The Effect of Air Pollution on Cardiovascular Mortality: Evidence from the 2008 Beijing Olympic Games

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Abstract

Exogenous air pollution variations induced by the 2008 Beijing Olympic Games provide a natural experiment to estimate the health effects of air pollution. This study finds that air pollution has a significant effect on cardiovascular mortality in China. A 10 $\mu g/m^3$ (10 percent) decrease in PM_{10} mean concentrations decreases monthly cardiovascular mortality by 13.6 percent, implying that more than 67,000 lives could be saved each year by a 10% reduction in PM_{10} concentrations. The estimates are robust to a variety of specifications.

Keywords: Air Pollution, Cardiovascular Mortality, particulate matter, 2008 Olympic Games,
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1 Introduction

To fulfill its international commitment to maintain high air quality during the 2008 Summer Beijing Olympic Games, the Chinese government implemented a series of stringent policies to reduce local and regional emissions in the greater Beijing metropolitan area. These aggressive regulations included setting higher emission standards, reducing traffic, halting large-scale construction projects, and shutting down polluting factories, etc. The combination of these measures resulted in dramatically improved air quality in Beijing and its neighboring cities.

This study explores this unique natural experiment to estimate the effects of this anthropogenic improvement in air quality during the Olympic Games on cardiovascular mortality in China. A city's regulatory status during the Olympic Games is used as the instrument for air quality, and the causal effect of air pollution on cardiovascular mortality is estimated using a fixed-effect instrumental variable model.

Over the last decade, a growing body of epidemiological and clinical evidence has led to heightened concern about the potential harmful effects of ambient air pollution on cardiovascular health (see Brook et al., 2004, Raun and Ensor, 2012 for literature reviews). In particular, researchers have found that air pollution is statistically significantly associated with cardiac arrest (Dennekamp et al., 2010, Ensor et al., 2013, Silverman et al., 2010). Cardiac arrest not only occurs to people with cardiac problems, it also occurs to people who have no pre-existing cardiac conditions. Thus, if air pollution causes death from cardiac arrest, reductions in life expectancy could be very large. In addition, researchers have found that air pollution is associated with other heart-related diseases such as cardiac arrhythmia (Peters et al., 2000), ventricular tachyarrhythmia (Dockery et al.,

2005), out-of-hospital coronary death (Forastiere et al., 2005), heart rate variability (de Hartog et al., 2009), and inflammation and thrombosis (Rich et al., 2012).

However, whether the estimates produced by these associational studies have causal implications is controversial. Time series studies, cross-sectional studies, and even longitudinal studies may all suffer from endogeneity bias (Chay and Greenstone, 2003b). Given the absence of random clinical trials, the 2008 Beijing Olympic Games provide a compelling opportunity to reduce potential omitted variable bias in estimating the impact of air pollution on cardiovascular mortality. The strict enforcement of air pollution regulations by the Chinese government during the period of November 2007 to September 2008 caused sharp variations in air pollution across Chinese cities that are likely to be orthogonal to city-district level changes in health outcomes except through their effects on air pollution. The present analysis compares changes in cardiovascular mortality rates in cities that experienced large reductions in air pollution with changes in cities that experienced no pollution reduction, using information before, during and after the Olympic Games.

Our empirical evidence suggests that this natural experimental research design provides a credible basis for evaluating the health impact of air pollution in severely polluted regions. We find a significant negative effect of air pollution on cardiovascular mortality rates at the city-district level, with a 10 point reduction in the API resulting in approximately 1.61 fewer cardiovascular deaths per 100,000 people per month (an elasticity of 2.02). When we recover PM_{10} concentrations from the API, we find that a 10 $\mu g/m^3$ (roughly 10%) reduction in PM_{10} concentrations results in approximately 0.81 fewer cardiovascular deaths per month (an elasticity of 1.36). Based on our results, more

than 67,000 lives in urban China could be saved from cardiovascular death if PM_{10} concentrations were to decrease by 10 $\mu g/m^3$ from current levels. Given the severe air pollution in China, our results suggest that reducing air pollution could have large welfare effects with respect to life expectancy.

Our estimates are robust across a variety of specifications. In particular, they are insensitive to the inclusion of weather conditions (rainfall and temperature), which are typically confounding factors in associational studies. A falsification test that uses injury death rates as the dependent variable indicates that our main findings are not due to city-specific death patterns. More importantly, our estimates are larger than those of previous studies. We compare our model with cross-sectional models, fixed-effect models, and epidemiological studies in the literature. We show that both cross-sectional and the fixed-effects models are likely to suffer from omitted variables bias and that the estimates are biased downward.

2 Health Effects of Air Pollution

The association between high levels of air pollution and human illness has been recognized for more than half a century.[4] Voluminous epidemiological studies have linked air pollution with morbidity and mortality, both in the short run and the long run (see Brook and Rajagopalan, 2003, Brunekreef and Holgate, 2002, Pope, 2000 for literature reviews). These studies can roughly be divided into the following categories: (1) time-series studies; (2) cross-sectional studies; (3) cohort-based and panel-data studies; and (4) natural- or quasi-experimental studies.

[4] 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).

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); and most of these studies find that temporary elevations in air pollution are associated with worse health outcomes (Dockery and Pope, 1996). However, sharp changes in air pollution levels are often driven by local weather conditions rather than by 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.[5] Moreover, because there are no control groups, it is difficult to rule out alternative explanations in nearly all 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. However, this research design is plagued by omitted variables bias. As people's health status and 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, estimates of the health effects of air pollution, using cross-sectional models, may be biased.

Cohort-based longitudinal studies (Dockery et al., 1993) may face problems similar to those of cross-sectional studies. In principle, longitudinal studies can accurately estimate the loss of life expectancy associated with higher levels of pollution because they collect

[5] For example, Beijing's thick smoggy days in 2013, which were intensively documented by the mass media, largely resulted from the combined effects of temperature, humidity, and wind. Most of these smoggy days occurred in conditions relatively low wind during winter. If people are more likely to die on cold or windy days, the association between air pollution and increased mortality can be misleading unless these factors are controlled.

data on long-term exposure to such conditions. However, to some extent, people self-select into different locations and are thus exposed to different levels of pollution. For example, 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 suggested by (Chay and Greenstone, 2003a, b), these associational approaches tend to produce unreliable estimates.

As more data become available, recent studies have used fixed-effects models (Currie and Neidell, 2005, Currie et al., 2009) to remove bias caused by time-invariant unobserved factors. Fixed effects models are particularly useful when time-invariant omitted factors explain most of the variation of 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 largely depend on factors similar to those that affect health outcomes (such as weather), this assumption may not hold.

In contrast, natural or quasi-experiments allow for more convincing identification strategies. Most existing studies of this kind focus on infant or child health, as infants and children are the most vulnerable segment of the population to air-borne diseases, and the effects are immediate. (Chay and Greenstone, 2003b) explored how air quality improvement induced by the 1981-1982 recession affected infant mortality in the United States, finding that a 1 percent reduction in Total Suspended Particulates (TSPs) resulted in a 0.35 percent decline in the infant mortality rate at the county level. (Chay and Greenstone, 2003a) also analyzed the effects of the Clean Air Act Amendments on infant

mortality, using nonattainment status as an instrument for TSP changes. They estimated that a 1 percent decline in TSPs resulted in a 0.5 percent decline in the infant mortality rate. Jayachandran (2009), in an analysis of the effects on infant and child mortality of changes in air quality (particulate matter) caused by wildfires in Indonesia, found that the increased pollution accounted for 15,600 missing children. Luechinger (2010) investigated the effect of SO_2 on infant mortality in Germany, utilizing a natural experiment created by the mandated desulfurization of power plants, with wind directions dividing counties into treatment and control groups. As for China, Tanaka (2010) estimated the effect of air pollution on infant mortality, using air quality variations induced by the SO_2 and acid rain control zones in the 1990s.

While nearly all associational studies have suggested that air pollution is positively and significantly associated with adult mortality rates, Chay, Dobkin and Greenstone (2003) challenged this consensus. Using the Clean Air Act as an instrumental variable for air quality, they found that, although regulatory status was associated with large reductions in TSPs, such reductions had little effect on either adult or elderly mortality. Chay, Dobkin and Greenstone (2003) may have reached this conclusion, however, due to over-aggregation of diseases. As some diseases are sensitive to air pollution, while others are not, it is very likely that air pollution only affects mortality caused by some diseases and not others.

With respect to morbidity, Schlenker and Walker (2011) estimated the health effects of air pollution induced by airline network delays in the United States, finding that carbon monoxide (CO) exposure led to significant increases in hospitalization rates for asthma and respiratory diseases and to 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.

3 Air Pollution Regulations during the Olympic Games

The air pollution controls instituted during the 2008 Beijing Olympic Games may be by far the largest efforts to control air quality in human history. To assure good air quality during the Olympic Games, the Chinese government implemented a series radical 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," which provided guidelines for the regulation of air quality before and during the Olympic Games. The Measures defined the period of November 1st, 2007 to July 20th, 2008 as the pre-Olympic Comprehensive Regulation period and the period of July 20th to September 20th, 2008 as the Olympic Games Temporary Pollution Control period.

During the pre-Olympic Comprehensive Regulation period, multiple measures were implemented simultaneously: (1) all coal-fired power plants in Beijing were required to install desulfurization, dust removal and denitrification facilities; (2) the public sector (public transit, environment and health agencies, etc.) replaced all heavy-emission vehicles; (3) oil-gas gathering units and recovery systems were installed in 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; (6) the Capital Steel Company was ordered to relocate, and its

production of steel fell from more than 600,000 tons per day to less than 200,000 tons per day.

Motor vehicle exhaust emissions are the largest air pollution source in large cities. To ensure good air quality, Beijing implemented temporary traffic controls 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 license plates were allowed on the roads only on odd-numbered days, and those with even-numbered license plates were allowed on the roads only on even-numbered days. Only a few exceptions, such as police vehicles, public transport and vehicles with Olympic passes, 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 news released by the committee of the Olympic Games and the State Environment Protection Agency in China (2008), total vehicle exhaust emissions decreased by more than 60 percent.[6] Traffic controls significantly decreased the concentration of fine particulates, ozone, nitrogen oxide and other pollutants generated by auto vehicles in Beijing.

At the same time, the government required that all power plants and chemical production plants to reduce their emissions by 30 percent from their previous emission levels, although these plants had already met the national emission standard. More than 20

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

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 Olympic Games.

Because air quality in Beijing was also affected by its neighboring areas, several cities and provinces (Tianjin, Hebei, Liaoning, Neimeng 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 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, Tianjian shut down many polluting factories before the Olympic Games and enforced temporary traffic control during the games. Shenyang replaced old buses prior to the Olympics and expanded green belts around construction sites. Qinhuangdao built shelter forests along piers to reduce dust, swept streets with water, and dumped garbage each day.

The combination of these regulations effectively improved air quality in and around Beijing. Our data show that the yearly API in Beijing decreased from 101 to 87 from 2007 to 2008, which is approximately equivalent to a decrease in concentration of particulate pollution (PM_{10}) from 152 $\mu g/m^3$ to 124 $\mu g/m^3$ (18 percent decrease). The improvement is particularly striking during the summer period (June-August). The average summer API in Beijing fell to 75 in 2008 from 98 one year previous, with a corresponding decrease in PM_{10}concentration from 145 $\mu g/m^3$ to 101 $\mu g/m^3$ (a 30 percent decrease). During the

17 days of the Olympic Games, all the indicators of air quality in Beijing met national standards. The findings are consistent with those of the United Nations Environment Programme (2009) and Chen et al. (2011). The United Nations Environment Programme (2009) examined the air pollution data provided by Beijing Environmental Protection Bureau and found that air quality in Beijing met the national standard less than 50 percent of the time in the period in 2000 and 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, Jin, Kumar and Shi (2011) evaluated the impact of the Olympics on Beijing's air quality, finding that the regulations effectively reduced the API in Beijing by 29.65 percent during the Olympic Games, compared with the same month one year previously. They also used satellite based Aerosol Optical Depth data to confirm that the improvement in air quality in Beijing was real.

4 Data

4.1 Air Quality Data

Air Quality data come from monitoring sites administered by the State Environment Protection Agency (SEPA), which has provided daily air quality information for 82 major urban cities in China from 2000 to the present. Air quality data include the following information: daily API and primary pollutant.

The API, an overall measure of ambient air quality, is an index used to report daily air quality to the general public. The higher the score of the API, the higher the level of air pollution. Three pollutants, PM_{10}, SO_2, and NO_2, are included in the API. Ideally, we would

obtain measures of concentrations of specific air pollutants. However, specific pollutant concentrations are not yet publicly available. Fortunately, the method used by the SEPA to construct the API allows us to recover concentrations of primary pollutants.[7] In the daily API data, PM_{10} is usually the primary pollutant (in 89.58 percent of daily samples). These particulates come from a variety of sources, including motor vehicles, industrial operations, construction, windblown soil, cooking, wild fires, and waste burning.

The reliability of official Chinese air quality data has been questioned by researchers. The government's unwillingness to publicize specific concentrations of pollutants imposes further difficulties for researchers seeking to verify the reliability of the data. Chen et al. (2012) assessed the quality of China's API data, finding 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, Zhe, Naresh and Guang (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 for cross-city and over-time variations in air pollution.

4.2 Mortality Data

Mortality data come from the Disease Surveillance Point System (DSPS) of China's Center for Disease Control and Prevention (CDC). The DSPS, initiated in 1978, covered 71 counties in 29 provinces from 1980 to 1989 and 145 counties in 31 provinces from 1990

[7] See appendix A on how to recover concentrations of primary pollutants from the API.

to 2000. The DSPS was overhauled following the SARS outbreak in 2003 and has covered 161 counties from 2003 to the present. To represent national population and mortality trends, the system adopts a multi-stage cluster population probability sampling method.

DSPS records nine categories of death: cancer, cerebrovascular diseases, digestive system diseases, cardiovascular diseases, injuries, perinatal diseases, respiratory system diseases, urine and procreative system diseases, and other diseases. Cardiovascular diseases are the third leading cause of death in China. In the sampled cities, the leading cause of death is cancer, which kills roughly 28 percent of people. The second leading cause of death is cerebrovascular diseases, accounting for 20 percent of the total. The share of deaths caused by cardiovascular diseases is 17 percent.

Our primary dependent variable is the monthly age-adjusted cardiovascular mortality rate, which is defined as the number of deaths caused by cardiovascular diseases per 100,000 people per month in a given city-district, adjusted by age distribution. Monthly age-adjusted cardiovascular mortality rates were calculated by age group from 2006 to 2010, based on death records. People are divided into 19 age groups: 0, 1, 2-5, 6-10, 11-15…, 75-80, 81-85, and older than 85.

The age-group specific mortality rate at a specific death surveillance point is calculated as:

$$Age_Spec_MR_{group_i} = \frac{100{,}000 * Death_{group_i}}{Total\ Population_{group_i}}$$

The age-adjusted mortality rate for a specific death surveillance location is calculated as:

$$Age_Adj_MR = \sum_k \left(Population\ Weight_{group_i} * Age_Spec_MR_{group_i}\right)$$

The population weights are calculated using China's 2000 Census. Age adjustment allows us to compare regions with different age structures.

We match cardiovascular mortality data with the API data at the monthly level. Thirty-four urban city-districts in China were successfully matched. This study focuses on a five-year window, from January 2006 to December 2010, and the sample covers approximately 76,000 deaths caused by cardiovascular diseases. The monthly average age-adjusted cardiovascular mortality rate is 5.94 deaths per 100,000 people per month, with a standard deviation of 3.37.

4.3 Weather Data

The data on rainfall and temperature are drawn from the Global Historical Climatology Network (GHCN) project. GHCN provides average monthly precipitation levels and temperatures for given longitudes and latitudes, with a minimum cell size of 0.5 degrees by 0.5 degrees.

We first identified the coordinates of the 34 city-districts in our sample. For each city-district, we collected rainfall and temperature data for its four nearest points in the GHCN data. We calculated weighted averages of precipitation and temperature, using inverse squared distances as weights. For example, the interpolated precipitation of location *j*, using the nearest four points, is given by:

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

where $Precip_j$ is precipitation at point *j*, and $Distance_{jk}^{-2}$ is the inverse squared distance between *j* and *k*.

Both rainfall and temperature may affect both air pollution levels and people's health status. Rainfall may be negatively correlated with air pollution, as rain can wash away pollutants in the air. The relationship between temperature and air pollution may be non-monotone, as air pollution will tend to increase on both extremely hot and extremely cold days, due to excessive energy consumption. In addition, people are more likely to die on extremely hot or cold days (Deschenes and Moretti, 2009). Summary statistics of the key variables are found in Table 1.

5 Research Design and Model

Our analysis compares changes in cardiovascular mortality rates in cities that experienced large reductions in the air pollution index (API) and particulate matter (PM_{10}) with changes in cities that experienced little or no reduction in pollution. We estimate the effects of air pollution on cardiovascular mortality, using a fixed-effects instrumental variable model:

$$Y_{it} = \delta_0 \hat{P}_{it} + X'_{it}\eta_0 + u_i + v_t + \varepsilon_{it} \quad (2)$$

$$P_{it} = \lambda_1 O_{it} + \lambda_2 T_{it} + X'_{it}\eta_0 + \tau_i + \pi_t + \xi_{it} \quad (1)$$

where P_{it} is the air pollution level in city i at time t, Y_{it} is the health outcome in city i at time t, X_{it} is a set of control variables, u_i and τ_i are city fixed effects, v_t and π_t are time fixed effects, and ε_{it} and ξ_{it} are unobservable disturbances. O_{it} is a regulation status indicator. If city *i* is regulated at time *t*, $O_{it} = 1$; otherwise, it is 0. T_{it} is the traffic control status indicator. If city i enforces traffic controls during the Olympic Games, $T_{it} = 1$; otherwise, it is 0. Both O_{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 controls were implemented beginning in November 2007, so we treat this month as the starting date of

the regulations. Some interventions, such as traffic control and temporary emission controls, were abandoned immediately after September 2008, while others, such as some factory shutdowns and relocations, became permanent. Because these regulations might have long-lasting effects on air quality, we chose December 2008, three months after the Olympics, as the ending date. Thus, $O_{it} = 1$, if a city was in a regulated city/province from November 2007 to December 2008; otherwise, it is 0.

Beijing and Tianjin faced more stringent regulations than other cities during the Olympics, with both cities enforcing temporary traffic controls during July-September 2008. To capture the treatment intensity differences, we include a traffic control dummy T_{it} (serving as another instrumental variable), which equals 1 during July-September 2008 for Beijing and Tianjin districts.[8]

In the first stage, we use Equation (1) to estimate how the air quality regulations affected air pollution. The coefficient λ_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. λ_2 has a similar interpretation. We expect both λ_1 and λ_2 to be negative. In the second stage of the IV regression, we use the estimate of pollution level $\hat{P}_{it}$ from the first stage in Equation (2). If air pollution negatively affects people's health,

[8] The point estimate and significance of the effect of air pollution on cardiovascular 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.

we expect that fewer people will die from cardiovascular diseases during periods of high-quality air in the regulated cities and that δ_0 will be positive.

Figure 1 shows trends in the monthly API for both the regulated (9 city-districts) and non-regulated cities (25 city-districts) during 2006-2010. "Being-regulated" is associated with a sharp decrease in the API in 2008. We observe strong seasonality in the trends of air quality for both treated and control groups, with air quality better in summer than in winter for both groups. In the control group, air quality is relatively stable from year to year. The average API in the treated group is higher than in the control group before 2008. In contrast, air quality improved significantly in 2008 for 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.

6 Results

6.1 The Effects of Air Pollution on Cardiovascular Mortality

We estimate the effects of air pollution on cardiovascular mortality using a fixed-effect instrumental variable model. The regression results are reported in Table 2. In the first stage, we estimate the effect of the two instruments (general air pollution regulation O_{it} and traffic control T_{it}) on the API using Equation (1) and controlling for both city-district fixed effects and month fixed effects. The results are summarized in columns 1-3. Both treatments are statistically significant at the 5% level. On average, O_{it} decreases monthly API by approximately 3.2 points, and T_{it} decreases monthly API by 11.5 points, conditional on city-districts fixed effect, month fixed effects, temperature, precipitation and their squares.

In the second stage, we estimate Equation (2), depicting the causal relationship between cardiovascular mortality and the API. The regression results, reported in columns 4-6 in Table 2, show a robust and statistically significant effect of the API on cardiovascular mortality. In the most restrictive specification (column 6), the estimated coefficient on the API is 0.161, with a 95 percent confidence interval of [0.015, 0.307]. If the API were to decrease by 10 points, monthly cardiovascular mortality would decrease by 1.61 per 100,000 people. Based on our estimates, the elasticity of cardiovascular mortality rate changes with respect to API changes is 2.02. Given that more than 690 million Chinese reside in urban areas, we estimate that more than 133,300 people would be saved from cardiovascular diseases each year if monthly API were to fall by 10 points.

For regulatory status to be a valid instrumental variable, regulations must affect cardiovascular mortality only through their effects on air pollution. Although this assumption cannot be directly tested, the regression results suggest that it is very likely to be true. In the first stage, including weather controls does not affect the point estimates of the two instruments but reduces their standard errors, suggesting that the two instrumental variables are not correlated with weather conditions. At the same time, the estimated effect of the API on cardiovascular mortality is stable and statistically significant in all three specifications. Including the weather controls decreases the estimates of the API only slightly. In other words, these control variables are not correlated with variations in the API induced by the regulations. In addition, the API coefficients are robust if we include a set of yearly social economic variables: per capita GDP, population density, the share of agriculture production, the share of manufacturing production, per capita investment in fixed-assets, per capita government expenditure, per capita government expenditure on

science and research, per capita government expenditure on education, and the per capita number of hospital beds.[9] However, due to endogeneity concerns associated with these yearly variables, we decided not to include them in the regressions. If air pollution variations induced by regulation are uncorrelated with these observable potential confounding factors, it is also likely that they are uncorrelated with other potential unobserved confounding factors.

6.2 Results for PM_{10}

Because the API is used only in China, it is difficult to compare our results with the findings of previous studies. We thus recover PM_{10} concentrations based on the API. PM_{10} is the primary air pollutant 89.5 percent of the time in daily samples (on 27 out of 30 days), so recovered monthly PM_{10} concentrations are usually accurate. The effects of PM_{10} on cardiovascular mortality are reported in Table 3.

Columns 1-3 report the results for the first-stage regressions. We find that both instruments are statistically significant at the 5 percent level. The relationship between PM_{10} and treatments are robust as we add more weather controls. Columns 4-6 report the effects of PM_{10} on cardiovascular mortality. The estimated coefficient of PM_{10} is 0.081 in the most restrictive model, a value that is statistically significant. The 95 percent confidence interval is [0.008, 0.154]. If PM_{10} concentrations were to decrease by 10 $\mu g/m^3$, monthly cardiovascular morality would decrease by approximately 0.81 per 100,000 people. Based on the estimates, the elasticity of cardiovascular mortality rate changes with respect to PM_{10} changes is 1.36. In other words, a 10% decline in PM_{10}

[9] See Appendix B for estimates that include district-level socio-economic variables.

approximately leads to a 13.6 percent reduction in the monthly cardiovascular mortality rate in China. The results imply that more than 67,000 lives in urban China could be saved from cardiovascular death every year if PM_{10} concentrations were to decrease by 10 $\mu g/m^3$ from current levels.

7 Robustness Checks

7.1 Internal Validity of the Main Results

To show that our results are not due to omitted factors that may have coincided with the reduction in air pollution since November 2007, we conduct a falsification test, using deaths caused by injuries. Injury mortality is an ideal alternative outcome variable because air quality levels should have no effect on it. If our findings are an artifact of unobserved omitted factors, significant and positive associations should also be found between air pollution and injury mortality rates.

In Table 4, we report the regression results for monthly injury mortality. The results for the API are summarized in columns 1-3, while the results for PM_{10} are summarized in columns 4-6. The estimated coefficients for both the API and PM_{10} are negative, close to zero and statistically insignificant in all specifications. The results indicate, as expected, that there is no relationship between air pollution and injury mortality.[10]

7.2 Confounding Activities during the Olympic Games

A potential threat to our main findings is that temporary factors may have affected cardiovascular mortality rates during the Olympic Games. Such factors may include

[10] To rule out extreme cases in which the Olympic Games might affect injury mortality (for example, traffic controls might reduce car accident deaths), we checked the same set of specifications excluding the traffic control months. The findings were unchanged.

increased exposure to outdoor air pollution of spectators of the Games, increased heart attacks associated with the excitement of sporting events, more timely medical treatment associated with improved traffic conditions, and greater availability of doctors owing to reduced numbers of patients coming from neighboring areas. The overall bias associated with these factors could be negative or positive.

The Olympics Games were held between August 8th and 24th. As shown in Figure 1, 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 the data from July and August 2008. If dropping these two months of data has a large impact (either positive or negative) on estimates of the API or PM_{10}, confounding factors associated with the period of the Games could potentially bias our results. Otherwise, we can feel confident about our estimates.

The regression results excluding observations for July and August of 2008 are reported in Table 5. The new coefficient estimates for the API (columns 1-3) and PM_{10} (columns 4-6) are only slightly smaller than those in Table 5 and Table 6. The coefficient estimates for the API range from 0.157 to 0.165 and are statistically significant at the 5 percent level in all three specifications. The coefficient estimates for PM_{10} range from 0.079 to 0.83 and are also statistically significant.

7.3 City-specific Trends

Another concern is that cardiovascular mortality and air quality in different cities may, for various reasons, follow different time trends, for example, differing economic growth rates. We include a set of city-specific time trends in the model to address this concern. However, inclusion of city-specific trends may unfavorably absorb too much variation in air

pollution, some of which was caused by the air pollution regulations. Thus, we must interpret these results with caution.

Table 6 reports the regression results. The relationship between cardiovascular mortality and air pollution is robust across different specifications, and the estimates are stable when more control variables are added. Because city-specific trends absorb too much variation in air pollution, the point estimates for the API and PM_{10} decrease by approximately 0.2 but remain statistically significant at the 10 percent level.

In addition, we conducted other robustness checks, for example, including cubic terms for temperature and precipitation as well as a set of yearly social-economic variables. The findings are robust to these specifications.

8 Comparison with Estimates from Associational Models

8.1 Cross-sectional Models and Fixed-Effects Models

The major concern in estimating the health effects of air pollution in the cross-sectional model and panel (fixed-effects) models is that air pollution may often be correlated with various omitted variables. We report the regression results for these models in this section.

A cross-sectional model can be written:

$$Y_{it} = \delta_0 P_{it} + X'_{it}\eta_0 + u_{it},\ u_{it} = v_i + \varepsilon_{it} \tag{3}$$

The effect of air pollution on health outcomes is captured by δ_0, if this effect 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 pollution (e.g., water pollution or hazardous waste), the estimates will be upwardly biased. However, if polluted areas are

relatively wealthy and have superior facilities and sanitation facilities, the cross-sectional estimates will be downwardly biased.

To check the robustness of the cross-sectional model, we first run regressions separately for each year, using three different specifications: without control variables, with temperature and its squared value, and with temperature, precipitation and their squared values. We then stack 5 years of data and estimate a pooled regression model.

The regression results are reported in Table 7. In the specification without control variables, the API is positively and statistically significantly associated with higher cardiovascular mortality rates in each year, with estimated coefficients ranging from 0.0288 to 0.0463. However, except in 2007, the coefficients become insignificant and the magnitudes of the estimates become much smaller when control variables are added. The results thus suggest that temperature and rainfall are correlated with air pollution levels and that they also affect mortality.

In the pooled regression, as seen in columns 16-18, the API is positively and statistically significantly associated with cardiovascular mortality in all three specifications. However, when precipitation and temperature are added, the estimated coefficient for the API decreases substantially (from 0.036 to 0.008). These results suggest that the cross-sectional model faces severe omitted variables problems: as we control for more confounding factors, the coefficient estimates for the API change dramatically. Hence, the API is highly unlikely to be exogenous. As a consequence, the cross-sectional estimates are likely to be smaller than the true effects.

Fixed-effects models remove bias from time-invariant factors and are useful when time-invariant factors explain much of the variation 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:

$$Y_{it} - \bar{Y}_i = \delta_0(P_{it} - \bar{P}_i) + (X'_{it} - \bar{X}'_i)\eta_0 + \varepsilon_{it} \quad (4)$$

where $\bar{Y}_i = \sum_{t=1}^{T} Y_{it}/T$, $\bar{P}_i = \sum_{t=1}^{T} P_{it}/T$, and $\bar{X}_i = \sum_{t=1}^{T} X_{it}/T$.

Table 8 summarizes the regression results for the fixed effects model. In columns 1-3, we control for city-district fixed effects. In columns 4-6, we additionally control for month fixed effects. If we only control for city-district fixed effects, the API is positively and statistically significantly associated with a higher cardiovascular mortality rate, with an estimated coefficient of 0.0263. When we also include month fixed effects, the estimated coefficient decreases to 0.0115 but remains statistically significant.

However, inclusion of the weather variables has a very large impact on the estimated effects of the API. Both in the city fixed effects model and the city and time fixed effects model, the significant relationship between the API and cardiovascular diseases disappears after controlling for precipitation and temperature. These results suggest that changes in air pollution fluctuations are correlated with changes in weather conditions and that changes in weather conditions may affect cardiovascular mortality directly or through channels other than air pollution. Thus, temperature plays a very important role in determining mortality in the fixed effects models. People are more likely to die on extremely hot and extremely cold days (Deschenes and Moretti, 2009).

The results for the fixed-effects models suggest that variations in the API over time and across regions cannot be treated as exogenous, even after controlling for city fixed

effects and month fixed effects. Because other unobserved variables (such as wind) may co-vary with both the API and cardiovascular mortality, estimates of the fixed-effects models may also be biased. In unreported results, the estimates of the effects of PM_{10} are also found to be sensitive to the inclusion of weather conditions in both cross-sectional and fixed effects models.[11]

8.2 Comparison with Epidemiological Studies

In this section, we compare our estimates with those of epidemiological studies. Many epidemiological studies have focused on short-term relationships between exposure to pollution and adverse health outcomes. Such studies often adopt time-series models to estimate the health effects of air pollution, using daily death and air pollution data.[12] As the present study evaluates the effects of relatively long-term exposure, quantitative comparisons between our study and daily time-series studies are difficult.[13] Furthermore, most studies focus on $PM_{2.5}$ rather than PM_{10}.

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, Pope, Xu, Spengler, Ware, Fay, Ferris Jr and Speizer (1993). In a cohort of 8,111 adults, with 14 to 16 years of follow-up, these 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

[11] The results are available upon request.
[12] See, for example, Sarnat et al. (2001) and Dominici et al. (2003) for the NMMAPS study; see Katsouyanni et al. (2001) for the APHEA2 study.
[13] Researchers have found that high-frequency time-series studies have reported substantially smaller health effects of air pollution than are indicated by the long-term cohort studies. For example, Schwartz (2000) showed 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.

accounting for the largest single category of difference in mortality. However, direct comparison between our results and these results is also difficult, as these authors used mortality rate ratios as their outcome variables.

Pope et al. (2002) conducted another large prospective cohort study of the long-term health effects of air pollution, using data from the ACS 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 each 10 $\mu g/m^3$ increase in the annual mean concentration 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 and 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 cardiovascular causes of death, a 10 $\mu g/m^3$ elevation in fine particulate matter was associated with an 8 to 18 percent increase in mortality risk, with greater risk observed for smokers than for nonsmokers.

9 Conclusion

This study has investigated the potential causal link between air pollution and cardiovascular mortality in China, using the 2008 Beijing Olympic Games as a natural experiment. Using monthly mortality, air quality and weather data from 34 urban cities in China from 2006 to 2010, we show that air pollution has a large and robust effect on cardiovascular mortality.

We estimate that a decrease in the API by 10 points will decrease monthly cardiovascular mortality by 1.61 per 100,000 people. We also estimate that a decrease in PM_{10} concentrations by 10 $\mu g/m^3$ will decrease monthly cardiovascular mortality by 0.81 per 100,000 people, corresponding to a 13.6 percent drop. That said, more than 67,000 lives will be saved in the urban areas of China from cardiovascular diseases each year if current PM_{10} pollution levels can be reduced by 10%.

We rule out the possibility that temporary migration or other factors related to the Olympic Games might confound our estimates by excluding data from July and August of 2008. In addition, we conduct a falsification test, finding that air pollution does not affect injury mortality. All these results indicate that the relationship between air pollution and cardiovascular mortality is likely causal.

In contrast, estimates from cross-sectional and fixed-effects models are not robust. Estimates from associational models are substantially smaller, indicating that they may underestimate the health effects of air pollution. Our findings are in keeping with Schlenker and Walker (2011), who also found that estimates of the health effects of air pollution obtained under a natural-experiment design were an order of magnitude larger than estimates obtained using non-experimental approaches.

Some researchers may be concerned about the issue of "harvesting," also called mortality replacement, which refers to the advancement of death by a few days or weeks of severely ill individuals. If elevated air pollution hastens the death of people who are already dying, gains in life expectancy as a result of slightly improved air quality will be small. However, this should not be a significant issue in the present study, as our treatment period is fairly lengthy (roughly one year).

The study has several limitations. First, we cannot obtain exact concentrations of different pollutants. Overall air quality is measured by the API, which is based on a calculation of concentrations of different air pollutants. Because different air pollutants decreased dis-proportionately during the regulation period,[14] it is unclear which air pollutants contribute most to cardiovascular diseases. While previous literature suggests that $PM_{2.5}$ and Ozone are risk factors for cardiovascular disease, we are unable to investigate these channels, due to data limitations. We estimate the effect of PM_{10} on cardiovascular mortality using concentrations of PM_{10} recovered from the API. The extent to which inaccuracy with respect to the concentrations of individual pollutants affects the results is unknown. Hence, the results should be interpreted with caution.

Second, we should emphasize that the estimated results are only locally valid. The effects of air pollution on cardiovascular mortality may be highly non-linear and negligible below a certain threshold. Our findings cannot be generalized to less polluted areas, for example, the rural regions of China.

Third, we ignore people's responses to changes in air quality and to the Olympic Games themselves. An individual's level of pollution exposure is determined by ambient 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

[14] For example, Rich et al. (2012) monitored daily air quality from July 20 to September 17 2008 in Beijing, observing differential reductions in the mean concentrations of different air pollutants from the pre-Olympic to the Olympic period: sulfur dioxide (−60 percent), carbon monoxide (−48 percent), nitrogen dioxide (−43 percent), elemental carbon (−36 percent), $PM_2.5$ (−27 percent), organic carbon (−22 percent), and sulfate (−13 percent). In contrast, ozone concentrations increased (24 percent). They also found that pollutant concentrations generally increased substantially from the Olympic period to the post-Olympic period for all pollutants (21 percent to 197 percent) except ozone (−61 percent) and sulfate (−47 percent).

particular, those at risk of being negatively affected by pollution may have relatively strong incentives to adopt compensatory/avoidance behaviors. For example, Neidell (2009) found that people responded to information about air quality, with smog alerts leading to significantly reduced attendance at major outdoor facilities in Los Angeles. Thus, the Olympic Games may have affected people's preferences between indoor and outdoor activities. The consequences of such behavioral changes on cardiovascular mortality require further investigation.

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Table 1. Summary Statistics

Variable	Mean	Std. Dev.	Min	Max
API	74.446	20.579	26.516	252.839
PM10 Concentration (ug/m^3)	99.254	39.661	26.516	386.987
Monthly Age-Adjusted Cardiovascular Mortality (per 100,000 People)	5.943	3.371	0	21.757
Precipitation (100mm)	0.726	0.794	0	5.700
Temperature (°C)	13.087	11.207	-20.298	31.012

Table 2. Fixed-Effects IV Models: Cardiovascular Mortality and API

	1st Stage, API			2nd Stage, Mortality		
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
O_{it} (Regulated)	-3.262**	-3.256**	-3.182**			
	(1.394)	(1.363)	(1.345)			
T_{it} (Traffic Control)	-11.182**	-12.466**	-11.547**			
	(4.634)	(4.523)	(4.325)			
API				0.192**	0.159**	0.161**
				(0.0838)	(0.0721)	(0.0743)
Temperature		-0.364	-0.420		-0.0176	-0.0118
		(0.507)	(0.499)		(0.0790)	(0.0805)
Temperature Sq.		0.031**	0.029**		-0.00681*	-0.00684*
		(0.013)	(0.014)		(0.00366)	(0.00369)
Precipitation			-3.225			-0.0343
			(2.700)			(0.447)
Precipitation Sq.			3.92e-5			4.25e-06
			(5.86e-5)			(7.86e-06)
City Fixed Effects	Y	Y	Y	Y	Y	Y
Month Fixed Effects	Y	Y	Y	Y	Y	Y
Observations	1,896	1,896	1,896	1,896	1,896	1,896
R-Squared	0.623	0.645	0.648	0.184	0.365	0.359

Notes: Standard errors are in parentheses and clustered at the city level. Asterisks, ***, ** and *, indicate the 1%, 5% and 10% significance level, respectively.

Table 3. Fixed-Effects IV Models: Cardiovascular Mortality and PM10

	1st Stage, PM 10			2nd Stage, Mortality		
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
O_{it} (Regulated)	-6.433**	-6.411**	-6.265**			
	(2.776)	(2.731)	(2.699)			
T_{it} (Traffic Control)	-22.613**	-24.855***	-23.049***			
	(9.216)	(9.035)	(8.617)			
PM10				0.0957**	0.0801**	0.0809**
				(0.0416)	(0.0363)	(0.0373)
Temperature		-0.706	-0.811		-0.0191	-0.0139
		(0.923)	(0.906)		(0.0746)	(0.0763)
Temperature Sq.		0.056**	0.053**		-0.00639*	-0.00643*
		(0.025)	(0.027)		(0.00341)	(0.00345)
Precipitation			-6.358			-0.0394
			(5.020)			(0.425)
Precipitation Sq.			8.07e-5			4.04e-06
			(1.10e-5)			(7.36e-06)
City Fixed Effects	Y	Y	Y	Y	Y	Y
Month Fixed Effects	Y	Y	Y	Y	Y	Y
Observations	1,896	1,896	1,896	1,896	1,896	1,896
R-Squared	0.628	0.648	0.650	0.228	0.385	0.380

Notes: Standard errors are in parentheses and clustered at the city level. Asterisks, ***, ** and *, indicate the 1%, 5% and 10% significance level, respectively.

Table 4. Falsification Test: Injury Mortality on Air Pollution

VARIABLES	(1) FEIV	(2) FEIV	(3) FEIV	(4) FEIV	(5) FEIV	(6) FEIV
API	-0.00877	-0.0127	-0.0205			
	(0.0260)	(0.0256)	(0.0273)			
PM10				-0.00427	-0.00634	-0.0103
				(0.0128)	(0.0128)	(0.0135)
Temperature		0.00771	4.77e-05		0.00786	0.000330
		(0.0367)	(0.0402)		(0.0365)	(0.0398)
Temperature Sq.		-0.00154*	-0.00153*		-0.00158*	-0.00158*
		(0.000911)	(0.000904)		(0.000899)	(0.000898)
Precipitation			-0.471***			-0.471***
			(0.159)			(0.154)
Precipitation Sq.			7.97e-06**			8.00e-06**
			(3.16e-06)			(3.11e-06)
City Fixed Effects	Y	Y	Y	Y	Y	Y
Month Fixed Effects	Y	Y	Y	Y	Y	Y
Observations	1,896	1,896	1,896	1,896	1,896	1,896
R-Squared	0.557	0.559	0.551	0.557	0.559	0.552

Notes: Standard errors are in parentheses and clustered at the city level. Asterisks, ***, ** and *, indicate the 1%, 5% and 10% significance level, respectively.

Table 5. Cardiovascular Mortality and API/PM10: 2008 July and August Data Excluded

VARIABLES	(1) FEIV	(2) FEIV	(3) FEIV	(4) FEIV	(5) FEIV	(6) FEIV
API	0.165**	0.157**	0.163**			
	(0.0715)	(0.0677)	(0.0716)			
PM10				0.0827**	0.0789**	0.0816**
				(0.0357)	(0.0338)	(0.0357)
Temperature		-0.0164	-0.00838		-0.0194	-0.0122
		(0.0740)	(0.0771)		(0.0694)	(0.0725)
Temperature Sq.		-0.00672*	-0.00684*		-0.00635*	-0.00646*
		(0.00348)	(0.00359)		(0.00326)	(0.00337)
Precipitation			0.0425			0.0418
			(0.445)			(0.423)
Precipitation Sq.			2.85e-06			2.48e-06
			(7.68e-06)			(7.23e-06)
City Fixed Effects	Y	Y	Y	Y	Y	Y
Month Fixed Effects	Y	Y	Y	Y	Y	Y
Observations	1,828	1,828	1,828	1,828	1,828	1,828
R-Squared	0.311	0.374	0.353	0.339	0.395	0.376

Notes: Standard errors are in parentheses and clustered at the city level. Asterisks, ***, ** and *, indicate the 1%, 5% and 10% significance level, respectively.

Table 6. Cardiovascular Mortality and API/PM10: with City-Specific Trends

VARIABLES	(1) FEIV	(2) FEIV	(3) FEIV	(4) FEIV	(5) FEIV	(6) FEIV
API	0.158*	0.137*	0.138*			
	(0.0868)	(0.0752)	(0.0769)			
PM10				0.0777*	0.0681*	0.0686*
				(0.0425)	(0.0374)	(0.0383)
Temperature		0.00376	0.00928		0.00377	0.00891
		(0.0702)	(0.0700)		(0.0661)	(0.0664)
Temperature Sq.		-0.00559*	-0.00561*		-0.00516*	-0.00519*
		(0.00297)	(0.00298)		(0.00272)	(0.00273)
Precipitation			-0.0801			-0.0688
			(0.375)			(0.355)
Precipitation Sq.			5.18e-06			4.64e-06
			(6.70e-06)			(6.27e-06)
City Fixed Effects	Y	Y	Y	Y	Y	Y
Month Fixed Effects	Y	Y	Y	Y	Y	Y
City-Specific Trends	Y	Y	Y	Y	Y	Y
Observations	1,896	1,896	1,896	1,896	1,896	1,896
R-Squared	0.391	0.491	0.489	0.429	0.511	0.509

Notes: Standard errors are in parentheses and clustered at the city level. Asterisks, ***, ** and *, indicate the 1%, 5% and 10% significance level, respectively.

Table 7. OLS Regressions: Cardiovascular Mortality and API

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
VARIABLES	2006	2006	2006	2007	2007	2007	2008	2008	2008
API	0.0383***	0.0128*	0.0120	0.0439***	0.0197**	0.0175**	0.0463***	0.0135	0.0126
	(0.00732)	(0.00747)	(0.00762)	(0.00691)	(0.00766)	(0.00779)	(0.00925)	(0.00957)	(0.00967)
Temp		-0.167***	-0.167***		-0.155***	-0.150***		-0.155***	-0.151***
		(0.0318)	(0.0319)		(0.0397)	(0.0405)		(0.0273)	(0.0285)
Temp Sq.		0.00283**	0.00299**		0.00308**	0.00359**		0.00158	0.00181
		(0.00114)	(0.00119)		(0.00140)	(0.00147)		(0.00106)	(0.00117)
Precip			-0.0106			-0.583			-0.278
			(0.565)			(0.546)			(0.546)
Precip Sq.			-4.69e-06			5.95e-06			3.71e-06
			(1.27e-05)			(1.19e-05)			(9.87e-06)
Obs.	372	372	372	408	408	408	408	408	408
R-squared	0.071	0.180	0.180	0.072	0.134	0.139	0.063	0.193	0.194

Table 7. (continued) OLS Regressions: Cardiovascular Mortality and API

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
VARIABLES	2009	2009	2009	2010	2010	2010	2006-10	2006-10	2006-10
API	0.0288***	0.00134	0.00130	0.0313***	0.00238	0.000267	0.0364***	0.00933**	0.00834**
	(0.0108)	(0.0116)	(0.0117)	(0.00898)	(0.00985)	(0.0101)	(0.00377)	(0.00386)	(0.00391)
Temp		-0.0910***	-0.0770***		-0.140***	-0.133***		-0.146***	-0.140***
		(0.0255)	(0.0260)		(0.0291)	(0.0306)		(0.0136)	(0.0139)
Temp Sq.		-0.000152	-0.000225		0.00191*	0.00204*		0.00198***	0.00215***
		(0.00102)	(0.00105)		(0.00111)	(0.00113)		(0.000508)	(0.000530)
Precip			-0.987**			-0.386			-0.425*
			(0.446)			(0.485)			(0.249)
Precip Sq.			3.16e-05***			5.83e-06			8.38e-06
			(9.56e-06)			(1.10e-05)			(6.22e-06)
Obs.	372	372	372	336	336	336	1,896	1,896	1,896
R-squared	0.024	0.115	0.127	0.030	0.152	0.154	0.047	0.149	0.150

Notes: Standard errors are in parentheses and clustered at the city level. Asterisks, ***, ** and *, indicate the 1%, 5% and 10% significance level, respectively.

Table 8. Fixed Effects Models: Cardiovascular Mortality and API

VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
API	0.0263***	-0.000387	-0.000486	0.0115***	0.00460	-0.000486
	(0.00381)	(0.00333)	(0.00349)	(0.00378)	(0.00417)	(0.00349)
Temperature		-0.0779***	-0.0762***		-0.0413**	-0.0762***
		(0.0133)	(0.0130)		(0.0195)	(0.0130)
Temperature Sq.		-6.99e-05	-9.74e-05		-0.00102**	-9.74e-05
		(0.000413)	(0.000427)		(0.000451)	(0.000427)
Precipitation			-0.119			-0.119
			(0.220)			(0.220)
Precipitation Sq.			4.27e-06			4.27e-06
			(5.50e-06)			(5.50e-06)
City-District Fixed Effects	Y	Y	Y	Y	Y	Y
Month Fixed Effects	N	N	N	Y	Y	Y
Observations	1,896	1,896	1,896	1,896	1,896	1,896
R-squared	0.578	0.617	0.617	0.641	0.649	0.617

Notes: Standard errors are in parentheses and clustered at the city level. Asterisks, ***, ** and *, indicate the 1%, 5% and 10% significance level, respectively.

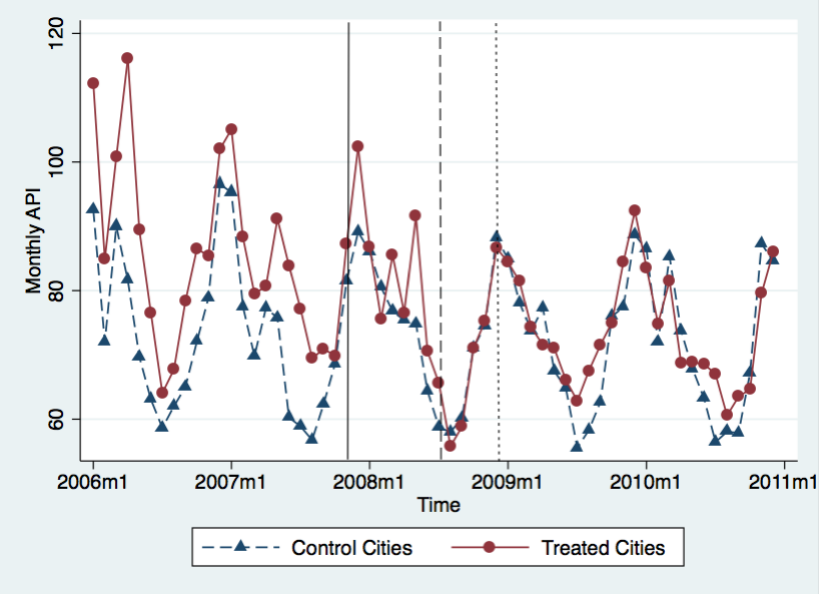


Figure 1. Air Pollution Index in the Treated and Control Group

Notes: The solid line indicates November 2007, when the pre-Olympic air quality interventions started; the dashed line indicates July 2008, when the Olympic Games temporary air quality interventions started. The dotted line indicates December 2008, when the air quality regulations ended.

Appendix A: API Calculation

The API score is constructed based on the concentrations of 3 atmospheric pollutants, namely sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and suspended particulates of 10 micrometers or less (PM_{10}) measured at the monitoring stations throughout each city. It is a proxy measure of the ambient air quality. API is calculated according to the maximum concentration of the three pollutants. Table A shows the relationship between API and the concentration of the five air pollutants.

Table A. The Relationship between API and Air Pollutant Concentrations

API	SO_2	NO_2	PM_{10}
50	0.05	0.08	0.05
100	0.15	0.12	0.15
200	0.8	0.28	0.35
300	1.6	0.565	0.42
400	2.1	0.75	0.5
500	2.62	0.94	0.6

Concentration is measured by mg/m^3.

The construction of API takes four steps. First, measure the daily average concentration of each pollutant. Second, for each pollutant, find out its corresponding concentration interval in Table A. Third, calculate the Pollution Index of each pollutant linearly. Finally, take the maximum of all pollution indices and define it as API.

For example, assume the concentrations of the three pollutants are: $C_{SO_2} = 0.07mg/m^3$, $C_{NO_2} = 0.10mg/m^3$, and $C_{PM_{10}} = 0.30mg/m^3$, then use Table 1 we find that the concentrations of SO_2, and NO_2 are in the interval [50,100] while the PM_{10} concentration falls into the interval [100,200]. Within each interval we can calculate pollution index of each pollutant linearly:

$$PI_{SO_2} = \frac{100 - 50}{0.15 - 0.05} * (0.07 - 0.05) + 50 = 60$$

$$PI_{NO_2} = \frac{100 - 50}{0.12 - 0.08} * (0.10 - 0.08) + 50 = 75$$

$$PI_{PM_{10}} = \frac{200 - 100}{0.35 - 0.15} * (0.30 - 0.15) + 100 = 175$$

Then $API = \max[PI_{SO_2}, PI_{NO_2}, PI_{PM_{10}}\} = 175,$, and PM_{10} is called the primary pollutant. According to the standard of SEPA, an API below 50 is defined as "excellent" air quality, 50-100 as "good", 100-200 as "slightly polluted", 200-300 as "moderately polluted" and above 300 as "severely polluted."

Appendix B: Regression Results with Social-Economic Controls

We include a rich set of social-economic controls to check the robustness of the results. Those variables are: per capita GDP, population density, the share of agriculture production, the share of manufacturing production, fixed-assets investment per capita, government expenditure per capita, government's expenditure on science and research per capita, government's expenditure on education per capita, and number of hospital beds per capita. These variables are collected from the statistical yearbooks in different provinces.

We find that the estimated effects of air pollution on cardiovascular mortality are slightly smaller when all the social-economic controls are included, but are still robust and statistically significant at 10% level. However, due to potential endogeneity of these yearly social-economic variables, the estimates may be biased. Moreover, the statistics for these variables are not available for all cities in all years. The smaller sample size may also partially cause changes in estimates and their significance.

Table B. Cardiovascular Mortality and API/PM10

	(1)	(2)	(3)	(4)	(5)	(6)
VARIABLES	FEIV	FEIV	FEIV	FEIV	FEIV	FEIV
API	0.152*	0.127*	0.126*			
	(0.0824)	(0.0674)	(0.0701)			
PM10				0.0757*	0.0633*	0.0633*
				(0.0408)	(0.0337)	(0.0352)
Temperature		-0.0124	-0.00909		-0.0154	-0.0126
		(0.0724)	(0.0717)		(0.0684)	(0.0680)
Temperature Sq.		-0.00600*	-0.00601*		-0.00577*	-0.00578*
		(0.00326)	(0.00326)		(0.00302)	(0.00302)
Precipitation			-0.128			-0.111
			(0.379)			(0.365)
Precipitation Sq.			5.17e-06			4.44e-06
			(6.81e-06)			(6.52e-06)
GDP pc	-0.488***	-0.458***	-0.456***	-0.487***	-0.457***	-0.455***
	(0.175)	(0.165)	(0.165)	(0.174)	(0.164)	(0.164)
Pop Density	-5.108	-4.682	-4.704	-5.189	-4.758	-4.777
	(3.181)	(3.127)	(3.173)	(3.172)	(3.116)	(3.160)
Ag. Share	0.190	0.150	0.150	0.194	0.154	0.154
	(0.409)	(0.423)	(0.424)	(0.407)	(0.421)	(0.421)
Manufacture Share	-0.111*	-0.115**	-0.115**	-0.110*	-0.115**	-0.114**
	(0.0586)	(0.0566)	(0.0568)	(0.0575)	(0.0558)	(0.0559)
Fixed-Asset Inv. pc	0.480*	0.448*	0.443*	0.488*	0.456*	0.452*
	(0.287)	(0.265)	(0.264)	(0.292)	(0.269)	(0.269)
Gov. Exp.	-4.63e-05	-5.68e-05	-5.56e-05	-7.00e-05	-7.58e-05	-7.47e-05
	(0.000207)	(0.000196)	(0.000195)	(0.000207)	(0.000197)	(0.000196)
Science Exp.	-8.29e-06	-0.000298	-0.000289	8.07e-05	-0.000219	-0.000210
	(0.000986)	(0.000935)	(0.000938)	(0.00102)	(0.000966)	(0.000969)
Edu Exp.	-0.000710	-0.000577	-0.000587	-0.000730	-0.000596	-0.000603
	(0.000972)	(0.000928)	(0.000937)	(0.000988)	(0.000943)	(0.000953)
Beds pc	0.0168	0.0131	0.0131	0.0185	0.0145	0.0145
	(0.0182)	(0.0173)	(0.0174)	(0.0185)	(0.0176)	(0.0177)
City Fixed Effects	Y	Y	Y	Y	Y	Y
Month Fixed Effects	Y	Y	Y	Y	Y	Y
Observations	1,788	1,788	1,788	1,788	1,788	1,788
R-squared	0.371	0.496	0.498	0.402	0.513	0.514

Notes: Standard errors are in parentheses and clustered at the city level. Asterisks, ***, ** and *, indicate the 1%, 5% and 10% significance level, respectively.