Log}[\mathrm{E}(\mathrm{Yt}|\mathrm{X})] &= S(\text{mean temperature},\ 3) + \mathrm{PM}_{10} + S(\text{time},\ 21)\\ &\quad + \mathit{factor}(\text{dow}) + \text{holiday} + S(\text{relative humidy},\ 3)\\ &= S(\text{mean temperature},\ 3) + \mathrm{PM}_{10} + \mathit{COVs}\end{aligned} \quad (1)$$

where t refers to the day of the observation; E(Yt|X) denotes estimated daily case counts on day t; $S()$ denotes the cubic smoothing spline. Given the wealth of worldwide findings of the association between population mortality and PM_{10} using the average concentration of lag of zero and 1 day (lag01), we also used lag01 PM_{10} (Kan et al., 2008; Schwartz, 2000; Vichit-Vadakan et al., 2008). Mean temperature and relative humidity denote the current day's temperature and relative humidity(Kan et al., 2008; Vichit-Vadakan et al., 2008). 7 degrees of freedom per year for time were selected so that little information from time scales longer than 2 months was included. This choice largely reduced confounding from seasonal factors and from longer-term trends (Dominici et al., 2000). Dow is day of week. Holiday is treated as a dummy variable (0 or 1 denotes not holiday or holiday).

Next, in order to estimate the potential interactive patterns of both PM_{10} and mean temperature on population mortality, we fitted a bivariate response surface model by thin-plate spline (Stafoggia et al., 2008; Wood, 2003). The degrees of freedom for the thin-plate spline were chosen using generalized cross-validation method.

$$\mathrm{Log}[\mathrm{E}(\mathrm{Yt}|\mathrm{X})] = S(\text{mean temperature}, \mathrm{PM}_{10}) + \mathit{COVs} \quad (2)$$

where S denotes thin-plate spline. We adjusted for the same covariates (COVs) as in Model 1.

Finally, a temperature-stratified parametric model was used for estimating the interaction of the temperature with ambient PM10 level on the population mortality. Through temperature stratification, this approach could examine the heterogeneity of PM_{10} effects so as to

Table 1
Summary statistics of health outcomes, air pollutants and meteorological conditions in Tianjin, China, 2007–2009.

Variable		Means	Minimum	P(25)	Media	P(75)	Maximum
Mortality	Non-accidental	101.4	56	90	100	113	175
	Cardiovascular	53.3	25	45	52	61	113
	Respiratory	8.6	0	6	8	11	24
	Cardiopulmonary	62	27	52	61	71	124
	Stroke	21.5	4	17	21	25	53
	IHD	26.6	6	21	26	31	71
Air pollution	PM_{10} (μg/m^3)	95	13	58	82	120	503
	SO_2 (μg/m^3)	60.2	8	29	43	80	312.5
	NO_2 (μg/m^3)	41.9	12.8	30.4	38.4	49.6	128
Meteorology	Temperature (°C)	13.3	-10.5	2.6	14.7	23.9	31
	Humidity (%)	58.3	15	45	60	73	95

Abbreviations: PM10: particulate matter with an aerodynamic diameter of <10 μm; SO_2: sulfur dioxide; NO_2: nitrogen dioxide; CI: confidence interval.

Table 2
Spearman correlation coefficients between air pollution and weather variables.

Variables	SO_2	NO_2	Mean temperature	Relative humidity
PM_{10}	0.295	0.506	0.113	0.129
SO_2	1	0.725	−0.735	−0.251
NO_2		1	−0.493	−0.038
Mean temperature			1	0.272

Abbreviations: PM10: particulate matter with an aerodynamic diameter of <10 μm; SO_2: sulfur dioxide; NO_2: nitrogen dioxide.

estimate the extent of the interaction. The choice of cut-offs was made using the results of Model 1 as a guide. This issue will be discussed further under Results. In this model, we categorized temperature into two levels (low and high) using the different cut-offs of temperature.

$$\mathrm{Log}[E(Yt|X)] = \beta_1 PM_{10} + \beta_2 \mathrm{temp} + \beta_3 PM_{10} : \mathrm{temp} + COVs \qquad (3)$$

where temp denotes levels of temperature at current day. β_1 signifies the main effects of PM_{10}, β_2 reflects the main effects of temperature, and β_3 is the interactive effect of PM_{10} and temperature. We adjusted for the same covariates (COVs) as in Model 1. The effect estimates of PM_{10} on low temperature days were obtained from the main effects of pollutant; the effect estimates of PM_{10} on high temperature days were obtained from the main effects of pollutant (β_1) and pollutant–temperature interaction effects (β_3) (Ren et al., 2008).

The estimated modifying effects were expressed as the increased percentage of the daily death counts with per 10 μg/m^3 increments in the daily mass concentrations of the pollutants. The age group was divided into ≥65 years and <65 years respectively for stratified analyses. We also examined copollutant model in the analyses. All model analyses were performed using R software, version 2.11.1, using the mgcv package, 1.6–2.

3. Results

There were 111,087 death records for our data analyses in 2007–2009. On average, there were approximately 101 non-accidental mortalities per day in our study area, of which 53 were due to cardiovascular mortality, 9 were due to respiratory mortality, 62 were due to cardiopulmonary mortality, 22 were due to stroke mortality, and 27 were due to IHD mortality (Table 1). The PM_{10} mean concentrations were below the China Grade II standard for ambient air quality (the PM_{10} 24-h average is 150 μg/m^3) (Table 1). However, the maximum daily mean PM_{10} concentration was well above the China Grade II standard, with approximately 10% of days during the study period exceeding the China Grade II standard. Table 2 shows Spearman correlations between weather variables and air pollutants

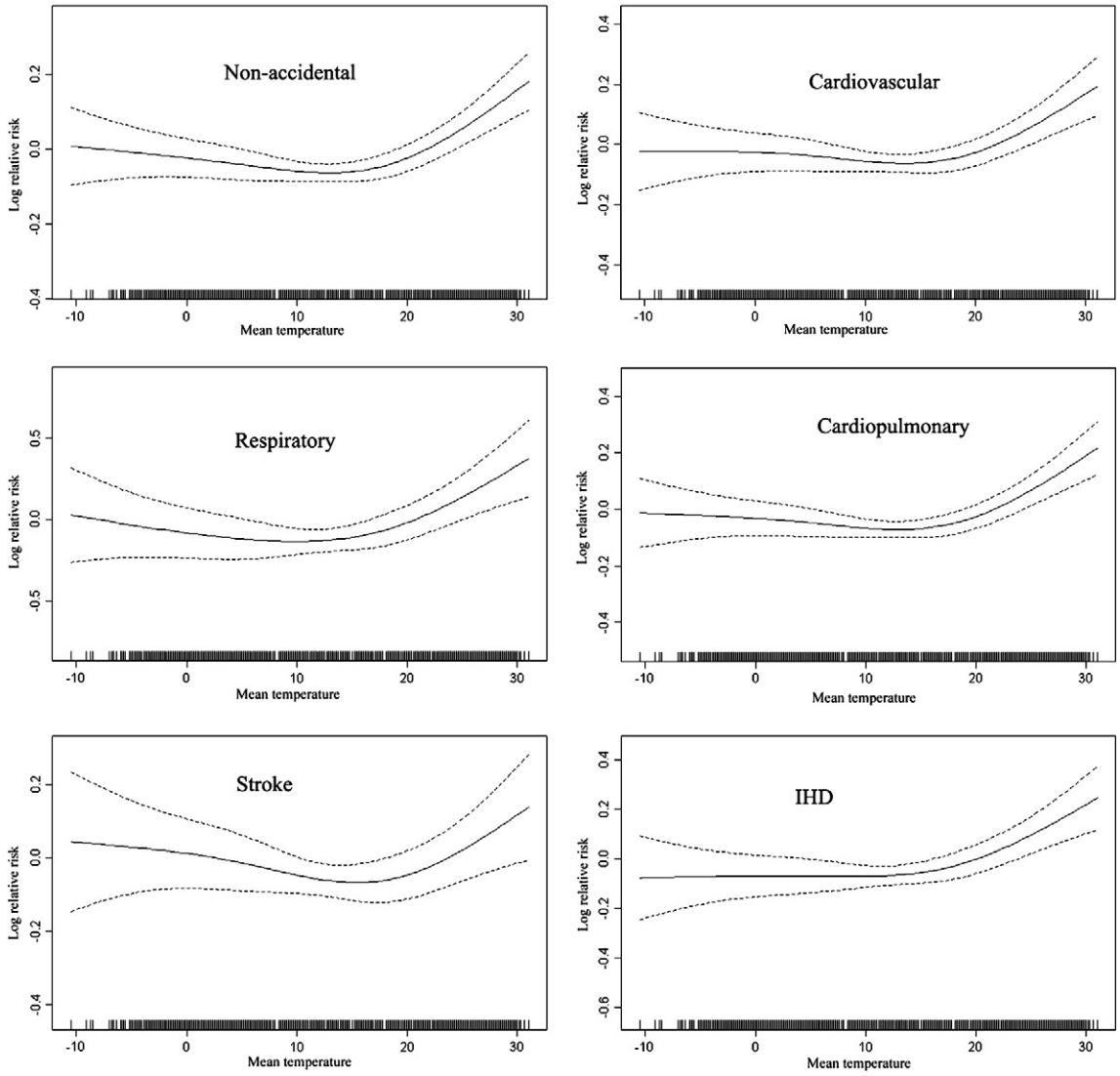

Fig. 1. Dose–response association of mean temperature with health outcomes in Tianjin, China, 2007–2009.

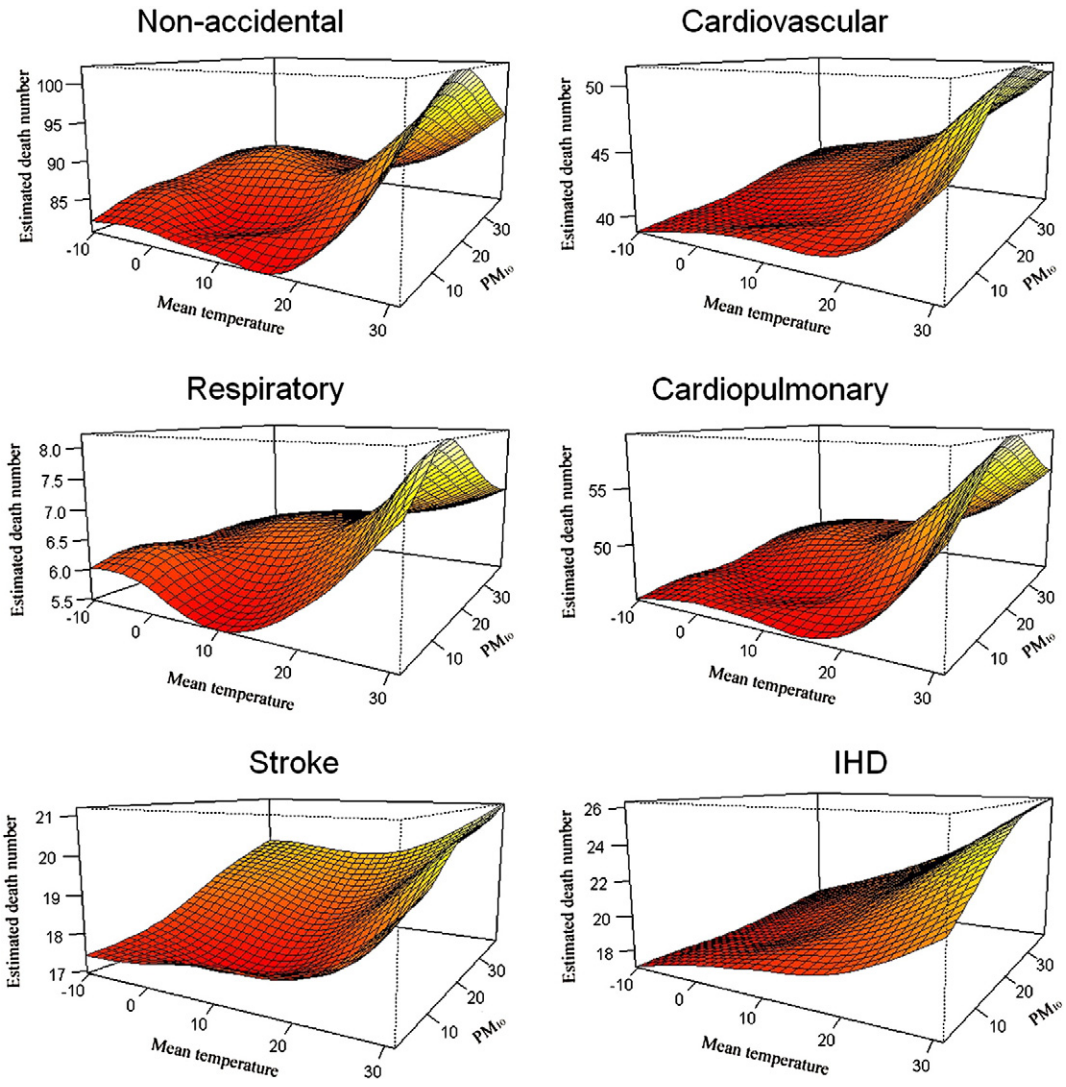

Fig. 2. Bivariate response surface of mean temperature and PM_{10} on health outcomes.

over the study period. PM_{10}, NO_2 and SO_2 had positive correlation coefficients with each other. NO_2 and SO_2 were negatively correlated with temperature and humidity respectively; conversely, PM_{10} had positive correlation coefficients with temperature and humidity respectively.

Fig. 1 graphically shows the exposure–response relationships for temperature–mortality relationships. Fig. 2 shows the potential interactive effects of PM_{10} and mean temperature on total and cause-specific mortality. Statistically significant relationships were observed for total and cause-specific mortality except that of stroke mortality. Based on the above results, we fitted temperature stratification into two levels. The median of temperature (14.65 °C) and visualized turning point 20 °C (Fig. 1) were chosen as cut-offs. Table 3 shows that the coefficients of the interactive terms were statistically significant for cardiovascular, cardiopulmonary, and IHD mortalities, but the magnitude of the interaction differed. The effects of PM_{10} on low/high temperature days using 20 °C cut-off were stronger than using 14.65 °C cut-off. For example, for a 10 μg/m^3 increment PM_{10}, cardiovascular deaths increased by 0.78% (95% CI: 041–0.14) at high temperature level, and 0.21% (−0.06–0.49) at low

Table 3
Estimates of the mean percentage of change (95% CI) in daily mortality per 10-μg/m^3 increase in PM_{10} concentration across temperature levels using different cut-offs.

Cut-off	Temperature	Non-accidental	Cardiovascular	Respiratory	Cardiopulmonary	Stroke	IHD
14.65 °C	Low	0.26 (0.04,0.49)	0.21 (−0.06,0.49)	0.45 (−0.17,1.07)	0.25 (−0.01,0.51)	0.11 (−0.29,0.52)	0.26 (−0.10,0.62)
	High	0.59 (0.30,0.88)	0.78 (0.41,1.14)[a]	0.73 (−0.15,1.62)	0.77 (0.42,1.12)[a]	0.53 (−0.01,1.07)	1.08 (0.61,1.55)[a]
20 °C	Low	0.28 (0.08,0.49)	0.25 (−0.01,0.50)	0.46 (−0.12,1.04)	0.28 (0.04,0.52)	0.14 (−0.24,0.51)	0.30 (−0.03,0.63)
	High	0.62 (0.27,0.97)	0.92 (0.47,1.36)[a]	0.74 (−0.33,1.82)	0.89 (0.47,1.32)[a]	0.65 (0.00,1.31)	1.20 (0.63,1.78)[a]

Abbreviations: PM10: particulate matter with an aerodynamic diameter of <10 μm; CI: confidence interval.
[a] The interaction effects between PM10 and temperature was significantly, $P<0.05$.

Table 4
Estimates of the mean percentage of change (95% CI) in daily mortality per 10-μg/m^3 increase in PM_{10} concentration by cause of death, temperature, and age, lag(0–1 day), in Tianjin, China, 2007–2009.

Age (years)	Temperature	Non-accidental	Cardiovascular	Respiratory	Cardiopulmonary	Stroke	IHD
<65	Low	0.29 (−0.03,0.62)	0.21 (−0.29,0.72)	−0.13 (−1.86,1.64)	0.18 (−0.30,0.67)	0.361 (−0.40,1.13)	−0.17 (−0.95,0.62)
	High	−0.34 (−0.90,0.21)	0.30 (−0.57,1.18)	−0.98 (−4.289,2.441)	0.21 (−0.63,1.06)	−0.64 (−2.05,0.79)	1.25 (−0.02,2.54)
≥65	Low	0.28 (0.05,0.51)	0.26 (−0.02,0.53)	0.52 (−0.08,1.13)	0.30 (0.04,0.55)	0.07 (−0.34,0.49)	0.39 (0.03,0.74)
	High	0.94 (0.54,1.33)[a]	1.07 (0.59,1.55)[a]	0.92 (−0.20,2.05)	1.05 (0.59,1.50)[a]	0.97 (0.25,1.68)[a]	1.19 (0.56,1.83)[a]

Abbreviations: PM10: particulate matter with an aerodynamic diameter of <10 μm; CI: confidence interval.
[a] The interaction effects between PM10 and temperature was significant, $P<0.05$.

temperature level when cut-off was 14.65 °C, and 0.92% (0.47,1.36) at high temperature level, and 0.25% (−0.01 to 0.50) at low temperature level when cut-off was 20 °C (Table 3).

Table 4 compares temperature modification effects on the health outcomes in different age groups (using 20 °C cut-off). For example, for a 10 μg/m^3 increment in PM_{10}, cardiopulmonary increased 1.05% (0.59–1.50) at high temperature levels for the elderly, but increased only 0.21%(−0.63 to 1.06) at high temperature levels for those younger than 65.

Table 5 provides the results of single pollutant and two-pollutant models. The effects of PM_{10} on total and cause-specific mortality did not alter much after adding SO_2 into the models; however, when adding NO_2, the interaction effects between PM_{10} and temperature on total mortality became significant.

4. Discussion

The present study was designed to analyze the interactive effect of particulate matter and temperature on the population mortality in Tianjin, China. We evaluated the hypothesis for modifying the effects of temperature on health effects of the ambient PM_{10} by adding linear interaction terms between PM_{10} and stratified temperature itself. The results showed that high temperature could enhance PM_{10} effect on the cause-specific population mortality, and there were significantly interactive effects on cardiovascular, cardiopulmonary, and IHD mortalities.

The interactions between the air pollutants and temperature have been explored in the epidemiologic studies (Katsouyanni et al., 1993, 2001; Qian et al., 2008; Ren and Tong, 2006). Katsouyanni et al. identified a strong interaction between sulfur dioxide and high temperature ((≥30 °C)) on mortality in the study in Athens, Greece, although the health effect of sulfur dioxide only on it was not significant (Katsouyanni et al., 1993). Ren et al. found that the temperature significantly modified the association between PM_{10} and non-accidental and cardiovascular mortalities in Brisbane, Australia between 1996 and 2001 (Ren and Tong, 2006). Qian et al. found that the effect estimates per 10 μg/m^3 increase in PM_{10} concentrations at the moving average of lags 0 and 1 day during hot temperature (>31.7 °C) were 2.20% for non-accidental, 3.28% (1.24–5.37) for cardiovascular, 3.31% (−0.22–6.97) for cardiac, and 3.02% (1.03–5.04) for cardiopulmonary mortalities, respectively (Qian et al., 2008).

For the analysis of interaction effects between PM_{10} and temperature, different statistical models were used. For example, Qian et al. added indicators of extreme temperature and the interaction terms between the indicators and PM_{10} in the basic model in order to analyze the modifying effects (Qian et al., 2008). Ren et al. employed bivariate response surface model and stratification parametric model to analyze the interactive effects (Ren and Tong, 2006; Ren et al., 2006). On the one hand, bivariate response surface model is flexible to visually examine whether potential interaction exists, but cannot provide a parametric estimate, because of a lack of linear assumption between independent and dependent variables. On the other hand, he thought that the stratification parametric model can provide parametric estimates and it is convenient to compare or pool estimates together in different subject cities. However, the selection of cut-off is still a challenge. In this study, cut-offs were selected according to median and dose–response curve between temperature and mortality (Model 1). We found that the turning point of dose–response curve might be a better choice for cut-offs compared with median temperature. Further studies were still needed to confirm this selection method.

Several explanations have been proposed for the synergistic effects of ambient PM_{10} pollution and high temperature on daily mortality. Meteorological variables such as temperature and humidity, play

Table 5
Copullant regression estimates of the mean percentage of change (95% CI) in daily mortality per 10-μg/m3 increase in PM_{10} concentration by cause, temperature, lag(0–1 day), in Tianjin, China, 2007–2009.

Pollutant	Temperature	Non-accidental	Cardiovascular	Respiratory	Cardiopulmonary	Stroke	IHD
PM_{10}	Low	0.28 (0.08,0.49)	0.25 (−0.01,0.50)	0.46 (−0.12,1.04)	0.28 (0.04,0.52)	0.14 (−0.24,0.51)	0.30 (−0.03,0.63)
	High	0.62 (0.27,0.97)	0.92 (0.47,1.36)[a]	0.74 (−0.33,1.82)	0.89 (0.47,1.32)[a]	0.65 (0.00,1.31)	1.20 (0.63,1.78)[a]
$PM_{10}+SO_2$	Low	0.18 (−0.03,0.41)	0.15 (−0.12,0.43)	0.41 (−0.21,1.05)	0.19 (−0.07,0.45)	0.02 (−0.38,0.43)	0.18 (−0.17,0.54)
	High	0.59 (0.24,0.94)	0.88 (0.44,1.33)[a]	0.72 (−0.34,1.81)	0.86 (0.44,1.29)[a]	0.62 (−0.03,1.27)	1.17 (0.59,1.75)[a]
$PM_{10}+NO_2$	Low	0.15 (−0.09,0.41)	0.08 (−0.23,0.4)	0.49 (−0.24,1.23)	0.13 (−0.16,0.44)	−0.13 (−0.6,0.34)	0.19 (−0.22,0.61)
	High	0.59 (0.24,0.95)[a]	0.89 (0.44,1.33)[a]	0.74 (−0.32,1.83)	0.87 (0.44,1.3)[a]	0.61 (−0.04,1.26)	1.18 (0.6,1.76)[a]

The current day of SO_2 and NO_2 were put into the model.
Abbreviations: PM10: particulate matter with an aerodynamic diameter of <10 μm; SO_2: sulfur dioxide; NO_2: nitrogen dioxide; CI: confidence interval.
[a] The interaction effects between PM10 and temperature was significant, $P<0.05$.

important roles in determining patterns of air quality over multiple scales in time and space (Macdonald et al., 2005). These linkages can operate through changes in air pollution emissions, transport, dilution, chemical transformation, and eventual deposition of air pollutants. The mechanisms of high temperature effects on mortality are also addressed in many studies. Keatinge et al. found that heat wave stress coupled with an increase in blood viscosity and cholesterol levels with high temperatures may increase the risk for cardio-respiratory deaths (Keatinge et al., 1986). Basu et al. and Bouchama et al. found that high temperature increases heat loss through the skin surface blood circulation which may be related to the increase in mortality (Basu and Samet, 2002; Bouchama and Knochel, 2002).

We have also observed that the effect estimates of population mortality for the older are stronger than for those younger than 65 years, which is consistent with other researchers' in the world (Aga et al., 2003; Bell et al., 2008; Katsouyanni et al., 2001; Vichit-Vadakan et al., 2008). A study in the subtropical city of Shanghai, China, also found that the elderly were most vulnerable to the effects of air pollution (Kan et al., 2008). Preexisting chronic disease in the elderly, such as respiratory and cardiovascular diseases, is more prevalent than in younger age group. Thus, older adults are generally more sensitive to PM_{10} pollution.

In our study, the health effects of ambient PM_{10} were decreased by NO_2 and SO_2 (Qian et al., 2007). Although the ambient PM_{10} level was associated with both the NO_2 and SO_2 levels (Table 2), the reduction of the effect estimates in two-pollutant model could not be due simply to confounders, the chemical component of PM_{10} might be responsible for the adverse health effect. Ostro et al. found that primary and secondary products of fuel combustion (EC, OC, SO_4 and NO_3) and other measures of mobile emissions (Cu, Fe,Ti, and Zn) in $PM_{2.5}$, exhibit the stronger with mortality (Ostro et al., 2008). Gold et al. reported that elevated level of black carbon (BC), which closely associated with the traffic pollution, increased risks of the ST-segment depression $\geq$0.5 mm of electrocardiogram (ECG) in an activity protocol study on older subjects ($\geq$60) (Gold et al., 2005). Lippmann et al. found that in the National Morbidity and Mortality Air Pollution Study (NMMAPS), daily mortality rates in the 60 cities with recent speciation data were significantly associated with average Ni of fine particulate matter (Lippmann et al., 2006). In addition, high concentrations of ambient particles can increase plasma viscosity, blood pressure, and changes of heart rate variability, triggering the onset of acute myocardial infarction (MI) (Guo et al., 2010; Peters et al., 2000, 2001).

Our study has several limitations. First, mortality misclassifications are possible for health outcomes. Because a relatively broad classification of diseases was used, we believe that misclassification for health outcomes is not likely to be substantial. Furthermore, all mortality data were validated again by China CDC for accurate coding. Second, this study is an ecologic design, in which bias from exposure measurement errors might occur to some degree due to lack of individual information. However, this kind of non-differential measurement error is known to cause a bias toward zero and underestimates the pollution effects (Zeger et al., 2000). There might have other important unknown and unmeasured factors. For example, socioeconomic status and ozone might play important roles as effect modifiers. Unfortunately, we currently do not have data on hand to explore the effects of these factors. Third, this study was carried out in a big city with temperate climate. Therefore, the results of this study may be difficult to generalize to other places. However, the present study included more than 100,000 deaths from one typical big city of China. The large quantity of data enabled us to explore relatively convincing results of the PM_{10}–temperature interaction.

5. Conclusions

In this time-series study, we found that the PM_{10} effects were more adverse at high temperature level days in Tianjin, China. These findings imply that it is important to control and reduce the emission of particles in Tianjin, particularly when temperature is high. In addition, further detailed studies, such as individual exposure level study, are needed to confirm these findings.

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