**Association between ambient particulate matter and daily cause-specific mortality in Tanggu,
Tianjin Binhai New Area, China**

Abstract

The aim of the study was to determine whether the area of Tanggu, Tianjin Binhai New Economic Developing Area, China, is subject to similar effects of PM₁₀ similar to other areas of China. This study was designed to investigate cause-specific mortality risks associated with air pollution in this geographical region. The present study used time-series analysis to explore the relationship between PM₁₀ and the cause-specific mortalities for non-accidental, cardiovascular and cardiopulmonary mortality from 1 January 2006 to 31 December 2010. A 10 µg/m³ increment of PM₁₀ was associated with a 1.02% (95% confidence interval (CI), 0.48%, 1.56%) increase in cardiovascular mortality, and a 0.88% (95% CI: 0.36%, 1.39%) increase in cardiopulmonary mortality. In addition, the effects from PM₁₀ appear to be consistent with multi-pollutant models. The results show there are strong associations between daily cardiovascular and cardiopulmonary mortality and ambient PM₁₀ exposure.

1. Introduction

Numerous epidemiologic studies during the past 20 years have confirmed that exposure to ambient pollution contributes to mortality and morbidity, both in China and in other countries (Kan et al., 2007, Samet et al., 2000). Among the air pollutants, particulate matter appears to show the most consistent association in the US (Schwartz et al., 2001), Europe (Pelucchi et al., 2009), Eastern Germany (Spix et al., 1993), China (Small and Luster, 1994, Li et al., 2002 Wong et al., 2008), Japan (Ueda et al., 2009), and Hong Kong (Wong et al., 2002). Some studies have shown that daily ambient air pollution variations in urban areas are associated with increased mortality, even when the fluctuations are below international standards (Katsouyanni et al., 2001, Brunekreef et al., 2009).

Generally, the estimated PM₁₀ effect from recent studies is in the range of 0.27–1.9% excess deaths per 10 $\mu\text{g}/\text{m}^3$ PM₁₀ increments in 24-hour average concentrations (Wong et al., 2008, Qian et al., 2007, Qian et al., 2008). Some studies show the estimated effects of PM₁₀ in Chinese cities were lower than those in Western countries (Wong et al., 2008, Kan et al., 2007). (Basu et al., 2008) Chinese studies report somewhat lower exposure-response coefficients compared to studies in Europe and the US (Aunan and Pan, 2004). A meta-analysis of Chinese studies found that each 10- $\mu\text{g}/\text{m}^3$ increase in PM₁₀ concentration was significantly associated with a 0.4% increase in cardiovascular mortality and 0.6% increase in respiratory mortality (Aunan and Pan, 2004). However, significant associations with respiratory and cardiovascular mortality were not found in Seoul, Korea (Goggins et al., 2011), or Hong Kong (Wong et al., 2001).

Several uncertainties remain regarding the impact of PM₁₀. For example, there is insufficient evidence about the curve of exposure-response associations between PM and daily cause-specific mortality and the mortality risk differences between China and Western countries remain unclear. The current study examined the associations of daily cause-specific mortality (non-accidental, cardiovascular and cardiopulmonary) with daily mean concentrations of PM₁₀ in Tanggu, Tianjin Binhai New Area, China.

2. Materials and methods

2.1 Study area and population

Tianjin Binhai New Area is a seaport city east of Tianjin. Tanggu, the core area of Tianjin Binhai New Area, is located on the Bohai Gulf in the Pacific Ocean, at 38° 44'–39° 13' north latitude, 117° 30'–117° 46' east longitude. Its population of approximately 500,000 resides in an area of 50 km². Tanggu has four distinct seasons, with cold, windy, dry winters, influenced by the vast Siberian anticyclone, and hot, humid summers, due to the monsoon. The major sources of air pollution in the area are particulates from burning coal and wind-blown dust, as well as the exhaust from trucks and a rapidly growing number of automobiles on the streets.

2.2 Mortality and environmental data

Daily counts of non-accidental deaths (A00-R99; International Classification of Diseases, 10th Revision), cardiovascular deaths (I00-I99) and cardiopulmonary deaths (I00-I99, J00-J99) were obtained from the Tanggu Center for Disease Control and Prevention in Tianjin Binhai New Area. The time series of daily death data available for analysis is from January 1, 2006 to December 31, 2010. All mortality data were checked against Public Security Bureau death report data and were validated each month by the same specialised public health worker to ensure consistency in data collection. In addition, a specialised data management person double checked missing data and data with errors to ensure the accuracy of data entry.

The daily mean temperature and relative humidity data were collected from the National Meteorological Information Center (CMA) of China. Daily air pollution data was from the Tianjin Municipal Environmental Monitoring Center. The air pollution data, including 24-hour average values of ambient PM₁₀, NO₂, and SO₂, were obtained from the Tianjin Environmental Monitoring Center. Daily mass concentrations for each pollutant were averaged from the available monitoring results of 11 areas in Tanggu under National Quality Control (see Figure 1, Map of Tanggu in Tianjin)

Figure 1 Map of

Tanggu in Tianjin and monitoring stations

Notes. Stars indicate monitoring stations

2.3 Statistical analysis

Spearman correlation analyses were conducted to evaluate the bivariate associations between the weather factors and the air pollutants during the period of study. A 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. It is generally assumed that the daily population mortality is associated with a small

probability event and had a Poisson distribution. The potential nonlinear effects of confounding factors (eg seasonal variation and weather conditions) on the dependent variable could be modelled with nonparametric smoothing functions in a GAM model. We used days of calendar time with a penalized 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 adjusted for as well, such as relative humidity and public holidays.

We examined the effect estimates for each pollutant at 0-, 1- and 2-day lags, and at lag 0–1 day average concentrations prior to the death events.

$$\begin{aligned} \text{Log}[E(Y_t|X)] &= S(\text{mean temperature}, 3) + \text{PM10} + S(\text{season}, 7*N) + \\ &\text{factor}(\text{DOW}) + \text{factor}(\text{holiday}) \\ &\quad + S(\text{mean humidity}, 3) \\ &= S(\text{PM10}, 3) + \text{COVs}, \end{aligned}$$

where “t” refers to the day of the observation, “E(Y_t|X)” denotes the estimated daily case counts on day t, and “S()” denotes the penalized smoothing spline. “Mean temperature” represents the current day’s mean temperature. Seven degrees of freedom per year for time were selected so that little information from time scales longer than two months was included (Dominici et al., 2000). This choice largely reduced confounding from seasonal factors and longer-term trends. “N” denotes the number of years, while “DOW” represents the day of the week. “Holiday” was treated as a dummy variable (0 or 1 denote “not a holiday” or “a holiday” respectively) (Li et al., 2011).

In general, the largest pollutant effects were observed at the lag 0–1, where pollution concentrations were evaluated at the average of the day of death (lag 0) and one day before death (lag 1). Several approaches were taken to investigate the validity of the linearity assumption on the relationship between PM10 and mortality. First, we replaced the linear term of the PM10 concentrations with a smooth function with 3 df using ps. The likelihood ratio test with 2 df (which compares the original model with the smoothed model) was used. Next, the visual inspection approach was used to assess whether the smoothed exposure-response curve resembles a straight line (Figure 1).

Sensitivity analyses were conducted by controlling for the lagged effect of meteorology when computing the lagged effect of air pollution (i.e., mean temperature lag 2 days, 3 + mean humidity lag 2 days, 3 + PM10 lag 2 days). These included adjustment for seasonality and long-term trends, the degree of freedom (df) of the nonlinear influence of air temperature and relative air humidity, and exposure lag. In the analysis, we used 2-day moving averages of air temperature and relative air humidity because the effects of PM10 were highest at lag01. The different degrees of freedom assigned to the smoothing function of time, air temperature and humidity (e.g., 6 df for time/year, 4 df for air temperature, and 4 df for humidity) was used. In addition, including and excluding air pollutant variables was also evaluated. These analyses did not result in any significant changes to the results.

The estimated effects were expressed as the increased percentage of the daily death counts per 10 µg/m³ increment in the daily PM10 concentration. All analyses

were performed using R software, version 2.11.1 (R Foundation for Statistical Computing, <http://cran.r-project.org/>), using the mgcv package in R.

3. Results

3.1 Data description

From January 1, 2006 to December 31, 2010, we identified 13,273 non-accidental deaths in the study population. On average, there were approximately 7.3 non-accidental deaths per day, including 3.5 from cardiovascular diseases and four from cardiopulmonary deaths (Table 1). Cardiopulmonary diseases accounted for 54.8% of the total deaths.

During our study period, the mean daily PM₁₀, SO₂, and NO₂ concentration was 99.2 µg/m³, 60 µg/m³, and 44 µg/m³. The mean daily average temperature and humidity were 13 °C and 59% (Table 1).

[Insert Table 1]

Table 2 shows the Spearman correlation coefficients between air pollutants and weather variables. PM₁₀, SO₂ and NO₂ were positively correlated. PM₁₀ had relatively lower correlations with SO₂ ($r = 0.30$), NO₂ had a strong positive correlation with PM₁₀ ($r = 0.70$), and SO₂ ($r = 0.54$). PM₁₀ had positive correlation with temperature and humidity respectively. Conversely, SO₂ was negatively correlated with temperature and humidity respectively, and NO₂ was negatively correlated with temperature.

[Insert Table 2]

3.2 Regression results

Table 3 summarizes the effect estimates of PM₁₀ on non-accidental and cause-specific mortality in a single pollutant model. Generally, we observed significant associations between PM₁₀ and daily cardiopulmonary mortality evaluated at the lag 0–1 days, and at the lag 0 day, but not at the lag 1 day or at the lag 2 days. The largest pollutant effects were observed at the lag 0–1 days. A 10 µg/m³ increment of PM₁₀ was associated with 1.02% (95% CI: 0.48%, 1.56%) increase of cardiovascular mortality and 0.88% (95% CI: 0.36%, 1.39%) increase of cardiopulmonary mortality. However, we did not observe any significant associations between PM₁₀ and non-accidental mortality.

[Insert Table 3]

Table 4 shows the results of effect estimates of PM₁₀ on non-accidental and cause-specific mortality in a multi-pollutant model at the lag 0–1 days. We observed a significant effect of PM₁₀ on daily cardiopulmonary mortality after adjustment for co-pollutants (SO₂, NO₂ or both). The PM₁₀'s effect on cardiopulmonary mortality

had weak change and remained statistically insignificant when SO₂ or NO₂ or both were added in the regression models ($p < 0.05$).

[Insert Table 4]

Figure 2 graphically shows the exposure-response curves for non-accidental and cause-specific mortality with PM₁₀ concentrations evaluated at lag 0–1 days in Tanggu, Tianjin Binhai New Area, China, 2006–2010. The curves approached linearity, and the confidence intervals are almost symmetrical, indicating the regression model was an appropriate approach to evaluate the PM₁₀ effects on non-accidental and cardiovascular mortality.

[Insert Figure 2]

In addition, the result of the likelihood ratio test shows there is no obvious difference when replacing the linear term of the PM₁₀ concentrations with a smooth function with 3 df using penalized smooth spline. Such results strengthened the assumption that the air pollution effects on the logarithm of mortality were linear.

4. Discussion

In our study, we found that the PM₁₀ concentration was much higher than the National Ambient Air Quality Standards (NAAQS) set by the US Environmental Protection Agency (EPA) (15 $\mu\text{g}/\text{m}^3$ for annual mean and 35 $\mu\text{g}/\text{m}^3$ for 24-hour mean) and the Global Air Quality Guidelines set by the World Health Organization (10 $\mu\text{g}/\text{m}^3$ for annual mean and 25 $\mu\text{g}/\text{m}^3$ for 24-hour mean). The PM₁₀ mean concentrations were below the China Grade II standard for ambient air quality (the PM₁₀ 24-hour average is 150 $\mu\text{g}/\text{m}^3$). However, the maximum daily mean PM₁₀ concentration was well above the China Grade II standard, with approximately 10% of the days during the study period exceeding this standard. The daily concentrations of SO₂ and NO₂ were higher in the winter and spring than in the summer and autumn.

The largest pollutant effect of PM₁₀ was observed at the lag 0–1 days, which was consistent with similar studies (Samoli et al., 2005, Wong et al., 2008). Our results (Figure 1) indicate that the spline curves for PM₁₀ with total cardiovascular and cardiopulmonary mortality were roughly linear, consistent with the absence of a threshold (Samoli et al., 2005). This is consistent with results from most North American and Western European studies (Samoli et al., 2005, Anderson et al., 2001, Samet et al., 2000) and supports the validity of the estimates from present studies (Lopez-Villarrubia et al., 2010). For example, Touloumi et al. (Touloumi et al., 1996) illustrated that a 10 $\mu\text{g}/\text{m}^3$ increase in PM₁₀ (lag 0–1 days) was associated with a 0.86% (95% CI: 0.53–1.19%) increase in cardiovascular mortality in seven European cities (Touloumi et al., 2005). Similarly, Samet (Samet et al., 2000) reported that the estimated increase in the relative rate of death from cardiopulmonary causes was 0.68% (95% CI: 0.20–1.16%) for each increase in the PM₁₀ level of 10 $\mu\text{g}/\text{m}^3$ in 20 of the largest cities and metropolitan areas in the US from 1987 to 1994.

Because previous findings (Park et al., 2011, Yu et al., 2011, Ma et al., 2011) reported that season and temperature can significantly modify the adverse effect of air pollution on mortality, we adjusted for the confounder of seasonality and short-term fluctuations by using a smoothing function of time and temperature, and day of the week as a factor. Adjustments were also made for other potential confounders, including sulphur dioxide (SO₂) and nitrogen dioxide (NO₂), relative humidity and public holidays. SO₂ and NO₂ exposure are significant contributor to mortality (Pryka et al., 1993). After adjusting for SO₂ and NO₂, the mortality effect of PM10 did not change markedly and remains statistically significant (Table 4).

We found significant association between PM10 and daily cause-specific mortality in the Tangu area. Without adjustment for SO₂ and NO₂, a 10 µg/m³ increment in the two-day moving average (lag 0–1 days) concentrations of PM10 corresponded to 1.02% (95% CI: 0.48%, 1.56%) and 0.88% (95% CI: 0.36%, 1.39%) increases of cardiovascular mortality and cardiopulmonary mortality respectively. Our estimates in Tangu were lower in magnitude than in another PM10 mortality study carried out in Bangkok (1.90% for cardiovascular mortality) (Wong et al., 2008), and higher than in some studies carried out in Shanghai (0.27% for cardiovascular mortality), Wuhan (0.57% for cardiovascular mortality), Hong Kong (0.61% for cardiovascular mortality), Europe (0.6% for cardiopulmonary mortality) and the US (0.69% for cardiovascular mortality) (Wong et al., 2008, Kan et al., 2007, Pope and Dockery, 2006, Daniels et al., 2000, Katsouyanni et al., 2001). Samoli et al. have explained the differences in terms of the chemical composition of PM10 (Samoli et al., 2005). PM10 represents a mixture, with varying chemical and physical characteristics, reflected in differing toxicity of its components. For example, Ostro et al. found that primary and secondary products of fuel combustion (EC, OC, SO₃ and NO₃) and other measures of mobile emissions (Cu, Fe, Ti and Zn) in fine particulate matter exhibit the stronger association with mortality (Ostro et al., 2000). Lippmann et al. found that daily mortality rates were associated significantly with average Ni of fine particulate matter (Lippmann et al., 2006). Inconsistent with majority of previous studies (Wong et al., 2008, Aunan and Pan, 2004, Kan et al., 2007, Pope and Dockery, 2006), after adjusting the confounding effect of seasonality, humidity, SO₂ and NO₂, the level of significant effect of PM10 remains.

The mechanism underlying the impact of ambient particle pollution on daily mortality remains unclear. Previous studies on biologic mechanisms have showed that air pollution was associated with aconitine-induced cardiac arrhythmia in blood pressure (Bartoli et al., 2009), hypertensive rats (Hazari et al., 2009), acute arterial vasoconstriction (Brook et al., 2002) and C-reactive protein (Pope et al., 2004). All those factors are directly or indirectly related to the function of the cardiovascular system. Brunekreef and Holgate hypothesized that air particles increase the risk of cardiopulmonary mortality through direct and indirect pathophysiologic mechanisms, including systemic and pulmonary inflammation, accelerated atherosclerosis, altered cardiac autonomic function and the increase of inflammatory cytokines in the heart (Brunekreef and Holgate, 2002). Our study showed a linear relationship between ambient particle and daily mortality. Although further research is needed these results

tend to support the assumption that PM₁₀ affects systemic and pulmonary inflammation, accelerated atherosclerosis, altered cardiac autonomic function and the increase of inflammatory cytokines in the heart (Brunekreef and Holgate, 2002).

Our study has several limitations. First, mortality misclassifications are possible for health outcomes because a relatively broad classification of diseases was used. However, we believe such misclassification for health outcomes is not likely to be substantial because all the doctors that issued the death certificates were trained by the same public health doctor, who also validated all the mortality data. Second, there may have been other important unknown and unmeasured factors. For example, socioeconomic status and ozone might play important roles as effect modifiers. Unfortunately, we do not have current data available to explore the effects of these factors. Third, this study was carried out in a city with population size around 500,000, generalization of these results to other regions should be treated with caution.

In conclusion, our results confirm those previously reported from the south of China, Europe and the US that associations between cardiopulmonary, cardiovascular mortality and ambient PM₁₀ are significant. The heterogeneity found in the different studies might be explained partly by factors such as the air pollution mix and population features. Further studies focusing on the composition of particles and decreasing air PM₁₀ concentration are needed to improve our understanding of the etiologic mechanism through which particles affect mortality in order to promote people health.

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Table 1. Summary statistics of health outcome, PM10 and meteorological conditions in Tianjin, China (2006–2010).

Variables	Means	Minimum	P(25)	Media	P(75)	Maximum
Non-accidental	7.3	1.0	5.0	7.0	9.0	19.0
Cardiovascular	3.5	1.0	2.0	3.0	5.0	11.0
Cardiopulmonary	4.0	1.0	3.0	4.0	5.0	12.0
PM10 ($\mu\text{g}/\text{m}^3$)	99.2	10.0	62.0	88.0	122.0	503.0
SO ₂ ($\mu\text{g}/\text{m}^3$)	60.2	8.0	28.0	44.0	78.0	339.0
NO ₂ ($\mu\text{g}/\text{m}^3$)	44.0	12.8	32.0	41.6	51.2	142.4
Temperature ($^{\circ}\text{C}$)	13.1	−14	2.1	14.1	23.8	32.0
Humidity (%)	58.8	15.0	45.0	60.0	73.0	97.0

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

Variables	PM10	SO ₂	NO ₂	Mean temperature	Relative humidity
PM10	1	0.30	0.54	0.11	0.12
SO ₂		1	0.70	−0.73	−0.20
NO ₂			1	−0.44	−0.01
Mean temperature				1	0.25
Relative humidity					1

Table 3. Estimates of the mean percentage of change in daily mortality per 10 $\mu\text{g}/\text{m}^3$ increase in concentrations of PM10 in Tanggu, Tianjin Binhai New Area, China.

Variables	Lag 0	Lag 1	Lag 2	Lag 0–1
Non-accidental	0.08(−0.25,0.41)	−0.08(−0.41,0.25)	0.03(−0.30,0.35)	0.01(−0.40,0.42)
Cardiovascular	0.88(0.44,1.31)*	0.44(0.01,0.87)*	0.21(−0.22,0.64)	1.02(0.48,1.56)*
Cardiopulmonary	0.73(0.31,1.15)*	0.40(−0.01,0.8111)	0.23(−0.18,0.64)	0.88(0.36,1.39)*

*p < 0.05.

Table 4. Co-pollutant regression estimates of the mean percentage of change (95% CI) in daily mortality per 10 $\mu\text{g}/\text{m}^3$ increases in concentrations of PM10 in Tanggu, Tianjin Binhai New Area, China.

Pollutant	Cardiovascular	Cardiopulmonary
PM10	1.02(0.48,1.56) *	0.87(0.36,1.39) *
PM10+SO ₂	1.07(0.47,1.66) *	0.92(0.35,1.49) *
PM10+NO ₂	0.91(0.26,1.56) *	0.79(0.17,1.42) *
PM10+SO ₂ +NO ₂	0.86(0.21,1.52) *	0.74(0.11,1.36) *

*p < 0.05.

Figure 2. Dose-response association of particulate matter with health outcomes in Tianjin, Binhai New Area in 2006-2010.