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

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Abstract

The 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 robust and 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. The estimates are larger than conventional estimates in the cross-sectional studies and fixed-effects studies.

Keywords: Air Pollution, Cardiovascular Mortality
JEL codes: Q53; I10

1 Introduction

To fulfill its international commitment to maintain high air quality during the 2008 Beijing Olympic and Paralympic Games, the Chinese government implemented a set of stringent policies to reduce local and regional emissions in the greater Beijing metropolitan area. These aggressive controls 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 how the anthropogenic improvement in air quality during the Olympic Games affects cardiovascular mortality in 34 urban city-districts of 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 with a fixed-effect instrumental variable model.

Over the last decade, a growing body of epidemiological and clinical evidence has led to a 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, searchers have found that air pollution is statistically significantly associated with cardiac arrests (for example, Silverman et al. 2010; Dennekamp et al. 2010; Ensor et al. 2013.) Cardiac arrests often occur to the people who have no pre-existing cardiac conditions. Thus, if air pollution actually caused people to die through cardiac arrests, the loss of life expectancy could be very large. Besides, researchers also found 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 deaths (Forastiere et al. 2005), heart rate variability (De Hartog et al. 2009), and inflammation and thrombosis (Rich et al. 2012), etc.

However, whether the estimates from those associational studies have causal interpretations is controversial. Time series studies, cross-sectional studies, and even longitudinal studies may all suffer from endogeneity issues (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. Thus, the relationship estimated using this natural experimental approach is very likely to be causal.

This study has several other attractive features. First, it uses less aggregated (monthly) data in many city-districts (34 cities) for a relative long period (5 years), which allowing us to check the robustness of different models. Second, presumably, the health effects of air pollution is highly nonlinear and very small below certain thresholds, so studies focusing on severely polluted regions, such as China, will help us understand the health effects of severe air pollution. Third, since we have cause-specific death information, we can use deaths that should not be affected by air pollution, the injury, to conduct convincing falsification tests.

This study finds 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.

The 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.

Section 2 discusses the literature on the health effects of air pollution. Section 3 summarizes the government's interventions on air pollution during 2008 Beijing Olympic Games. Section 4 describes the datasets and provides summary statistics. Section 5 discusses the research design and estimation strategy. Section 6 presents the main results, and Section 7 checks the robustness. Section 8 compares the results with associational models and previous non-experimental estimates, and Section 9 concludes.

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 centur. [2] So far several hundred published epidemiological studies have linked air pollution with morbidity and mortality both in the short run and in the long run (see Brunekreff and Holgate, 2002; Pope, 2000; and Brook et al., 2003 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-experiments 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), and most of these studies find that temporary elevation in air pollution is associated with worse health outcomes (see 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 actually caused by elevated air pollution or by other risk factors.[3] Moreover, since there is no an adequate control group, it is difficult to rule out alternative explanations in nearly all time-series studies.

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

[3] For example, Beijing's thick smoggy days in 2013, which were intensively documented by the mass media, largely resulted from the combined effects temperature, humidity and wind. Most of these smoggy days occurred in conditions relatively low wind during the winter. If people are more likely to die on cold or windy days, without controlling for these factors, the association between air pollution and increased mortality can be misleading unless these factors are controlled.

Cross-sectional studies compare the health outcomes across locations and examine how air pollution is associated with the health outcomes after controlling for potential confounding factors. However, this type of 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-economic factors, a correlation between air pollution and health status does not necessarily indicate that there is a causal relationship. In practice it is infeasible to control for all potential confounding factors, so the estimates of the health effect of air pollution using cross-sectional models may be biased.

Cohort-based longitudinal studies (Dockery et al, 1993) may suffer from similar problems 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 data on long-term exposure to air pollution. However, to some extent, exposure to different levels of pollution is an outcome of people's selection process. People migrate and endogenously choose which a level of air pollution. For example, wealthy people, whose health status is good for other reasons, can migrate to clean regions while poor people have to stay in the polluted regions. The observed association between air quality and mortality may be the consequence of factors other than air pollution. As suggested by Chay and Greenstone (2003a, 2003b), these associational approaches tend to produce unreliable estimates.

As data becomes more available, recent studies have used fixed-effects models (Currie and Neidell, 2005; Currie et al., 2009) to remove permanent sources of bias. Fixed effects models are particularly useful when time-invariant 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 largely depend on similar factors as health outcomes do (such as weather), this assumption may not hold as well.

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 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. They found 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 how Clean Air Act Amendments affected infant mortality. They used nonattainment status as an instrument for TSPs changes and estimated that a 1 percent decline in TSPs resulted in a 0.5 percent decline in the infant mortality rate. Jayachandran (2009) analyzed how air quality (particulate matter) changes caused by the wildfires in Indonesia affected infant and child mortality. She found that the pollution led to 15,600 missing children. Luechinger (2010) investigated the effect of SO_2 on infant mortality in Germany. He studied the natural experiment created by the mandated desulfurization at power plants, with wind directions dividing counties into treatment and control groups. As for China, Tanaka (2012) estimated the effect of air pollution on infant mortality, using air quality variations induced by the SO_2 and acid rain control zone in the 1990s.

While nearly almost all the associational studies have suggested that air pollution is positively and significantly associated with higher adult mortality rates, Chay et al. (2003) challenged this consensus. Using the Clean Air Act as an instrumental variable for air quality, they found that even though the regulatory status was associated with large reductions in total suspended particulates, such reductions had little effect on either adult or elderly mortality. The reason that Chay et al. (2003) could not find the effects of air pollution on adult mortality may be 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 some specific disease mortality.

With respect to morbidity, Schlenker and Walker (2011) estimated the health effects of air pollution induced by airline network delays in the United States. They found that carbon monoxide (CO) exposure led to significant increases in hospitalization rates for asthma and respiratory diseases, and heart related emergency room admissions that were an order of magnitude larger than conventional estimates. The effects were statistically significant for infants, the elderly and the adult population.

3 Air Pollution Regulations during the Olympics

The air pollution controls instituted during the 2008 Beijing Olympic Games are perhaps by far the largest interventions in air quality in human history. In order 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 the "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 Games. The Measures defined

November 2007 - July 20th 2008 as the pre-Olympic Comprehensive Regulation period, and July 20th – September 20th in 2008 as the Olympic Games Temporary Pollution Control period.

During the pre-Olympic Comprehensive Regulation period, multiple measures were implemented simultaneously: (1) all the 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 the heavy-emission vehicles; (3) oil-gas gathering units and recovery system were installed on gas stations, oil storage facilities and tankers; (4) The Second Beijing Chemical Plant, Beijing Eastern Petrochemical Company and several other polluting factories were completely shut down; (5) the government raised the gas prices in November 2007 and June 2008 respectively to discourage auto vehicle usage; (6) the Capital Steel Company was required to relocate, and its production of steel decreased from more than 600,000 tons per day to less than 200,000 tons per day.

Motor vehicle exhaust emissions are the major air pollution sources in large cities. To ensure good air quality, Beijing implemented temporary traffic control during the Olympic Games Temporary Pollution Control period. From July 1 to September 20, 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 no longer allowed on the roads. From July 20 July to September 20, vehicles with plates ending with odd numbers were allowed on the road only on odd dates and those with plates ending with even

numbers were allowed only on even dates. Only a few exceptions, such as police vehicles, public transports, vehicles with Olympic passes were exempted from the odd-even plate rule. More than two million auto vehicles were pulled off Beijing's roads depending on their license plate number every day. According to the report of the committee of the Olympic Games and the State Environment Protection Agency in China (2008), the total vehicle exhausts emissions reduced by more than 60 percent.[4] Traffic controls significantly decreased the concentration of fine particulate, ozone, nitrogen oxide and other pollutants generated by auto vehicles in Beijing.

At the same time, the government required that all the power plants and chemical production plants reduced their emissions by 30 percent from their previous emission levels, even though these plants had already met the national emission standard. 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 the particulate matter pollution, the Chinese government also halted all construction projects during the Games.

Because air quality in Beijing was affected by its neighbors, 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 the power plants and 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 soccer games were held in Tianjin, Shenyang and Qinhuangdao, similar air pollution controls

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

were implemented in these cities as well. For example, Tianjian shut down many polluting factories before the Games and enforced temporary traffic control during the games. Shenyang, replaced all the old buses prior to the Olympics and expanded green belts around construction facilities. Qinhuangdao built shelter forests along the piers to reduce dust, swept streets with water and dumping 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 UNEP (2009) and Chen et al. (2011). UNEP (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 approximately 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, UNEP (2009) showed that *CO* and *NO₂* concentrations decreased significantly, with *NO₂* levels, for example, falling by 50 percent from 2005 to 2008. Chen et al. (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

This study combines several data sets to address the research question for China. We are able to trace out the evolution of cardiovascular mortalities and air quality across cities over time, linked with local weather conditions.

4.1 Air Quality Data

Air Quality data come from the monitoring sites in the State Environment Protection Agency (SEPA). It provides daily air quality information for 82 major urban cities in China from 2000 to present. Air quality data include the following information: daily air pollution index (API), primary pollutant and air quality level.

API is an overall measure of ambient air quality. A higher value of API indicates a worse air quality. Ideally we hope to obtain the concentrations of specific air pollutants. However, the pollutant concentrations are not yet public available. Fortunately, the method used by SEPA to construct API allows us to approximately recover the concentration of the primary pollutant.[5]

In the daily data, PM_{10} is the primary pollutant in most of the time (89.58 percent); SO_2 is the primary pollutant in 10.19 percent of all time; and NO_2 is the primary pollutant in 0.23 percent of all time. The other two pollutants, CO and O_3, are never primary pollutants. Due to the dominant share of PM_{10} as the primary pollutant, we can approximately recover the concentration of PM_{10}.

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[5] See appendix A on how to recover the concentration of the primary pollutant using API.

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Airborne particulate matter consists of a heterogeneous mixture of solid and liquid particles suspended in air. Primary particles are emitted directly into the air, such as diesel soot, whereas secondary particles are created through physicochemical transformation of gases. There are many sources of particulate matter, such as motor vehicle emissions, power generation and other industrial combustion, smelting and other metal processing, construction, wood burning, forest fires and combustion of agricultural debris, etc.

The reliability of the Chinese official air quality data have been questioned by both mass media and researchers. The government's unwillingness to publicize specific concentrations of different pollutants further imposes difficulties for the researchers to verify its reliability. Chen et al. (2012) assessed the quality of China's API data and found that there exists a discontinuity at the threshold of 100 because China defines a day with an API at or below 100 as a blue-sky day and the local government has incentives to manipulate the data around this threshold. Nevertheless, Chen et al. (2012) found that API data ha strong correlations with NASA's Aerosol Optical Depth data and China Meteorological Administration's visibility data, and such correlations do not change significantly when the API is closely below or above 100. They concluded that although count of blue-sky days may be subject to data manipulation, the reported API does contain useful information for cross-city and cross-time variations of air pollution.

4.2 Death Data

Death data come from the Disease Surveillance Point System (DSPS) in China Center for Disease Control and Prevention (CDC). DSPS was initiated in 1978, covered 71 counties in 29 provinces 1980 to 1989, and 145 counties in 31 provinces from 1990 to 2000, and

161 counties from 2003 to now. The system adopts a multi-stage cluster population probability sampling method in order to represent the population and death trends countrywide.

DSPS recorded nine categories of death causes: cancer, cerebrovascular diseases, digestive system diseases, cardiovascular diseases, injuries, perinatal diseases, respiratory system diseases, urine and procreative systems diseases, and other diseases.

The primary dependent variable is monthly age-adjusted cardiovascular mortality, which is defined as the number of deaths caused by cardiovascular diseases per month at a given city-district per 100,000 people, adjusted by age distribution.

We calculated monthly number of deaths caused by cardiovascular diseases and by age group from 2006 to 2010 based on their 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.

Age-group specific mortality rate in a specific death surveillance point is calculated as:

$$Age\_Spec\_MR_{group_i} = \frac{100{,}000 * Death_{group_i}}{Total\ Population_{group_i}}$$

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

$$Age\_Adj\_MR = \sum_k (Population\ Weight_{group_i} * Age\_Spec\_MR_{group_i})$$

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

Cardiovascular mortality data and API data are matched at the monthly level. Thirty-four urban city-districts of China were found in both datasets. This study focuses

on a five-year window, from January 2006 to December 2010. Cardiovascular diseases are the No. 3 cause of death in China. In the sampled cities, the leading cause of deaths is cancer. Roughly 28 percent people die from cancer. The second leading cause of death is cerebrovascular diseases, accounting for 20 percent of all deaths. The share of deaths caused by cardiovascular diseases is 17 percent. The sample covers roughly 76,000 deaths caused by cardiovascular diseases. The monthly average age-adjusted cardiovascular mortality is 5.94 deaths per month per 100,000 people, 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 monthly average precipitation and temperature for given longitudes and latitudes with the minimum cell size of 0.5 degree by 0.5 degree.

We first identified the coordinates of the 34 sampled city-districts. For each city-district, we collected rainfall and temperature data for its 4 nearest points in the GHCN data. We calculated a weighted average precipitation and temperature using the inverse squared distance as the weights. For example, an interpolated precipitation of location j using the nearest four points k, k =1, 2, 3, 4 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 the precipitation at point j, and $Distance_{jk}^{-2}$ is the distance between j and k.

Both rainfall and temperature may affect the air pollution level as well as 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. Air pollution will increase in both extreme hot and cold days due of excessive energy consumption. People are also more likely to die on extreme hot or cold days. The summary statistics of the key variables are in Table 1.

5 Research Design and Model

We estimate the effects of air pollution on cardiovascular mortality using a fixed-effects instrumental variable model:

$$P_{it} = \lambda_1 O_{it} + \lambda_2 T_{it} + X'_{it}\eta_0 + u_i + v_t + \xi_{it} \qquad (1)$$

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

where P_{it} is the air pollution levels in city i at time t, Y_{it} is the health outcome. O_{it} is a regulation status indicator. If city i was regulated at time t, $O_{it} = 1$, otherwise it is 0. T_{it} is the traffic control status indicator. If city i enforced traffic control during the Olympic Games, $T_{it} = 1$, otherwise it is 0. X_{it} is a set of control variables, u_i is city fixed effects, v_t is time fixed effects, and ξ_{it} are unobservable disturbances.

As discussed in the previous sections, radical air pollution controls were implemented from 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, were permanent. Because the effects of these regulations might have long-lasting impact 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 province from November 2007 to December 2008, otherwise it is 0.

Beijing and Tianjin faced more stringent regulations than other cities during the Olympics. Both cities enforced temporary traffic controls from July-September, 2008. To capture the treatment intensity differences, we include a traffic control dummy T_{it} (served as another instrumental variable), which is equal to 1 during July-September 2008 for districts in Beijing and Tianjin.[6]

In the first stage, we estimate how the air quality regulations affect air pollution, using Equation (1). The coefficient λ_1 is essentially a Difference-in-Difference estimator, capturing the difference in the changes in air pollution levels in regulated periods (November 2007 to December 2008) and non-regulated periods (January 2006 to October 2007, January 2009 to December 2010), between the localities that are and are not regulated. λ_2 has similar interpretations. We expect both λ_1 and λ_2 to be negative. In the second stage of the IV regression, we take the estimated pollution level $\hat{P}_{it}$ from the first stage into Equation (2). If air pollution harms people's health, we expect that less people die from cardiovascular diseases in the good air quality episodes in the regulated cities and that δ_0 is positive.

Figure 1 shows the trends of the monthly average pollution index (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 average pollution index (API) in 2008. We observe strong seasonality in the trends of air quality for both treated

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

and control group. In both groups, air quality is better in summer than in winter. In the control group, air quality from year to year is relatively stable. In contrast, air quality was significantly improved in 2008 in the treated group. The average API in the treated group is higher than that in the control group, but both groups follow similar trends before 2008. Compared to 2008, air quality in 2009 and 2010 in the treated group became slightly worse, suggesting that the effects of the regulations on air pollution diminished as time elapsed.

6 Results

6.1 Air Pollution on Cardiovascular Mortality

We estimate the effect of air pollution on cardiovascular mortality in 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 treatments (general air regulation O_{it} and traffic control T_{it}) on API using Equation (1), after controlling for both city-district fixed effects and fixed effects for each month (59 dummies for 60 months). The results are summarized in columns 1-3. Both treatments are statistically significant at 5% level. On average O_{it} decreases monthly API by about 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), the relationship between cardiovascular mortality and API. The regression results are reported in columns 4-6 in Table 2. We find a robust and statistically significant relationship between cardiovascular mortality and API. In the most restrictive specification, column 6, the estimated coefficient of API is 0.161, with a 95 percent confidence interval [0.015, 0.307]. If we

decrease API by 10 point, monthly cardiovascular mortality would decrease by 1.61 per 100,000 people.

China has more than 690 million urban people; a rough calculation suggests that each year more than 133,300 people would be saved from cardiovascular diseases if monthly API increases by 10 point.

For the regulatory status to be a valid instrumental variable, we require that these regulations affect cardiovascular mortality only through its impacts on air pollution. Even though this assumption cannot be directly tested, the regression results suggest it is very likely to be true. In the first stage, including the weather controls does not affect the point estimates of the two instruments but reduces their standard errors, suggesting that those two instrumental variables are not correlated with weather conditions. At the same time, the estimated effect of API on cardiovascular mortality is statistically significant in all three specifications. Including the weather controls only slightly decreases the estimates of API, and the change of magnitude is negligible. In other words, these control variables are not correlated with API variations induced by the regulation. If air pollution variations induced by the regulation are not correlated with these observable potential confounding factors, it is also more likely that they are not correlated with other potential unobserved confounding factors.[7]

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[7] The results 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. However, due to concerns of endogeneity of these yearly variables, we decide not to include them in the regressions.

6.2 Results for PM10

API is an index and is only used in China, making it very hard to compare the estimates with findings in other studies. We thus recover the PM_{10} concentrations based on API. PM_{10} is the primary air pollutant for 89.5 percent of the time in the sample, so the recovered PM_{10} concentration is accurate for most of the time. The effects of PM_{10} on cardiovascular mortality are reported in Table 3.

Columns 1-3 report the first-stage regressions. We find that both treatments are statistically significant at 5 percent level. The relationship between PM_{10} and treatments are robust as we include 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 and is statistically significant. The 95 percent confidence interval is [0.008, 0.154]. If PM_{10} concentration decreases by $10\ \mu g/m^3$, monthly cardiovascular morality would roughly decrease by 0.81 per 100,000 people. In other words, a 10 $\mu g/m^3$ decline in PM_{10} roughly leads to a 13.6 percent reduction in monthly cardiovascular mortality in China. Based on this estimation, more than 67,000 lives in urban China can be saved from cardiovascular death if PM_{10} concentration decreases by $10\ \mu g/m^3$ from its current level.

6.3 Falsification Test Using Deaths by Injuries

We conduct a falsification test using deaths caused by injuries. Injury mortality is an ideal comparison group because air quality levels should not affect injuries. If our findings are an artifact resulting from the unobserved overall death patterns, similar results should be found in other mortalities as well.

In Table 4 we report the regression results for monthly injury mortality. The results for API are summarized in columns 1-3 and the results for PM_{10} are in columns 4-6. The estimated coefficients of both API and PM_{10} are close to zero and not statistically significant in all the specifications. The estimated coefficients of air pollution on injury mortality are negative and the standard errors are large. The results indicate that there is no relationship between air pollution and injury mortality. Just as expected, air pollution does not affect injury mortality.[8]

7 Robustness Checks

7.1 Migration and Other Potential Threats

One potential threat of the findings is temporary migration. If there exists temporary migration during the Olympic Games, our estimates may be biased. The bias may go both ways. On the one hand, if healthier people migrate into Beijing and other regulated cities during the Olympics (perhaps to watch the games), the health effects of air pollution will be over-estimated. On the other hand, the Olympics Games created more jobs in the construction and service industries, and thus it could attract more rural migrant workers to migrate in. Presumably, people in the rural areas are less healthy in China. If more unhealthy people migrate into the regulated cities, the health effect of air pollution will be under-estimated.

We check the robustness of our results by excluding the data in 2008 July and August. As shown in Figure 1, the largest air quality improvement occurred in July, and

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[8] To rule out the extreme case in which the Olympic Games somehow still affected injury mortality (for example, traffic controls might reduce car accident deaths), we check the same set of specifications excluding the traffic control months and the findings are the same.

air quality was the best in August. The Olympics Games were held between August 8th and August 24th.

If there was no migration at all or the migration didn't affect the population health structure, we should expect fewer deaths in these two months because of the improved air quality. Dropping the two months data would result in slightly smaller estimated coefficients of API or PM_{10}, since some observations that are prone to identify a larger effect are missing.

In contrast, if dropping the July-August sample has a large impact (either positive or negative) on the estimates, migration or other factors that occurred during the Olympics might potentially confound our results.

The regression results excluding 2008 July and August observations are reported in Table 5. In columns 1-3 we summarize the results for API; and in columns 4-6 we summarize the results for PM_{10}. As expected, the estimated coefficients of API and PM_{10} are slightly smaller than those in the Table 5 and Table 6. The estimated coefficient of API ranges from 0.157 to 0.165 and is statistically significant in all three specifications at 5 percent level, and the estimated coefficient of PM_{10} ranges from 0.079 to 0.83 and is statistically significant, too.

The findings suggest that even if there might exist temporary migration during the Olympic Games, this migration does not have a large impact on our estimates. The robust results also ruled out the possibility that other temporary activities during the Olympics had a large effect on cardiovascular mortality. Such possibilities include, for example, people's exposure to outdoor air pollution being changed during the Games due to

watching Games, people being too excited and thus experiencing more heart attacks, and medical treatment becoming more available because of less traffic.

7.2 City-specific Trends

Another concern is that cardiovascular mortality and air quality in different cities may have different trends. We check if our findings are sensitive to including city-specific trends. In Table 6 we report the regression results.

Once again, the relationship between cardiovascular mortality and air pollution is robust. The estimated coefficients are stable when we include more control variables. They are still statistically significant at 10 percent level.

Since this set of specifications is too restrictive, the point estimates of API and PM_{10} decreases by about 0.2. The reason is that including city-specific trends unfavorably absorbs too much variation in air pollution, some of which were caused by the air pollution regulations.

Even though not reported, we also conducted other robustness checks such as including the cubic terms for the temperature and precipitation, and including some yearly social-economic variables. The findings are the same.

8 Comparison with Estimates from Associational Models and Previous Studies

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 be often correlated with a number of omitted variables. We report the regression results from these models in this section.

A cross-sectional model can be written as:

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

δ_0 captures the effect of air pollution on the health outcome if it is not correlated with the unobservable disturbance, $E[P_{it}u_{it}] = 0$. However, since air quality is not randomly assigned across locations, this condition my not hold. For instance, if higher air pollution is associated with higher level of other pollution (water pollution, hazardous waste), the estimates will be upwardly biased. On the other hand, if polluted areas are also wealthier and have better medical treatment and sanitation, the cross-sectional estimates will be downwardly biased.

To check the robustness of cross-sectional models, we first run regressions separately for each year with three different specifications: without any control variables, with temperature and its square, and with temperature, precipitation and their squares. Then we stack the 5 years of data and estimate a pooled regression model.

The regression results are reported in Table 7. Without any control variables, API is positively and statistically significantly associated with higher cardiovascular mortality rates in each year. The estimated coefficient ranges from 0.0288 to 0.0463. However, they become insignificant as we include the control variables except in 2007. The estimated coefficients are very sensitive to the weather variables, suggesting that temperature and rainfall are correlated with air pollution levels, and they also affect mortality.

In the pooled regression, columns 16-18 show that API is positively and statistically significantly associated with the cardiovascular mortality rate in all three specifications. As we include the weather controls, the estimated coefficients of API

change substantially (from 0.036 to 0.008). These results suggest that cross-sectional models face severe omitted variables problems: as we control for more confounding factors, the estimates of API are affected. API is very unlikely to be exogenous.

Fixed-effects models remove permanent sources of bias and are particularly useful when time-invariant factors explain much 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, too.

In a fixed-effects model, we estimate:

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

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 fixed effects models. In columns 1-3, we control for city-district fixed effects. In columns 4-6, we also control for fixed effects for each month.

If we only control for city-district fixed effects, API is positively and statistically significantly associated with higher cardiovascular mortality, and the estimated coefficient is 0.0263. When we also include the fixed effects for each month, the estimated coefficient decreases to 0.0065 and becomes statistically insignificant.

Inclusion of the weather variables has a very large impact on the estimated effects of the API in the city fixed effects model. The significant relationship between the API and cardiovascular diseases disappears after controlling for precipitation and temperature. In the city and time fixed effects model, API becomes statistically significant at 10% level after controlling for temperature. The statistical significance disappears when we

23

further control for precipitation. 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. Temperature plays a very important role in determining mortality in the fixed effects models. People are more likely to die on cold days.

The results from the fixed-effects models suggest that API variations from month to month and from location to location cannot be treated as exogenous even after we control for city fixed effects and month fixed effects. Since there may exist other unobserved variables (such as winds) that co-vary with both API and cardiovascular mortality, the estimates from fixed-effects models may be biased as well.

Though not reported, the estimated coefficient of PM_{10} is also sensitive to the inclusion of weather conditions in both cross-sectional and fixed effects models.[9]

8.2 Comparison with Previous Studies

We compare our estimates with epidemiological studies in this section. Many epidemiological studies have focused on short-term relationships between pollution exposure and adverse health outcomes. These studies often adopt time-series models to estimate acute health effects of air pollution using daily death and air pollution data.[10] Since our study evaluated relatively long-term exposure, quantitative comparisons with daily time-series studies are difficult to make.[11] Instead, we focus on several noteworthy long-term cohort studies here.

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[9] The results are available upon requests.

[10] See for example, Samet et al. (2000) and Dominici et al. (2003) for the NMMAPS study; Katsouyanni et al. (2001) for the APHEA2 study.

[11] Researchers have found that high-frequency time-series studies have reported substantially smaller health effect of air pollution than are indicated by the long-term

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 to 16 years of follow-up, they found that the adjusted overall mortality rate ratio for the most-polluted city versus the least-polluted city was 1.26, and cardiovascular deaths accounted for the largest single category of the increase mortality. However, direct comparison between our results with theirs is also hard to make since they used mortality rate ratios as 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. In approximately 500,000 adults in all 50 states in the United States, chronic exposure to multiple air pollutants was linked to mortality statistics for a 16-year window. They showed that each $10 \, \mu g/m^3$ increase in annual fine particulate matter ($PM_{2.5}$) mean concentration 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 looked into the association between specific cardiopulmonary diseases to explore potential mechanistic pathways linking exposure and mortality. They found that long-term particulate matter exposures were 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 8 percent to 18 percent increases in mortality risk, with larger risk being observed for smokers relative to nonsmokers.

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cohort studies. For example, Schwartz (2000) showed that as data became more aggregated the effects of air pollution on ischemic heart disease mortality and all-death mortality became larger, suggesting a greater effect of long term exposure, possibly due to development of chronic disease.

Clancy et al. (2002) analyzed the effect of air pollution control on death rates in Dublin, Ireland. In September 1990, the Irish Government banned the marketing, sale and distribution of bituminous coals within the city of Dublin. The effect of this intervention resulted in an immediate and permanent reduction in particulate concentrations. They found that the average black smoke concentration in Dublin declined by $35.6 \, \mu g/m^3$ after the ban; and that the reduction of air pollution is associated with 10.3 percent decline of standardized cardiovascular death rates.

In our natural-experimental design, we estimate that a $10 \, \mu g/m^3$ decline in PM_{10} results in a 13.6 percent reduction in monthly age-adjusted cardiovascular mortality in China. Giving that $PM_{2.5}$ concentration is only a fraction of PM_{10} concentration, our estimate is substantially larger, compared to previous cohort studies. This result suggests that associational studies may have underestimated the effect of air pollution on cardiovascular mortality.

9 Conclusion

This study investigates 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 decreasing API by 10 points will decrease monthly cardiovascular mortality by 1.61 per 100,000 people. We also estimate that decreasing PM_{10} concentration 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 being 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 deducted by 10%.

We rule out the possibility that temporary migration or other activities during the Olympic Games might confound our estimates by excluding the 2008 July and August data. We conduct a falsification test and show that air pollution does not affect injury mortality. All these results show that the relationship between air pollution and cardiovascular mortality is very likely to be causal.

In contrast, estimates from cross-sectional and fixed-effects models are not robust. The estimates from these associational models are substantially smaller, indicating they may underestimate the health effect of air pollution. Our findings are in keeping with Schlenker and Walker (2011) study. Schlenker and Walker (2011) also found that estimates from natural-experimental design are an order of magnitude larger than the non-experimental estimates.

Some researchers may concern about the "harvesting" issue. "Harvesting" is also called mortality replacement, which refers to the advancement of death by a few days or weeks for severely ill individuals. If elevated air pollution hastens the death of people who are already dying, life expectancy saved from slightly better air pollution will be rather small. However, it should not be a big issue in this study because our treatment period is fairly long (roughly one year).

This study has several limitations. First, we do not have the exact concentrations of different pollutants. The overall air quality is measured by API, which is based on a calculation of concentrations of different air pollutants. Different air pollutants decreased

dis-proportionally in the regulated period. [12] So it is not clear which air pollutants contribute most to cardiovascular diseases. Previous literature mostly suggests that $PM_{2.5}$ and Ozone are risky factors of cardiovascular diseases. However, due to data limitation, we are not able to investigate the channels. We recover the concentrations of PM_{10} based on API and estimate the effect of PM_{10} on cardiovascular mortality. However, how the inaccuracy would affect the results are unknown, so 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 can be highly non-linear and negligible under certain threshold. The findings cannot be generalized to less polluted areas, such as the rural areas in China.

Third, we ignore people's response to changes in air quality and the Olympics itself. An individual's level of pollution exposure is determined by ambient air quality, indoor air quality and the time split between indoor and outdoor activities. People may adjust their behaviors in response to changes in air pollution. Those who are at risk of being negatively affected by pollution usually have stronger incentive to adopt compensatory/avoidance behaviors. For example, Neidell (2009) found that people responded to information about air quality, and that smog alerts significantly reduced the

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[12] For example, Rich et al. (2012) monitored daily air quality from July 20 to September 17 in 2008 in Beijing, and observed differentiated reductions in the mean concentration of different air pollutants: 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) from the pre-Olympic to the during-Olympic period. In contrast, ozone concentrations increased (24 percent). They also found that pollutant concentrations generally increased substantially from the during- to post-Olympic period for all the pollutants (21 percent to 197 percent) except ozone (−61 percent) and sulfate (−47 percent).

attendance at major outdoor facilities in Los Angeles. The Olympic Games might have changed people's preferences between indoor and outdoor activities. The consequences of this behavior change 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

VARIABLES	1st Stage, API			2nd Stage, Mortality		
	(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

Standard Errors clustered at the city level in parentheses
*** p<0.01, ** p<0.05, * p<0.1

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

VARIABLES	1st Stage, PM 10			2nd Stage, Mortality		
	(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

Standard Errors clustered at the city level in parentheses
*** p<0.01, ** p<0.05, * p<0.1

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

Standard Errors clustered at the city level in parentheses
*** p<0.01, ** p<0.05, * p<0.1

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

Standard Errors clustered at the city level in parentheses
*** $p<0.01$, ** $p<0.05$, * $p<0.1$

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* (0.0868)	0.137* (0.0752)	0.138* (0.0769)			
PM10				0.0777* (0.0425)	0.0681* (0.0374)	0.0686* (0.0383)
Temperature		0.00376 (0.0702)	0.00928 (0.0700)		0.00377 (0.0661)	0.00891 (0.0664)
Temperature Sq.		-0.00559* (0.00297)	-0.00561* (0.00298)		-0.00516* (0.00272)	-0.00519* (0.00273)
Precipitation			-0.0801 (0.375)			-0.0688 (0.355)
Precipitation Sq.			5.18e-06 (6.70e-06)			4.64e-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

Standard Errors clustered at the city level in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 7. OLS Regressions: Cardiovascular Mortality and API

VARIABLES	(1) 2006	(2) 2006	(3) 2006	(4) 2007	(5) 2007	(6) 2007	(7) 2008	(8) 2008	(9) 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

Robust standard errors in parentheses, *** p<0.01, ** p<0.05

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

VARIABLES	(1) 2009	(2) 2009	(3) 2009	(4) 2010	(5) 2010	(6) 2010	(7) 2006-10	(8) 2006-10	(9) 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

Robust standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1

Table 8. Fixed Effects Models: Cardiovascular Mortality and API

VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
API	0.0263***	-0.000387	-0.000486	0.0065	0.00822*	0.00731
	(0.00381)	(0.00333)	(0.00349)	(0.0045)	(0.00450)	(0.0045)
Temperature		-0.0779***	-0.0762***		-0.0765*	-0.0803*
		(0.0133)	(0.0130)		(0.0388)	(0.0397)
Temperature Sq.		-6.99e-05	-9.74e-05		-0.00227***	-0.0025***
		(0.000413)	(0.000427)		(0.00081)	(0.00081)
Precipitation			-0.119			-0.579**
			(0.220)			(0.229)
Precipitation Sq.			4.27e-06			1.13e-05**
			(5.50e-06)			(4.49e-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	0.647	0.654	0.656

Standard Errors clustered at the city level in parentheses, *** p<0.01, ** p<0.05, * p<0.1
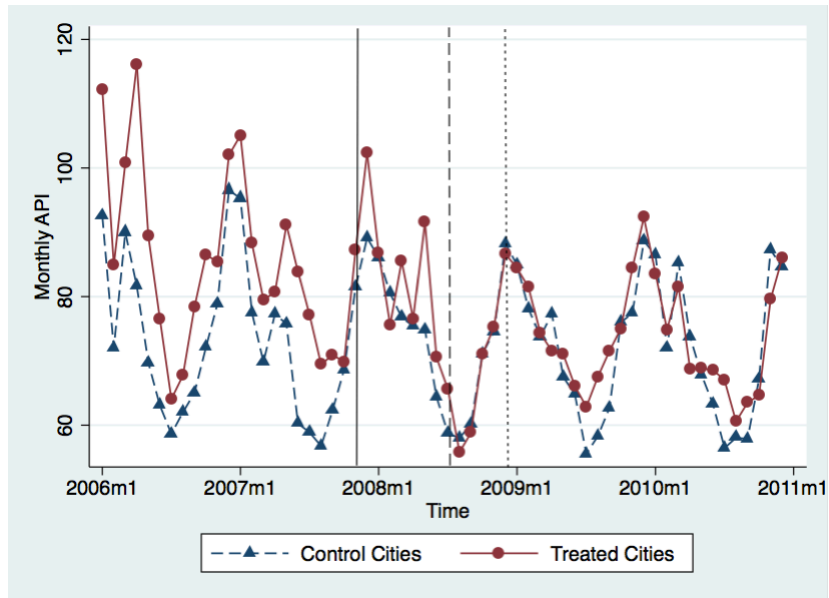
Figure 1 Air Pollution Index in the Treated and Control Group

Note: 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 5 atmospheric pollutants, namely sulfur dioxide (SO_2), nitrogen dioxide (NO_2), suspended particulates of 10 micrometers or less (PM_{10}), carbon monoxide (CO), and ozone (O_3) measured at the monitoring stations throughout each city. API is calculated according to the maximum concentration of these pollutants. It is a proxy measure of the ambient air quality. 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}	CO	O_3
50	0.05	0.08	0.05	5	0.12
100	0.15	0.12	0.15	10	0.2
200	0.8	0.28	0.35	60	0.4
300	1.6	0.565	0.42	90	0.8
400	2.1	0.75	0.5	120	1
500	2.62	0.94	0.6	150	1.2

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 5 pollutant are: $C_{SO_2} = 0.07mg/m^3$, $C_{NO_2} = 0.10mg/m^3$, $C_{PM_{10}} = 0.30mg/m^3$, $C_{CO} = 8mg/m^3$ and $C_{O_3} = 0.18mg/m^3$; then use Table 1 we find that the concentration of SO_2, NO_2, CO, and O_3 is 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:

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

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

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

$$PI_{CO} = \frac{100 - 50}{10 - 5} * (8 - 5) + 50 = 80$$

$$PI_{O_3} = \frac{100 - 50}{0.2 - 0.12} * (0.18 - 0.12) + 50 = 87.5$$

Then $API = \max\{PI_{SO_2}, PI_{NO_2}, PI_{PM_{10}}, PI_{CO}, PI_{O_3}\} = 175$, , 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."