



The Association Between Particulate Air Pollution and Respiratory Mortality in Beijing Before, During, and After the 2008 Olympic and Paralympic Games

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Specialty section:

This article was submitted to
Toxicology, Pollution and the
Environment,
a section of the journal
Frontiers in Environmental Science

Received: 02 November 2020

Accepted: 12 April 2021

Published: 23 July 2021

Citation:

Breitner S, Su C, Franck U,
Wiedensohler A, Cyrus J, Pan X,
Wichmann H-E, Schneider A and
Peters A (2021) The Association
Between Particulate Air Pollution and
Respiratory Mortality in Beijing Before,
During, and After the 2008 Olympic
and Paralympic Games.
Front. Environ. Sci. 9:624180.
doi: 10.3389/fenvs.2021.624180

To improve ambient air quality during the 2008 Summer Olympic and Paralympic Games, the Chinese Government and Beijing's municipal government implemented comprehensive emission control policies in Beijing and its neighboring regions before and during this period. The goal of this study was to investigate the association between particulate air pollution and cause-specific respiratory mortality before, during and after the period of the Olympic Games. Further, we wanted to assess whether changes in pollutant concentrations were linked to changes in respiratory mortality. We obtained daily data on mortality due to respiratory diseases (coded as J00-J99 according to the International Classification of Diseases and Related Health Problems 10th revision [ICD10]) and pneumonia (ICD10: J12–18), meteorology, particulate matter less than 10 μm or 2.5 μm in diameter (PM₁₀, PM_{2.5}) and particle number size distribution from official monitoring networks and sites located on the Peking University campus between May 20 and December 1, 2008. We assessed the effects of particulate air pollution on daily respiratory mortality using confounder-adjusted Quasi-Poisson regression models. Furthermore, we estimated air pollution effects for three periods—before (May 20 to July 20, 2008), during (August 1 to September 20, 2008) and after (October 1 to December 1, 2008)—by including interaction terms in the models. We found associations between different particle metrics and respiratory and pneumonia mortality, with more pronounced effects in smaller particle size ranges. For example, an interquartile range increase of 7,958 particles/cm³ in ultrafine particles (particles <100 nm in diameter) led to a 16.3% (95% confidence interval 4.3%; 26.5%) increase in respiratory mortality with a delay of seven days. When investigating the sub-periods, results indicate that a reduction in air pollution during the Olympics resulted in reduced (cause-specific) respiratory mortality. This reduction was especially pronounced for pneumonia mortality. The findings suggest that even a short-term reduction in pollution concentrations may lead to health benefits and that

smaller particles in the ultrafine size range may be particularly important for respiratory health.

Keywords: particulate air pollution, ultrafine particles, respiratory mortality, accountability study, Beijing 2008 Olympic and Paralympic games

INTRODUCTION

Numerous epidemiological studies have shown a link between exposure to elevated mass concentrations of particulate matter (PM) and increased cardiorespiratory morbidity and mortality (Thurston et al., 2017). Findings have been the most robust for fine particles (PM_{2.5}) (Frampton and Rich, 2016). Besides PM, particle number concentrations (PNC) in different size fractions may provide a more suitable exposure metric regarding the toxicity of PM exposure (Hennig et al., 2018; Chen et al., 2020). Particularly, ultrafine particles (UFP, particles <100 nm in diameter) may contribute to the observed health effects due to the small size, high total particle number concentration, large active surface area, and a high pulmonary deposition efficiency (HEI, 2013; Ohlwein et al., 2019; Schraufnagel, 2020). However, the epidemiological evidence for health effects of UFP is limited and inconsistent (Stafoggia et al., 2017; Ohlwein et al., 2019). Especially in China, the availability of appropriate exposure data is limited despite high particle concentrations.

To significantly improve the air quality during the 2008 Summer Olympic and Paralympic Games, the Chinese Government and Beijing's municipal government implemented comprehensive pollution control measures in Beijing and the neighboring regions before and during the Olympics. Main measures aimed at controlling vehicular emissions; for example, stringent vehicle emission standards were established, and high-emitting vehicles were banned from entering the city. Further measures included the shutdown of manufactories with high air pollutant emissions during the Olympics period or the relocation of polluting industries. Also, more than 16,000 small coal-fired boilers were converted to cleaner natural gas (Fang et al., 2009; Schleicher et al., 2012; Su et al., 2015). These regulations resulted in significant decreases in levels of PM and several gaseous co-pollutants in Beijing during August and September 2008 (Wang et al., 2009; Sun et al., 2011; Schleicher et al., 2012).

These pollution control efforts provided a unique opportunity to evaluate whether a short-run improvement in air quality would also result in acute changes in human health outcomes (Peel et al., 2010; Su et al., 2015). Using this quasi-experimental design, a number of studies have been conducted that explored the association between air pollution and health in Beijing (Wu et al., 2010; Lin et al., 2011; Huang et al., 2012; Rich et al., 2012; Zhang et al., 2013; Gong et al., 2014; Mu et al., 2014; Su et al., 2015). For example, Rich et al. (2012) reported an association between the change in air quality during the Beijing Olympics and short-term changes in inflammatory and thrombotic biomarkers in healthy young participants. Biomarkers of pulmonary and systemic inflammation showed similar results (Lin et al., 2011; Huang et al., 2012; Zhang et al., 2013; Gong et al., 2014). However, these studies were mostly

limited to PM_{2.5} and cardiorespiratory biomarkers; none of them was related to respiratory mortality.

We aimed at investigating the association between particulate air pollution and cause-specific respiratory mortality in Beijing before, during, and after the period of the Olympic Games from May 20, to December 1, 2008. Further, we wanted to assess whether changes in pollutant concentrations were linked to changes in respiratory mortality.

METHODS

We conducted this study in the urban area of Beijing, China. In 2008, this area had a land area of about 1,368 km², consisting of eight districts with approximately 12.3 million registered inhabitants (<http://www.bjstats.gov.cn/tjnj/2009-tjnj/>).

In our main analysis, we investigated the data in three specific periods: pre-Olympic period (May 20, to July 20–62 days), during-Olympic period (August 1, to September 20–51 days), and post-Olympic period (October 1, to December 1–62 days) (Su et al., 2015). We further specified a 10-days transition interval between each sub-period. Also, we examined data for the period May 20, to December 1, 2008 (“whole period”).

Data Collection Mortality Data

We obtained mortality data from the Beijing Center for Disease Control (CDC). Regarding death outcomes, we particularly investigated mortality data of respiratory diseases (International Classification of Diseases and Related Health Problems 10th revision [ICD10]: J00–J99) and pneumonia (ICD10: J12–18). For overall respiratory mortality, we also investigated different sex (male vs. female) and age groups (age <75 years vs. age ≥75 years).

Air Pollution and Meteorological Data

Hourly PM_{2.5} concentrations were obtained at an urban background monitoring site located in the Haidian district, about 5 km north from Peking University (PKU) (**Supplementary Figure S1**). Monitoring was done using a TEOM(R) RP1400A air sampler (Su et al., 2015). We calculated daily averages of PM_{2.5} only if at least 75% of the hourly data was available. There were no missing values for the daily PM_{2.5} concentrations.

Particle size distribution data was measured on top of a 6-floor building on the PKU campus, also located in the Haidian District (**Supplementary Figure S1**). The campus is located in a primarily residential and commercial area with no industrial or agricultural sources. Main local emission sources within a radius of 1000 m included road traffic emissions, fuel combustion for domestic cooking and heating, and construction (Wehner et al., 2008). An earlier examination on the spatial variability showed similar

average particle number size distributions during summer at the PKU site and another regional monitoring site, located around 50 km to the south (Yue et al., 2009). Therefore, we consider the PKU site to be an urban background station (Breitner et al., 2011). The setup of the measurement devices has been described in detail elsewhere (Wehner et al., 2004; Wehner et al., 2008). Briefly, a Twin Differential Mobility Particle Sizer was used to sample the size range from 3 – 800 nm (mobility diameter). Data were corrected for losses due to diffusion and sedimentation within the inlet line, as described by Wehner et al. (2004). The instruments were calibrated regularly, and the data were quality checked by the Leibniz Institute for Tropospheric Research.

We calculated 24 h average particle number concentrations (PNC) for the following size fractions (nm): 10–30, 30–50, 50–100, 100–300, 300–1,000, and also for UFP (10–100 nm). We selected these size ranges based on particle behavior, origin, and deposition in the respiratory tract (Gu et al., 2012; Su et al., 2015). About 4% of the data were missing.

We obtained data on PM with a diameter of less than 10 μm (PM_{10}) and the gaseous air pollutant nitrogen dioxide (NO_2) from the Beijing Municipal Environmental Monitoring Center's network. This network included eight measurement sites spread over the urban area of Beijing (Supplementary Figure S1). Sites are "mandated not to be in the direct vicinity of traffic or of industrial sources and not to be influenced by local pollution sources, thus also avoiding buildings or housing and large emitters such as coal-, waste-, or oil-burning boilers; furnaces; and incinerators" (Chen et al., 2012). We obtained daily average concentrations from the eight monitoring locations. If there were missing air pollutant concentrations from one station on a given day, we used data of the remaining sites to calculate the average concentration (Su et al., 2015).

Data on 24 h mean air temperature and relative humidity were available from a station of the China Meteorological Data Sharing Service, located in the southeastern part of Beijing within the Daxing District (Su et al., 2015).

We further obtained data on air mass history by use of the Hybrid Single-Particle Lagrangian Integrated Trajectory model (version 4.8, <http://ready.arl.noaa.gov/HYSPLIT.php>). We generated 72 h back-trajectories to determine the origin of air masses (Breitner et al., 2011; Su et al., 2015). We classified the back-trajectories by cluster analysis resulting in two clusters: the first cluster consisted of air masses arriving from the neighboring (mainly southern) regions of Beijing with low wind speed to stagnant conditions, whereas the second cluster represented air masses mainly coming from north-western directions with average to high wind speed.

Statistical Analysis

We calculated descriptive statistics for each particle metric (size-segregated PNC, $\text{PM}_{2.5}$, PM_{10}) and NO_2 , air temperature, relative humidity, and the different mortality outcomes for the whole reporting period, and for the three sub-periods. We also used Poisson regression models to estimate the between-period changes in mortality (Zhang et al., 2013; Su et al., 2015).

In the next step, we evaluated the relationship between air pollution and daily (cause-specific) respiratory death counts using confounder-adjusted Quasi-Poisson regression models. As potential confounders, we included same-day air temperature (penalized

TABLE 1 | Descriptive statistics of particle metrics, meteorology and respiratory mortality for the period May 20 to December 1, 2008.

	Whole period				
	(May 20–December 1, 2008, 175 days)				
	IQR	Mean	SD	Min	Max
PNC					
10–30 nm (cm^{-3})	3,502	5,050	2,542	1,672	13,474
30–50 nm (cm^{-3})	1,758	3,002	1,115	985	5,942
50–100 nm (cm^{-3})	2,154	4,719	1,578	985	10,947
10–100 nm (cm^{-3}) (UFP)	7,958	13,785	4,822	4,782	25,285
100–300 nm (cm^{-3})	2,901	4,578	2,268	462	15,206
300–1000 nm (cm^{-3})	791	893	626	54	4,275
PM and NO_2					
$\text{PM}_{2.5}$ ($\mu\text{g}/\text{m}^3$)	59.7	70.1	42.4	4.2	213.5
PM_{10} ($\mu\text{g}/\text{m}^3$)	77.0	112.4	83.2	15.0	563.0
NO_2 ($\mu\text{g}/\text{m}^3$)	29.5	46.9	22.9	14.0	142.0
Meteorology					
Air temperature ($^{\circ}\text{C}$)	10.6	19.7	7.7	−0.4	29.9
Relative humidity (%)	23.0	60.3	17.3	16.0	90.0
Respiratory mortality					
Total (J00–J99)	5	11	3	3	22
Male	3	6	2	0	13
Female	3	5	2	1	11
Age 0–74 years	2	3	2	0	8
Age ≥ 75 years	4	8	3	2	15
Pneumonia (J12–J18)	3	4	2	0	10

IQR: interquartile range; SD: standard deviation; PNC: particle number concentration; UFP: ultrafine particles; PM: particulate matter; PM_{10} : PM < 10 μm in diameter; $\text{PM}_{2.5}$: PM < 2.5 μm in diameter; NO_2 : nitrogen dioxide.

spline with four degrees of freedom (df)), a three-day average of relative humidity (spline with three df), and dummy variables for the three sub-periods, weekday, public holidays, and the heating period (heating period: October 20–December 1; non-heating period: May 20–October 19). Similarly to Su et al. (2015), the df of the weather parameters were selected according to the Generalized Cross-Validation (GCV) criterion. For the "whole period" analysis (May 20 to December 1, 2008), we investigated associations between respiratory mortality and air pollutants with different lags, from lag 0 (exposure on the event day) to lag 10 (exposure eight days before the event), according to previous publications (Stafoggia et al., 2017). Moreover, air pollution effects were estimated for the three sub-periods by the use of two dummy variables and corresponding interaction terms in the models.

We also investigated the effects of different particle metrics on respiratory mortality in sex and age groups. We further assessed the modification of particle effects by air mass origin using an interaction term to estimate the particle effects of the corresponding cluster. Two-pollutant models were used to examine the independent effects of PM, UFP, and NO_2 .

We conducted several sensitivity analyses on the confounder model, for example, varying the df for meteorological variables to test the robustness of the exposure-response function.

TABLE 2 | Description of particle metrics, meteorological variables and respiratory mortality pre, during, and post the Olympic Games 2008 in Beijing.

	Pre		During		Post	
	(May 20–July 20, 2008, 62 days)		(August 1–September 20, 2008, 51 days)		(October 1–December 1, 2008, 62 days)	
	Mean	SD	Mean	SD	Mean	SD
PNC						
10–30 nm (cm ⁻³)	4,880	2,354	3,836	2,028	6,514	2,573
30–50 nm (cm ⁻³)	2,966	1,044	2,422	994	3,641	996
50–100 nm (cm ⁻³)	4,700	1,414	3,955	1,159	5,528	1,775
10–100 nm (cm ⁻³) (UFP)	12,548	4,107	10,790	4,084	18,188	3,704
100–300 nm (cm ⁻³)	4,445	1,835	3,582	1,325	5,936	2,772
300–1000 nm (cm ⁻³)	898	529	649	421	1,106	794
PM and NO₂						
PM _{2.5} (µg/m ³)	87.4	41.0	52.6	27.0	67.2	47.7
PM ₁₀ (µg/m ³)	151.9	106.1	69.7	35.3	108.0	65.8
NO ₂ (µg/m ³)	43.2	12.5	28.3	7.0	65.8	25.3
Meteorology						
Air temperature (°C)	24.6	2.8	24.8	2.4	10.5	3.1
Relative humidity (%)	62.6	12.2	69.3	9.6	50.6	17.9
Respiratory mortality						
Total (J00–J99)	11	4	10	3	12	3
Male	6	3	6	2	7	2
Female	5	2	4	2	5	2
Age 0–74 years	3	2	2	2	3	2
Age ≥75 years	8	3	8	3	9	3
Pneumonia (J12–J18)	3	2	3	2	5	2

IQR: interquartile range; SD: standard deviation; PNC: particle number concentration; UFP: ultrafine particles; PM: particulate matter; PM₁₀: PM < 10 µm in diameter; PM_{2.5}: PM < 2.5 µm in diameter; NO₂: nitrogen dioxide.

TABLE 3 | Spearman rank correlation coefficients between daily mean values of air pollutants and meteorological variables for the period May 20 to December 1, 2008.

	PNC 30–50 nm	PNC 50–100 nm	PNC 10–100 nm	PNC 100–300 nm	PNC 300–1,000 nm	PM _{2.5}	PM ₁₀	NO ₂	T	RH
PNC 10–30 nm	0.83	0.48	0.90	-0.12	-0.34	-0.30	-0.12	0.33	-0.53	-0.70
PNC 30–50 nm	1	0.81	0.97	0.24	-0.03	-0.11	-0.04	0.48	-0.51	-0.54
PNC 50–100 nm		1	0.78	0.66	0.32	0.16	0.14	0.66	-0.44	-0.35
PNC 10–100 nm			1	0.23	-0.07	-0.14	-0.03	0.50	-0.58	-0.63
PNC 100–300 nm				1	0.84	0.61	0.48	0.67	-0.27	0.05
PNC 300–1,000 nm					1	0.81	0.64	0.51	-0.09	0.32
PM _{2.5}						1	0.80	0.39	0.16	0.24
PM ₁₀							1	0.51	0.04	0.06
NO ₂								1	-0.53	-0.19
T									1	0.31
RH										1

PNC: particle number concentration; PM: particulate matter; PM_{2.5}: PM < 2.5 µm in diameter; PM₁₀: PM < 10 µm in diameter; NO₂: nitrogen dioxide; T: air temperature, RH: relative humidity.

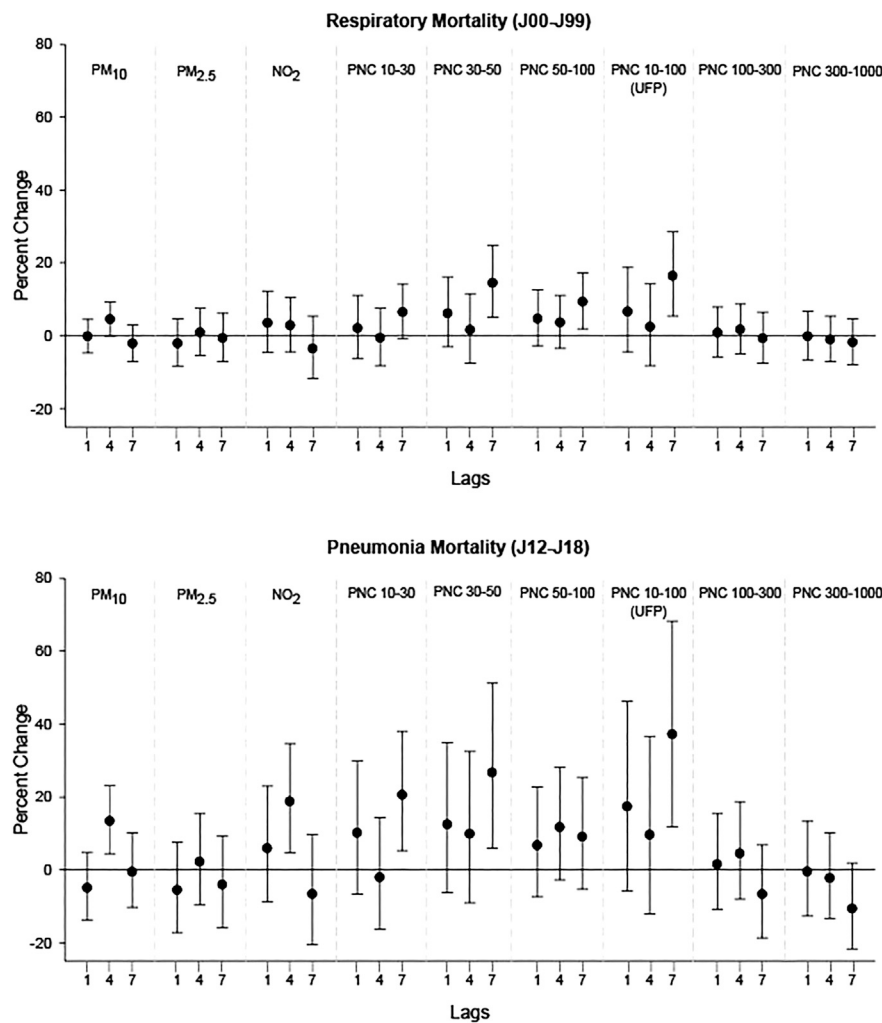
RESULTS

Descriptive Statistics

Table 1 and Table 2 summarize the descriptive statistics of the different particle metrics, NO₂, meteorology, and (cause-specific) respiratory mortality for the whole study period and the three sub-periods. Air pollutants showed lowest concentrations during the Olympics period. Pollutant levels decreased when comparing

the pre-to during-Olympics period, and increased again after the Olympic and Paralympic Games. The number of death counts due to respiratory diseases and pneumonia were similar or slightly lower in the pre-Olympics period, while mortality was higher afterward in comparison with the period during the Games (**Supplementary Figure S2**).

PM mass was moderately to highly correlated with the PNC size fractions 100–300 nm and 300–1000 nm and NO₂, but not



PM: particulate matter; PM₁₀: PM<10 μ m in diameter; PM_{2.5}: PM<2.5 μ m in diameter; NO₂: nitrogen dioxide; PNC: particle number concentration in different size-ranges.

FIGURE 1 | Percent change (95%-confidence interval) in respiratory mortality (**top**) as well as pneumonia mortality (**bottom**) per an interquartile range increase in air pollutants at lags 1, 4, and 7 in the urban area of Beijing for the period May 20 to December 1, 2008.

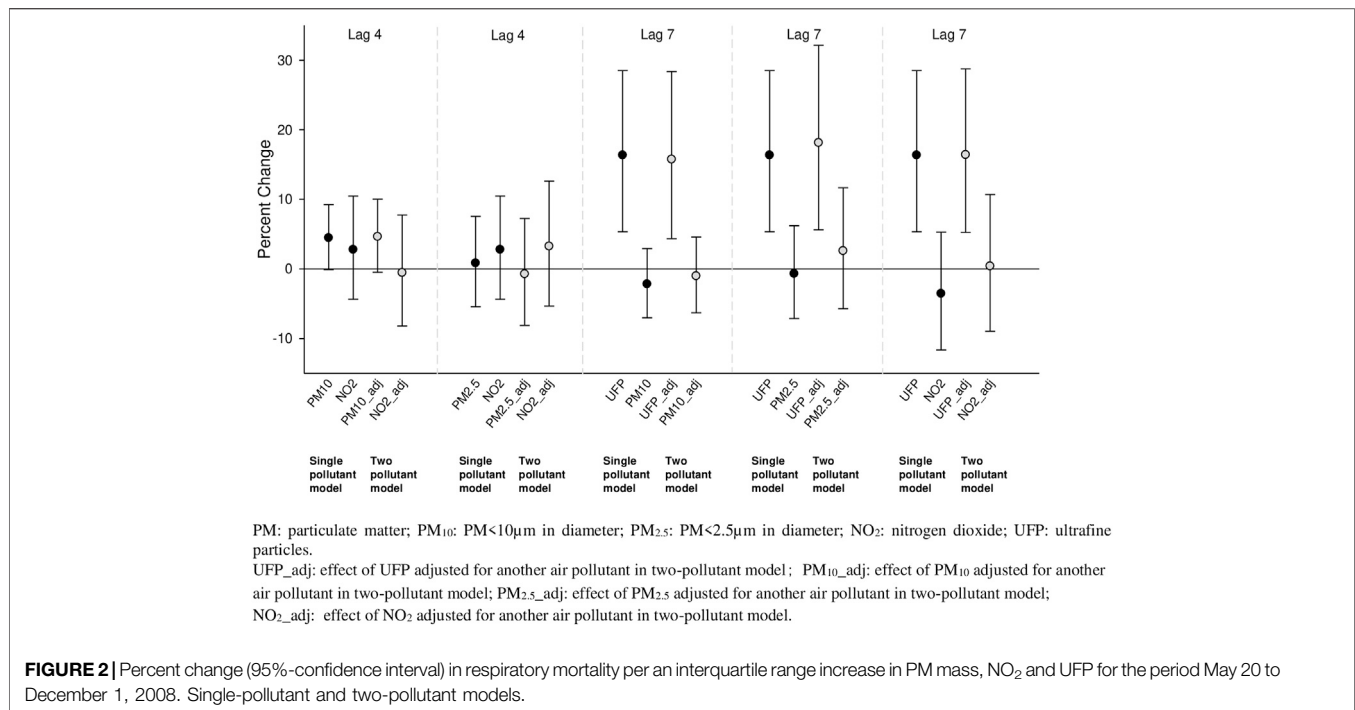
correlated with PNC size fractions below 100 nm (Table 3). PNC in the smallest size fraction 10–30 nm as well as PNC in the size class 30–50 nm were highly correlated with each other, as were PNC in the size fractions 30–50 nm and 50–100 nm. PNC in the smaller size fractions showed high correlations with UFP, negative correlations with meteorological variables, and no correlations with PNC size fractions above 300 nm, PM mass, or NO₂.

Effects of Particulate Air Pollution on Respiratory Mortality for the Whole Study Period

Associations between respiratory mortality and air pollution were investigated for exposure lags of 0–10 days. As the effects declined after a lag of seven days, we only present selected lags reflecting

immediate (lag1) and delayed associations (lag4 and lag7).

Figure 1 shows the associations between air pollutants and overall respiratory and pneumonia mortality for the whole study period, respectively. The effects are given as percent changes in mortality per an interquartile range (IQR) increase in the respective pollutant. We observed strongest effects of PM mass and NO₂ on respiratory mortality and pneumonia mortality at a lag of four days. For the different PNC size fractions, the strongest effects were observed with a delay of seven days. Effects on respiratory mortality were more pronounced for the size ranges 30–50 nm, and 50–100 nm as well as 10–100 nm (UFP); for pneumonia mortality, the effects were stronger in size ranges 10–30 nm, 30–50 nm, and 10–100 nm (UFP). In our age- and gender-specific analyses, we did not find a consistent pattern (Supplementary Figure S3).



Our two-pollutant models showed that the effects of UFP stayed relatively stable when adjusting for PM₁₀, PM_{2.5}, and NO₂ (Figure 2). However, PM₁₀, PM_{2.5}, and NO₂ effects all slightly increased when additionally considering another pollutant in the models.

Further, we assessed the potential modification of particle effects by air mass origin; this approach indirectly considers possible variation within the chemical composition of PM. Results showed that associations between respiratory mortality and air pollutants were (slightly) stronger for air masses from areas south of or around Beijing (cluster 1) compared with the air masses arriving from north-western directions with average to high wind speed (cluster 2) (Supplementary Figure S4); however, the differences between the two clusters were not statistically significant.

Effects of Particulate Air Pollution on Mortality in the Sub-periods

We assessed potential effect modifications by period (pre vs. during vs. post Olympics and Paralympics) only for selected lags—for PM mass and NO₂ with a lag of four days and the different PNC fractions at lag7. We overall found decreases in the effects of particulate air pollution over the sub-periods (from the pre-to post-Olympic period), except for the larger PNC size ranges (Figure 3). There, associations were lowest during the Olympic Games, and increased again afterward. Regarding pneumonia mortality, we observed that the risks were lowest during the Olympic period when strictest control measures have been applied. These differences were especially pronounced for PM_{2.5} and the PNC size fractions 100–300 nm

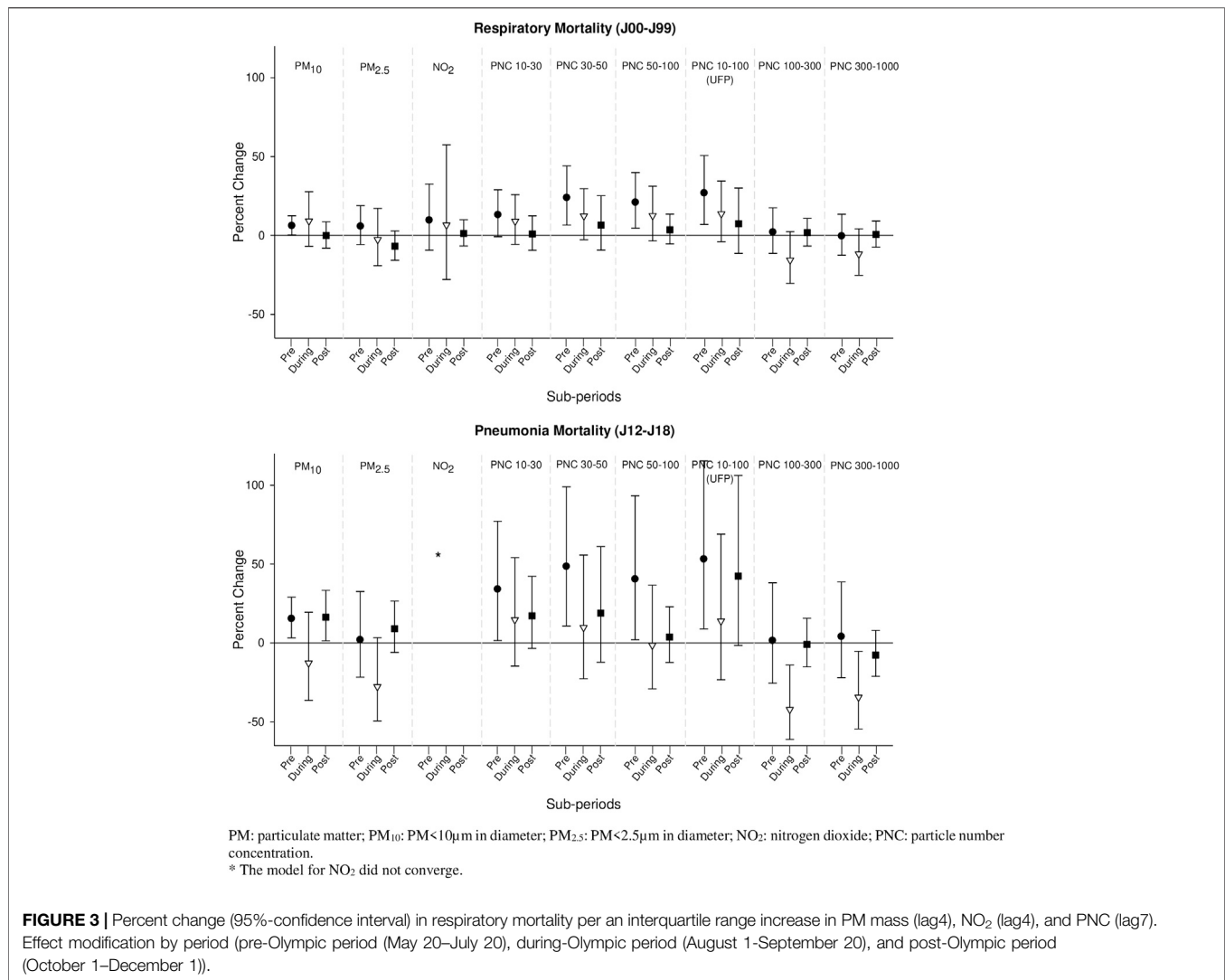
and 300–1000 nm; for the latter, effect modifications reached significance.

Sensitivity Analyses

Applying an alternative definition of the sub-periods (Zhang et al., 2013) or using different df for air temperature and relative humidity did not result in notable changes in the air pollution effects. We also did not observe deviations from linearity of the exposure-response relationships (data not shown).

DISCUSSION

Air quality improved during the 2008 Olympic and Paralympic Games in Beijing—we observed large concentration decreases in all of the examined air pollutants. However, after the relaxation of the emission control measures in October 2008, air pollution concentrations increased again; some air pollutants even reached or exceeded the levels observed before the Olympic and Paralympic Games. For the whole study period (May 20 to December 1, 2008), we observed an increase in respiratory and pneumonia events in association with PM mass and different PNC size ranges—effects were more pronounced in smaller particle size ranges (10–30 nm, 30–50 nm and 50–100 nm). When assessing effect modification by sub-periods, findings indicate that improved air quality during the Olympic and Paralympic Games resulted in decreased respiratory and pneumonia mortality. Respiratory mortality continued to be decreased even in the post-Olympics period.



Decreases in air pollution concentrations during the 2008 Olympic and Paralympic Games have already been reported (Wang et al., 2009; Schleicher et al., 2012; Schleicher et al., 2011; Sun et al., 2011). For example, a study applying a mobile laboratory to measure PM, black carbon, and gaseous pollutants along Beijing's Fourth Ring Road on selected days before, during, and after the Olympics found large reductions in pollutants' concentrations during the Games period (Wang et al., 2009). This study also observed concentration increases after the relaxation of the emission control measures (after September 20), which is in agreement with our results. Further, Schleicher et al. (2011) and Schleicher et al. (2012) examined size-segregated mass concentrations of particles before, during, and after the period of the Olympic Summer Games and also found considerably reduced air pollution levels.

In this study, a 7,958 particles/cm³ in UFP led to a 16.3% increase (95% confidence interval [CI]: 4.3%; 26.5%) in respiratory mortality with a delay of seven days. UFP effects were also seen on pneumonia mortality. Epidemiologic evidence on short-term association between UFP and respiratory mortality is still

limited mostly due to the limited availability of appropriate measurement data (Ohlwein et al., 2019). In one of the very first studies conducted in Erfurt, Germany, researchers found an increase of 15.5% (95% CI: 5.5%; 26.4%) in respiratory mortality per 12,690 particles/cm³ increase in UFP one day after exposure (Wichmann et al., 2000), a result later confirmed in the same city (Stölzelet al., 2007). Moreover, Atkinson and colleagues found excess risks for respiratory mortality one day after PNC exposure in London, with an estimate of 2.3% (95% CI: 0.1%; 4.8%) per 10,166 particles/cm³ increase in PNC (Atkinson et al., 2010). A study conducted in the Ruhr Area, Germany, between March 2009 and December 2014, found a 4.51% (95% CI: 0.37%; 8.81%) increase in respiratory mortality with a delay of six days in association with 4,900.2 particles/cm³. Further, in a study conducted in five Central-Eastern European cities, results suggested a delayed association between UFP and respiratory mortality (9.9% [95% CI: -6.3%; 28.8%] increase in association with an increase of 2,750 particles/cm³ in the 6-days average of UFP) (Lanzinger et al., 2016). However, studies conducted in Helsinki, Finland (Halonen et al., 2009), Prague, Czech Republic (Branis et al., 2010), and a

multi-city study in eight European urban areas (Stafoggia et al., 2017) failed to show associations between PNC and respiratory mortality.

Whereas earlier studies on UFP/PNC and respiratory mortality pointed to more immediate effects with UFP/PNC (Wichmann et al., 2000; Atkinson et al., 2010), more recent studies—including this study—pointed to stronger effects with a longer delay up to seven days (Lanzinger et al., 2016; Stafoggia et al., 2017; Hennig et al., 2018). Possible explanations for the change in the lag pattern might be changes in the particle sources, physical or chemical properties.

Furthermore, we also observed effects of PM_{10} at a lag four days, but no associations between $PM_{2.5}$ and respiratory or pneumonia mortality. This finding is somehow contradictory to previous studies; for example, a recent global multi-city study reported that $PM_{2.5}$ accounted for a larger proportion of the effects of PM_{10} and $PM_{2.5}$ combined (Liu et al., 2019). However, our findings are in line with a very recent systematic review and meta-analysis (Orellano et al., 2020).

In our study, particles in the size range of 30–50 nm might have a strong role. It has been shown that traffic emissions strongly influence Aitken mode particles (defined as particles with a diameter between 30 and 100 nm) such as soot, which is also the case in Beijing (Breitner et al., 2011). These soot particles may have adverse effects on human health as they may contain a high amount of polycyclic aromatic hydrocarbons (PAH) and toxic metals (Branis et al., 2010; Breitner et al., 2011). However, Leitte et al. (2012) investigated the association between PNC and respiratory mortality in Beijing from March 2004 until August 2005 and only observed increases in respiratory mortality for PNC in the size range of 300–1000 nm. Possible explanations for the inconsistent results might be changes in the particle sources. Further, Halonen et al. (2009) also found stronger associations between accumulation mode particles (AMP, 0.1–0.29 μm) and mortality due to respiratory causes and pneumonia. The authors hypothesized that their finding of a stronger association between AMP and respiratory mortality may be related to a better exposure assessment of these particles compared to particles in other size fractions (including UFP) (Halonen et al., 2009).

The biological mechanisms proposed in the literature by which particulate air pollution—including UFP—could cause, exacerbate, or accelerate the progression of respiratory diseases are mainly related to altered immune system response, oxidant-mediated cellular damage (including the production of reactive oxygen species (ROS) and oxidative stress) and exaggerated local and systemic inflammatory response (Leikauf et al., 2020; Pompilio and Di Bonaventura, 2020). Thereby, particles may induce an inflammatory response in the lung and modify immune response to subsequent stimuli, such as infectious pathogens. Once an infection occurs, increased pulmonary and systemic inflammation may also contribute to, for example, greater pneumonia (Pirozzi et al., 2017; Croft et al., 2019).

Accountability studies are defined as “a subset of epidemiologic studies that attempt to exploit policy-related, planned, or controlled interventions that result in changes (usually reductions) in exposure and/or exposure variability,” according to a publication by the Health Effects Institute (HEI)

(Pope 2009). Examples are studies on the ban of the sale of coal in Dublin, Ireland (Dockery et al., 2013), or traffic restrictions during the 1996 Summer Olympic Games in Atlanta, Georgia, United States (Peel et al., 2010) or the 2002 summer Asian games in Busan, Korea (Lee et al., 2007). The current study fits this description: The Chinese government and Beijing’s municipal government launched strict emission control policies before and during the Olympic period which resulted in a substantial reduction of levels of air pollutants. In the analysis of the modification effects by periods (pre-during-post Olympic Games), we observed improved air quality during the Olympic and Paralympic Games which resulted in a reduction in respiratory mortality. Respiratory mortality continued to be decreased even in the post-Olympics period, although levels of air pollution concentrations were increasing again. One possible explanation for this finding might be a delayed benefit from air pollution reductions in addition to a prompt reduction in risk during the Olympics. Regarding pneumonia mortality, we observed that the risks were lowest during the period of the Olympic Games where strongest control measures have been applied. These differences were especially pronounced for $PM_{2.5}$ and the PNC size fractions 100–300 nm and 300–1000 nm.

Strengths and Limitations

Accountability studies are considered to be the “closest epidemiologic equivalent to controlled experimental studies,” since “there is more exogeneity with regard to the changes in air pollution exposures and, thus, less opportunity for confounding” (Pope 2009). In this study, we defined three sub-periods according to when the emission control measures came into force. We also specified a transition interval of ten days between each of the sub-periods to allow enough time for a change in the air pollution levels after the measures came into force (Su et al., 2015). Moreover, we adjusted for air temperature, relative humidity—both known risk factors of mortality—and day of the week as well as public holidays as potential confounders (Welty and Zeger, 2005). We also considered the heating season during which increasing air pollution concentrations were observed. Our final models therefore can be considered to be conservative and robust, as indicated by additional sensitivity analyses.

There were also concerns about a change in the population during the Olympics period, which could lead to changes in the rates of respiratory mortality. Although millions of visitors from across the globe visited Beijing because of the Olympics, we used mortality data that based on records for registered permanent residence in Beijing; therefore, we can assume a probably stable population.

In this study, particle number size distribution data was measured at one fixed monitoring site; therefore, exposure misclassification might be possible due to greater spatial variability, especially for UFP with higher spatial variability compared to fine PM mass concentrations. However, our monitoring site could be classified as an urban background station (Breitner et al., 2011; Leitte et al., 2012); moreover, mass concentrations of fine PM were shown to be evenly distributed over the urban area of Beijing (Cheng et al., 2007; Wang et al., 2010). Hence, it can be assumed that our monitoring

site collected the average exposure of the study area (Su et al., 2015). Furthermore, concurrent measurements of UFP at different sites within one city/urban area have shown good correlations regarding day-to-day variations despite differing magnitudes in space (Cyrus et al., 2008; Meier et al., 2015). Therefore, if the background site is carefully chosen, it might well represent UFP exposures of the average population for short-term studies (Cyrus et al., 2008; Meier et al., 2015). Unfortunately, we did not have particle number size distribution data for a longer post-Olympic period to see if the particle effects on respiratory mortality increased again later on. Moreover, we were not able to investigate the effects of particulate air pollution on cause-specific respiratory mortality, as we did not have enough power for other cause-specific respiratory mortality than pneumonia. We also cannot rule out that inherent immunity might have influenced the results of this study. However, studies have suggested that inhalation of particulate air pollution may also modulate the innate immune system and affect respiratory innate immune responses (Bauer et al., 2012; Mishra et al., 2020). Further, we do not have information on previous illnesses of the deceased persons.

CONCLUSION

In summary, we observed large concentration decreases in all measured air pollutants during the 2008 Summer Olympic Games and Paralympics in Beijing. Particulate air pollution showed associations with respiratory mortality, with more pronounced effects in the smaller particle size ranges (10–30 nm, 30–50 nm, and UFP). Results from the specific sub-periods analysis indicated a reduction in respiratory mortality due to a reduction of the air pollution concentrations during the Olympics. The findings further suggest that ultrafine particles may be particularly adverse for human health, and the effect is largely independent of other airborne pollutants. At present, administrative air quality measurements of national networks, not only in China, measure only particle mass concentrations such as PM₁₀ or PM_{2.5}. In the future, regular air pollution observations should be extended by measurements of particle number size distributions.

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DATA AVAILABILITY STATEMENT

The datasets presented in this article are not readily available because data cannot be made available due to data security reasons. Requests to access the datasets should be directed to susanne.breitner@helmholtz-muenchen.de.

AUTHOR CONTRIBUTIONS

SB contributed to data collection, data management, data analysis, and drafted the manuscript; CS contributed to data collection and data management, analyzed the data, and drafted the manuscript; UF was lead partner of the project and contributed to data management and manuscript editing; AW contributed to data collection, data management, and manuscript editing; JC contributed to data collection, data management, and manuscript editing; XP. was lead partner of the project and contributed to data collection and manuscript editing; H-EW coordinated the project, contributed to data analysis and manuscript editing; AS contributed to data management, data analysis, and manuscript editing; AP contributed to data collection and manuscript editing.

FUNDING

This work was supported by the German Research Foundation (DFG) (WI 621/16-1, FR 1417/4-1, HE 2805/3-1; WI 1449/18-1). Parts of this work were funded by a scholarship being awarded to Chang Su (File no.2011601062) under the State Scholarship Fund by the China Scholarship Council (CSC).

SUPPLEMENTARY MATERIAL

The Supplementary Material for this article can be found online at: <https://www.frontiersin.org/articles/10.3389/fenvs.2021.624180/full#supplementary-material>

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