Journal of Environmental Economics and Management 79 (2016) 18–39

Contents lists available at ScienceDirect

Journal of Environmental Economics and Management

journal homepage: www.elsevier.com/locate/jeem

# The effect of air pollution on mortality in China: Evidence from the 2008 Beijing Olympic Games ☆

Guojun He [a], Maoyong Fan [b], Maigeng Zhou [c]

[a] Division of Social Science, Division of Environment, and Department of Economics, The Hong Kong University of Science and Technology, Hong Kong

[b] Department of Economics, Ball State University, USA

[c] National Center for Chronic and Noncommunicable Disease Control and Prevention, Chinese Center for Disease Control and Prevention, China

ARTICLE INFO

*Article history:*
Received 23 March 2015
Available online 4 May 2016

*JEL classification:*
Q53
I15
I18

*Keywords:*
Air pollution
Mortality
Particulate matter
2008 Beijing Olympic Games

ABSTRACT

By exploiting exogenous variations in air quality during the 2008 Beijing Olympic Games, we estimate the effect of air pollution on mortality in China. We find that a 10 percent decrease in $PM_{10}$ concentrations reduces the monthly standardized all-cause mortality rate by 8 percent. Men and women are equally susceptible to air pollution risks. The age groups for which the air pollution effects are greatest are children under 10 years old and the elderly.



## Introduction

Air pollution imposes significant health risks on humans in developing countries where the levels of pollution are often several orders of magnitude higher than those in developed countries (Chen et al., 2013a; Ebenstein et al., 2015; Greenstone and Hanna, 2014). Accurately estimating the health effects of air pollution is critical for the environmental regulation debate and optimal environmental policy design. Overstating the effects will lead to over-regulation and hinder economic growth, while underestimating the effects will leave a large number of people unprotected and create significant and unnecessary welfare losses.

This study uses a natural experiment to estimate the causal effects of air pollution on mortality in China. To ensure that the air for the 2008 Beijing Olympic Games (BOG08) was relatively clean, the Chinese government enforced a series of stringent air pollution regulations in Beijing and its neighboring cities from late-2007 through late-2008. These regulations resulted in a sudden and significant improvement in air quality in the regulated cities. By comparing the mortality rates in

☆ We are indebted to Jeffrey Perloff, Michael Anderson, Meredith Fowlie, Peter Berck, William Dow, Elisabeth Sadoulet, Sofia Villas-Boas, Maximillian Auffhammer, Christian Treager, Philip Coelho, and seminar participants at UC Berkeley, Harvard, University of Chicago, HKUST, NBER, IUPUI, and 88th WEAI dissertation workshop for comments and suggestions. Guojun He also thanks the Pamela and Kenneth Fong Fellowship and IBER dissertation grant for financial support. All errors are our own.

*E-mail addresses:* gjhe@ust.hk (G. He), mfan@bsu.edu (M. Fan), maigengzhou@126.com (M. Zhou).

http://dx.doi.org/10.1016/j.jeem.2016.04.004
0095-0696/

the regulated cities with those in the unregulated cities before, during, and after the regulation period, we find that improved air quality during the regulation period significantly reduced the mortality rates in the regulated cities.

We make three primary contributions to the existing literature. First, while many previous natural experimental designs investigate the effects of permanent policy changes on air pollution (Chay et al., 2003; Chay and Greenstone, 2003b), our study explores the air quality variations triggered by temporary and strictly-enforced regulations. The enforcement of permanent air pollution regulations, such as the Clean Air Act in the United States, might be endogenous because, as citizens become aware of the potential health consequences of air pollution, they put political pressure on government to create specific policy instruments to respond to their concerns. In cities where people are more health conscious, the enforcement of air quality regulations might be stricter and the subsequent health improvements might be larger. In contrast, the strong pressure to improve air quality during the BOG08 came from the international community rather than within China. The level of air pollution in Beijing was the biggest concern of the International Olympic Committee in the bidding process for the 2008 Summer Olympic Games. The commitment to ensure good air quality in the Beijing metropolitan area and co-hosting cities during the BOG08 was key to winning the bid; and it became an important political task for the Chinese government. Starting in late 2007, the Chinese government implemented a series of stringent policies to reduce local and regional emissions in the greater Beijing metropolitan area to ensure good air quality during the BOG08. Among the aggressive regulations were setting higher emission standards, reducing traffic, halting large-scale construction projects, and shutting down polluting factories. The enforcement of the BOG08 regulations was strict and likely to be exogenous. The combination of these radical regulations led to a dramatic improvement in air quality in Beijing, its neighboring cities, and the co-host cities. For example, our results show that, during the BOG08 period, monthly $PM_{10}$ concentrations in Beijing were reduced by approximately 30 percent in Beijing.

Second, to our knowledge, this study is the first to estimate the sub-chronic health effects of $PM_{10}$ pollution on a national scale in China. Previously, a large number of epidemiological studies have examined the short-term associations between air pollution and mortality using daily data (see Aunan and Pan, 2004; Lai et al., 2013; Lu et al., 2015; Shang et al., 2013 for literature review). The estimates from these high-frequency time-series data offer insights on the acute effects of air pollution. For the long-run effect, Chen et al. (2013a) estimated the impact of air pollution on life expectancy using China's winter heating policy as a natural experiment. This study complements both lines of research by providing insights on how monthly variations in air pollution affect mortality. We find that air pollution has a significant impact on monthly mortality rate, with a 10 percent reduction in $PM_{10}$ concentrations resulting in an 8 percent decrease in all-cause mortality rate. A back-of-envelope calculation shows that more than 285,000 premature deaths in urban China could be averted annually if $PM_{10}$ concentrations were to decrease by 10 percent.

Third, by analyzing the most comprehensive monthly air pollution and mortality data ever assembled in China, we are able to estimate the heterogeneous effects of $PM_{10}$ pollution by the cause of death, gender, and age groups. Our analysis shows that men and women are equally susceptible to air pollution risks. Air pollution has a larger impact on the most vulnerable groups: children under 10 years old and the elderly (ages 75 years and over). We also find that increased air pollution causes more old people to die from cardio-cerebrovascular and respiratory (CVR) diseases but not from non-CVR diseases. For infants and young children, we find the opposite: deaths from non-CVR diseases drive the main results.

We conduct a variety of robustness checks and find that they do not alter our conclusions. Weather conditions (temperature and precipitation) and socio-economic characteristics, which are typically confounding factors in associational studies, have little effects on our estimates. We conduct falsification tests using air-pollution-irrelevant (such as cancer and injury) mortality and show that the main findings are not due to our model choice or the underlying overall death patterns. The evidence suggests that our research design provides a credible basis for evaluating the air pollution effect.

The remainder of this paper is structured as follows. Section "Health effects of air pollution" reviews the literature on estimating the health effects of air pollution. Section "Air pollution regulations during the BOG08" discusses the air pollution regulations during the BOG08. Section "Data" describes the air quality data, mortality data and meteorological data. Section "Research Design and Model" addresses our research design and model. Section "Results" summarizes the main results, and Section "Robustness checks" checks their robustness. Section "Comparison with estimates from associational models" compares our results with those of traditional models and previous studies, and Section "Health benefits of air pollution reduction" provides a range of estimates on the monetary value of the averted deaths. Section "Conclusions" concludes.

## Health effects of air pollution

The association between air pollution and human health has been recognized for more than half a century.[1] The majority of previous studies fall into the following categories: (1) time-series studies; (2) cross-sectional and cohort-based studies; (3) panel (fixed-effects) studies; and (4) natural experimental studies.

Time-series studies investigate whether daily or weekly fluctuations in air pollution are associated with changes in health outcomes (such as hospital admissions or deaths). Time series models are widely used in epidemiological studies.

[1] For example, during the London fog incident of 1952, extreme elevations of air pollution were found to be associated with markedly increased mortality rates (Logan, 1953).

Most of these studies find that temporary elevations in air pollution are associated with worse health outcomes (see Aunan and Pan, 2004; Lai et al., 2013; Lu et al., 2015; Shang et al., 2013 for literature review). However, sharp changes in air pollution levels are often driven by local weather conditions rather than changes in polluting activities (Chay et al., 2003). If weather conditions cause health problems through other channels, it is unclear whether the poorer health outcomes are caused by elevated air pollution or by other risk factors.[2] Moreover, because there are no appropriate control groups, it is difficult to rule out alternative explanations in most time-series studies.

Cross-sectional studies compare health outcomes across locations, examining how air pollution is associated with health outcomes after controlling for potential confounding factors. This type of research design is plagued by omitted variables bias. As people's health status and the local air quality are usually simultaneously determined by many other social and economic factors, a correlation between air pollution and health status does not necessarily indicate a causal relationship. In practice, it is infeasible to control for all potential confounding factors; thus, the health effect of air pollution estimated from cross-sectional models may be biased.

Cohort-based longitudinal studies (e.g. Dockery et al., 1993; Zhang et al., 2011) may face problems similar to those of cross-sectional studies. In principle, longitudinal studies can accurately estimate the reduction in life expectancy associated with higher levels of pollution because they collect data on long-term exposure (Chay et al., 2003). However, to some extent, people self-select into different locations; thus, their exposure to different levels of pollution is endogenous. Wealthy people, whose health status tends to be good for other reasons, can migrate to clean regions, while poor people may be confined to polluted areas. Hence, the observed association between air quality and mortality may result from factors other than air pollution. As pointed out by Chay and Greenstone (2003b), these observational/associational approaches tend to produce unreliable estimates due to endogeneity issues.

As longitudinal data become increasingly available, recent studies have used fixed-effects models to deal with potential bias caused by time-invariant unobserved factors (Currie and Neidell, 2005; Currie et al., 2009). Fixed-effects models are particularly useful when time-invariant omitted factors explain most of the variations in an outcome variable. The assumption required for identification is that there are no unobserved shocks to air pollution levels that co-vary with unobserved shocks to health outcomes. However, because changes in air quality often depend on factors similar to those affecting health outcomes (such as weather), this assumption may not hold. In addition, measurement error of air pollution is common in applied research. It may attenuate the marginal impact of air pollution and inflate the equation error variance in the fixed-effects models (Wansbeek and Meijer, 2000).

In contrast, natural or quasi-experiments provide compelling identification strategies (Chay and Greenstone, 2003a,b). Most quasi-experimental evidence on the health impact of air pollution has been based on settings in developed countries, and the majority of economic research focuses on the health of infants rather than the whole population. Chay and Greenstone (2003a) analyzed the effects of the Clean Air Act Amendments on infant mortality, using nonattainment status as an instrument for Total Suspended Particulates (TSPs) changes. They estimated that a 1-percent decline in TSPs resulted in a 0.5-percent decline in the infant mortality rate. Chay and Greenstone (2003b) also explored how air quality improvement as a result of the 1981–1982 recession affected infant mortality in the United States. They find that a 1-percent reduction in TSPs resulted in a 0.35-percent decline in the infant mortality rate at the county level. Currie and Walker (2011) analyzed the introduction of electronic toll collection devises in New Jersey and Pennsylvania and found that it reduced auto emissions in the vicinity of a toll plaza. As a result, infant health improved in areas immediately adjacent to the toll plaza. Luechinger (2014) investigated the effect of $SO_2$ on infant mortality in Germany, utilizing a natural experiment made possible by the mandated desulfurization of power plants, with wind directions dividing counties into treatment and control groups.

The results for adult health from natural experimental studies are mixed. For example, using the Clean Air Act as an instrumental variable for air quality, Chay et al. (2003) found that, although air pollution regulations were associated with large reductions in TSPs, such reductions had little effect on either adult or elderly mortality. In contrast, Schlenker and Walker (2011) estimated the health effects of air pollution due to airline network delays in the United States and found that carbon monoxide (CO) exposure led to significant increases in hospitalization rates for asthma and respiratory diseases and in heart-related emergency room admissions that were an order of magnitude higher than conventional estimates. The effects were statistically significant for infants, the elderly and the adult population.

The evidence from developed countries has limited external validity for several reasons. First, the dose–response relationship between pollution and health might be non-linear, and is still not clear to researchers (Arceo-Gomez et al., 2012). Estimates derived from the United States and other developed countries may not be a good reference for developing countries' regulations. Second, avoidance behavior can mitigate the air pollution effect. The costs associated with avoidance behavior are relatively higher in the developing countries (Graff Zivin and Neidell, 2009; Moretti and Neidell, 2011), so the air pollution effect may differ. Third, the air pollution effect might be related to socioeconomic gradients in health (Jayachandran, 2009). Lower income, poorer medical services and less immediate emergency care may all contribute to a different (perhaps larger) air pollution effect in developing countries.

[2] For example, Beijing's smoggy days in 2013, which were intensively reported by the mass media, largely resulted from the combined effects of temperature, humidity, and wind. Most of these smoggy days occurred on cold days with light wind. If people are more likely to die from cold weather, the association between air pollution and increased mortality can be misleading unless these factors are fully controlled for.

Studies linking air pollution exposure to health in the developing countries are relatively rare. The major challenge is to collect reliable data on both pollution and mortality. Good quality air pollution and health data are difficult to come by in developing countries because of limited financial support for data collection, less coordinated government efforts, and fragmented data storage and computerization. Jayachandran (2009) dealt with this obstacle by using satellite data to track smoke from wildfires in Indonesia in 1997. She found that that increased pollution from wildfires accounted for 15,600 missing children. Foster et al. (2009) also used satellite data to derive pollution levels in Mexico, and they estimated the air pollution effect using a voluntary pollution reduction program as an instrumental variable. Greenstone and Hanna (2014) studied India's pollution regulations, and found that the most successful air pollution regulation resulted in a modest and statistically insignificant decline in infant mortality. In another study focusing on Mexico, (Arceo-Gomez et al., 2012) used temperature inversion as an instrumental variable for air pollution. They found that CO has a larger impact on infant mortality in Mexico than United States. Cesur et al. (2015) explored a quasi-experimental design provided by natural gas expansion in Turkey, and found that the improved air quality, due to switching from coal to natural gas, significantly decreased infant mortality. For China, Rich et al. (2012) compared the health status of young adults in Beijing before and during the BOG08 and found that their health status improved during the Olympic Game air regulation period. Chen et al. (2013a) found that an increase in TSP concentrations by 100 $\mu g/m^3$ led to a reduction of approximately three years in life expectancy at birth and concluded that China's winter heating policy might have caused the 500 million residents of Northern China to lose more than 2.5 billion years of life. Tanaka (2015) explored China's acid rain policies and found air pollution has a significant impact on infant mortality.

## Air pollution regulations during the BOG08

The air pollution regulations enforced during the BOG08 may be by far the largest efforts made in human history to control air quality within a short period of time (Chen et al., 2013b). To ensure good air quality during the BOG08, the Chinese government implemented a series of radical pollution regulations starting in late 2007.

In October 2007, the State Council of China issued “Measures to Ensure Good Air Quality in the 29th Beijing Olympics and Paralympics.” The Measures defined the period from November 1, 2007 to July 20, 2008 as the pre-Olympic Comprehensive Regulation period and the period from July 20 to September 20, 2008 as the Olympic Games Temporary Pollution Control period.

During the pre-Olympic Comprehensive Regulation period, multiple regulations were implemented simultaneously: (1) all coal-fired power plants in Beijing were required to install desulfurization, dust removal, and denitrification facilities; (2) the public sectors (public transit, environment and health agencies, etc.) replaced all heavy-emission vehicles; (3) oil–gas gathering units and recovery systems were installed at gas stations, oil storage facilities, and tankers; (4) the Second Beijing Chemical Plant, the Beijing Eastern Petrochemical Company, and several other polluting factories were completely shut down; (5) the government raised gas prices twice, in November 2007 and June 2008, to discourage auto vehicle usage; and (6) the Capital Steel Company was ordered to relocate, and its production of steel fell from more than 600,000 t per day to less than 200,000 t per day.

Motor vehicle exhaust emissions are the primary air pollution source in large cities. To further ensure good air quality, Beijing implemented temporary traffic control during the Olympic Games Temporary Pollution Control period. From July 1 to September 20, 2008, vehicles with yellow environmental labels (vehicles that failed to meet the European No. I standards for exhaust emissions) were banned from Beijing's roads. As a consequence, more than 300,000 heavy-emission vehicles (mostly trucks, tractors, low-speed cargo trucks, tri-wheeled motor vehicles, and motorcycles) were not allowed on the roads. From July 20 to September 20, 2008, vehicles with odd-numbered (even-numbered) license plates were allowed on the roads only on odd-numbered (even-numbered) days. Police vehicles, public transport, vehicles with Olympic passes, and a few others were exempted from the odd–even plate rule. This policy reduced the number of vehicles on the public roads of Beijing by two million vehicles per day. According to the committee of the BOG08 and the Ministry of Environmental Protection (MEP) in China (2008), total vehicle exhaust emissions decreased by more than 60 percent.[3] Traffic control significantly decreased the concentrations of fine particulates, ozone, nitrogen oxide, and other pollutants generated by auto vehicles in Beijing.

At the same time, the government required all power plants and chemical production plants to reduce their emissions by 30 percent from previous levels even though these plants were within the national emission standard to begin with. More than 20 cement production factories, more than 140 concrete mixing plants and more than 100 lime production sites were completely shut down. To further reduce particulate matter pollution, the Chinese government also halted all construction projects during the BOG08.

Because air quality in Beijing was affected by its neighboring areas, several cities and provinces (Tianjin, Hebei, Liaoning, Inner Mongolia, and Shanxi) around Beijing were also required to enforce the central government's emission control plans. All these provinces were required to retire outdated production facilities in power plants and to install desulfurization facilities. Factories were forced to reduce their production or temporarily shut down if they could not meet the national

[3] http://www.bj.xinhuanet.com/bjpd_2008/2008-09/22/content_14462703.htm.

standard before June 2008. As some Olympic soccer games were held in Tianjin, Shenyang and Qinhuangdao, similar air pollution controls were implemented in these cities as well. For example, Tianjin shut down many polluting factories before the BOG08 and enforced temporary traffic control during the BOG08. Shenyang replaced old buses prior to the Olympics and expanded green belts around construction sites. Qinhuangdao built shelter forests along the piers to reduce dust, hosed down the streets with water, and transported garbage to the landfills/incinerators every day.

The combination of these regulations effectively improved air quality in and around Beijing. Our data show that the yearly $PM_{10}$ concentrations in Beijing decreased from 152 $\mu g/m^3$ to 124 $\mu g/m^3$ (an 18-percent decrease). The improvement was particularly striking during the summer period (June-August). The summer monthly $PM_{10}$ concentrations decreased from 145 $\mu g/m^3$ in 2007 to 101 $\mu g/m^3$ in 2008, which corresponds to a 30-percent change in monthly air pollution levels. During the 17 days of the BOG08, all indicators of air quality in Beijing met national standards.

The air quality improvements have been confirmed by multiple studies, such as the United Nations Environment Programme (2009) and Chen et al. (2013b). The United Nations Environment Programme (2009) examined the air pollution data provided by the Beijing Environmental Protection Bureau and found that air quality in Beijing met the national standard less than 50 percent of the time in 2000 but more than 75 percent of the time in 2008. Through analysis of satellite images during the month of August from 2005 to 2008, the United Nations Environment Programme (2009) showed that CO and $NO_2$ concentrations decreased significantly, with $NO_2$ levels, for example, falling by 50 percent from 2005 to 2008. Chen et al. (2013b) evaluated the impact of the BOG08 on Beijing's air quality and found that the regulations effectively reduced the Air Pollution Index (API) by 30 percent in Beijing during July and August of 2008 (the BOG08 period) from a year ago. They also confirmed the improvement in air quality in Beijing using satellite-based Aerosol Optical Depth data.

## Data

### *Air quality data*

Air quality data come from monitoring sites administered by the MEP in China, which has been providing daily air quality information for 82 major cities in China since 2000. Our air quality data include information on the daily API and the primary pollutant. The API is an index for reporting daily air quality to the general public.[4] It is an overall measure of ambient air quality. A higher API score indicates a higher level of air pollution. Three individual pollutants, $PM_{10}$, $SO_2$ and $NO_2$, are used to construct the API. Ideally, we would obtain the concentrations of each air pollutant. However, specific pollutant concentrations are not publicly available. Fortunately, the method used by the MEP to construct the API allows us to recover the concentrations of the primary pollutant. In the daily API data, $PM_{10}$ is the primary pollutant for 90 percent of our daily samples. As a consequence, we are able to recover the average monthly $PM_{10}$ concentrations from the API with high accuracy. The details of calculating monthly $PM_{10}$ concentrations using the API are presented in Appendix A.

### *Mortality data*

Mortality data come from the Disease Surveillance Point System (DSPS) of the Chinese Center for Disease Control and Prevention (CDC). The DSPS was established by the Chinese government in 1978 to provide timely information on the cause and number of deaths for a sampled population. For a selected surveillance point (either a county or a city–district), the DSPS collects data on all deaths in hospitals or at home for the resident population. To represent national population and mortality trends, the DSPS adopts a multi-stage cluster population probability sampling method. The main objectives of the DSPS are to: (1) identify the number of deaths related to each disease and provide basic mortality information about the deceased for public health officials; and (2) provide feedback to evaluate the impacts of the public health interventions. The DSPS initially covered 71 counties and city–districts in 29 provinces; then it was expanded to 145 counties and city–districts in 31 provinces in 1990. Following the SARS outbreak in 2003, the DSPS was overhauled and 161 counties and city–districts are designated in the system from 2004 to the present. Currently the DSPS covers more than 81.5 million people or approximately 6 percent of the Chinese population.

According to the epidemiological literature, we divide all deaths into two large categories by their causes: air-pollution-related deaths and air-pollution-irrelevant deaths. Air-pollution-related deaths are those caused by cardio-cerebrovascular diseases and respiratory (CVR) diseases, and air-pollution-irrelevant deaths are those caused by cancer, digestive diseases,

[4] The reliability of official Chinese air quality data has been questioned by researchers. The government's unwillingness to publicize specific concentrations of pollutants makes it even more difficult for researchers to verify the reliability of the data. Chen et al. (2012) assessed the quality of China's API data and found a discontinuity at the threshold of 100 owing to the fact that a day with an API value of 100 or less is called a "blue-sky day," a threshold that local governments are incentivized to manipulate their data around. Nevertheless, Chen et al. (2012) found that the API is strongly correlated with NASA's Aerosol Optical Depth data and the China Meteorological Administration's visibility data; and such correlations do not change significantly when the API is just above or below 100. They concluded that, although the number of blue-sky days may be subject to data manipulation, the reported API did contain useful information on cross-city and over-time variations in air pollution.

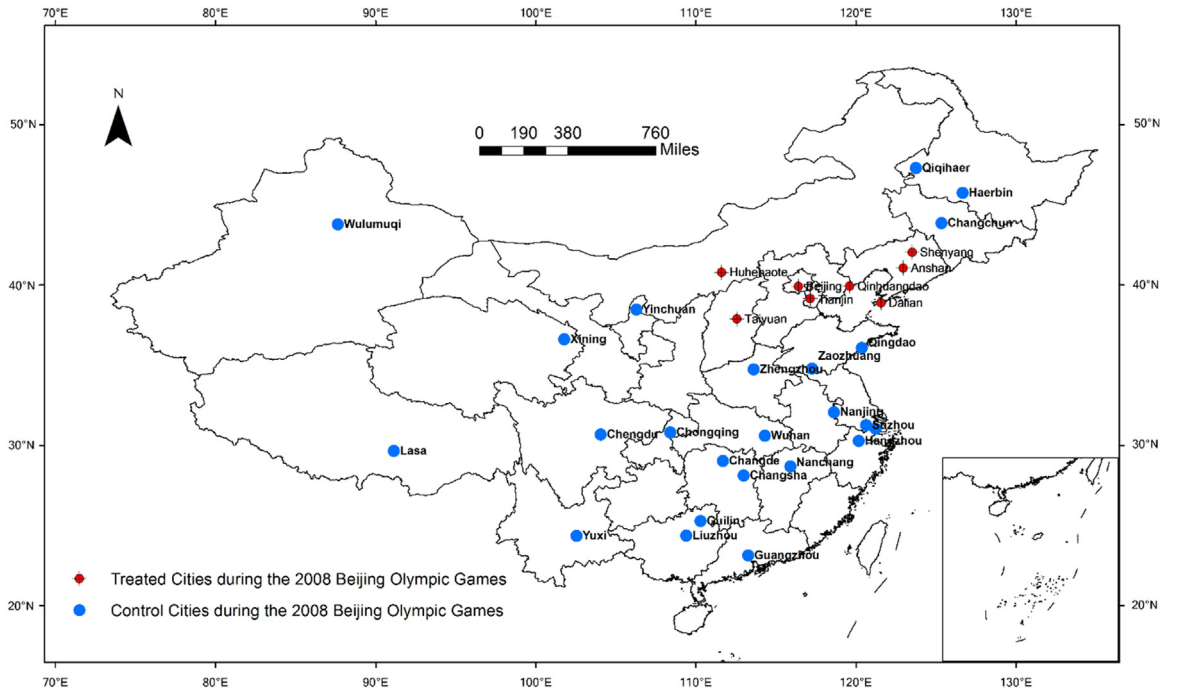


**Fig. 1.** Geographical distribution of sampled urban cities in China. Notes: this graph shows geographical locations of the treatment and control cities in our sample.

injuries, and other diseases.[5] The monthly-standardized mortality rate is defined as the number of deaths per 10,000 people per month in a designated DSPS area, adjusted by the age distribution.[6]

We match mortality data with air pollution data at the monthly level and end up with 34 urban city–districts in both data sets. Fig. 1 shows the geographical locations of these cities; they are dispersed over 26 provinces and encompass much of China's geography. The population of all these cities totals more than 200 million people. The red marks are treatment cities, while the blues marks are control cities. This study focuses on a five-year window, from January 2006 to December 2010. The average monthly standardized mortality rate is 4.12 per 10,000 people, with a standard deviation of 1.61. The monthly standardized CVR mortality rate is 1.85 per 10,000 people, with a standard deviation of 0.89.

### *Weather data*

Data on temperature and precipitation are drawn from the Global Historical Climatology Network (GHCN) project.[7] GHCN provides average monthly temperatures and precipitation levels for given longitudes and latitudes, with a minimum cell size of 0.5° by 0.5°.

For each city–district in our sample, we identify four nearest points out of the GHCN gridded data points for the entire globe based on geographical distance. We then calculate weighted averages of temperature and precipitation, using inverse squared distances as weights. For example, the precipitation of location $j$, using the nearest four points, is calculated as follows:

$$Precip_j = \sum_{k=1}^{4} \frac{Precip_k * Distance_{jk}^{-2}}{\sum_{k=1}^{4} Distance_{jk}^{-2}}$$

where $Precip_j$ is precipitation at point $j$, and $Distance_{jk}^{-2}$ is the inverse squared distance between $j$ and $k$. Summary statistics of the key variables are provided in Table 1.

[5] Even though some cancers are associated with exposure to air pollution, we think cancer mortality still works as a valid placebo because the gestation periods for most cancers are much longer than the time period covered by our study.

[6] See Appendix B on how to calculate the monthly standardized mortality rate.

[7] The data are downloaded from: http://www.ncdc.noaa.gov/data-access/land-based-station-data/land-based-datasets/global-historical-climatology-network-ghcn.

**Table 1**
Summary statistics of the main variables.

| | Mean | Std. dev. | Min | Max |
|---|---|---|---|---|
| $PM_{10}$ ($\mu g/m^3$) | 97.99 | 36.22 | 24.73 | 283.44 |
| Standardized All-Cause Mortality Rate (per 10,000) | 4.12 | 1.61 | 0.28 | 14.12 |
| Standardized CVR Mortality Rate (per 10,000) | 1.85 | 0.89 | 0.05 | 7.05 |
| Standardized Non-CVR Mortality Rate (per 10,000) | 2.27 | 0.98 | 0.18 | 11.82 |
| Standardized Cancer Mortality Rate (per 10,000) | 1.04 | 0.34 | 0.00 | 2.69 |
| Standardized Injury Mortality Rate (per 10,000) | 0.29 | 0.22 | 0.00 | 2.72 |
| Precipitation (100 mm) | 75.87 | 83.77 | 0.00 | 809.92 |
| Temperature (°C) | 13.50 | 11.10 | −20.30 | 30.89 |

Notes: All variables are measured at the monthly level. CVR stands for cardio-cerebrovascular and respiratory diseases. $PM_{10}$ concentrations are calculated from the API (see Appendix A for detailed discussion). We use the age structure in 2000 Census of China to calculate the standardized mortality rates.

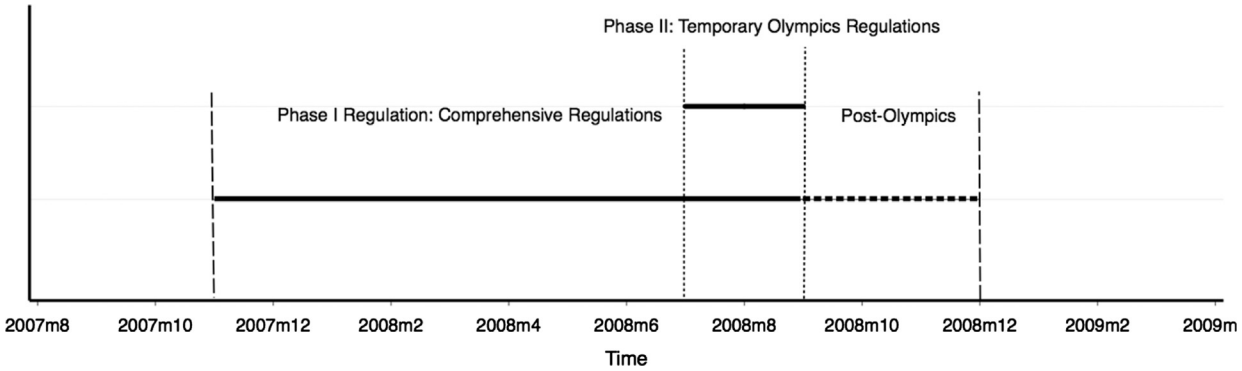


**Fig. 2.** The timeline of air pollution regulations during the BOG08. Notes: the graph indicates the starting and end time of the two air pollution regulations. The comprehensive regulations started in November of 2007 and ended in December of 2012. The traffic control started in July of 2008 and ended in September of 2008.

## Research design and model

Our analysis compares changes in mortality rates in cities that experienced large reductions in $PM_{10}$ with those in cities that experienced little or no reduction in pollution. We estimate the effects of air pollution on mortality rates using a fixed-effects instrumental variable model:

$$Y_{it} = \delta \hat{P}_{it} + X'_{it}\gamma + u_i + v_t + \varepsilon_{it} \tag{1}$$

$$P_{it} = \lambda_1 R_{it} + \lambda_2 T_{it} + X'_{it}\theta + \tau_i + \pi_t + \xi_{it} \tag{2}$$

where $P_{it}$ is the air pollution level in city $i$ at time $t$, $Y_{it}$ is the logarithm of the monthly mortality rate per 10,000 in city $i$ at time $t$. $X_{it}$ is a set of control variables, $u_i$ and $\tau_i$ are city fixed effects, $v_t$ and $\pi_t$ are year–month fixed effects, and $\varepsilon_{it}$ and $\xi_{it}$ are unobservable disturbances.$R_{it}$ is a regulation status indicator. If city $i$ is regulated at time $t$, $R_{it} = 1$; otherwise, it is 0. $T_{it}$ is the traffic control status indicator. If city $i$ enforces traffic control during the BOG08, $T_{it} = 1$; otherwise, it is 0. Both $R_{it}$ and $T_{it}$ are instrumental variables that cause changes in air pollution without directly affecting mortality.

As discussed in the previous sections, radical air pollution regulations started in November 2007, so we treat this month as the starting month of the regulation period. Some interventions, such as traffic control and temporary emission controls, were abandoned immediately after September 2008, while others, such as halt of construction projects, can have a lasting effect in the next couple of months. Since these regulations might have lasting effects on air quality, we choose December 2008, three months after the Olympics, as the ending point.[8] Thus, the regulation dummy, $R_{it}$, takes the value of 1 if a city was regulated due to the BOG08 from November 2007 to December 2008 and 0 otherwise. We also experiment with various ending points and present the results in Section "Robustness checks".

Beijing and Tianjin faced more stringent regulations than other treated cities with both cities enforcing temporary traffic control during July–September 2008. To capture the treatment intensity differences, we include a traffic control dummy $T_{it}$ as a second instrumental variable, which equals 1 during July–September 2008 for Beijing and Tianjin and 0 otherwise.[9] Fig. 2 shows the timeline of different regulations.

[8] Please refer to Section "Robustness checks" for reasons why December 2008 is chosen as the end time for our empirical analysis.

[9] The point estimate and significance of the effect of air pollution on mortality is essentially unchanged if we use only one instrumental variable, $O_{it}$. However, using two instrumental variables improves the significance of the first stage of the regression analysis.

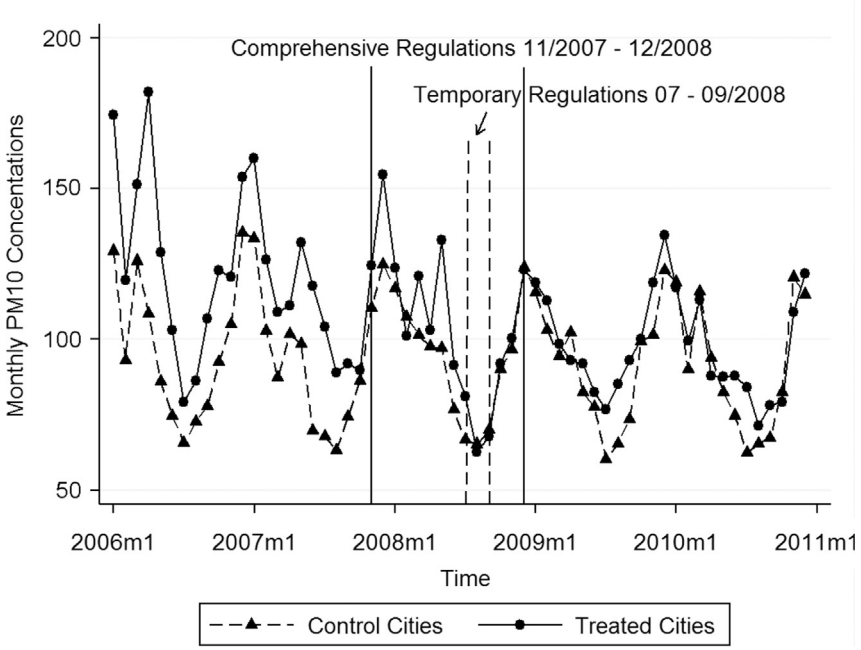


**Fig. 3.** Monthly $PM_{10}$ concentrations in treatment group and control group. Notes: the figure depicts the time trend of Monthly $PM_{10}$ concentrations for the treatment and control cities separately. The solid line represents the treated cities and the dashed line represents the control cities. November 2007 to December 2008 is the comprehensive regulation period (R), and July–September 2008 is the traffic control period (T). This figure shows that air quality in the regulated cities during the regulation period was significantly improved, while the air pollution levels in the control cities were similar across years.

In the first stage, we estimate Eq. (2) to understand how the air quality regulations affected air pollution during BOG08. The coefficient $\lambda_1$ is essentially a difference-in-difference estimator, capturing differences in the changes in air pollution levels during regulation periods (November 2007 to December 2008) and non-regulation periods (January 2006 to October 2007, January 2009 to December 2010) between the locations that were regulated and those that were not. $\lambda_2$ has a similar interpretation. We expect both $\lambda_1$ and $\lambda_2$ to be negative. In the second stage of the IV regression, we estimate the effect of air pollution on mortality. If air pollution negatively affects people's health, we expect that fewer people will die in the regulated cities than in the unregulated cities during periods of low air pollution and that $\delta_0$ will be positive. Fig. 3 shows trends in the monthly average $PM_{10}$ concentrations for both the regulated (9 city–districts) and non-regulated cities (25 city–districts) during 2006–2010. We observe a strong seasonality pattern in the trends of air quality for both treated and control groups, with better air quality in summer than in winter for both groups. In the control group, air quality is relatively stable from year to year. The average $PM_{10}$ concentrations are higher in the treated group than in the control group before 2008. In contrast, air quality improved significantly in 2008 in the treated group. Air quality in 2009 and 2010 for the treated group became slightly worse than in 2008, suggesting that the effects of the regulations on air pollution diminished over time.

## Results

In this section we present estimates of the effect of particulate matters on mortality. We first estimate the effect of $PM_{10}$ concentrations on all-cause mortality; then we differentiate the effect by cause of death, gender and age group; and finally we discuss the validity of the instrumental variables.

### *The effects of air pollution on mortality*

Table 2 presents the results from estimating Eqs. (1) and (2) using two-stage least square regression. The first three columns are estimates from the first stage. In column 1 we estimate the effect of the two instruments (the general comprehensive Olympic air pollution regulation $O_{it}$ and traffic control $T_{it}$) on $PM_{10}$ concentrations using Eq. (2). Column 1 includes no weather controls. Columns 2–3 add weather controls gradually. We include temperature, squared temperature and precipitation as weather controls because they are typical confounding factors in the associational studies. Air pollution tends to increase on both extremely hot and cold days, due to excessive energy consumption. At the same time, people are more likely to die on both extremely hot and cold days (Deschenes and Moretti, 2009). Rain can wash away pollutants in the

**Table 2**
The effect of $PM_{10}$ on the monthly standardized mortality rate.

| | First stage $PM_{10}$ ($\mu g/m^3$) | | | Second stage Mortality (log, per 10,000) | | |
|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| Regulated (R) | −5.64** | −5.58*** | −5.53*** | | | |
| | (2.23) | (2.15) | (2.09) | | | |
| Traffic control (T) | −18.61*** | −20.20*** | −19.18*** | | | |
| | (4.34) | (4.52) | (4.39) | | | |
| PM10 (10 $\mu g/m^3$) | | | | 0.0961** | 0.0819** | 0.0836** |
| | | | | (0.0443) | (0.0385) | (0.0396) |
| Temp and Sq. | N | Y | Y | N | Y | Y |
| Precipitation | N | N | Y | N | N | Y |
| City FE | Y | Y | Y | Y | Y | Y |
| Year–month FE | Y | Y | Y | Y | Y | Y |
| Observations | 1930 | 1930 | 1930 | 1930 | 1930 | 1930 |
| *F*-statistics | 27.64 | 25.73 | 25.18 | | | |
| $R^2$ | 0.67 | 0.69 | 0.69 | 0.190 | 0.361 | 0.349 |

Notes: This table reports the instrumental variable regression coefficients and standard errors. Air regulations and traffic control are used as the instrumental variables for monthly PM10 concentrations. The dependent variable is the logarithm of monthly standardized mortality rate per 10,000 people. Columns 1–3 report the first stage regression results, and columns 4–6 report the second stage results. The variance–covariance matrix allows for arbitrary autocorrelation within each city.

*** Asterisks, indicate the 1 percent significance level.

** Asterisks, indicate the 5 percent significance level.

air, so it is often negatively correlated with air pollution. Rainfall might influence mortality because it changes the humidity and disease environment. All specifications adjust for both city–district fixed effects and year–month fixed effects.

Both general regulations and traffic control are estimated to have strong effects on air pollution. The overall model explains approximately 70 percent of the variations in $PM_{10}$ concentrations. The estimated coefficients of the instrumental variables are remarkably stable across all three specifications.[10] Together, general regulations and traffic control reduce monthly $PM_{10}$ concentrations by approximately 26 $\mu g/m^3$ $\mu g/m^3$ in a short period. The results suggest that air quality regulations had a large effect on the level of $PM_{10}$ concentrations.

The next three columns show the results of air pollution effect on mortality. The control variables and city and year–month fixed effects are the same as in the first stage. We use the logarithm of the mortality rate as the dependent variable. The pollution variable, $PM_{10}$, is estimated to have strong effects on mortality and are robust to controlling for weather. The estimates are statistically significant at the 5 percent level and imply that a 10-$\mu g/m^3$ decline in monthly $PM_{10}$ concentrations results in 8.36–9.61 percent reduction in monthly mortality. The overall model explains 19–36 percent of the variations in mortality. The results provide causal estimates of the effect of particulate matter pollution on standardized mortality in China's urban areas. In Appendix Table C.1, we find similar results using the API as the air pollution measure.

*Heterogeneous effect of air pollution*

As has been documented in the literature (Chen et al., 2013a; Ebenstein et al., 2015; Pope et al., 1995), particulate air pollution has heterogeneous impacts on different diseases. Deaths caused by CVR diseases are found to be associated with both short- and long-term exposure to particulate air pollution. In contrast, associations are rarely found between particulate matters and non-CVR mortality. Thus, we estimate the effect of $PM_{10}$ on different mortality rates due to different causes of death. Table 3 presents the estimation results of $PM_{10}$ on CVR, non-CVR, cancer, and injury mortality based on the most restrictive specification in Table 2. The estimates in the first column implies that a 10-$\mu g/m^3$ reduction in monthly $PM_{10}$ concentrations leads to an 8.78 percent drop in the CVR mortality rate. The estimated coefficients of air pollution on non-CVR and cancer mortality in columns 2 and 3 are much smaller and statistically insignificant. In column 4, the estimate for injury mortality is statistically insignificant and close to zero in magnitude. The results using the API are consistent with those using $PM_{10}$, as reported in Appendix Table C.2.

To account for the gender difference we analyze males and females separately. Table 4 presents the gender-specific estimates for all-cause, CVR, and non-CVR mortality. The impacts of air pollution on gender-specific mortality rates are very similar to what we find using the full sample, with the estimated effects of a 10-$\mu g/m^3$ reduction in monthly $PM_{10}$ concentrations leading to a 8.47 and a 8.28 percent decrease in male and female mortality. The decrease in mortality is primarily driven by fewer CVR deaths for both sexes.

[10] We also experiment with higher order terms for temperature and precipitation. These specifications appear to have little impact on the estimates. More robustness checks are in Section VII.

**Table 3**
The effect of PM10 on disease-specific mortality rates.

| | CVR (1) | Non-CVR (2) | Cancer (3) | Injury (4) |
|---|---|---|---|---|
| $PM_{10}$ (10 μg/m$^3$) | 0.0878** | 0.0461 | 0.0341 | −0.00364 |
| | (0.0384) | (0.0338) | (0.0230) | (0.0128) |
| Weather | Y | Y | Y | Y |
| City FE | Y | Y | Y | Y |
| Year–month FE | Y | Y | Y | Y |
| Observations | 1930 | 1930 | 1930 | 1930 |

Notes: This table reports the instrumental variable regression coefficients and standard errors. Air regulations and traffic control are used as the instrumental variables for monthly PM10 concentrations. The dependent variables are the logarithm of monthly standardized mortality rates per 10,000 people for four different types of causes of death: cardio-cerebrovascular and respiratory (CVR) diseases, non-CVR diseases, cancer, and injury. The variance–covariance matrix allows for arbitrary autocorrelation within each city.
** Indicate the 5 percent significance level.

**Table 4**
The effect of $PM_{10}$ on male and female mortality rates

| | Male | | | Female | | |
|---|---|---|---|---|---|---|
| | (1) All-Cause | (2) CVR | (3) Non-CVR | (4) All-Cause | (5) CVR | (6) Non-CVR |
| PM10 (10 $\mu g/m^3$) | 0.0847** | 0.0871** | 0.0474 | 0.0828** | 0.0897** | 0.0425 |
| | (0.0414) | (0.0387) | (0.0367) | (0.0392) | (0.0410) | (0.0319) |
| Weather | Y | Y | Y | Y | Y | Y |
| City FE | Y | Y | Y | Y | Y | Y |
| Year-Month FE | Y | Y | Y | Y | Y | Y |
| Observations | 1930 | 1930 | 1930 | 1930 | 1930 | 1930 |

Notes: This table reports the instrumental variable regression coefficients and standard errors. Air regulations and traffic control are used as the instrumental variables for monthly PM10 concentrations. The dependent variables are the logarithm of monthly standardized mortality rates per 10,000 people for all causes, cardio-cerebrovascular and respiratory (CVR) diseases, and non-CVR diseases. Columns 1–3 and 4–6 show estimates for males and females separately. The variance-covariance matrix allows for arbitrary autocorrelation within each city.
** Indicate the 5 percent significance level.

We investigate the nonlinear effects across age groups by examining the impact of air pollution on mortality rates separately for different age groups. The overall findings are summarized in Fig. 4 which shows a U-shaped effect: air pollution has a greater impact on the most vulnerable groups (i.e. infants, children, and the elderly) than other age groups. The corresponding regression results are reported in Table 5. Column 1 of Table 5 shows that the largest air pollution impact is on children younger than 5 years of age. A 10-μg/m$^3$ decrease in monthly $PM_{10}$ concentrations reduces the monthly mortality rate for age group 0–4 by 19 percent.[11] The air pollution effect is also statistically significant on children ages 5–9. For the elderly ages 75–80, the results show that the impacts of a 10-μg/m$^3$ change in $PM_{10}$ concentrations range from 15 to 17 percent, with a greater effect observed among those ages 75–79. For those ages 10-74, we fail to find statistically significant impact on them, possibly due to their higher tolerance to exposure and/or their stronger body condition. Columns 2–3 report the regressions results separately for CVR and non-CVR mortality rates. For the two young age groups, the impacts of air pollution are through non-CVR diseases rather than CVR diseases. In contrast, air pollution effect on the elderly is due to CVR diseases. This difference is reasonable because infants and young children's immune system and other body systems are not well developed yet. Thus, they are more likely to die from acute diseases such as infections, rather than chronic diseases such as chronic obstructive pulmonary diseases, heart attack, or stroke. In our data, the most common cause of death for the infant group is neonatal and perinatal diseases (which include a variety of complications of diseases); and for the children group, many of them die from infections, injuries, and congenital malformations. Based on these findings, we cautiously conclude that air pollution might affect infants and children in ways that are different from the elderly. In Appendices C.3 and C.4, we find similar results using the API as the air pollution indicator.

[11] The air pollution effect for infants and young children should be interpreted with caution because the data quality of this age group is not as good as that of other age groups. The subpar data quality is due to the social culture and birth control policies in China. For example, infant death is considered to be a big misfortune to a family so some people are unwilling to disclose the information. For another example, due to the son preference and one child policy, some infants' births and deaths have no official records. Even though we have adjusted for the under-reporting issue when calculating the mortality rate, these adjustments might be inadequate for infant and young children.

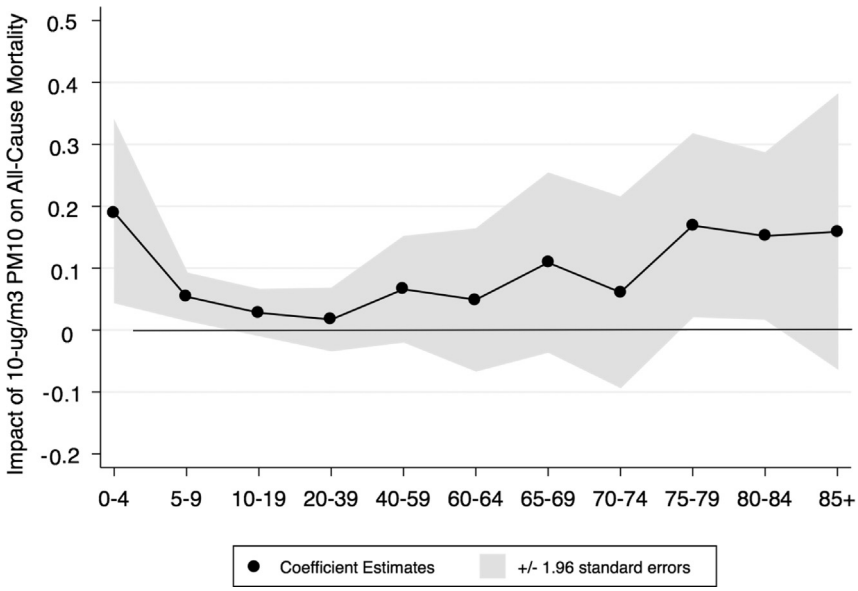


**Fig. 4.** The Effect of $PM_{10}$ (10 μg/$m^3$) on Mortality by Different Age Groups. Notes: The figure depicts the effects of a 10-μg/$m^3$ change in monthly $PM_{10}$ concentrations on by-age-group mortality rates (log, per 10,000). Each dot is an estimated impact of a 10-μg/$m^3$ change in $PM_{10}$ concentrations on the percent change in mortality rate for an age group.

**Table 5**
The effect of $PM_{10}$ on age-specific mortality rates.

| | All-cause (1) | CVR (2) | Non-CVR (3) |
|---|---|---|---|
| Age 0–4 | 0.190*** | -0.003 | 0.192*** |
| | (0.074) | (0.011) | (0.073) |
| Age 5–9 | 0.054*** | 0.013 | 0.041** |
| | (0.019) | (0.008) | (0.018) |
| Age 10–19 | 0.028 | 0.004 | 0.022 |
| | (0.019) | (0.006) | (0.020) |
| Age 20–39 | 0.017 | 0.011 | 0.009 |
| | (0.026) | (0.013) | (0.022) |
| Age 40–59 | 0.066 | 0.056 | 0.041 |
| | (0.043) | (0.034) | (0.038) |
| Age 60–64 | 0.049 | 0.021 | 0.048 |
| | (0.058) | (0.048) | (0.060) |
| Age 65–69 | 0.109 | 0.160* | 0.035 |
| | (0.073) | (0.082) | (0.071) |
| Age 70–74 | 0.061 | 0.099 | 0.013 |
| | (0.078) | (0.086) | (0.085) |
| Age 75–79 | 0.169** | 0.204** | 0.083 |
| | (0.075) | (0.085) | (0.072) |
| Age 80–84 | 0.152** | 0.141* | 0.096 |
| | (0.068) | (0.078) | (0.063) |
| Age 85+ | 0.159 | 0.209** | 0.019 |
| | (0.113) | (0.107) | (0.134) |

Notes: This table reports the instrumental variable regression coefficients and standard errors. Each cell represents a separate regression of monthly age-specific mortality rates (log, per 10,000 people) on $PM_{10}$ concentrations (10-μg/$m^3$). Air regulations and traffic control are used as the instrumental variables for monthly PM10 concentrations. The dependent variables are the logarithm of monthly age-specific mortality rates per 10,000 people for all causes, cardio-cerebrovascular and respiratory (CVR) diseases, and non-CVR diseases. The specification corresponds to the column 6 specification in Table 2. The variance–covariance matrix allows for arbitrary autocorrelation within each city.

*** Indicate the 1 percent significance level.

** Indicate the 5 percent significance level.

* Indicate the 10 percent significance level.

**Table 6**
The effect of air pollution regulations on mortality rates.

| | All-cause (1) | CVR (2) | Non-CVR (3) | Cancer (4) | Injury (5) |
|---|---|---|---|---|---|
| Regulated ($R$) | −0.0807 | | | −0.0884* | −0.0468 |
| 0.00690 | | | | | −0.0404 |
| | (0.0478) | (0.0477) | (0.0394) | (0.0173) | (0.0267) |
| Traffic control ($T$) | −0.0649 | −0.0580 | −0.0293 | −0.00686 | −0.00548 |
| | (0.0443) | (0.0434) | (0.0420) | (0.0202) | (0.0313) |
| $F$-Stat. ($H_0$: $R=T=0$) | 3.12* | 4.57** | 0.91 | 0.11 | 1.26 |
| Weather | Y | Y | Y | Y | Y |
| City FE | Y | Y | Y | Y | Y |
| Year–month FE | Y | Y | Y | Y | Y |
| Observations | 1932 | 1932 | 1932 | 1932 | 1932 |

Notes: This table reports the reduced-form coefficients and standard errors. The dependent variables are the logarithm of monthly age-specific mortality rates per 10, 000 people for all causes, cardio-cerebrovascular and respiratory (CVR) diseases, non-CVR diseases, cancer, and injury. The variance-covariance matrix allows for arbitrary autocorrelation within each city.

** Asterisks, indicate the 5 percent significance level.

* Asterisks, indicate the 10 percent significance level.

*Validity of instrumental variables*

The biggest concern about the identification strategy is that the regulations during the BOG08 might have led to not only air quality improvement, but also other factors affecting health. For example, it could be that medical treatments became more available in the regulated cities during the BOG08, thus less people died. For air pollution regulations to be valid instruments, they must affect mortality only through their effects on air pollution. We address the validity of the instrumental variables in two ways.

First, we check whether the results are robust to the inclusion of typical confounders in air pollution studies. If controlling for other factors changes the estimates, the validity of the instruments would be questionable and the estimates might be biased. Weather conditions are typical confounders in estimating the health effects of air pollution because they change air pollution levels and also affect human health. In cross-sectional and time-series studies, including weather controls will substantially change the air pollution effects. The first-stage results in Table 2 show that the estimated coefficients of the two instruments are statistically significant in all three specifications and including weather controls has little effect on them. This suggests that the two instrumental variables are not correlated with weather conditions. Furthermore, the coefficient of $PM_{10}$ only slightly decreases when the weather controls are included. In other words, these weather control variables are not correlated with variations in air pollution induced by the regulations. In the next section (Section "Robustness checks"), we also conduct sensitivity analysis to show that the results are still robust when we include regional trends and a set of yearly socioeconomic variables. In conclusion, these results show no evidence that air pollution variations induced by regulations are correlated with unobserved potential confounding factors.

Second, our empirical results suggest that the reduced mortality during the BOG08 is likely only caused by the improved air quality rather than other factors. If the regulations not only improved air quality, but also significantly changed other health-influencing factors, such as availability of medical services, the air-pollution-irrelevant mortality rates during the BOG08 would decrease as well. However, the IV regression results in Table 3 show that non-CVR, cancer, and injury mortality rates are not affected by the air pollution regulations.

We further estimate the effect of air pollution on mortality using a reduce-from model to show that these regulations only affect air-pollution-related deaths:

$$Y_{it} = \lambda_1 R_{it} + \lambda_2 T_{it} + X'_{it}\theta + u_i + v_t + \varepsilon_{it} \tag{3}$$

Table 6 reports the results. The regulations have negative impact on all-cause and CVR mortality, suggesting that fewer people died in the regulated cities during the regulated period than in the unregulated period. We test the joint significance of the two instrumental variables and reject the null hypothesis at the 10 percent level for all-cause mortality (column 1) and at the 5 percent level for CVR mortality (column 2). In contrast, we fail to reject the null hypothesis that both instruments are jointly zero for non-CVR, cancer, and injury mortality. In other words, the BOG08 regulations only decreased CVR mortality, but not non-CVR, cancer, and injury mortality, in regulated cities.

Since both city fixed effects and year–month fixed effects are included in Eq. (3). The coefficients reported in Table 6 are, in essence, differences-in-differences (DID) estimates. Fig. 5 depicts the differences in mortality rates between the treatment and control cities in different years. As the whole year of 2008 was regulated for the treatment cities, we compare differences in CVR, non-CVR, cancer, and injury mortality rates between the treated and control cities in 2008 with those in 2006 and 2010. In Panel A, the thick solid line, which represents differences in CVR mortality rate between the treated and control cities in 2008, is lower than the other two lines representing 2006 and 2010 respectively. This suggests that the treatment had a large impact on CVR mortality rate. In contrast, in panels B–D, the differences in non-CVR, cancer, and injury mortality rates between the treated and control cities are similar in all years. These patterns are consistent with our natural

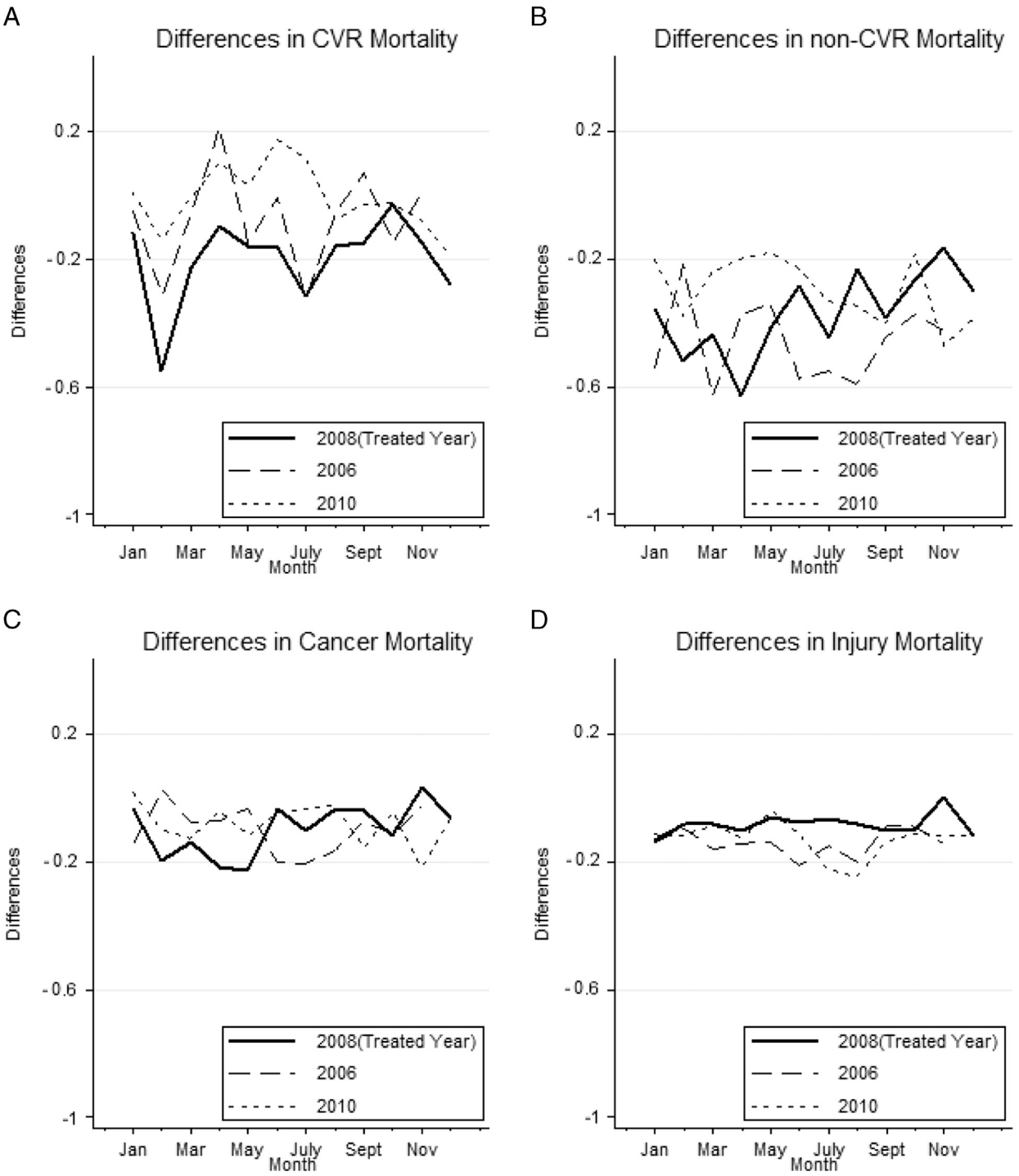


**Fig. 5.** Mortality rate differences between the treatment cities and control cities. Notes: The figure shows the differences in the mortality rates between treated and control cities in different years. The solid line indicates the mortality differences between treated and control cities in 2008. Panel A shows that, compared with other years, cardio-cerebrovascular and respiratory (CVR) mortality difference was the lowest in 2008. Panels B–D show that the differences in non-CVR mortality, injury mortality and cancer mortality between treated and control cities in 2008 were similar to other years.

experimental design that during the regulation period, the treated cities experienced a significant improvement in health outcomes than the control cities.

The underlying identification assumption for DID models is that the pre-treatment trends in mortality for the treatment and control groups are parallel. We test this assumption by including a set of leads and lag(s) of the treatment (air quality

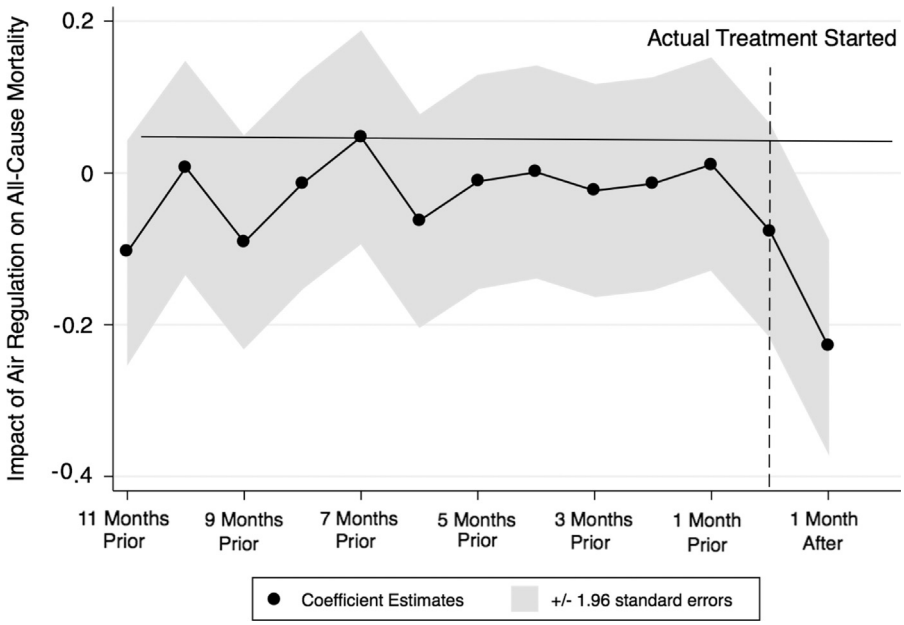


**Fig. 6.** Estimated effect of implied Olympic regulations on monthly standardized mortality rate (log, per 10,000) for Months before, during and after Regulation. Notes: This figure shows the estimated effect of implied Olympic regulations on monthly standardized mortality rate (log, per 10,000). Each dot is an estimated coefficient of a dummy variable indicating the number of month(s) before or after the actual treatment took place. We include 11 leads and 1 lag of the treatment indicator dummies in the regression. All the leads of treatment indicators are statistically indifferent from zero, suggesting that mortality trends between the regulated cities and control cities are parallel during the pre-treatment periods. The (one-month) lagged treatment variable is negative and statistically significant, implying that overall mortality started to decrease in the treatment group one month after the regulations took place.

regulations).

$$Y_{it} = \sum_{j=-n}^{m} \lambda_j D_{it}(t = k_i - j) + X'_{it}\theta + u_i + v_t + \varepsilon_{it} \tag{4}$$

where $k_i$ denotes the time at which the treatment is being switched on in city $i$ (November of 2007), and the "treatment variables" $D_{it}(t = k_i - j)$ take on the value 1 in city $i$ and period $k_i - j$ (or $j$th period before the start of the treatment in city $i$) and is zero elsewhere. We include $m$ leads and $n$ lags treatment dummies in the regression. $\lambda_j$ is the coefficient on the $j$th lead or lag. A test of the parallel trend assumption is $\lambda_j = 0$ for all $j > 0$. In other words, the coefficients on all leads of the treatment indicator should be statistically indifferent from zero. We include up to 11 leads ($m = 11$) and 1 lag ($n = 1$) "treatment variables" in the regression. The estimated coefficients and their corresponding 95% confidence intervals are plotted in Fig. 6. Each dot is an estimated coefficient of a treatment dummy variable indicating the number of month(s) prior or after the actual treatment took place. We find that none of the coefficients of the 11 leads is statistically significant. The results indicate that we fail to reject the parallel trend assumption in this DID setting. The (one-month) lagged treatment variable is negative and statistically significant, implying that overall mortality started to decrease in the treatment group one month after the regulations took place.

## Robustness checks

In this section, we provide a variety of robustness checks for our main results. First, we experiment with different end times of air pollution regulations. Second, we drop the data of July and August 2008 to further address the concern that our results may be driven by some temporary factors during the traffic control period such as more timely medical treatment and improved traffic conditions. Third, we control for a set of regional time trends to account for heterogeneous health conditions across regions. Fourth, we include a set of socio-economic characteristics in the model. Lastly, we exclude cities that are too far from the treated cities.

### *Choices of the end time*

As we discussed in Section III, there is no official announcement of the end of the air quality regulations. Some regulations, such as traffic control and temporary emission restrictions, were abandoned immediately after the BOG08; while

**Table 7**
Robustness check I: choices of different end times.

| | Overall (1) | CVR (2) | Non-CVR (3) |
|---|---|---|---|
| *Different End Times of the Treatment Period* | | | |
| September of 2008 | 0.0734** | 0.0730** | 0.0413 |
| | (0.0313) | (0.0298) | (0.0276) |
| October of 2008 | 0.0784** | 0.0776** | 0.0451 |
| | (0.0351) | (0.0328) | (0.0309) |
| November of 2008 | 0.0814** | 0.0827** | 0.0460 |
| | (0.0376) | (0.0358) | (0.0324) |
| **December of 2008** | **0.0836**** | **0.0878**** | **0.0461** |
| | **(0.0396)** | **(0.0384)** | **(0.0338)** |
| January of 2009 | 0.0801** | 0.0849** | 0.0434 |
| | (0.0386) | (0.0370) | (0.0339) |
| February of 2009 | 0.0783** | 0.0860** | 0.0396 |
| | (0.0390) | (0.0374) | (0.0341) |
| March of 2009 | 0.0758** | 0.0857** | 0.0363 |
| | (0.0381) | (0.0355) | (0.0335) |
| April of 2009 | 0.0693* | 0.0791** | 0.0333 |
| | (0.0361) | (0.0326) | (0.0322) |

Notes: This table reports the instrumental variable regression coefficients and standard errors. Air regulations and traffic control are used as the instrumental variables for monthly PM10 concentrations. Each cell represents a separate regression of the monthly standardized mortality rate (log, per 10,000 people) on $PM_{10}$ concentrations (10-$\mu g/m^3$). The dependent variables are the logarithm of monthly standardized mortality rates per 10,000 people for all causes, cardio-cerebrovascular and respiratory (CVR) diseases, and non-CVR diseases. Each row indicates a different choice of end time for the air pollution regulation period. The specification corresponds to the column 6 specification of Table 2. The variance–covariance matrix allows for arbitrary autocorrelation within each city.

** Asterisks, indicate the 5 percent significance level.

* Asterisks, indicate the 10 percent significance level.

others, such as plant renovation and relocation, may have longer impact on air quality than temporary controls. Therefore, our findings might be sensitive to the end time of the treatment period.

To address this concern, we experiment with eight different end times from September 2008 to April 2009 using the most restrictive specification in Table 2. Table 7 summarizes the estimates. Most coefficients are statistically significant at 5 percent levels and are remarkably similar across various end times. The estimated coefficients of $PM_{10}$ range from 6.9 to 8.4 percent for all-cause mortality and from 7.3 to 8.8 percent for CVR mortality. In our main analysis, we choose December 2008 as the end time for our main specification because (1) the end of the calendar year is often the end of temporary policies; and (2) three months after the BOG08 should be long enough for normal production activities to resume.

*Ruling out temporary confounding factors during the BOG08*

A potential threat to our findings is that the effect of air pollution on mortality might be driven by temporary factors during the BOG08. Such factors include increased exposure to outdoor air pollution of spectators during the BOG08, increased incidences of heart attack associated with the excitement of sporting events, more timely medical treatment associated with traffic control, and greater availability of doctors owing to a reduced number of patients due to restriction on entering the treatment cities during the BOG08 period. The overall bias associated with these factors could be negative or positive.

The BOG08 were held between August 8th and 24th and the traffic control was enforced in both July and August. As shown in Fig. 3, the largest air quality improvement occurred in July, and air quality peaked in August. To eliminate bias caused by potential confounding factors during the 17-day period, we exclude July and August 2008 from the sample. If dropping these two months has a large impact (either positive or negative) on the estimates of $PM_{10}$, confounding factors correlated with these two-month periods could potentially bias our results.

The regression results excluding observations of July and August 2008 are reported in the first panel of Table 8. The first three columns present estimates for all-cause mortality and the next three columns are for CVR mortality. The new coefficient estimates for $PM_{10}$ are only slightly smaller than those in Table 2 and Table 3. For all-cause mortality, the estimated coefficients of $PM_{10}$ range from 7.2 to 7.5 percent and are statistically significant at the 10 percent level. For CVR mortality, the coefficients of $PM_{10}$ are also statistically significant at the 5 percent level in all specifications. Thus, excluding data of July and August 2008 has a negligible impact on the effects of air pollution, ruling out the possibility that our findings are due to temporary activities during the BOG08.

*Regional trends*

China is a vast country and health-related conditions vary substantially across regions. The mortality rates and air quality in different regions may, for various reasons, follow different trends. For example, some regions may experience epidemics

**Table 8**
Robustness check II: $PM_{10}$ and mortality rate.

| | Overall mortality | | | CVR mortality | | |
|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| *A. Eliminating the game period—July and August of 2008* | | | | | | |
| $PM_{10}$ | 0.0747* | 0.0722* | 0.0744* | 0.0806** | 0.0784** | 0.0814** |
| | (0.0413) | (0.0396) | (0.0412) | (0.0394) | (0.0384) | (0.0401) |
| Observations | 1,860 | 1,860 | 1,860 | 1,860 | 1,860 | 1,860 |
| $R^2$ | 0.363 | 0.422 | 0.407 | 0.400 | 0.452 | 0.433 |
| *B. Controlling for regional trends* | | | | | | |
| $PM_{10}$ | 0.0982** | 0.0813** | 0.0830** | 0.104** | 0.0867** | 0.0890** |
| | (0.0493) | (0.0411) | (0.0422) | (0.0485) | (0.0415) | (0.0427) |
| Observations | 1,930 | 1,930 | 1,930 | 1,930 | 1,930 | 1,930 |
| $R^2$ | 0.174 | 0.368 | 0.356 | 0.221 | 0.410 | 0.393 |
| *C. Controlling for yearly socioeconomic characteristics* | | | | | | |
| $PM_{10}$ | 0.0784** | 0.0688** | 0.0699** | 0.0749** | 0.0656** | 0.0669** |
| | (0.0314) | (0.0285) | (0.0297) | (0.0333) | (0.0305) | (0.0318) |
| Observations | 1,822 | 1,822 | 1,822 | 1,822 | 1,822 | 1,822 |
| $R^2$ | 0.238 | 0.369 | 0.361 | 0.458 | 0.554 | 0.547 |
| *D. Sub-sample analysis (cities in Tibet, Yunnan and Guangxi are excluded)* | | | | | | |
| $PM_{10}$ | 0.0850* | 0.0668* | 0.0665* | 0.0883** | 0.0712** | 0.0716** |
| | (0.0455) | (0.0343) | (0.0346) | (0.0409) | (0.0329) | (0.0336) |
| Observations | 1,668 | 1,668 | 1,668 | 1,668 | 1,668 | 1,668 |
| $R^2$ | 0.183 | 0.391 | 0.393 | 0.353 | 0.521 | 0.518 |
| Temp. and its Square | N | Y | Y | N | Y | Y |
| Precipitation | N | N | Y | N | N | Y |
| City FE | Y | Y | Y | Y | Y | Y |
| Year–month FE | Y | Y | Y | Y | Y | Y |

Notes: This table reports the instrumental variable regressions coefficients and standard errors for four robustness checks. Each panel indicates a different robustness check to our main results in column 6 of Table 2. Air regulations and traffic control are used as the instrumental variables for monthly PM10 concentrations. Each cell represents a separate regression of the monthly standardized mortality rate on $PM_{10}$ concentrations (10-$\mu g/m^3$). The dependent variable is the logarithm of monthly standardized mortality rate per 10, 000 people for all causes and cardio-cerebrovascular and respiratory (CVR) diseases. Weather controls are added gradually. The variance–covariance matrix allows for arbitrary autocorrelation within each city.

** Asterisks, indicate the 5 percent significance level.

* Asterisks, indicate the 10 percent significance level.

that lead to extra deaths while others do not; or some regions may experience decreases in mortality rates due to faster economic growth than others. To address these concerns, we control for a set of regional time trends in the model.

We categorize the 34 city–districts into five groups based on the treatment status and the regional economic development according to the Development Research Center of the State Council. These five groups are regulated cities, northeastern cities, coastal cities, inland central cities, and inland western cities. The second panel of Table 8 presents the results with regional trends. The estimated coefficients of $PM_{10}$ are robust across different specifications. In the most restrictive specification, the estimated $PM_{10}$ coefficients are 8.3 and 8.9 percent for all-cause mortality and CVR mortality, respectively, which are nearly identical to the corresponding estimates in Table 2 and Table 3. This analysis, again, shows that the instrumental variables are likely to be exogenous.

### *Socio-economic characteristics*

Given the data constraints, we do not have socio-economic characteristics measured at the monthly level. Instead, we include a set of yearly socioeconomic controls to check if our findings are sensitive to the socioeconomic differences between the treatment and control cities. We include per capita GDP, the share of manufacturing production, gross fixed-assets investment per capita, and the number of hospital beds per capita in the regressions.[12] Per capita GDP and gross fixed-assets investment per capita capture mortality differences across cities due to different economic conditions. The share of manufacturing production represents the economic structure of the local economy. The number of hospital beds per capita serves as a measure of the availability of local medical resources. Socioeconomic characteristics are missing for several control cities. As a consequence, the sample size of the robustness check decreases to 1822 from 1930.

[12] The socioeconomic variables are collected by authors from China's city statistical yearbooks from 2006 to 2010.

Results are presented in the third panel of Table 8. We find that the estimated coefficients of $PM_{10}$ are slightly smaller when we control for socioeconomic characteristics. They are statistically significant at the 5 percent level for all-cause mortality and at the 10 percent level for CVR mortality. The smaller coefficients of air pollution might be caused by the sample difference. This analysis shows that air pollution effects are still robust even after controlling for important socio-economic factors that would mitigate the health effect of a non-random change in air pollution. We conclude that air pollution variations induced by regulations are also unlikely to be correlated with other potential unobserved confounding factors.

*Sub-sample analysis*

As shown on the map (Fig. 1), our treatment cities are clustered in the northern part of China. However, some control cities are located far away from the treatment cities, such as Lhasa in Tibet, Yuxi in Yunan province, and Guilin in Guangxi province. To address this concern, we exclude these cities and examine whether our findings remain the same without these distant and perhaps not very comparable cities. In panel D of Table 8, we show that the results of the sub-sample regression are comparable to those of the full-sample and robust in different specifications. In Appendix Table C.4, we report the robustness check using the API.

## Comparison with estimates from associational models

*Cross-sectional models and fixed-effects models*

In this section we compare our results with those in associational studies. The major concern in estimating the health effects of air pollution in the cross-sectional and panel (fixed-effects) models is that air pollution may be correlated with various omitted variables.

A cross-sectional model can be written as follows:

$$Y_{it} = \delta P_{it} + X_{it}'\eta + u_{it}, u_{it} = v_i + \varepsilon_{it} \tag{5}$$

The effect of air pollution on health outcomes is captured by $\delta$ if air pollution is uncorrelated with the unobserved disturbance, $E[P_{it}u_{it}] = 0$. However, as air quality is not randomly assigned across locations, this condition may not hold. For example, if air pollution is positively associated with other types of unobserved pollution (e.g., water pollution or hazardous waste), the estimates will be biased upward. If polluted areas are relatively wealthy and have superior medical and sanitation facilities, the cross-sectional estimates will be biased downward.

In Table 9, we estimate the cross-sectional model. We first estimate the association between $PM_{10}$ and mortality separately for each year, using three different specifications with different weather controls, then we stack five years of data and estimate a pooled OLS regression model.

In the specification without weather control variables (the first column), the estimated effects of $PM_{10}$ vary widely across the years. The estimates vary from an insignificant $-0.002$ in 2009 to a significant 0.01 in 2010. And none of the estimated coefficients is statistically significant except for the 2010 sample. Columns 2 and 3 present the results from specifications that include temperature and precipitation. Controlling for weather conditions has a large impact on the estimates. When precipitation and temperature are controlled for, the coefficients of air pollution variables become negative and statistically significant using the entire sample. The OLS estimates are very sensitive to the year analyzed and the set of variables used as controls, suggesting that the omitted variables may play an important role in cross-sectional models.

Fixed-effects models remove bias from time-invariant factors and are useful when time-invariant factors explain most of the variations in an outcome variable. However, if changes in air pollution are correlated with changes in other unobserved factors (such as temperature, humidity, and other pollutants) that also affect health outcomes, the fixed-effects estimates will be biased as well.

In a fixed-effects model, we estimate

$$Y_{it} = \delta P_{it} + X_{it}'\eta + u_i + v_t + \varepsilon_{it} \tag{6}$$

Table 10 summarizes the regression results for the fixed-effects model. In the first column, we only control for city fixed effects $u_i$, and the effect of air pollution is positively and statistically significantly associated with a higher mortality rate at the 1 percent level. When we also include weather variables (the second column), the estimated coefficients decrease by more than two-thirds and become statistically insignificant. That is, including the weather variables in the city fixed-effects model still substantially affects the estimated effects of air pollution. In the third column, we control for both city fixed effects $u_i$ and year–month fixed effects $v_t$. The estimated air pollution effect becomes insignificant at the conventional level and close to zero in magnitude. The estimates for CVR mortality follow a similar pattern. The results suggest that air pollution variations are correlated with fluctuations in weather conditions which may affect mortality directly or through channels other than air pollution. The insignificant and close-to-zero estimates suggest that attenuation bias is magnified in fixed effects models.

**Table 9**
Associations between $PM_{10}$ and the mortality rate.

| Year | Overall mortality | | |
|---|---|---|---|
| | (1) | (2) | (3) |
| 2006 | 0.0098** | −0.0079 | −0.0080 |
| | (0.0042) | (0.0050) | (0.0051) |
| 2007 | 0.0058 | −0.0064 | −0.0071 |
| | (0.0041) | (0.0051) | (0.0052) |
| 2008 | 0.0073 | −0.0101* | −0.0097* |
| | (0.0045) | (0.0051) | (0.0052) |
| 2009 | −0.0020 | −0.0147*** | −0.0147*** |
| | (0.0043) | (0.0048) | (0.0049) |
| 2010 | 0.0099*** | 0.0003 | 0.0002 |
| | (0.0037) | (0.0043) | (0.0044) |
| 2006–2010 | 0.0074*** | -0.0060*** | −0.0062*** |
| | (0.0019) | (0.0023) | (0.0023) |
| Temp and Sq. | N | Y | Y |
| Precipitation | N | N | Y |

Notes: This table reports the OLS coefficients and standard errors. Each cell represents an OLS regression of the monthly standardized mortality rates (log, per 10,000 people) on $PM_{10}$ concentrations (10-μg/$m^3$). The variance–covariance matrix allows for arbitrary autocorrelation within each city.
*** Asterisks, indicate the 1 percent significance level.
** Asterisks, indicate the 5 percent significance level.
* Asterisks, indicate the 10 percent significance level.

**Table 10**
Fixed effects estimates between $PM_{10}$ and the mortality rate.

| | Overall mortality | | | | CVR mortality | | | |
|---|---|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| $PM_{10}$ | 0.013*** | 0.003 | 0.001 | 0.003 | 0.018*** | 0.003 | 0.002 | 0.004 |
| | −0.002 | −0.003 | −0.003 | −0.002 | −0.003 | −0.003 | −0.003 | −0.003 |
| Weather | N | Y | N | Y | N | Y | N | Y |
| City FE | Y | Y | Y | Y | Y | Y | Y | Y |
| Month FE | N | N | Y | Y | N | N | Y | Y |
| Observations | 1930 | 1930 | 1930 | 1930 | 1930 | 1930 | 1930 | 1930 |
| $R^2$ | 0.595 | 0.616 | 0.643 | 0.653 | 0.638 | 0.680 | 0.695 | 0.703 |

Notes: This table reports the coefficients and standard errors of the fixed effect models. The dependent variable is the logarithm of monthly standardized mortality rate per 10, 000 people for all causes and cardio-cerebrovascular and respiratory (CVR) diseases. Control variables are added to the fixed effect model gradually. The variance-covariance matrix allows for arbitrary autocorrelation within each city.
*** Asterisks, indicate the 1 percent significance level.

As such, we conclude that the variations in air pollution over time and across regions cannot be treated as exogenous, even after controlling for city fixed effects and year–month fixed effects. Because other unobserved variables (particular weather conditions) may co-vary with both air pollution and mortality rates, estimates of fixed-effects models may also be biased. The estimated coefficients in associational models are substantially smaller than the instrumental variable estimates, suggesting associational models may under-estimate the air pollution effects. This finding is in keeping with Schlenker and Walker (2011), who also find that estimates of air pollution effects obtained using a natural experiment design were much larger than estimates obtained through non-experimental approaches.

*Comparison with time-series studies*

Most contemporary research of air pollution's health effects uses either time-series or cohort approaches. Time-series studies assess the short-term relationships between exposure to pollution and adverse health outcomes (“acute effect”). Such studies often adopt generalized linear or generalized additive models to estimate the health effects of air pollution, using daily death and air pollution data. Among the voluminous time-series studies, we compare our results with two influential multi-site time-series studies: the Air Pollution and Health: A European Approach (APHEA) project, and the National Morbidity, Mortality and Air Pollution Study (NMMAPS).

The APHEA project, supported by the European Commission, studied the short-term effects of air pollution on human health in 12 European cities (Katsouyanni et al., 1997). APHEA researchers estimated that a 10-μg/$m^3$ increase in daily $PM_{10}$ was associated with a 0.4 percent increase in total daily mortality for western European cities and a 0.8 percent increase in total daily mortality for central eastern European cities. Katsouyanni et al. (2001), the follow-up APHEA2 project, examined

mortality and pollution in 29 European cities and found that a 10-$\mu g/m^3$ increase in daily $PM_{10}$ was associated with a 0.6 percent increase in total daily mortality.

The NMMAPS examined the effect of $PM_{10}$ on mortality in U.S. cities (Samet et al., 2000). The NMMAPS research team first investigated 20 large U.S. cities; then extended their analysis to 90 U.S. cities. The results showed that a 10-$\mu g/m^3$ increase in daily $PM_{10}$ was associated with a 0.21 percent increase in all-cause mortality, and a 0.31 percent increase in CVR mortality.

There is also a growing number of time-series studies focusing on short-term associations between air pollution and mortality in China (see Aunan and Pan, 2004; Lai et al., 2013; Lu et al., 2015; Shang et al., 2013 for literature review). For example, Lu et al. (2015) conducted a meta-analysis and concluded that the excess risks of mortality due to cardiovascular disease and mortality due to respiratory disease were 0.36 percent, and 0.42 percent for a 10-$\mu g/m^3$ increase in $PM_{10}$.

Compared with the past studies using time-series approach, our estimates are substantially larger. Our results in the most restrictive specification show that a 10-$\mu g/m^3$ change in monthly average $PM_{10}$ concentrations would lead to an 8.36 percent change in all-cause mortality, and an 8.78 percent change in CVR mortality.

*Comparison with cohort studies*

Cohort studies assess the association between air pollution and mortality using data with a much longer time scale ("chronic effect"). Substantially larger health effects of air pollution have been reported in the long-term cohort studies than in the high-frequency time-series studies.[13] The first large cohort study that demonstrated an adverse health impact of long-term air pollution exposure was the Harvard Six Cities study by Dockery et al. (1993). In a cohort of 8111 adults, with 14–16 years of follow-up, the authors found that the adjusted ratio of the mortality rate of the most-polluted city to that of the least-polluted city was 1.26, with cardiovascular deaths accounting for the single largest category of difference in mortality. The relative risks in all-cause mortality were associated with a difference in ambient fine particles concentrations: a 10-$\mu g/m^3$ difference in $PM_{2.5}$ concentrations was associated with an approximately 14 percent difference in the all-cause mortality rate.

Pope et al. (2002) conducted another large prospective cohort study of the long-term health effects of air pollution, using data from the American Cancer Society Cancer Prevention II project. Among approximately 500,000 adults in 50 states in the United States, chronic exposure to multiple air pollutants was linked to mortality statistics over a 16-year window. They showed that a 10-$\mu g/m^3$ increase in the annual mean concentrations of fine particulate matter ($PM_{2.5}$) was associated with increases in all-cause, cardiopulmonary, and lung cancer mortality of 4 percent, 6 percent, and 8 percent, respectively. Pope et al. (2004) further examined the association between air pollution and specific cardiopulmonary diseases to explore potential mechanistic pathways linking exposure to mortality. They found that long-term particulate matter exposure was most strongly associated with mortality attributable to ischemic heart disease, dysrhythmias, heart failure, and cardiac arrest. For these causes of death, a 10-$\mu g/m^3$ elevation in $PM_{2.5}$ was associated with an 8–18 percent increase in mortality risks, with greater risk observed for smokers than nonsmokers.

Estimates of the long-term associations between $PM_{10}$ and mortality in China were reported by a few cohort studies (Dong et al., 2012; Zhang et al., 2014; Zhang et al., 2011). The estimates of China's cohort studies are found to be greater than our IV estimates as well as those derived from western countries. For example, Zhang et al. (2014) showed that a 10-$\mu g/m^3$ increase in the annual average concentrations of $PM_{10}$ over a ten-year period corresponded to a 24 percent increase in all-cause mortality and a 23 percent increase in cardiovascular mortality.

Our estimates are not directly comparable to those reported in Dockery et al. (1993), Pope et al. (2002), and Pope et al. (2004) because data on $PM_{2.5}$ concentrations in China were not available during our sample period. In Beijing and a few other Chinese cities, $PM_{2.5}$ usually accounts for 50–70 percent of $PM_{10}$ (Yang et al., 2002; Yu et al., 2004). If we use 60 percent for a back-of-envelope calculation, we estimate that a 10-$\mu g/m^3$ elevation in monthly $PM_{2.5}$ concentrations would lead to a 14 percent increase in the monthly standardized mortality rate and a 15 percent increase in the CVR mortality rate. Compared with China's cohort studies, our estimates are smaller. This is reasonable because the long-term exposure to air pollution is likely to have a greater impact on mortality than the short-term exposure.

## Health benefits of air pollution reduction

As pointed out in Dominici et al. (2014), a critical question of particulate matter research is to identify the magnitude of public health benefits from reduction of particulate matters. In this section, we assess the benefits of improving China's air quality and provide a range of estimates on the monetary value of the averted deaths.

According to the 2010 Census, China has 690 million urban inhabitants. Assuming our estimates apply to all cities and using the most conservative IV estimate in column 6 of Table 2, a back-of-envelope calculation shows that 285,190

[13] For example, Schwartz (2000) shows that, as data become more aggregated, the effects of air pollution on ischemic heart disease mortality and all-death mortality increase, suggesting larger effects of long-term exposure, possibly due to development of chronic diseases.

**Table 11**
Valuation of the health benefits associated with air quality improvement

| | Location | Approach | VSL (1000 USD) | Reduce $PM_{10}$ by 10 $\mu g/m^3$ (Billion USD) | Reduce $PM_{10}$ to WHO guideline level (Billion USD) |
|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) |
| Hammitt and Zhou (2006) | Beijing | CV | 77.4 | 22.1 | 176.5 |
| Krupnick et al. (2006) | Shanghai, Chongqing | CV | 225.8 | 64.3 | 514.8 |
| Wang and Mullahy (2006) | Chongqing | CV | 46.1 | 13.1 | 105.1 |
| Guo and Hammitt (2009) | National | HW | 38.7～129 | 11.0-36.8 | 88.2～294.1 |
| Qin et al. (2013) | National | HW | 619.4 | 176.5 | 1412.2 |
| World Bank (2007) | N/A | CV | 161.3 | 46.0 | 367.8 |

Notes: This table reports the back-of-envelope calculation of the health benefits from air pollution reduction in China. Column 3 shows various estimates of the value of a statistical life in six past studies. We transfer the Chinese Yuan to U.S. dollars using an exchange rate of 6.2:1. Column 3 multiplies the estimates in column 6 of Table 2 to calculate the monetary value of averted deaths associated with a 10-μg/m$^3$ reduction of $PM_{10}$ concentrations. Column 5 includes the monetary value of averted deaths associated with the reduction of $PM_{10}$ concentrations to the WHO guideline level (20 μg/m$^3$). CV stands for contingent valuation approach, and HW stands for hedonic wage approach.

(=4.12*0.0836*12*690,000,000/10,000) deaths per year could be avoided among China's urban population in one year if $PM_{10}$ concentrations were to decrease by 10 μg/m$^3$.

The WHO (2014) sets an annual mean of 20 μg/m$^3$ as the guideline for $PM_{10}$ concentrations based on a summary of risk assessments. If we assume that the air pollution effect is linear, a back-of-envelope calculation shows that more than 2.28 million (=4.12*0.086*8*690,000,000/100,00) pre-mature deaths in urban China can be attributed to air pollution ($PM_{10}$ level above 20 μg/m$^3$).

A comprehensive evaluation of the health benefits of air pollution requires measurements of a variety of health outcomes, such as mortality, incidence of chronic bronchitis, respiratory and cardiovascular hospital admission, work loss days, and worker productivity. Among all these outcomes, averted premature deaths are associated with the highest economic value. The mortality risks are typically valued using the value of a statistical life (VSL)—the amount of money that people would pay to reduce their risk of dying. The VSL is often estimated from contingent valuation surveys or through the hedonic wage approach.

Several studies have been conducted in China to value the mortality risk reduction. We focus on five most recent studies published after 2006. Among them, three use the contingent valuation approach and the other two use the hedonic wage approach. The VSL estimates in these studies are summarized in Table 11. The estimated VSL in China ranges from 38.7 to 619.4 thousand US dollars, depending on when and where the data were collected and which approach was used. We calculate the monetary value of averted pre-mature deaths by multiplying the VSL by the number of predicted lives saved (285,000) for the urban population associated with a 10-μg/m$^3$ $PM_{10}\mu g/m^3 PM_{10}$ reduction and report the results in column 4 of Table 11. The estimated benefits range from 11.0 to 176.5 billion dollars. Column 5 shows the benefits if the $PM_{10}$ concentrations decrease to the WHO guideline level from the current level (about 100 μg/m$^3$). The lowest benefits associated with the abatement are approximately 88.2 billion dollars, and the highest benefits are more than 1.4 trillion dollars. If we use the World Bank's VSL to calculate the health benefits of reducing pollution to the WHO guideline level in China, the total health benefits will exceed 367 billion dollars, which amounts to 6 percent of China's GDP in 2010.

Our calculations show that policies aiming to reduce air pollution can have large benefits in terms of averted pre-mature deaths. However, the monetary values in columns 4 and 5 should be interpreted with caution because (1) evidence from studies in China and Western countries shows that the exposure-response relationship may be non-linear as particulate pollution levels increase (Almond et al., 2009; Samoli et al., 2001); (2) in order to extrapolate the treatment effect to the rest of the country, we assume the estimated air pollution effects apply to all cities in China; (3) we ignore other health costs such as hospitalizations and avoidance behaviors; and (4) air pollution has large impacts on the elderly and our calculation does not discount the VSL for the elderly.

## Conclusions

This study investigates the causal link between air pollution and mortality in China, using the BOG08 as a natural experiment. We find that air pollution has a large and robust effect on mortality. Our results show that a 10-μg/m$^3$ (roughly 10 percent) decrease in $PM_{10}$ concentrations results in an 8.36 percent drop in the all-cause mortality rate. Based on our estimates and China's urban population, more than 285,000 premature deaths could be averted each year if the current levels of $PM_{10}$ concentrations decrease by 10 $\mu g/m^3$.

Our analysis also shows that the effects of air pollution are primarily driven by deaths from CVR diseases. A 10-$\mu g/m^3$ reduction in $PM_{10}$ concentrations decreases the monthly CVR mortality rate by 8.78 percent. The elderly are particularly vulnerable to air pollution shocks, and they are more likely to die from CVR diseases when air quality deteriorates. Our results also show that the air pollution has large impacts on infant and child mortality, implying a much greater loss in life years. The results for males and females are very similar to each other.

Associational approaches tend to underestimate the health effects of air pollution. In both cross-sectional and fixed-effects models, we find that the estimated coefficients are sensitive to weather controls and model specifications, and that the estimated impacts are substantially smaller than our instrumental variable regression estimates. A comparison with studies in public health literature shows that our estimates are larger than those in time-series studies and smaller than those in long-term cohort studies.

We are aware of three caveats in this study that call for cautious interpretation of our results. First, our results might only be locally valid and should not be generalized to lower air pollution levels because the dose–response function might be non-linear and perhaps negligible below a certain threshold. We also warn readers not to generalize our findings to rural areas in China. Rural residents are less likely to adopt certain avoidance behaviors, such as wearing masks or installing air filters, in response to air pollution attacks. They may also be more vulnerable to air quality shocks because their *ex ante* health status is worse than that of urban residents. Consequently, the health impact of air pollution may be greater for rural residents than urban residents. For example, Zhou et al. (2015) found that the smog episodes were associated with a higher mortality rate in rural areas but not in urban areas. Future studies are warranted to evaluate the differential health effects of air pollution in China's urban and rural populations.

Second, people's behavioral changes during the study period are not examined due to lack of data. An individual's level of pollution exposure is determined by outdoor air quality, indoor air quality and how one divides one's time between indoor and outdoor activities. People may adjust their behaviors in response to changes in air pollution. In particular, those at risk of being negatively affected by pollution may have relatively strong incentives to adopt avoidance behaviors. For example, Neidell (2009) find that people respond to information about air quality, with smog alerts leading to significantly reduced attendance at major outdoor facilities in Los Angeles. Thus, the BOG08 may have affected people's preferences between indoor and outdoor activities. The consequences of such behavioral changes on mortality merit further investigation.

Lastly, “harvesting” effect (Schwartz, 2000) is not examined. We observe a mortality inversion during the post-Olympic periods despite that air pollution level is lower than the pre-Olympic periods. However, due to data limitation, we are unable to identify whether this is simply because the deaths of the most susceptible were postponed by several months or because air pollution has an asymmetric impact on health. These questions are of great importance and beg for further investigation.

## Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j. jeem. 2016.04.004.

## References

Almond, D., Chen, Y., Greenstone, M., Li, H., 2009. Winter heating or clean air? Unintended impacts of China's Huai River policy. Am. Econ. Rev. 99, 184–190.
Arceo-Gomez, E.O., Hanna, R., Oliva, P., 2012. Does the effect of pollution on infant mortality differ between developing and developed countries? Evid. Mex. City 18349, 2012.
Aunan, K., Pan, X.-C., 2004. Exposure-response functions for health effects of ambient air pollution applicable for China – a meta-analysis. Sci. Total. Environ. 329, 3–16.
Cesur, R., Tekin, E., Ulker, A., 2015. Air pollution and infant mortality: evidence from the expansion of natural gas infrastructure. Econ. J..
Chay, K., Dobkin, C., Greenstone, M., 2003. The Clean Air Act of 1970 and adult mortality. J. Risk Uncertain. 27, 279–300.
Chay K.Y., Greenstone M., 2003a. Air Quality, Infant Mortality, and the Clean Air Act of 1970. NBER Working Paper No. 10053.
Chay, K.Y., Greenstone, M., 2003b. The impact of air pollution on infant mortality: evidence from geographic variation in pollution shocks induced by a recession. Q. J. Econ. 118, 1121–1167.
Chen, Y., Ebenstein, A., Greenstone, M., Li, H., 2013a. Evidence on the impact of sustained exposure to air pollution on life expectancy from China's Huai River policy. Proc. Natl. Acad. Sci. 110, 12936–12941.
Chen, Y., Jin, G.Z., Kumar, N., Shi, G., 2013b. The promise of Beijing: evaluating the impact of the 2008 Olympic Games on air quality. J. Environ. Econ. Manag. 66, 424–443.
Chen, Y., Zhe, J.G., Naresh, K., Guang, S., 2012. Gaming in air pollution data? Lessons from China. BE J. Econ. Anal. Policy 13, 1–43.
Currie, J., Neidell, M., 2005. Air pollution and infant health: what can we learn from California's recent experience? Q. J. Econ. 120, 1003–1030.
Currie, J., Neidell, M., Schmieder, J.F., 2009. Air pollution and infant health: lessons from New Jersey. J. Health Econ. 28, 688–703.
Currie, J., Walker, R., 2011. Traffic Congestion and Infant Health: evidence from E-ZPass. Am. Econ. J.: Appl. Econ. 3, 65–90.
Deschenes, O., Moretti, E., 2009. Extreme weather events, mortality, and migration. Rev. Econ. Stat. 91, 659–681.
Dockery, D.W., Pope, C.A., Xu, X., Spengler, J.D., Ware, J.H., Fay, M.E., Speizer, F.E., 1993. An association between air pollution and mortality in six US cities. N. Engl. J. Med. 329, 1753–1759.
Dominici, F., Greenstone, M., Sunstein, C.R., 2014. Particulate matter matters. Science 344, 257–259.
Dong, G.-H., Zhang, P., Sun, B., Zhang, L., Chen, X., Ma, N., Lee, Y.L., 2012. Long-term exposure to ambient air pollution and respiratory disease mortality in Shenyang, China: a 12-year population-based retrospective cohort study. Respiration 84, 360–368.

Ebenstein, A., Fan, M., Greenstone, M., He, G., Yin, P., Zhou, M., 2015. Growth, pollution, and life expectancy: China from 1991–2012. Am. Econ. Rev. 105, 226–231.
Foster, A., Gutierrez, E., Kumar, N., 2009. Voluntary compliance, pollution levels, and infant mortality in Mexico. Am. Econ. Rev. 99, 191–197.
Graff Zivin, J., Neidell, M., 2009. Days of haze: environmental information disclosure and intertemporal avoidance behavior. Journal. Environ. Econ. Manag. 58, 119–128.
Greenstone, M., Hanna, R., 2014. Environmental regulations, air and water pollution, and infant mortality in India. Am. Econ. Rev. 104, 3038–3072.
Guo, X., Hammitt, J.K., 2009. Compensating wage differentials with unemployment: evidence from China. Environ. Resour. Econ. 42, 187–209.
Hammitt, J., Zhou, Y., 2006. The economic value of air-pollution-related health risks in china: a contingent valuation study. Environ. Resour. Econ. 33, 399–423.
Jayachandran, S., 2009. Air quality and early-life mortality evidence from Indonesia's wildfires. J. Human. Resour. 44, 916–954.
Katsouyanni, K., Touloumi, G., Samoli, E., Gryparis, A., Tertre, A.L., Monopolis, Y., Schwartz, J., 2001. Confounding and effect modification in the short-term effects of ambient particles on total mortality: results from 29 European cities within the APHEA2 Project. Epidemiology 12, 521–531.
Katsouyanni, K., Touloumi, G., Spix, C., Schwartz, J., Balducci, F., Medina, S., Bacharova, L., 1997. Short-term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: results from time series data from the APHEA project. Air Pollution and Health: a European Approach. Br. Med. J. 314, 1658–1663.
Krupnick, A., Hoffmann, S., Larsen, B., Peng, X., Tao, R., Yan, C., McWilliams, M., 2006. The Willingness to Pay for Mortality Risk Reductions in Shanghai and Chongqing, China. Resources for the Future. The World Bank, Washington, DC.
Lai, H.-K., Tsang, H., Wong, C.-M., 2013. Meta-analysis of adverse health effects due to air pollution in Chinese populations. BMC Public Health, 13.
Logan, W., 1953. Mortality in the London fog incident, 1952. Lancet 261, 336–338.
Lu, F., Xu, D., Cheng, Y., Dong, S., Guo, C., Jiang, X., Zheng, X., 2015. Systematic review and meta-analysis of the adverse health effects of ambient PM2.5 and PM10 pollution in the Chinese population. Environ. Res. 136, 196–204.
Luechinger, S., 2014. Air pollution and infant mortality: A natural experiment from power plant desulfurization. J. Health Econ. 37, 219–231.
Moretti, E., Neidell, M., 2011. Pollution, health, and avoidance behavior evidence from the ports of Los Angeles. J. Hum. Resour. 46, 154–175.
Neidell, M., 2009. Information, avoidance behavior, and health the effect of ozone on asthma hospitalizations. J. Hum. Resour. 44, 450–478.
Pope, C.A., Burnett, R.T., Thun, M.J., Calle, E.E., Krewski, D., Ito, K., Thurston, G.D., 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. J. Am. Med. Assoc. 287, 1132–1141.
Pope, C.A., Burnett, R.T., Thurston, G.D., Thun, M.J., Calle, E.E., Krewski, D., Godleski, J.J., 2004. Cardiovascular mortality and long-term exposure to particulate air pollution epidemiological evidence of general pathophysiological pathways of disease. Circulation 109, 71–77.
Pope, C.A., Dockery, D.W., Schwartz, J., 1995. Review of epidemiological evidence of health effects of particulate air pollution. Inhal. Toxicol. 7, 1–18.
Qin, X., Li, L., Liu, Y., 2013. The value of life and its regional difference in China. China Agric. Econ. Rev. 5, 373–390.
Rich, D.Q., Kipen, H.M., Huang, W., Wang, G., Wang, Y., Zhu, P., Diehl, S.R., 2012. Association between changes in air pollution levels during the Beijing Olympics and biomarkers of inflammation and thrombosis in healthy young adults. J. Am. Med. Assoc. 307, 2068–2078.
Samet, J.M., Dominici, F., Curriero, F.C., Coursac, I., Zeger, S.L., 2000. Fine particulate air pollution and mortality in 20 U.S. cities, 1987–1994. N. Engl. J. Med. 343, 1742–1749.
Samoli, E., Schwartz, J., Wojtyniak, B., Touloumi, G., Spix, C., Balducci, F., Bacharova, L., 2001. Investigating regional differences in short-term effects of air pollution on daily mortality in the APHEA project: a sensitivity analysis for controlling long-term trends and seasonality. Environ. Health Perspect. 109, 349–353.
Schlenker W., Walker W.R., 2011. Airports, Air Pollution, and Contemporaneous Health. NBER Working Paper No. 17684.
Schwartz, J., 2000. Harvesting and long term exposure effects in the relation between air pollution and mortality. Am. J. Epidemiol. 151, 440–448.
Shang, Y., Sun, Z., Cao, J., Wang, X., Zhong, L., Bi, X., Huang, W., 2013. Systematic review of Chinese studies of short-term exposure to air pollution and daily mortality. Environ. Int. 54, 100–111.
Tanaka, S., 2015. Environmental regulations on air pollution in China and their impact on infant mortality. J. Health Econ. 42, 90–103.
United Nations Environment Programme, 2009. Independent Environmental Assessment: Beijing 2008 Olympic Games, vol. 2015.
Wang, H., Mullahy, J., 2006. Willingness to pay for reducing fatal risk by improving air quality: a contingent valuation study in Chongqing, China. Sci. Total. Environ. 367, 50–57.
Wansbeek, T.J., Meijer, E., 2000. Measurement Error and Latent Variables in Econometricss. Elsevier, Amsterdam.
WHO, Ambient (outdoor) air quality and health, 2014, http://www.who.int/mediacentre/factsheets/fs313/en/.
World Bank, 2007. Cost of Pollution in China: Economic Estimates of Physical Damages. World Bank, Washington, DC.
Yang, F., He, K., Ma, Y., Zhang, Q., Yu, X., 2002. Variation characteristics of $PM_{2.5}$ concentration and its relationship with $PM_{10}$ and TSP in Beijing. China Environ. Sci. 22, 506–510.
Yu, J., Yu, T., Wei, Q., Wang, X., Shi, J., Li, H., 2004. Characteristics of mass concentration variations of $PM_{10}$ and $PM_{2.5}$ in Beijing area. Res. Environ. Sci. 17, 45–47.
Zhang, L.-w., Chen, X., Xue, X.-d., Sun, M., Han, B., Li, C.-p., Tang, N.-j., 2014. Long-term exposure to high particulate matter pollution and cardiovascular mortality: a 12-year cohort study in four cities in northern China. Environ. Int. 62, 41–47.
Zhang, P., Dong, G., Sun, B., Zhang, L., Chen, X., Ma, N., Lee, Y.L., 2011. Long-term exposure to ambient air pollution and mortality due to cardiovascular disease and cerebrovascular disease in Shenyang, China. Plos. One 6, e20827.
Zhou, M., He, G., Fan, M., Wang, Z., Liu, Y., Ma, J., Liu, Y., 2015. Smog episodes, fine particulate pollution and mortality in China. Environ. Res. 136, 396–404.