Essays on the Health Effects of Pollution in China

By

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A dissertation submitted in partial satisfaction of the

requirements for the degree of

Doctor of Philosophy

in

Agricultural and Resource Economics

in the

Graduate Division

of the

University of California, Berkeley

Committee in charge:

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Fall 2013

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Abstract

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This dissertation consists of three chapters that analyze the health effects of pollution in China. The first chapter investigates the effect of air pollution on cardiovascular mortality in the urban areas of China. The second chapter estimates the effect of water pollution on infant mortality. The third chapter studies the relationship between water pollution and cancer among the elderly.

The first chapter entitled "The Effect of Air Pollution on Cardiovascular Mortality: Evidences from the Beijing Olympic Games". I explore the exogenous air pollution variations induced by the 2008 Olympic Games to estimate the effects of air pollution on cardiovascular mortality in China. I use the regulation status during the Olympic Games as an instrument for air pollution. In the fixed-effects instrumental variable model, I find that air pollution has a robust and significant effect on cardiovascular mortality. In contrast, estimates from the conventional associational models are not robust. I estimate that decreasing current PM_{10} concentration by 10% will save more than 67,000 lives (from cardiovascular diseases) in the urban areas in China each year.

China's surface water system has been severely polluted in the process of rapid industrialization. The second chapter investigates how this water pollution affects infant mortality. I find that surface water pollution has a significant, nonlinear effect on infant mortality. As surface water quality deteriorates, infant mortality first increases and then decreases. Moderate levels of pollution are the most dangerous. People's avoidance behavior may explain the results: as water becomes more polluted people reduce the consumption of surface water. The ordered-probit selection model is applied to estimate the effects, and precipitation and wastewater dumping are used as the instruments for surface water quality.

China also witnessed a dramatic increase in cancer rate in the past thirty years. In the third chapter, I investigate whether this high cancer rate is caused by water pollution. The difficulty in estimating the long-run health effects of pollution is that the lifetime

exposure to pollution is hard to measure. However, China provides an ideal setting to estimate the long-run health effects of pollution because the Household Registration System (Hukou) effectively stopped people from migrating for many years. I focus on the elderly people (Age>60) because their mobility is extremely restricted by the System, so their life-time exposure to water pollution is more likely captured by the water quality data in recent years. I find that water pollution has large, significant, positive effects on all cancer mortality rate, digestive cancer mortality rate, urinary cancer mortality rate, liver and stomach cancer mortality rate. I also find that water pollution has no impact on cancer mortality rates for the younger adults (Age from 20-50), which may partially justify our argument that pollution exposure for the younger people cannot be accurately measured because they migrate.

For my beloved parents
Guangyou He and Zhumei Wang

献给我深爱的
父亲何光友，母亲王竹美

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Acknowledgements

First and foremost, I owe an enormous debt of gratitude to my advisor Professor Jeffrey Perloff for his tireless support throughout my graduate education in Berkeley. I couldn't image a more supportive source of guidance in the past five years and believe he is the best mentor a research student can possibly hope for. He has deep insights on a variety of issues and is willing to support any research topic I want to explore. He is understanding, accommodating and possesses a witty sense of humor. I feel blessed and very proud to be one of Jeff's students. Working with Jeff has been enlightening as well as exciting and I look forward to many more years of fruitful collaboration.

I am also very grateful to Michael Anderson. Michael's challenging questions always make me think, write, learn and push myself a little harder. He introduced me to the field of applied econometrics, and I was fascinated by the "magical" ways to identify causal effects in social sciences. I am very fortunate to have Michael in my dissertation committee and as the oral exam chair.

I'd like to extend my special thanks to Maoyong Fan. He graduated from ARE a few years ago and now is an assistant professor in Ball State University. We collaborate on a variety of research projects and I have learnt a lot from him.

Special thanks are also due to William Dow, Peter Berck, Meredith Fowlie for their help in the process of developing research ideas, writing and revising the papers. This dissertation certainly would not have been completed without their help.

I also thank other faculty members in ARE. I thank Elisabeth Sadoulet, Meredith Fowlie, David Roland-Holst, Maximilian Auffhammer, Jeremy Magruder, Sofia Berto Villas-Boas for their valuable suggestions on different stages of my research.

I could not have asked for a better set of classmates. You guys made my Berkeley years so much more enjoyable: Lydia Ashton, Maria Bowman, Siwei Cao, Andrew Dustan, Kate Foreman, Kiran Torani, Diana Lee, Stanislao Maldonado, Ayako Matsuda, Tiffany Shih.

I also appreciate the excellent research assistance from Jennifer Kao, Yan Panassie, Lin Wang, Yingyue Hu, and Qilun Zhou. I thank the Pamela and Kenneth Fong Fellowship and IBER grant for providing financial support. I thank Maigeng Zhou in China CDC for providing research data.

Without the help of administrative staff, the academic life can be a mess. In particular, I would like to thank Gail Vawter and Diana Lazo for their warm and kind assistance over the past five years.

My friends have made my journey much less stressful. I enjoyed every moment with you: Di Zeng, Bohan Ye, Zhen Sun, Liangpeng Guo, Ying Zheng, Haoyuan Li, Peijia Lin, Shipeng Guo, Yi Rao, Wei Ye, Liangfeng Wang, Libei Tian, the Chinese graduate students in ARE and Econ, and the young undergraduates in Tennis at Cal. Special thanks to Swupnil Sahai for playing tennis with me over the past four years.

The constant support of my family was invaluable. They encourage me, believe in me, and always support me with their whole hearts. I love you. There is an individual who has witnessed all my progress in recent years that I'd like to keep hidden from everyone else that I've named so far. I wish you the best of luck and a happy life.

Pursuing an academic life is not easy, especially for a student coming from a less-developed region of China. I was the only student admitted by Qujing No. 1 Middle School from my middle-school class, three years later I was the only student admitted by Peking University from my high school class, four years later I was the only student admitted by UC Berkeley from my college class. Now I have earned a Ph.D. and will head to Harvard University to continue my post-doctoral research life. I feel I am very blessed and fortunate all the way along. I'd like to thank all the people who helped or intended to help me in the past years.

Despite my efforts above, it is impossible to repay the debt that I owe in a few paragraphs. I hope to accomplish that in the future.

Chapter 1

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

1.1 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 include 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.

In this study, I use a fixed-effects instrumental variable model 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. In the first stage, I estimate how the regulation affects air quality after controlling for a set of local characteristics. Then in the second stage, I estimate how air quality influences cardiovascular mortality in urban China.

This research has several attractive features. First, the Olympic-induced regulatory pressure is very likely orthogonal to city-district level changes in health outcomes, except through its impact on air pollution. Thus the relationship estimated using the instrumental variable approach has a causal interpretation. Second, this study uses less aggregated (monthly) data in many city-districts (34 cities) for a relative long period (5 years). It allows me to check the robustness of different models. Third, 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.

Section 1.2 discusses the literature on the health effects of air pollution. Section 1.3 summarizes the government's interventions on air pollution during 2008 Beijing Olympic Games. Section 1.4 discusses the research design and estimation strategy. Section 1.5 describes the datasets and provides summary statistics. Section 1.6 presents the main results. Section 1.7 checks the robustness, Section 1.8 compares our results with previous non-experimental studies, and Section 1.9 concludes.

1.2 Health Effects of Air Pollution

An association between high levels of air pollution and human illnesses has been known for more than half a century. For example, during the London fog incident of 1952, extreme elevations of air pollution were found to be associated with markedly increased mortality rates. 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 be roughly 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, whether the association in time-series studies indicates causality is controversial. 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 not clear whether the worse health outcomes are actually caused by elevated air pollution or other risk factors. For example, Beijing's thick smoggy days in 2013, which were intensively documented by the mass media, largely resulted from the impacts of a combination of temperature, humidity and wind. Most of these smoggy days happened on less windy days during the winter. If people are more likely to die on the cold or windy days, without controlling for temperature and other risk factors, the association between air pollution and increased mortality can be misleading. Moreover, there is no an adequate control group, it is difficult to rule out alternative explanations in virtually all time-series studies.

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 with omitted variables bias. Since 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 usually infeasible to control for all potentially confounding factors. As a consequence, the estimates of the health effect of pollution may be biased.

Cohort-based longitudinal studies (Dockery et al, 1993) may suffer from similar problems as 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.

Many recent studies have utilized fixed-effects models. Fixed-effects models (Currie and Neidell, 2005; Currie et al., 2009) remove permanent sources of bias and are particularly useful when time-invariant factors explain much variation 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, since changes in air quality depend on similar factors as health outcomes do (such as temperature, humidity, and factory polluting activities), this assumption may not hold. Moreover, fixed effects model tends to exacerbate measurement error bias (Griliches and Hausman, 1986).

In contrast, natural or quasi- experiments provide more convincing identification strategies. Among them, most existing studies focus on infant or child health because they are the most vulnerable to air-borne diseases and the effects are immediate. For example, Chay and Greenstone (2003a) 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 (2003b) 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.

Even though almost all the associational studies suggested that air pollution is positively and significantly associated with adult mortality rates, Chay et al. (2003) challenged these results. Again, they used the Clean Air Act as an instrumental variable for air quality, and found that even though the regulatory status was associated with large reduction in total suspended particulates but 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 on diseases. Since some diseases are sensitive to air pollution while others are not, it is very likely that air pollution only affects some specific disease mortality. As for 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.

This paper relates air pollution to cardiovascular mortality in China. 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 diseases.[1] For example, Peters et al. (2000) found that air pollution is associated with cardiac arrhythmia. Dockery et al. (2005) found that air pollution is associated with

[1] For recent reviews, see Brook et al. 2004; Raun and Ensor, 2012.

increased incidence of ventricular tachyarrhythmia. Forastiere et al. (2005) found that air pollution was associated with out-of-hospital coronary deaths. Xu et al. (2008) found that bad air quality was associated with more cardiorespiratory hospitalizations. De Hartog et al. (2009) found associations between $PM_{2.5}$ and heart rate variability. For China, Guo et al. (2010) found that air pollution was associated with cardiovascular mortality in Tianjin. Rich et al. (2012) found association between air pollution levels during the Beijing Olympics and biomarkers of inflammation and thrombosis in healthy young adults.

Many studies found that air pollution is statistically significantly associated with cardiac arrests, which almost always lead to deaths without prompt intervention. Silverman et al. (2010) conducted a case-cross-over analysis of air pollution and out-of-hospital cardiac arrests (OHCA) and found that $PM_{2.5}$ was associated with an increase in OHCA. Dennekamp et al. (2010) found similar association between $PM_{2.5}$ and OHCA. Ensor et al. (2013) found a significant association between OHCA and ozone. Since many cardiac arrests happened to the people who have no pre-existing cardiac conditions, if air pollution actually caused them to die, the loss of life expectancy could be very large.

1.3 Air Pollution Regulations during the Olympics

Beijing started to regulate its air pollution in the 1990s. In 1998, Beijing initiated its first Environmental Cleaning Plan. Four years later, by the end of 2002, the government reported that it had converted approximately 1,500 coal furnaces into clean fuel, retired more than 23,000 old automobiles, decreased the industrial emission by 30,000 tons and increased the green area by 100 square kilometers (Chen, et al., 2011).

In the following years, Beijing continued its efforts to improve air quality, especially after being selected to host the 2008 Olympic Games. On July 13, 2003, the International Olympic Committee granted Beijing the privilege of hosting the 2008 Games. The Chinese government immediately established the Beijing Organizing Committee for the Games of the XXIX Olympiad (BOCOG) and "Clean the Air" became a major task. From 2003 to 2004, it was reported that industrial coal use decreased by 10 million tons; in 2005 and 2006, desulfurization, dust removal and denitrification facilities were installed in two of the largest power plants in Beijing: the Beijing Thermal Power Plant and Power Plant of the Capital Steel.

In order to guarantee good air quality in 2008, radical interventions were implemented 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". The Measures provided guidelines to regulate 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 Emission Control period.

During the pre-Olympic Comprehensive Regulation period, multiple regulations were implemented simultaneously. For example, all the coal-fired power plants in Beijing installed desulfurization, dust removal and denitrification facilities. Even though these plants had already met the national emission standards, they were required to reduce their emissions by 30 percent from their previous emission levels. More than 150 polluting

factories had to restrict their production substantially to meet the new emission standards. The Second Beijing Chemical Plant, Beijing Eastern Petrochemical Company, 27 Cement Production factories, more than 140 Concrete Mixing Plants, and more than 100 lime production factories were completely shut down. In late 2007, the largest plant relocation action took place in the Capital Steel Company. Its production of steel decreased to 200,000 tons per month, which was less than 1/3 of its normal production level.

Besides, the public sector (public transit, environment and health agencies, etc.) replaced all the heavy-emission vehicles. In November 2007 and June 2008, the government increased gas prices to discourage the use of private vehicles. Motor vehicle exhaust emissions are the major air pollution sources in big cities. To ensure good air quality, Beijing Municipal Government implemented temporary traffic control before and during the Games. From July 1 to September 20, vehicles with yellow environmental label (vehicles that failed to meet the European No. I standards for exhaust emissions) or test license plates 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, motorcycles) were no longer allowed on the roads.

From July 20 July to September 20, vehicles with plates ending with odd numbers were only allowed on the road on odd dates and those with plates ending with even numbers were only allowed on even dates. Only a few exceptions, such as police vehicles, public transports, vehicles with Olympic passes, etc. 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 Ministry of Environment Protection in China (2008), the total vehicle exhaust emissions reduced by more than 60 percent. Traffic controls significantly decreased the concentration of fine particulate, ozone, nitrogen oxide and other pollutants generated by auto vehicles in Beijing. To further reduce the particulate matter pollution, the Chinese government also required that all construction projects were halted during the Games.

Since air quality in Beijing was affected by its neighbors, several cities and provinces (Tianjin, Hebei, Liaoning, Neimeng, 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 Jun 2008. Some soccer games were held in Tianjin, Shenyang and Qinhuangdao, similar air pollution controls were implemented in those 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 before the Olympics and increased greenbelt in the construction facilities. Qinhuangdao built shelter forests along the piers to reduce dust, swept streets with water and dumping garbage everyday.

The combination of these regulations effectively improved air quality in and around Beijing. For example, during the 17 days of the Olympic Games, all the indicators of air quality in Beijing met the national standards.

Figure 1.1 shows the monthly average pollution index (API) in Beijing from 2006 to 2010. A higher API indicates a worse air quality. The yearly average pollution index (API) in Beijing decreased from 101 to 87, which approximately corresponds to a fall in the concentration of particulate matters (PM_{10}) from 152 $\mu g/m^3$ to 124 $\mu g/m^3$ (an 18 percent decrease). The air quality improvement is particularly striking during the summer period (June-August). In 2007 the average summer API in Beijing was 98; one year later in 2008, average summer API decreased to 75. The corresponding PM_{10} concentration decreased from 145 $\mu g/m^3$ to 101 $\mu g/m^3$ (a 30 percent decrease) during the same period.

1.4 Research Design and Model

The major concern in estimating the health effects of air pollution is that air pollution may be often correlated with a number of omitted variables in the cross-sectional model and even in some panel (fixed-effects) models.

Many studies relied on estimates from cross-sectional models. In our setting, a cross-sectional model can be written as:

$$Y_{it} = \delta_0 P_{it} + X'_{it}\eta_0 + u_{it}, u_{it} = u_i + \varepsilon_{it} \quad (1)$$

where Y_{it} is a health outcome in city i at time t, P_{it} is the air pollution levels and X_{it} is a set of control variables. u_{it} are unobservable disturbances.

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.

In a fixed-effects model, we impose time independent effects for each city that are possibly correlated with air pollution. Then Equation (1) becomes:

$$Y_{it} - \bar{Y}_i = \delta_0(P_{it} - \bar{P}_i) + (X'_{it} - \bar{X}'_i)\eta_0 + \varepsilon_{it} \quad (2)$$

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

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, water pollution and hazardous waste) that also affect health outcomes, the fixed-effects estimates will be biased, too. Moreover, since our measure of city-district level monthly air quality is averaged across different monitoring sites and aggregated from daily data, we may suffer from slight measurement error bias. This bias will be exacerbated in fixed effects models.

The biases in the cross-sectional model and fixed-effects model can be alleviated if there exists an instrumental variable (IV) that is correlated with changes in air quality but have no direct impact on the health outcome. Such a variable would purge the biases caused by both measurement error and omitted variables.

Our instrument is a city's air quality regulatory status during the Olympics. In particular, we estimate the follow two equations:

$$Y_{it} = \delta_0 \hat{P}_{it} + X'_{it}\eta_0 + u_i + v_t + \varepsilon_{it} \quad (3)$$

$$P_{it} = \lambda_1 O_{it} + \lambda_2 T_{it} + X'_{it}\eta_0 + u_i + v_t + \xi_{it} \quad (4)$$

where O_{it} is a regulation status indicator of city *i* at time *t*. 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.

We focus on a 5-year window, from 2006 January to 2010 December. As discussed in the previous section, radical air pollution controls were implemented from November 2007, so we treat this month as the starting point of the regulation.

Even though most of the air pollution regulations were abandoned after the Olympic Games, the good air quality could last for a few more months longer, so we also include the last 3 months (October, November, and December) in 2008 as the regulation period. 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 air quality regulations than other cities. 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.[2]

"Being-regulated" is associated with a sharp decrease in average pollution index (API) in 2008. Figure 1.2 presents the trends of the monthly average pollution index (API) for both the regulated (9 city-districts) and non-regulated cities (25 city-districts) in our sample during 2006-2010. We observe strong seasonality in the trends of air quality for both treated 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.

In the first stage, we estimate how the air quality regulations affect air pollution, using Equation (4). The coefficient λ_1 is 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 (3). 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.

[2] 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.

1.5 Data and Descriptive Statistics

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

1.5.1 Air Quality Data

Air Quality data comes 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. Our air quality data has the following information: daily air pollution index (API), primary pollutant and air quality level.

Unfortunately we do not have information on the concentrations of various air pollutants. However, the way that SEPA calculates API allows us to approximately recover the concentration of the primary pollutant.

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 1.1 shows the relationship between API and the concentration of the five air pollutants.

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 1.1. 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 1.1 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."

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}.

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. Largely because of the complex nature of PM, it has been measured and regulated based primarily on mass within defined size ranges.

We aggregate daily API to monthly and match it with the death data. In our sample, the average monthly API is 74.45, with a standard deviation of 20.58. The average API for regulated cities is 78.98, with a standard deviation of 19.81. In contrast, the average API in the control cities is lower, 72.81, with a standard deviation of 20.61

The recovered concentration of PM_{10} for the regulated, control, and the whole sample is 108.10, 96.07, and 99.25, respectively. The corresponding standard deviations are 39.39, 39.28 and 39.66.

The reliability of the Chinese official air quality data has been questioned by both mass media and researchers, and the government's unwillingness to publicize specific concentrations of different pollutants further imposes difficulties for the researchers to verify its reliability. Wang et al. (2009) compared measures of pollutant concentrations collected by themselves with SEPA's, and found that there is very strong association between their own air quality data with the official data, but the absolute value of the official data is about 30 percent lower. So it is likely that Chinese government manipulated the data to some extent. However, the evidences in Wang et al. (2009) and similar studies also have their own drawbacks: the sampling methods and the choice of monitoring locations are not identical to SEPA's, so the discrepancy does not necessarily indicate a true data manipulation. Moreover, in our quasi-experimental approach, the estimations will be based on differences instead of levels. So even if the Chinese government underrates the pollution level, as long as the bias is proportional, our estimates shouldn't be affected.

Chen et al. (2011) evaluated the impact of the Olympics on Beijing's air quality. They found that the regulations effectively reduced API in Beijing by 29.65 percent during the Games as compared to one year before any Olympic-motivated action. They also used the satellite based AOD data, acquired from NASA, confirms that air quality improvement in Beijing during 2008 was real.

1.5.2 Death Data

Death data comes 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.

Our primary dependent variable is the 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.

We aggregated daily air quality data to monthly level and matched it with the death records data. Thirty-four city-districts in urban cities of China were matched using the two datasets, sampled from January 2006 to December 2010.

The 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.

Our study focuses on the cardiovascular mortality. Cardiovascular diseases are the No. 3 cause of death in China. In our sample, 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 in our sample, with a standard deviation of 3.37.

1.5.3 Weather and social-economic 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. More importantly, rainfall changes the concentration of water pollutants, which may contribute to worse health outcomes. The relationship between temperature and air pollution may be non-monotone. Air pollution may increase in both extreme hot and cold days due of excessive energy consumption. People are more likely to die on extreme hot or cold days. The summary statistics of the key variables are in Table 1.2.

1.6 Main Results

1.6.1 Results from Cross Sectional Models

We first estimate the relationship between API and cardiovascular monthly mortality rate using cross-sectional models. We run cross-sectional 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 1.3. Without any control variables, API is positively and statistically significantly associated with higher cardiovascular mortality rates in each year. However, they become insignificant as we include the control variables in most years. The estimated coefficients are 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 we face omitted variables problems: as we control for more confounding factors, the estimates of API are affected so API is very unlikely to be exogenous.

1.6.2 Results from Fixed Effects Models

In Table 1.4, we summarize 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 (59 dummies for 60 months).

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 0.0115 but is still statistically significant.

However, including the weather variables has a very large impact on the estimates of API. Both in the city fixed effects model and the city and time fixed effects model, the significant relationship between API and cardiovascular diseases disappears after we control for weather conditions. These results suggest that changes in air pollution fluctuations are still correlated with the weather conditions and that weather conditions may affect cardiovascular mortality directly or through channels other than affecting air pollution. Temperature plays a very important role in determining mortality in the fixed effects model. More people died on the 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. The results are very sensitive to including weather conditions. There may exist other unobserved variables (such as winds) that co-vary with both API and cardiovascular mortality. The estimates from fixed-effects models are very likely to be biased as well.

1.6.3 Results from Fixed-Effects Instrumental Variable Model

We now turn to the natural experiment research design. We estimate the relationship between air pollution and cardiovascular mortality using a fixed-effects instrumental variable model.

In the first stage, we estimate the relationship between the regulatory status and API using Equation (4). Then we take the predicted air pollution index into Equation (3) and estimate the relationship between cardiovascular mortality and API. In both stages, we control for both city-district fixed effects and fixed effects for each month. We report the regression results in Table 1.5.

The regulatory status variable is a valid instrument if (1) the regulatory status affects air pollution levels, and (2) these regulations affect cardiovascular mortality only through its impacts on air pollution.

The first condition can be justified by looking at the relationship between the instrumental variables and API. In columns 1-3, we report the effect of the two treatments (general air regulation O_{it} and traffic control T_{it}) on API. Both instruments are statistically significantly associated with API at 5% level. On average O_{it} decreases monthly API by about 3.2 ug/m^3, and T_{it} decreases monthly API by 8.56 ug/m^3, conditional on city-districts fixed effect, month fixed effects, temperature, precipitation and their squares.

Even though we cannot directly test the second condition, the regression results suggest both instruments are likely to be exogenous. 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 exogenous. At the same time, the estimated effect of API on cardiovascular mortality is statistically significant in all three specifications. Without including any weather or social-economic controls, the estimated coefficient of API on cardiovascular mortality is 0.192, as reported in column 4 of Table 1.5. Including the weather controls slightly decreases the

estimates of API, and the change of magnitude is small. In other words, these control variables are not correlated with API variations induced by the regulation. If the API variations induced by the regulation are not correlated with these observable potential confounding factors, it is also likely that they are not correlated with other potential unobserved confounding factors.

In the most restrictive specification, where we include city fixed effects, month fixed effects and weather conditions, the effect of API on cardiovascular mortality is 0.161 and the relationship is statistically significant at 5 percent level. The 95 percent confidence interval is [0.015, 0.307]. If we increase API by 10 point, monthly cardiovascular mortality would increase by 1.61 per 100,000 people. The estimates from this natural experiment design are substantially larger than the estimates from cross-sectional and fixed-effects models.

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.

1.6.4 Results for PM10

Since API is an index, it is very hard to compare our estimates with findings in other studies. We thus recover the PM_{10} concentrations based on API. PM_{10} is the primary pollutant for most of the time (roughly 90 percent), so the recovered concentration should be a good proxy for the actual PM_{10} concentrations.

Though not reported, we find that estimated coefficient of PM_{10} is sensitive to the inclusion of weather conditions in both cross-sectional and fixed effects models.[3] However, in the instrumental variable setting, the relationship is robust. The effects of PM_{10} on cardiovascular mortality are reported in Table 1.6.

The estimated coefficient of PM_{10} on cardiovascular mortality is 0.081 in the most restrictive model and is statistically significant. The 95 percent confidence interval is [0.008, 0.154]. Given that the average monthly PM_{10} concentration is 99.25 $\mu g/m^3$, if PM_{10} concentration decreases by 10 percent (approximately 10 $\mu g/m^3$), monthly cardiovascular morality would roughly decrease by 1 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 our estimation, more than 67,000 lives in urban China may be saved from cardiovascular death if PM_{10} concentration decreases by 10 $\mu g/m^3$ from its current level.

1.6.5 Falsification Test Using Deaths by Injuries

Last, 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 death patterns, similar patterns should occur on other mortalities as well.

In Table 1.7, we report the regression results for monthly injury mortality. We summarize the results for API in columns 1-3 and the results for PM_{10} in columns 4-6.

[3] The results are not reported and are available upon requests.

We find that the estimates 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. In other words, air pollution does not affect injury mortality. In case the Olympic Games somehow affected injury mortality (for example, traffic controls might reduce car accident deaths) during the Olympic Games, we check the same set of specifications excluding the traffic control months and the findings are the same.

1.7 Robustness Checks

1.7.1 Migration and Other Potential Threats

One 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 1.1 and 1.2, the largest air quality improvement occurred in July, and 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 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 1.8. 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 1.5 and Table 1.6. 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.

1.7.2 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 1.9 we report the regression results. Once again, the relationship between cardiovascular mortality and air pollution is robust and is statistically significant at 10 percent level.

This set of specifications is more restrictive. However, including city-specific trends would unfavorably absorb too much variation in air pollution, some of which were caused by the air pollution regulations. Consequently, including city-specific decreased the point estimates of API and PM_{10} by about 0.2.

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 that are not apparently endogenous.[4] Our findings are the same and the results are robust.

1.8 Comparison with Previous Estimates

The relationship observed in this study between air pollution and cardiovascular mortality is consistent with findings in many epidemiological time-series and cross sectional studies. We compare our results with previous 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.[5] Since our study evaluated relatively long-term exposure, quantitative comparisons with daily time-series studies are difficult to make. [6] Instead, we compare our results with several noteworthy long-term cohort studies here.

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.

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[4] The results are essentially unchanged when we include per capita GDP, per capita fixed-asset investment, the share of agricultural production, and the share of industrial production. However, due to concerns of endogeneity of these yearly variables, we decide not to include them in the regressions.

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

[6] 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 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.

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.

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.

1.9 Conclusion

We investigate the potential causal relationship between air pollution and cardiovascular mortality in China. We use the 2008 Beijing Olympic Games as a natural experiment, and estimate the effect of air pollution on cardiovascular mortality.

We use death data from China CDC and air quality data from SEPA. We collect social economic data and weather data from various sources. We matched these datasets and investigate how air pollution is associated with cardiovascular mortality in 34 urban cities in China. We show that results from the traditional cross-sectional models and fixed effects models are not robust, suggesting that there exist omitted variable problems in those approaches.

Instead, we use a fixed-effects instrumental variable model. We split the matched 34 city-districts into two groups based on their regulatory status during the Olympic Games. In the first stage, we estimate how the regulations affect air quality using a difference in difference model (control for both city-district fixed effects and fixed effects for each month). In the second stage we estimate the relationship between cardiovascular mortality and the predicted air pollution levels.

We find that air pollution has a robust and significant effect on cardiovascular mortality. Increasing API by 10 points is associated with 1.61 increase in monthly cardiovascular mortality. We also estimate the effect of PM_{10} on cardiovascular mortality. Our measure of PM_{10} is an approximate measure of the actual PM_{10} concentration. If PM_{10} mean concentration decreases by 10 $\mu g/m^3$, more than 67,000 lives will be saved in the urban areas of China each year.

Our results are not sensitive to including weather and social-economic control variables, suggesting that the instrumental variable is not correlated with the observed factors. We rule out the possibility that temporary migration or other activities during the Olympic Games might confound our estimates by examining the data without 2008 July and August. 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.

Our estimate of the effect of air pollution on cardiovascular mortality is larger than the conventional estimates in previous studies, indicating the associational studies underestimate the health effect of air pollution. The findings are in keeping with Schlenker and Walker (2011) study. Schlenker and Walker (2011) found that estimates from natural-experimental design are much larger than the conventional estimates.

In the literature, some researchers concerns about "harvesting". "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. Our measurement of air quality, API, is based on a calculation of different pollutant concentrations. We estimate the effect of PM_{10} on cardiovascular mortality using the recovered approximate concentration. However, the magnitude of the inaccuracy is unknown, so the results should be interpreted with caution.

Second, different air pollutants decreased dis-proportionally in the regulated period.[7] 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 how different pollutants affect cardiovascular mortality.

Third, 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. We cannot generalize the results to less polluted areas, and to rural areas in China.

[7] 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).

Fourth, 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 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.

Table 1.1 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.

Table 1.2 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 1.3 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, * p<0.1

Table 1.3 (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 1.4 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

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

Table 1.5 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

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

Table 1.6 Fixed-Effects IV Models: Cardiovascular Mortality and PM10

	1st Stage, PM 10			2nd Stage, Mortality		
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
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)
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)			
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 1.7 Falsification Test: Injury Mortality on API

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 1.8 Robustness Check: 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 1.9 Robustness Check: 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

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

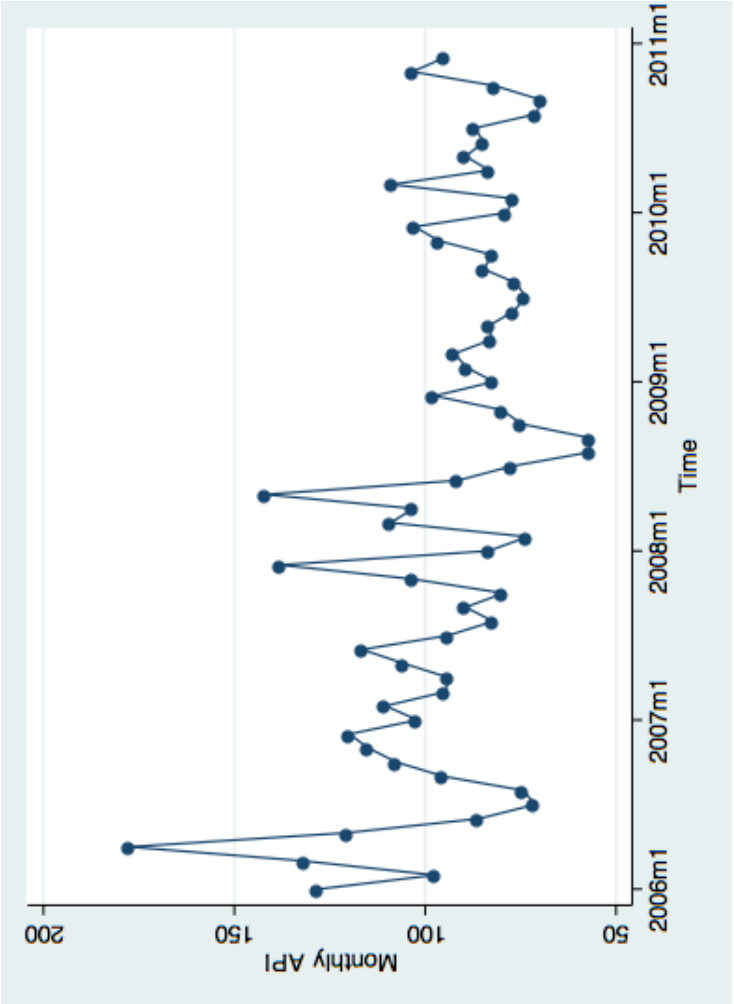

Figure 1.1 Monthly Air Pollution Index in Beijing

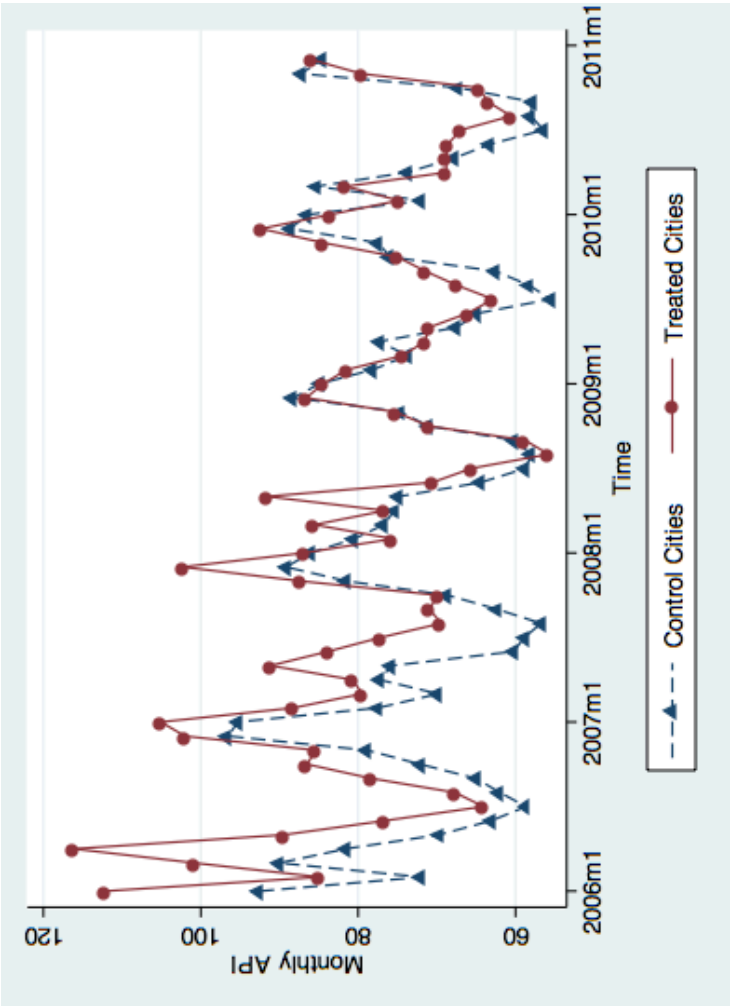

Figure 1.2 Air Pollution Index in the Treated and Control Group

Chapter 2

Death By Ignorance? Surface Water Quality and Infant Mortality in China

2.1 Introduction

China's rapid industrialization, which brought significant economic prosperity over the last thirty years, led to severe water pollution in many areas due to massive industrial wastewater discharges and extensive use of agricultural fertilizer. This water pollution endangers everyone's health, especially infants. Even though about 60 to 70 percent of river water is unsafe for human consumption (World Bank, 2006), many people in poor areas rely on the surface water systems for daily use, including drinking. Using surface water quality data from monitoring sites in 19 provinces and county-level infant mortality data from the 2000 Census, we estimate the effects of surface water quality on infant mortality in China.

We hypothesize that if surface water becomes slightly degraded, people do not notice the pollution and continue consuming it. Consequently, infant mortality initially rises as water pollution increases. However as the pollution gets worse, people begin to notice the pollution using visual and other clues and reduce their consumption of surface water. As water pollution becomes very pronounced, infant mortality falls.

We discuss the literature and background on the effects of surface water quality on health in Section 2.2. In Section 2.3, we describe the dataset, define the key variables and provide summary statistics. In Section 2.4, we estimate the effects of surface water quality on infant mortality using an ordered-probit selection model. The selection rule in based on surface water quality: people choose where they live and hence the local level of water pollution. We use wastewater dumping and precipitation as instruments for surface water quality, and estimate the selection model using a two-step approach. Section 2.5 concludes.

2.2 The Effects of Water Pollution on Health

Studying the effects of water pollution on people's health has been an important research topic in environmental science, epidemiology, environmental economics and health economics for decades if not centuries. The earliest research in this area dates to the 1850s (see Freedman, 1991). Using a natural experiment on water supply distribution in London, John Snow demonstrated that unsanitary water caused cholera outbreaks. He found that the death rates for dirty-water users were over eight times higher than that for the clean-water users.

Many recent studies have found various connections between water pollution and diseases and other public health measures. Some studies focused on water pollution and water-borne diseases, such as typhoid (Cutler and Miller, 2005) and diarrhea (Jalan and Ravalion, 2003). Other studies explored the relationship between water pollution and cancer, such as Cantor (1997), Davis and Masten (2004), Chen et al. (2005), and Ebenstein (2012).

Much of the research focuses on infant and child mortality. Galiani et al. (2005) found that privatizing water service improves water quality, and reduces child mortality. Merrick (1985), Lavy et al. (1996), and Lee et al. (1997) found positive associations between water quality and infant health. Greenstone and Hanna (2011) investigated the effects of air quality and water quality on infant mortality in India, and found no significant relationship between infant mortality and water quality. Brainerd and Menon (2012) found a negative effect of fertilizer agrichemicals in water on infant and child health in India. Currie et al. (2013) found that contaminated drinking water has large and statistically significant effects on birth weight and gestation of infants born to less educated mothers.

This study investigates the relationship between surface water quality and infant mortality in China. The surface water system broadly covers rivers, lakes and reservoirs. China's surface water system has been severely polluted in the process of industrialization. However, none of the existing studies has explored how this pollution affects infant's health. We argue that the relationship between surface water pollution and infant health status can be non-monotonic. In the face of water pollution, people's avoidance behaviors may greatly mitigate the pollution's negative impacts on human health. As surface water becomes very polluted, people stop using it and search for alternatives, such as bottled water. The avoidance behaviors have been empirically supported by a few previous studies. For example, Neidell (2009) found that when smog alerts were issued, attendance at major outdoor facilities in Los Angeles would significantly decrease. As for water, Zivin et al. (2011) found that bottled water sales would significantly increase as a result of tap water violations in Northern California and Nevada.

In China, the overall surface water quality is graded based on various chemical pollutant indicators, which include the pH-value and the concentrations (measured by mg/L) of dissolved oxygen, biochemical oxygen demand, ammonia, and nitrogen, etc. The overall surface water quality is graded on a 6-degree scale, where Type I water is the best quality water and Type VI is the worst.

According to the China Ministry of Water Resources, Type I water is an "Excellent" source of potable water. Type II water is a "Good" source of potable water. Type III water is "Fair." Pathogenic bacteria and parasites ova can sometimes be found in Type II and III water so drinking it may cause diseases. Thus, Type II and III water should be purified and treated (such as by boiling) before drinking. Type IV water is polluted and unsafe to drink without advanced treatment, which is only possible at water supply plants. Type V is seriously polluted and can never been used for human consumption. Type VI water is called "Worse than Type V Water," and any direct contact with it is harmful to humans.

Individuals can easily distinguish clean water from heavily polluted water, such as Types V and VI. Very polluted water is murky and smelly and sometimes has algal blooms on the surface. However, distinguishing "excellent" water from "good" or "fair" water can be challenging. Figure 2.1 gives a visual illustration of different types of water. People may not be aware of the potential health risks as water quality falls from "excellent" to "good" or "fair", so it is not surprising that people consume good or fair water and their health status is damaged by the ignorance. As water quality further

deteriorates, people typically avoid consuming polluted water, avoid serious health risks. Thus the relationship surface water quality on infant mortality may be non-monotonic.

Not all the harms from consuming polluted water are immediately apparent. Some effects occur at once, while others appear only after toxins have accumulated in the body. For example, consuming contaminated water can cause malaria outbreaks within days, but it may take decades for water pollution to cause cancers. In this study, we focus on infant mortality, which includes only fatal effects that occur within one year.

Infant mortality rate is the annual number of deaths among infants less than one year old per thousand live births. Neonatal death is the major component of infant deaths. In China, about 60 to 70 percent of infant deaths occurred within the first month after they were born. Most of the deaths during the neonatal period are due to endogenous causes (inherited defects), such as congenital anomalies, gestational immaturity, birth complications, and other physiological problems. Lack of proper care during the pregnancy can cause neonatal deaths. For example, if pregnant women drink polluted water, the neonatal mortality may rise. The post-neonatal mortality rate is the death rate of infants from one month to one year. The vast majority of post-neonatal deaths are from exogenous causes, such as injuries, environmental and nutritional factors, especially as they interact with infectious disease like gastroenteritis and pneumonia. Thus water pollution may cause both neonatal and post-neonatal mortality.

To consistently estimate the effects of water pollution on health, we need to avoid sample selection bias. It would be inhuman to randomly assign one group of people to live in a polluted area while assigning another group to a less polluted area. Even if we could assign people to various areas, we could not stop people from moving. Because poor people are less able to move from the polluted area than are rich people, it is possible that poor people, whose health status is very bad for other reasons, gather in polluted areas, and rich people, whose health status is already good, stay in clean areas. Many previous studies have ignored this potential selection problem, so their estimates may be biased.

Ebenstein (2012) suggests that China provides an ideal context to estimate the health effects of water pollution because of the Household Registration System (Hukou). This system prevents people from moving from rural to urban areas. It also makes it relatively difficult to move within rural and urban areas. A variety of benefits, such as health care and social security, are associated with the Household Registration System. If the system has effectively stopped people from migrating, we may treat people's location and thus water quality as predetermined and estimate the effects of water pollution using ordinary least squares.

However, this argument may have held in the past, the share of people migrating has increased by an order of magnitude over the last two decades. According to the Census, only 0.66 percent of the total population migrated in 1982 compared to 7.9 percent in 2000. Given that many rural migrant workers are of childbearing age, if the migration decision is correlated with water pollution levels, a selection problem may exists.

We formally address the potential sample selection problem using a Heckman-like selection model. In the first step, we estimate the relationship between the endogenous water pollution on the instrumental variables. Conditional on the estimates

from the first step, we estimate the effects of water pollution on infant mortality in the second step.

We use wastewater dumping and precipitation as instruments for surface water quality. Industrial wastewater dumping degrades surface water quality. We use the amount of untreated wastewater discharged into the water system at the prefectural level as an instrument for the water quality at a given county. We treat the prefecture-level wastewater dumping as an exogenous variable at the county level because a county takes the total amount of discharged wastewater as given.[8]

Precipitation is one of the most important factors that influence surface water quality. Where rainfall is heavy, surface water quality is typically good. Precipitation affects surface water quality through two primary channels. First, rain directly dilute the concentration of water pollutants, and thus improves water quality. Second, rain causes the river to flow faster, which carries away water pollutant quickly and makes the river less prone to eutrophication (Zhong et al., 2005).

China has seven major river basins: Changjiang (Yangtze) River Basin, Huang River Basin, Zhujiang River Basin, Huai River Basin, Songhuajiang River Basin, Liao River Basin and Hai River Basin. Surface water quality in the southern river basins is better than that in the northern river basins because southern China has more rain. In our sample, the average yearly precipitation in the northern provinces (Beijing, Tianjin, Hebei, Liaoning and Ningxia) is 458 mm, with a standard deviation of 148 mm. In contrast, the average precipitation in the rest of the provinces is 1,286 mm with a standard deviation of 417 mm.

In Figure 2.2, we show the proportion of the river segments that were seriously polluted (Type IV, V, and VI) from 1991−2005. Roughly 60 to 70 percent of rivers in northern China (Huai River, Hai River, Songhuajiang River, Liao River) were severely polluted. In contrast, in southern river basins, such as the Changjiang River basin and Zhujiang River basin, the proportion of polluted river segments was much lower.

Although precipitation has a large effect on surface water quality, for it to be a valid instrument, it must affect infant mortality only through its impact on surface water quality. Our primary concern is that rainfall fluctuations may affect infant health through other channels besides surface water effects. For example, some studies have argued that an increase in rainfall increases agricultural production, lowering food prices, and increasing nutrient intake and hence health.[9] This is a potential threat to the validity of the rainfall instrument. However, after careful investigation, this argument does not seem hold in this study.

First, small shocks in agricultural production induced by rainfall variations do not necessarily cause infant to die, unless the households are extremely poor and heavily depend on the food or the resulting income to survive. We summarize the main results from a series of recent studies that directly evaluate the relationship between rainfall and health outcomes in Table 2.1. Not surprisingly, we find a mixture of positive, negative,

[8] A prefecture usually includes dozens of counties. Ideally, we would like to use the total wastewater discharged in all other counties within the same prefecture as the instrumental variable for a particular county. Unfortunately, we cannot obtain county-level wastewater data.

[9] The link between rainfall and agricultural income has been investigated in the literature. For example, Levine and Yang (2006) showed that more rainfall increases rice output in Indonesia. Duflo and Udry (2004) looked at how men and women's income and spending change when the yields of different crops vary due to their different sensitivity to rainfall.

and non-significant results. More importantly, the studies that found a significant effect of rainfall fluctuations (within its usual range) on infant mortality always focused on poor and arid or semiarid regions. In our study, the sampled counties are all located near large rivers, lakes and reservoirs, which by nature are relatively water-abundant. The irrigation process mainly relies on the surface water system instead of rainfall. So it is unlikely that rainfall affects infant mortality through its impact on agricultural production. When we regress agriculture production per capita on precipitation, the estimated coefficient on precipitation is –0.245 (a 1 mm increase in precipitation is associated with a 0.245 Yuan decrease in per capital agricultural production) with a t-statistic of -0.99. Thus, we conclude that rainfall is not correlated with agriculture production in our sample.

Second, even in arid and semiarid regions, where water scarcity reduces agricultural production, the primary channel by which rainfall affects infant mortality is not through its impact on agriculture production. For example, Bhalotra (2007) found that in rural areas of India income shocks have significant negative effects on infant mortality. However, when he controlled for rainfall, the income effect did not change, suggesting that the effect of aggregate income on rural infant mortality is not driven by agriculture income. In Rocha and Soares's (2012) study, irrespective of how they introduced agricultural production in the regression, the impact of rainfall variations on health at birth was not affected, also suggesting that agricultural income does not affect infant mortality. Instead, they found that the negative impact of rainfall on infant mortality would be greatly reduced by using piped water. Thus if people no longer use contaminated water, rainfall itself would not harm infant health.

Ebenstein (2012) investigated the effects of water pollution on digestive cancer in China and found that precipitation has only a very weak relationship with other disease mortality rates except for digestive and lung cancer rates, and found almost no relationship between rainfall and cancer rates in areas with high rate of tap water. His results also showed that the effect of rainfall variations on people' health is primary due to its impact on the surface water.

In this study, if we include per capita agriculture production as a control variable, we find that it is not statistically significant and that the estimates of the effect of water quality on infant mortality are unchanged. Thus, we believe that precipitation is a valid instrument and that rainfall affects infant health through its impact on surface water quality. We also verified that in 1999 and 2000 no catastrophic natural disaster such as severe droughts or floods occurred in the sampled counties.

2.3 Data and Summary Statistics

China established a nationwide water quality monitoring system in the 1980s. Each year, the Ministry of Water Resource publishes the China National Water Resource Yearbook, which provides water quality information for major lakes, rivers and reservoirs. Many provinces also publish province-level water-body quality measures. The national and provisional publications are the sources of water quality data used in this paper.

We identified each water quality monitoring site's location and matched it with the 2000 Census data at the county-level.[10] Our sample includes 461 counties in 19

[10] There are five administrative levels in China. The highest level is the provincial level, which include provinces, autonomous regions, direct-controlled municipality, and special administrative regions (Hongkong and Macao). The second highest level of government is the prefecture level, which includes prefectures, autonomous prefectures, prefecture-level cities and Leagues. Next is the county level, which includes counties, autonomous counties, county-

provinces. The sampled provinces include Anhui, Beijing, Chongqing, Fujian, Guangxi, Guizhou, Hianan, Hebei, He'nan, Jiangsu, Jiangxi, Liaoning, Ningxia, Shandong, Shanghai, Sichuan, Tianjin, Yunnan and Zhejiang. Guangxi and Ningxia are in the Autonomous Region; Beijing, Chongqing, Shanghai and Tianjin are directly controlled municipalities; and the rest are governed at the provincial level. Northern provinces are relatively under-sampled because we could not find water quality data for several Northern provinces.

The distribution of the six types of water quality is presented in Table 2.2. Nearly half (47 percent) of the rivers and other bodies of water in our sample are seriously polluted (Types IV, V, and VI).

The overall Chinese infant mortality rate has been decreasing at a rapid pace over the past forty years, from about 150 per thousand in the 1960s to around 20 per thousand in the 2000s. In our sample, the average infant mortality across counties is 19.2 per thousand. The variation in infant mortality rates is large, with a standard deviation of 15.8. The distribution of infant mortality rate is skewed to the left, with a few counties out in the tail with considerably higher infant mortality rates than the average. The lowest infant mortality rate in our data is less than 1 per thousand, while the highest infant mortality rate is 81.2 per thousand. The summary statistics of infant mortality are presented in Table 2.3.

In general, female infants are more likely to die than male infants in China. Female infant mortality is about 22 per thousand, and male infant mortality is about 17 per thousand. The difference is striking because male infants are usually more vulnerable and thus more likely to die than female infants on purely medical grounds. This difference is consistent with the popular argument that rural Chinese people prefer boys to girls so they invest more on male infants' health. The variance of female infant mortality is also higher than that of male infant mortality.

To isolate the effect of water pollution on health, we include a set of explanatory variables from the 2000 Census of China: the percentage of the population that is non-agricultural, illiteracy rate, average rooms per home, and per capita housing area (square meters). We also include a few social-economic variables in the 2000 China Statistical Yearbook: per capita GDP (Chinese Yuan), per capita government expenditure, and the number of beds in medical institutions per 10,000 people. We show the summary statistics of these variables in Table 2.4.

The percentage of the population that is non-agricultural is the share of the population (including the temporary and mobile population) that lives in non-agricultural areas to the total population. It measures the degree of urbanization. Urbanization has both positive and negative health effects, and the net impact on population is not obvious (see, for example, Van de Poel, O'Donnell and Van Doorslaer, 2009). It is generally believed that the positive consequences of urbanization outweigh the negative ones for infants' health. Urbanization is associated with better sanitation and medical treatments, easier access to tap water and infant care, all of which play important roles in improving infant health. So we expect a negative association between the percentage of non-agricultural population and infant mortality.

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level cities, and city districts. Below the county is the township level and the (informal) village level. The Census data are not available for these last two levels.

The illiteracy rate variable is the share of the total population over 15 who are illiterate (have not completed an elementary-school education). Many studies have found that infants are less likely to die the more educated are their parents, especially their mothers (Delgado et al. 2002; Behrman et al. 2003; Basu and Stephenson, 2005; Frost et al. 2005; Miguel, 2005; Boyle, 2006; Cowell, 2006; Cutler and Lleras-Muney, 2006).
Poor housing and overcrowding are negatively associated with infant health (Martin, 1967; Brennan and Lancashire, 1978; Victora, et al, 1988). We expect that as either of our measures of living conditions—the average number of rooms per household and the per capita housing area (square meters)—increases, infant mortality falls.

We expect that per capita county GDP to be negatively correlated with infant mortality. We use per capital government expenditure to approximate the local government's investments on social welfare programs, such as public health insurance, sanitation maintenance, tap water provision, waste management, and pollution treatment. We expect it to be negatively correlated with infant mortality. We use the number of beds per 10,000 people in medical institutions as a measure of the availability of medical treatment. In regions where hospitals are readily available, infant mortality should be lower.

The reliability of the data collected by Chinese government is often questioned by academic researchers. Particularly because local governments in China have been criticized for hiding news from the public about water pollution accidents, we might question the reliability of the water quality data. Consequently, we examined the consistency of the data in three ways.

In China, multiple agencies (e.g. the Ministry of Water Resource, the Environment Protection Agency, and the Center for Disease Control and Prevention) collected data on surface water quality. Since we don't know the raw data sources of different water quality reports, we first checked for consistency between reports of water quality data between national water resources reports and provincial water resources. We found that 98 percent of these paired reports are consistent, and the few differences are all within 1 water quality level. In the few case where they differ, we rely on the data from the provincial water resource reports.[11]

Second, we compared a subsample of the 2004 water quality data from the yearbooks with 2004 water quality data provided by the World Bank, which is used in Ebenstein (2012) paper. The data are almost always identical for comparable monitoring sites. Third, several monitoring sites' data are reported in the River Basin Water Quality Reports. Again, we found no substantial difference between these and other sources.

Thus, the various reports on water quality are consistent. Hopefully, they are accurate. In the past several years, the government has trumpeted its efforts to promote the transparency of surface water quality information. For example, the central government started to release weekly water quality report for 100 national monitoring sites to the public in 2004, and it started to publicize real-time water quality data in 2009.

[11] If we drop these observations, we obtain the same qualitative results as reported below.

2.4 Models and Estimations

2.4.1 Ordered-Probit Selection Model

We use a two-step sample selection model to estimate infant mortality conditional on surface water quality. In the first step, we estimate the water quality using an ordered probit model. If people move in response to the local water quality, the water pollution level is endogenous.

Because the number of observations of Type I water is relatively small (Table 2.2), we aggregate Type I and Type II water into a single group, which leaves us with five water quality categories. Each County *i* has water quality in Category 1 (Type I and Type II), Category 2 (Type III), …, or 5 (Type VI), ranging from good quality to bad, We estimate the water quality using an ordered-probit:

$$water_i^* = \alpha' z_i + u_i \tag{1}$$

$$water_i = \begin{cases} 1 \ if -\infty < water_i^* \le \mu_1, \\ 2 \ if \quad \mu_1 < water_i^* \le \mu_2, \\ 3 \ if \quad \mu_2 < water_i^* \le \mu_3, \\ 4 \ if \quad \mu_3 < water_i^* \le \mu_4, \\ 5 \ if \quad \mu_4 < water_i^* < \infty \end{cases} \tag{2}$$

where $water_i^*$ is the unobserved latent selection variable (actual water quality), z_i are a set of variables that affect water quality, u_i is a normal disturbance; $water_i$ is the observed 5-degree water quality scale, and the unobserved cutoffs satisfy $\mu_1 < \mu_2 < \mu_3 < \mu_4$.

The observed infant mortality rate IMR_i is a linear function of observed independent variables x_i, the demographic, social-economic variables, conditional on the observed water quality level. We estimate separate coefficients of x_i for each category $water_i$:

$$IMR_i = \begin{cases} \beta_1' x_i + \epsilon_{i1} \ \ if\ water_i = 1, \\ \beta_2' x_i + \epsilon_{i2} \ \ if\ water_i = 2, \\ \beta_3' x_i + \epsilon_{i3} \ \ if\ water_i = 3, \\ \beta_4' x_i + \epsilon_{i4} \ \ if\ water_i = 4, \\ \beta_5' x_i + \epsilon_{i5} \ \ if\ water_i = 5 \end{cases} \tag{3}$$

where for each water quality category j, ϵ_{ij} has mean 0 and variance σ_j^2, and is bivariate normal with u_i. The correlation between ϵ_{ij} and u_i is ρ_j for group j. We assume that ϵ_{ij} and u_i are independently and identically distributed across observations.

We estimate this model using a two-step estimation procedure that has been described by Greene (2002) and is a generalization of Heckman's (1979) estimator for the binary case. [12] Define:

[12] There are two popular approaches in estimating the probit selection model: the full information maximum likelihood (FIML) approach and the two-step approach. In a binary selection case, Puhani (2000) found that FIML is usually more efficient than the two-step estimator. However, in an ordered probit selection model, such as we use, Chiburis and Lokshin (2007) found that the two-step estimator is more robust and is the better choice for almost all practical applications.

$$\lambda_i \equiv E(u_i|water_i, z_i) = \frac{\int_{\mu_j}^{\mu_{j+1}} (water_i^* - \alpha' z_i)\phi(water_i^* - \alpha' z_i)\, dwater_i^*}{\Phi(\mu_{j+1} - \alpha' z_i) - \Phi(\mu_j - \alpha' z_i)} = \frac{\phi(\mu_j - \alpha' z_i) - \phi(\mu_{j+1} - \alpha' z_i)}{\Phi(\mu_{j+1} - \alpha' z_i) - \Phi(\mu_j - \alpha' z_i)} \quad (4)$$

where ϕ is the standard normal density function, and Φ is the standard normal cumulative distribution function. Then the expectation of infant mortality rate, conditional on all the observed factors, is

$$E[IMR_i|water_i, z_i, x_i] = \beta_j' x_i + E\left(\epsilon_{ij} \middle| water_i = j, z_i\right) = \beta_j' x_i + \rho_j \sigma_j \lambda_i \quad (5)$$

Thus if we only regress IMR_i on x_i over the subsample {i: $water_i = j$}, without taking into account of λ_i, the estimation will be inconsistent if $\rho_j \neq 0$.

For the ordered-probit selection model to be identified, z must contain at least one variable that is not in x.[13] That is, we must have at least one instrument z for the selection variable $water$ (observed water quality) that is a significant determinant of water quality yet satisfies the exclusion restriction $Cov(z, \epsilon_j) = 0$ for all j. We use wastewater dumping, rainfall, and their squares as instruments for water quality level.

In the first step, we estimate (2) by an ordered probit of $water$ on z, yielding the consistent estimates $\hat{\alpha}$ and $\hat{\mu}_j$. Define $\widehat{water}_i^* = \hat{\alpha}' z_i$. Using (4), we consistently estimate λ_i by

$$\hat{\lambda}_i = \frac{\phi(\hat{\mu}_j - \widehat{water}_i^*) - \phi(\hat{\mu}_{j+1} - \widehat{water}_i^*)}{\Phi(\hat{\mu}_{j+1} - \widehat{water}_i^*) - \Phi(\hat{\mu}_j - \widehat{water}_i^*)} \quad (6)$$

for $j = water_i$.

By using the observations i for which $water_i = j$, we can consistently estimate β_i' with OLS regression of IMR on x and $\hat{\lambda}$.

Moreover, σ_j can be estimated by:

$$\hat{\sigma}_j \equiv \frac{1}{n_j}\left(RSS_j - \hat{C}_j^2 \sum_{i:j=j} \frac{\partial \hat{\lambda}_i}{\partial \widehat{water}_i^*}\right)$$

$$= \frac{RSS_j}{n_j} - \frac{\hat{C}_j^2}{n_j}\left\{\frac{(\hat{\mu}_j - \widehat{water}_i^*)\phi(\hat{\mu}_j - \widehat{water}_i^*) - (\hat{\mu}_{j+1} - \widehat{water}_i^*)\phi(\hat{\mu}_{j+1} - \widehat{water}_i^*)}{\Phi(\hat{\mu}_{j+1} - \widehat{water}_i^*) - \Phi(\hat{\mu}_j - \widehat{water}_i^*)}\right\} \quad (7)$$

where n_j is the number of observations in which equation j is observed, $\hat{C}_j$ is the coefficient on $\hat{\lambda}$, and RSS_j is the residual sum of squares for the regression. Since $\hat{C}_j$ is a consistent estimator for $\rho_j \sigma_j$, we have a consistent estimator for ρ_j:

$$\hat{\rho}_j \equiv \frac{\hat{C}_j}{\hat{\sigma}_j} \quad (8)$$

2.4.2 Estimations

Table 2.5 reports the mean values of selected variables by water quality type. We see pronounced water type patterns. Infant mortality is the highest in the Type III regions. In the polluted regions, infant mortality is substantially lower. These summary statistics already show a non-monotonic relationship between infant mortality and water pollution. We also see that in more polluted regions, the share of non-agricultural population is higher, suggesting that pollution is accompanied by urbanization. Type IV and V water is associated with the highest per capita GDP and government expenditure. These patterns suggest that water quality is not randomly assigned to different regions and cannot be

[13] Otherwise the identification will solely rely on the functional form of the ordered-probit model.

treated as exogenous. People may sort into different regions and thus a regression of infant mortality on water pollution will give a biased estimate.

We report the regression results of the ordered-probit selection model in Table 2.6. In the first step, we regress water quality on the explanatory variables z_i, which include our four instrumental variables (precipitation, wastewater dumping and their squares) and all the demographic, social-economic x_i. The estimated coefficients of the four instrumental variables are all statistically significant. Both precipitation and wastewater dumping have strong effects on surface water quality. In the second step, we estimate the relationship between IMR_i and the independent variables x_i, taking into account the first-step estimates $\hat{\lambda}$.

The estimated coefficients of x_i vary across different groups. The percentage of non-agricultural population is statistically significant in Type I or II, Type IV and Type V areas. It is negatively correlated with infant mortality, as expected. On average a 10 percent increase in non-agricultural population is associated with roughly a 1.9 per thousand drop in infant mortality in Type I or II areas. The estimated coefficients for Type IV and V areas are 1.6 and 2.4 per thousand, respectively.

A higher illiteracy rate statistically significantly increases infant mortality in all regressions. As the illiteracy rate increases by 1 percent, infant mortality falls by roughly 1 per thousand.

Housing and living conditions are typically negatively correlated with infant mortality. Both the average number of rooms per household and the per capita housing area are statistically significantly associated with infant mortality. For example, if each household in Type I or II areas has one more room at home, infant mortality will fall by 3.8. If per capita housing area in Type I or II areas increases by 10 square meters, infant mortality will decrease by 4.0 per thousand. As per capita GDP goes up, infant mortality goes down. An increase of one thousand Yuan (about 150 US dollars) in per capita GDP is associated with a 0.17 to 0.64 per thousand fall in infant mortality, depending on which group is chosen.

The importance of taking into account selection bias can be seen from the statistical significance of the selection terms ($\boldsymbol{\lambda_i}$). These significant selection effects (in the Type IV and VI regressions) indicate that if we did not explicitly treat the levels of exposure to water pollution as endogenous, our estimates of the effects of water pollution on infant mortality using OLS would be biased.

We can predict the expected infant mortality $\widehat{IMR_i} = \hat{\beta}_j' x_i$ for each category (water type), and calculate the unconditional averages and differences of the predicted infant mortality for each category. This is a prediction of infant mortality without taking into account the influence of the selection. Using the estimates in Table 2.5, we find the average predicted infant mortality $\overline{\hat{y}}$ for the five types of water are respectively 20.3, 24.0, 14.9, 12.1 and 5.8 per thousand. That is, the highest infant mortality is associated with Type III water. In the cleanest areas (Type I or II) and most polluted areas (Type IV V and VI), infant mortality is lower. The relationship between water quality and infant mortality is non-monotonic, and the most polluted areas (Type VI) have the lowest infant mortality. On average, infant mortality in the Type I or II areas is 3.7 per thousand lower than that in the Type III areas; and infant mortality in the Types IV, V and VI areas are respectively 9.1, 11.9, and 18.2 per thousand lower than that in the Type III areas.

Our goal is to estimate the effects of water quality on infant mortality. An alternative, perhaps better, method is to estimate the counterfactual $\widehat{IMR}_j$ for the given equation *j*, if all observations were to switch to category *j*, but taking into account the category that was actually chosen. Particularly, conditional on sorting into Type *k* water quality, the infant mortality equation in county *i* can be written as:

$$IMR_{ik} = \beta_k' x_i + \rho_k \sigma_k \lambda_{ik} + \epsilon_{ik} \quad (9)$$

If observation *i* in category *k* were to switch to category *j*, conditional on that category *k* is actually chosen, the predicted counterfactual infant mortality is:

$$\widehat{IMR}_{ij} = \hat{\beta}_j' x_i + \hat{\rho}_j \hat{\sigma}_j \hat{\lambda}_{ik} \quad (10)$$

where $\hat{\lambda}_{ik}$ is calculated as in (6), using the actual $water_{ik}$.

We repeat this calculation for all the observations in category *k*, where $k \neq j$, to get the counterfactual infant mortality rate if these observations were to switch to category *j*. Then we repeat the process for all *j*, *j* = 1,2,...,5.

The predicted counterfactual average infant mortalities are reported in row 1 in Table 2.7. If all observations were to switch to the Type I or II area, the average infant mortality would be 18.7 per thousand; if all were to switch to the Type III area, the average infant mortality would be 24.8 per thousand; the value would be 16.9 per thousand if all were to switch to the Type IV area; it is 15.1 for Type V area and 4.6 for Type VI area.

We use the Type III areas as the reference group, and compare its infant mortality with that in other groups. The differences are reported in row 2 in Table 2.7, and they tell us the estimated effects of water quality on infant mortality in the selection model. If water quality changes from Type I or II to Type III, infant mortality will increase by about 6.0 per thousand. Changing water quality from Type III to Type IV decreases infant mortality by 7.9 per thousand. The lowest infant mortality is found in the most polluted areas (Type VI). If water quality deteriorates from Type III to Type VI, infant mortality would drop by 20.2 per thousand.

We also estimated the ordered probit selection model by gender. We calculated the counterfactual average infant mortality for each category and the differences between them, using Type III areas as the reference group. The results for male infants are reported in rows 4-5 in Table 2.7. Changing water quality from Type I or II to Type III would increase male infant mortality by roughly 9.7 per thousand. As water quality deteriorates from Type III to Type IV, V and VI, male infant mortality will drop by 7.1, 8.1 and 15.1 per thousand, respectively. The estimates for female infants are reported in rows 7-8 in Table 2.7. The results are slightly different. Changing water quality from Type I or II to Type III wouldn't significantly increase female infant mortality; the difference is only about 1.1 per thousand. However, as water quality becomes more polluted, changing from Type III to Type IV, V and VI, female infant mortality would drop dramatically, with the respective estimated magnitudes of -8.8, -11.6 and -26.3 per thousand. The estimates of water pollution on female infant mortality are larger than male infant mortality. Female infants benefit more than males from water quality falling from fair to polluted.

We compared our sample-selection model results to those estimated using OLS. In the OLS regressions, we estimate the following model:

$$IMR_i = \beta_0 + \beta_1 D1_i + \beta_2 D2_i + \beta_3 D3_i + \beta_4 D4_i + \beta_5' x_i + \varepsilon_i \quad (11)$$

where IMR_i is infant mortality rate in county i, x_i is a vector of covariates, D1,…,D4 are dummies indicating whether a county has Type I or II water (D1=1), Type IV water (D2=1), Type V (D3=1) water, and Type VI (D4=1) water. Type III water is still used as the reference group.

The regression results from OLS are reported in Table 2.8. The non-monotonic relationship between water quality and infant mortality is robust in the OLS regressions. Based on the OLS estimates, changing water quality from Type I or II to Type III is associated with 4.0 per thousand drop in infant mortality; and as water quality deteriorates from Type III to Type IV, V, and VI, the associated infant mortality would drop by 5.7, 7.7 and 8.0 per thousand, respectively.

The OLS model underestimates the effects of water pollution on infant mortality, especially for the more polluted areas. For example, as water quality deteriorates from Type III to Type VI, OLS indicates the infant mortality would only decrease only by 8 per thousand, whereas it would drop by 20 per thousand in the ordered probit selection model.

Assuming that people cannot perceive water quality changes between Type I or II and Type III, the associated 6.0 per thousand increase in infant mortality can be interpreted as the pure health effect of water pollution. In other words, we find that one-degree deterioration of water quality leads to about 30% increase in infant mortality. Is this plausible? To answer this question, we conduct a crude calculation on the magnitude to water quality changes from Type I or II to Type III.

According to the Environmental Surface Water Quality Standard in China, a deterioration of water quality from Type I to Type III approximately corresponds to (using Type I as reference) a 33-percent decrease in the concentration of dissolved oxygen (DO), a 200-percent increase in the concentration of Potassium permanganate (KMnO4), a 33-percent increase in the concentration of chemical oxygen demand (COD), a 560-percent increase in the concentration of Ammonia Nitrogen (NH3-N), a 900-percent increase in the concentration of Total phosphorus (TP), a 400-percent increase in the concentration of Total nitrogen (TN). The changes in the concentrations for other pollutants are even more pronounced. For example, the maximum allowed number of fecal coliform for Type I water is 200 per liter, and it is 2,000 per liter for Type II water, and this maximum number increases to 10,000 per liter for Type III water. Thus, the actual change in water quality from Type I to Type II to Type III, even though cannot be easily detected by eye-browsing, is significant and potentially harmful.

The large effect of water pollution on infant mortality in China is in-keeping with the findings in several studies. In particular, Cutler and Miller (2005) argued that the adoption of clean water technologies such as filtration and chlorination was responsible to up to 75 percent of infant mortality in early twentieth century America. Galiani et al. (2005) found that privatization of water supply in Argentina reduced the mortality of children under age 5 by 26 percent. Brainerd and Menon (2012) found that 10 percent increase in the average of fertilizer chemicals in water in the month of conception increased infant mortality by 4 percent, and neo-natal mortality by 7 percent.

2.5 Conclusion and Policy Implication

The relationship between surface water quality and infant mortality is not monotonic in China. As surface water quality deteriorates, the infant mortality increases at first then

decreases. Infant mortality is the highest in areas where surface water quality is neither too good nor too bad. This pattern is robust to a variety of specifications and models.

Our explanation is that, as surface water deteriorates, initially it is difficult to detect a quality change, so it harms infants. As the water pollution becomes more obvious, people reduce their consumption of polluted water so their health status improves. We find evidences supporting this argument. In both the OLS and the orderd-probit selection models, the regression results show that infant mortality is the highest when water quality is fair. In both cleaner and more polluted regions, infant mortality is lower. The lowest infant mortality is associated with the most severe water pollution.

The selection issue has been well recognized in labor economics and other related fields, however, few studies investigating the health effects of pollution considered this problem. We formally address the endogeneity of water pollution using a generalized Heckman selection model. Explicitly incorporating sorting across water pollution levels into the infant mortality equations allows us to produce unbiased estimates of differentials in infant mortality with respect to water pollution levels. Furthermore, by running the infant mortality functions separately for different water types we allow the structure of the infant mortality functions vary across water pollution level.

The policy implication is straightforward. Since moderate level of water pollution is most dangerous, public provision of clean drinking water is most urgent in these regions. The government should pay more attention to these regions, instead of the most polluted areas. We find the statistically significant relationship between illiteracy rate and infant mortality for all water quality types, suggesting that providing health program about water pollution may be an economically effective way to reduce infant mortality. Better housing conditions also reduce infant mortality, which may primarily due to better sanitation.

We find an interesting differential effect of water pollution on male and female infants. Male infants are more likely to die if water quality changes from Type I or II to Type III; however, as water quality further deteriorates, female infants seem to benefit more from the avoidance behaviors. The gender-preference may partially explain this pattern; however, this relationship clearly requires further investigation and more evidences.

The avoidance behavior argument will be much more compelling if we know the share of people consuming/contacting surface water in each county. Unfortunately, due to data availability, we cannot test this hypothesis directly. In the past years, water pollution accidents took places from time to time in China. A direct consequence of these water pollution accidents is the "panic purchasing" of bottled water. If we search "water pollution", "bottled water", "panic purchasing" in Chinese on the largest Chinese search engine, Baidu.com, it gives us more than 133,000 related entries. If similar inquiries are done through Google.com, more than 292,000 related entries are found.[14] Thus the avoidance behaviors in response to water pollution are very common.

One drawback of this study is that we cannot control for other pollutants, such as air pollution. Since other pollutants are presumably correlated with water pollution levels, our estimated effects for surface water may reflect the combined effects of exposure to a range of environmental pollutants and risks. However, the non-monotonic relationship

[14] These inquires were made in May 2013.

between surface water quality and infant mortality makes it less likely that the effects of water and other pollutants are confounded. People have fairly easy and low costs ways to protect themselves from being harmed by polluted water (such as by using boiled water, tap water and bottled water); while it is not easy to avoid the harm from other pollutants, such as air-borne diseases and toxin accumulation through food chain. Since most of these pollutants are positively correlated, if what we captured was the combined effects of all environmental contaminants, we should not expect to see infant mortality falling in areas with poor water quality.

Table 2.1 The Effects of Rainfalls on Infant Mortality

Study	Outcome	Explanatory Variables	Data	Conclusions
Aguilar and Vicarelli (2011)	Cognitive tests, anthropometric variables, health indicators	Exogenous excessive rainfall shocks.	Individual survey data from 506 rural communities in Mexico	Children born in years and regions affected by excessive rain experience slower anthropometric growth and cognitive development.
Baird, et al. (2007)	Infant Mortality	Rainfalls, per capita GDP, characteristics of women, conflict, quality of institution, etc.	Birth data in 59 developing countries	Rainfall shocks have no significant effect on infant mortality.
Friedman (2010)	Infant Mortality	Daily temperature and rainfalls	Birth data in 47 South African countries	Excess rainfall is both detrimental and protective depending on the timing in which it occurs.
Kovats and Wilkinson (2004)	Morality by Age and by Cause of Death	Daily rainfalls, temperature, and season, trend, holidays and air pollution.	Birth data in New Delhi	Any increase in rainfall increases the risk of infectious disease mortality in the near term.
Kim (2010)	Infant Mortality	Monthly Rainfalls, mother's characteristics, child's characteristics, religion, region, etc.	Birth data in 9 West African countries	There is no association between rainfall and infant mortality on average. But rainfall shocks have an adverse effect on the survival of young children that were born in the rainy season.
Kudamastsu, et al. (2010)	Infant Mortality by Area, by Household Type	Monthly rainfalls, season, drought indicator, household type, area (endemic, epidemic, non-malarious, rainy and arid)	Birth data in 28 African countries	Increased rainfall is associated with higher mortality by malaria in epidemic, but not in endemic areas.
Rocha and Soares (2012)	Gestation, Birth Weight and Infant Mortality	Monthly rainfall, drought indicator, temperature, trend, etc.	Birth data in Semiarid Northeast Brazil	Negative rainfall fluctuations lead to higher incidences of low birth weight, preterm gestation and infant mortality rates, in particular due to intestinal infections and malnutrition.
Skoufias et al. (2011)	Child height-for-age	Rainfall, household and individual characteristics.	Mexico Family Life Survey and National Nutrition Survey of Mexico	The effects of rainfall on height-for-age is heterogeneous. A positive rainfall shock during the wet season is associated with shorter children in the North, but not in the Centre/South regions.

Table 2.2 Water Quality Distributions

Water Quality	Type I	Type II	Type III	Type IV	Type V	Type VI	Total
Frequency	23	132	90	60	59	97	461
Percent	4.99	28.63	19.52	13.02	12.80	21.04	100

Table 2.3 Summary Statistics of Infant Mortality

	Infant Mortality (per thousand)		
	All	Male	Female
Mean	19.23	16.97	21.97
Std. Dev.	15.82	13.44	20.42
25% Quantile	8.64	7.89	8.61
50% Quantile	14.62	13.63	15.17
75% Quantile	25.31	21.81	29.47

Table 2.4 Summary Statistics of the Explanatory Variables

	Mean	Std. Dev.	Min	Max
Non-Agricultural Population (%)	19.23	15.83	0.89	81.24
Illiteracy Rate (%)	16.98	13.45	0.00	76.00
Average Rooms per Household	21.98	20.42	0.00	125.00
Housing Area per capita (sq.m.)	30.16	26.55	4.38	97.40
GDP per capita (1000 Yuan)	9.52	6.15	1.57	48.34
Government Expenditure per capita	2.60	0.58	1.50	4.92
Hospital Beds per 10,000 people	23.47	6.48	9.47	45.64

Table 2.5 Mean Values of the Key Variables by Water Quality Type

	Type I and II	Type III	Type IV	Type V	Type VI
Infant Mortality (per thousand)	21.26	23.82	16.47	13.60	16.88
Male Infant Mortality (per thousand)	17.75	21.41	14.84	12.16	15.88
Female Infant Mortality (per thousand)	25.61	26.71	18.35	15.31	18.09
Non-Agricultural Population (%)	25.23	27.25	36.74	36.40	32.87
Illiteracy Rate (%)	9.78	9.84	9.94	8.00	9.48
Average Rooms per Household	2.57	2.69	2.56	2.55	2.61
Housing Area per capita (sq.m.)	24.21	24.45	24.73	22.67	21.10
GDP per capita (1000 Yuan)	7.87	9.92	12.02	11.01	8.06
Government Expenditure per capita	0.60	0.72	1.09	1.07	0.75
Hospital Beds per 10,000 people	27.55	36.97	35.14	33.61	33.12

Table 2.6 Ordered-Probit Selection Model: Infant Mortality and Water Quality

First Step (Water Quality)		Second Step (Infant Mortality by Water Type)				
		I or II	III	IV	V	VI
Precipitation (100mm)	-0.271***	-	-	-	-	-
	(0.048)	-	-	-	-	-
Precipitation Sq.	0.006***	-	-	-	-	-
	(0.002)	-	-	-	-	-
Wastewater Dumping	0.033**	-	-	-	-	-
	(0.015)	-	-	-	-	-
Wastewater Dumping Sq.	-0.001***	-	-	-	-	-
	(0.0003)	-	-	-	-	-
Non-Ag Population (%)	0.006**	-0.185***	-0.007	-0.164*	-0.243***	-0.023
	(0.003)	(0.065)	(0.110)	(0.094)	(0.072)	(0.057)
Illiteracy Rate (%)	0.022**	0.925***	1.330***	1.053***	1.296***	1.000***
	(0.011)	(0.199)	(0.349)	(0.278)	(0.368)	(0.183)
Ave. Rooms per Household	0.236**	-3.813*	1.005	0.251	-5.964**	-0.734
	(0.113)	(2.310)	(3.121)	(3.932)	(2.602)	(2.063)
Housing Area per capita	-0.008	-0.401***	-0.597**	-0.580*	-0.586**	-0.330
	(0.011)	(0.150)	(0.280)	(0.305)	(0.256)	(0.306)
GDP per capita (1000 Yuan)	0.021*	-0.323	-0.644*	-0.260	-0.169	-0.463*
	(0.011)	(0.212)	(0.368)	(0.328)	(0.144)	(0.282)
Gov. Exp. per capita	0.004	-2.223	2.134	-1.585	-0.934	-0.872
	(0.080)	(2.449)	(5.502)	(2.638)	(0.970)	(2.072)
Hospital Beds per 10,000 people	-0.002	0.040	-0.025	0.204	0.224**	0.008
	(0.003)	(0.054)	(0.104)	(0.138)	(0.109)	(0.069)
λ_i	-	-0.994	-0.106	9.552***	3.309	10.20***
	-	(3.044)	(2.935)	(3.397)	(2.058)	(2.781)
Number of Observations	460	ρ_0=-0.091	ρ_1=-0.008	ρ_3=0.661	ρ_4=0.373	ρ_5=0.788

Note: *** Significant at 1% level;
** Significant at 5% level;
* Significant at 10% level

Table 2.7 The Effects of Water Quality on Infant Mortality

			Type I or II	Type III	Type IV	Type V	Type VI
Overall	(1)	Counterfactual Average	18.733	24.758	16.894	15.093	4.570
	(2)	Differences	-6.025	-	-7.864	-9.666	-20.188
	(3)	OLS Estimates	-3.964	-	-5.682	-7.683	-7.956
Male Infant	(4)	Counterfactual Average	12.793	22.458	15.364	14.403	7.375
	(5)	Differences	-9.666	-	-7.095	-8.055	-15.083
	(6)	OLS Estimates	-4.552	-	-5.304	-6.699	-6.018
Female Infant	(7)	Counterfactual Average	26.426	27.548	18.674	15.956	1.284
	(8)	Differences	-1.122	-	-8.874	-11.592	-26.264
	(9)	OLS Estimates	-3.102	-	-6.187	-8.895	-10.28

Table 2.8 OLS Regression Results on Infant Mortality

	Infant Mortality		
	Overall	Male	Female
Type I or II	-3.964**	-4.552***	-3.102
	(1.742)	(1.556)	(2.258)
Type IV	-5.682***	-5.304***	-6.187**
	(2.194)	(1.987)	(2.729)
Type V	-7.683***	-6.699***	-8.895***
	(1.980)	(1.661)	(2.686)
Type VI	-7.956***	-6.018***	-10.28***
	(1.883)	(1.738)	(2.270)
Non-Agricultural Population (%)	-0.128***	-0.081***	-0.187***
	(0.028)	(0.024)	(0.038)
Illiteracy Rate (%)	1.054***	1.026***	1.066***
	(0.130)	(0.112)	(0.167)
Ave. Rooms per Household	-2.678**	-1.883**	-3.586***
	(1.016)	(0.907)	(1.391)
Housing Area per capita	-0.356***	-0.166**	-0.587***
	(0.084)	(0.071)	(0.122)
GDP per capita (1000 Yuan)	-0.356***	-0.352***	-0.367***
	(0.087)	(0.079)	(0.112)
Gov. Exp. per capita (1000 Yuan)	-0.339	-0.226	-0.439
	(0.561)	(0.514)	(0.706)
Hospital Beds per 10,000 people	0.007	0.033	-0.023
	(0.026)	(0.021)	(0.039)
F-Statistics	36.81	31.76	35.28
R Square	0.493	0.484	0.428
Number of Observations	460	460	460

Note: Robust Standard errors are in the parenthesis.
** Significant at 1% level;
** Significant at 5% level;
* Significant at 10% level

Type I **Type II** **Type III**

Type IV **Type V** **Type VI**

Figure 2.1 Different Types of Water Quality

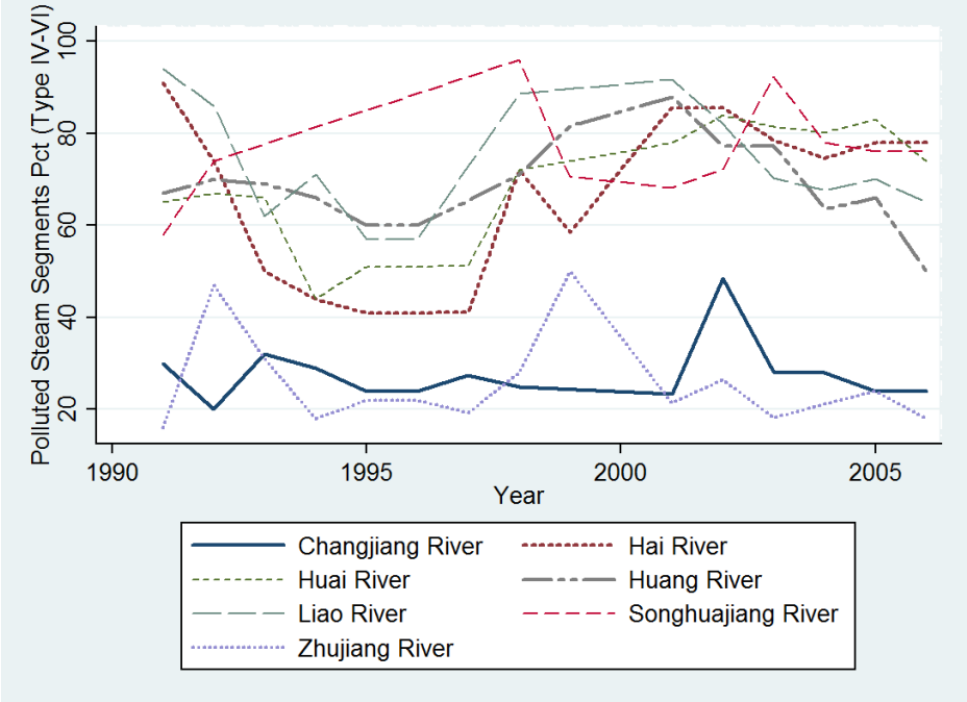

Figure 2.2 Polluted Segment of the Main Water System in China

Chapter 3

Surface Water Pollution and Cancer Mortality among the Elderly in China

3.1 Introduction

Massive industrial wastewater discharges and extensive use of agricultural fertilizer have destroyed the surface water system in most places of China in the past thirty years. At the same time, China witnessed a dramatic increase in cancer rate. For example, according to the National Bureau of Statistics of China, the total number of new cancer cases increased by 14.6 percent from 2000 to 2005. By 2009 cancer has become the leading cause of death in urban and Rural China, and roughly a quarter of all deaths countrywide died from cancer. It is believed that the rise of cancer rates in China is driven by the deteriorations of the environment. The emergence of "cancer villages" in recent years has shown that the relationship between water pollution and cancer is very likely to be causal. These communities—where an unusually high number of residents are struck by the same types of cancer—tend to cluster in poorer areas along polluted waterways or downstream from industrial parks (Liu, 2010).

The poisoned water and soil are devastating to local residents in the poor areas since most villages are largely self-sufficient. The young and healthy people can leave and seek income elsewhere. Those too old, too poor, or too sick to leave have to stay.

Many recent studies have found various connections between water pollution and diseases and other public health measures. Some studies focused on water pollution and water-borne diseases, such as typhoid (Cutler and Miller, 2005) and diarrhea (Jalan and Ravallion, 2003). Some studies investigated how water pollution affects infant and child mortality, such as Galiani et al. (2005), Merrick (1985), Lavy et al. (1996), and Lee et al. (1997), Greenstone and Hanna (2002), and Brainerd and Menon (2012). As for China, He (2012) found that water pollution has a non-monotone impact on infant mortality and that slightly polluted water is the most dangerous to infants.

A limited but growing literature has linked water pollution to particular cancer types such as liver cancer (Lin et al., 2000) or gastric cancer (Morales-Suarez-Varela et al., 1995). Similar studies investigating water pollution and cancer include Cantor (1997), Davis and Masten (2003), and Chen et al. (2005). Ebenstein (2012) was the first study trying to estimate the causal relationship between water pollution and digestive cancer in China. He argued that China provides an ideal context to estimate the health effects of water pollution because of the Household Registration System (Hukou). Since a variety of benefits, such as health care and social security, were associated with the Household Registration System, this system prevented people from moving from Rural to urban areas, and also made it relatively difficult to move within Rural and urban areas. Therefore, he claimed "the location of residents at the time of observation in the data will likely reflect their true lifetime surface water pollution exposure". Based on this

assumption, he estimated that a deterioration in water quality by a single grade increases the digestive cancer death rate by 9.7 percent in China.

Our study differs from Ebenstein (2012) in several important ways. First, Ebenstein used death data from 1991-2000 but water quality data in 2004. The inconsistency of these two datasets creates substantial measurement errors. That is, water quality in 2004 cannot represent water quality during the 1991-2000. Figure 2.2 shows the proportion of the river segments that were seriously polluted (Type IV, V and VI) from 1991-2005. It is obvious that water quality is not the same in different years, and that water quality in 2004 is not an ideal measure of the long-term water quality. Thus his estimates might be biased, and the magnitude of the bias was unknown. In our study, we use matched datasets so that the deaths data and the water quality data cover the same period.

Second, while the migration restriction argument clearly held in the past, in the last two decades, the share of people migrating has increased dramatically. According to the Census, only 0.66 percent of the total population migrated in 1982 compared to 7.9 percent in 2000, and 17 percent in 2010. The results based on the overall population can be misleading because young and healthy people in the rural areas migrate to the urban cities. The level of pollution exposure for the total population has changed because of migration. In our paper, we focus on the elderly people, whose mobility is extremely restricted. Thus their life-time exposure to water pollution will be more accurately captured by our water quality data.

Third, to isolate the effect of water pollution on cancer, we collect comprehensive control variables from the statistical year books at the county level, such as per capita GDP, the share of agricultural output, the share of manufacture output, per capita government expenditure, per capita investment, and number of doctors per 10,000 people, etc.[15]

The remainder of this paper is structured as follows. Section 3.2 discusses the details of our data. To investigate the effects of water pollution on cancer, we combine rich datasets on water quality, death, weather and social-economic conditions. Section 3.3 presents the model and estimation. Section 3.4 presents the main results and robustness checks. Section 3.5 concludes.

3.2 Data and Summary Statistics

3.2.1 Water Quality Data

The surface water system broadly covers rivers, lakes and reservoirs. The overall surface water quality is graded using the concentrations of different chemical pollutants indicators, which include the pH-value and the concentrations (measured by mg/L) of dissolved oxygen, biochemical oxygen demand, ammonia, and nitrogen. The overall surface water quality is graded on a 6-degree scale, where Type I water is the best quality water and Type VI is the worst. According to the China Ministry of Water Resources, Type I water is an “Excellent” source of potable water. Type II water is a “Good” source

[15] In comparison, for example, Ebenstein (2012) used four dummies (from poorest to richest) to measure the relative richness of difference counties.

of potable water. Type III water is "Fair." Pathogenic bacteria and parasites ova can sometimes be found in Type II and III water so drinking it may cause disease. Thus, Type II and III water should be purified and treated (such as by boiling) before drinking. Type IV water is polluted and unsafe to drink without advanced treatment, which is only possible at water supply plants. Type V is seriously polluted and can never been used as for human consumption. Type VI water is called "Worse than Type V Water," and any direct contact with it is harmful to humans.

China established a nationwide water quality monitoring system in the 1980s. Both the State Environmental Protection Agency (SEPA) and the Ministry of Water Resource (MWR) in China collect surface water quality data. Our datasets come from both agencies.

Starting from 2004, SEPA publishes weekly water quality readings for its 100 monitoring sites countrywide on their website. This is the primary water quality data we use in this study. We aggregate the weekly data to yearly.

We collected water quality data for 59 major water sources from MWR. Water sources are usually reservoirs with good water quality. They provide the population in major cities with clean water (after treatment). The water quality readings for water sources are reported every 10 days, and are available from 2001 to 2009. We also aggregate the data to yearly.

For the year of 2004, we obtained yearly water quality data through the World Bank.[16] The dataset was collected from MWR's 484 monitoring points. The number of observations is significantly larger. We use this dataset for cross-validation and robustness check.

3.2.2 Cancer Mortality Data

The cancer data comes from the Disease Surveillance Point System (DSPS) in Center for Disease Control and Prevention of China. 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. Roughly 70 million people are covered by DSPS.

Seven categories of diseases were recorded in the DPSP data set: cancer, cerebrovascular diseases, respiratory system diseases, heart diseases, digestive system diseases, urine and procreative systems diseases, and prenatal diseases. Cancer death rate among the old (age>60), adjusted by age distribution, is our primary dependent variable. People older than 60 are divided into six age groups: 60-64, 65-69, 70-74, 75-79, 80-84, and older than 85. Age-group specific mortality rate in a specific death surveillance location is calculated as:

$$Age\ Specific\ Mortality\ Rate_{Age\ group_i} = \frac{100000 \times Death_{Age\ group_i}}{Total\ Population_{Age\ group_i}}$$

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

[16] This dataset was previously used in Ebenstein (2012).

$$Age\ Adjusted\ Mortality\ Rate = \sum_i Population\ Weight_{Age\ group_i} \times Age\ Specific\ Mortality\ Rate_{Age\ group_i}$$

Table 3.1 shows the age-adjusted mortality rate among the senior citizens in China. The average all-cancer mortality rate is 1157 per 100,000 people per year from 2006-2009. Female cancer mortality rate (737 per 100,000) is substantially lower than male cancer mortality rate (1642 per 100,000). The average digestive cancer mortality rate is 724 per 100,000 people, urinary cancer mortality rate is 26 per 100,000 people, and respiratory cancer mortality rate is 373 per 100,000 people. In particular, we calculate liver cancer mortality and stomach cancer mortality, which are 202 and 300 per 100,000 people, respectively.

3.2.3 Precipitation Data

We draw rainfall data from the Global Historical Climatology Network (GHCN) project. GHCN provides average precipitation and temperature in mm for given longitudes and latitudes with the minimum cell size of 0.5 degree by 0.5 degree.

For each county, we identified its coordinates, then calculated a weighted average precipitation using the inverse squared distance as the weights. For example, an interpolated precipitation of grid 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 $Distance_{jk}$ is the distance between j and k.

3.2.4 Control Variables

We collect a rich set of control variables from the statistical yearbooks at the county level: per capita GDP, the share of agricultural output, the share of manufacture output, per capita government expenditure, per capita investment, and number of doctors per capita. The summary statistics of these control variables are reported in Table 3.2.

We match the DSPS data with water quality data by water basins supplemented by distance. That is, we first match water quality data with DSPS data based on whether they locate at the same river basin. If not, we match the DSPS point to its nearest water quality monitoring site.

3.3 Model and Estimation

3.3.1 OLS Regression

A typical panel-data model estimates the following equation

$$y_{it} = \beta_0 + \beta_1 Q_{it} + X_{it}'\delta + v_i + \varepsilon_{it} \quad (1)$$

where y_{it} is cancer death rate among the elderly (age>60) in county i at year t, Q_{it} is the level of water quality in county i at year t. X_{it} is a set of control variables, v_i is county-specific fixed effects and ε_{it} is noises.

Before making the assumptions necessary for estimation, we know that whatever the properties of v_i and ε_{it}, if Equation (1) is true, it must also be true that

$$\bar{y}_i = \beta_0 + \beta_1 \bar{Q}_i + \bar{X}_i' \delta + v_i + \bar{\varepsilon}_i \quad (2)$$

where $\bar{y}_i = \sum_t y_{it} / T_i$, $\bar{Q}_i = \sum_t Q_{it} / T_i$ and $\bar{X}_i = \sum_t X_{it} / T_i$.

Subtracting Equation (2) from Equation (1), we get

$$(y_{it} - \bar{y}_i) = \beta_0 + \beta_1 (Q_{it} - \bar{Q}_i) + (X_{it}' - \bar{X}_i') \delta + \varepsilon_{it} \quad (3)$$

These three equations provide the basis for estimating β_1. In particular, Equation (3) is known as the fixed-effects estimator, and Equation (2) is the between estimator. The random-effects estimator is a weighted average of the estimates produced by the fixed-effects estimator and the between estimator, which is equivalent to estimation of

$$(y_{it} - \theta \bar{y}_i) = (1-\theta)\alpha + \beta_1 (Q_{it} - \theta \bar{Q}_i) + (X_{it}' - \theta \bar{X}_i') \delta + ((1-\theta) v_i + (\varepsilon_{it} - \theta \bar{\varepsilon}_i)) \quad (4)$$

where θ is a function of σ_v^2 and σ_ε^2.

In most cases, the fixed-effects estimator, Equation (3), is preferred, since it removes all permanent bias v_i. The estimation of β_1 utilizes variations on time. That is, β_1 is the effect of changes in water quality on changes in cancer rates. However, since it may take decades for water pollution to cause cancer, the year-to-year variations in water quality may only have limited/negligible impact on cancer death rates. The fixed-effects model unfavorably absorbs all the cross-sectional variations and thus cannot be applied to this (long-term relationship) study.

The between estimator of Equation (2), on the contrary, utilizes the cross-sectional variations and completely discards the over-time variations. To consistently estimate β_1, we require that conditional on $\bar{X}_i$, v_i and $\bar{Q}_i$ are not correlated: $E(v_i, \bar{Q}_i | \bar{X}_i) = 0$. Since the elderly cannot easily migrate to other places in the face of severe water pollution, the water pollution exposure (measured by $\bar{Q}_i$) is exogenous to them. Thus $\bar{Q}_i$ is likely not correlated with other (unobservable) variables v_i. Even though this condition cannot be directly tested, we can check this condition by including a rich set of control variables $\bar{X}_i$: were v_i and $\bar{Q}_i$ correlated, the inclusion of extra control variables $\bar{X}_i$ will change the point estimate of β_1. If including extra control variables don't have substantial impact on the estimation of β_1, it is likely that $\bar{Q}_i$ is not correlated with v_i.

The random-effects estimator of Equation (4) requires the same no-correlation assumption. Compared to the between estimator, the random-effects estimator produces more efficient results. The between estimator is less efficient because it completely discards the over-time information in the data; the random-effects estimator uses both the within and the fixed-effects information.

3.3.2 Instrumental variable Estimator

We compare our results from OLS regressions with that from using the between-estimator instrumental variable (IV) regression. In the IV model, we estimate the following equations

$$\bar{y}_i = \beta_0 + \beta_1 \bar{Q}_i + \bar{X}_i' \delta + \epsilon_i \quad (5)$$

$$\bar{Q}_i = \alpha_0 + \alpha_1 \bar{Z}_i + \bar{X}_i' \gamma + \omega_i \quad (6)$$

In the first stage of IV regression, Equation (6), we estimate how the instrument $\bar{Z}_i$ affect water quality $\bar{Q}_i$. In the second stage, we take the estimated water quality $\hat{Q}_i$ from the first stage into Equation (5). The identification assumption is that $\bar{Z}_i$ affects $\bar{y}_i$ only through its impact on water quality: $E(\bar{Z}_i \epsilon_i) = 0$ and $E(\bar{Z}_i \bar{Q}_i) = 0$.

We use precipitation as an instrument for water quality. Precipitation is one of the most important factors that influence surface water quality. Where rainfall is abundant, surface water quality is typically good. For example, southern China has more rain than northern China. As shown in Figure 2.2, the proportion of the river segments that were seriously polluted (Type IV, V, and VI) is much higher in Northern China rivers basins (Huai River, Hai River, Songhuajiang River, Liao River) than that in Southern China river basins (the Changjiang River basin and Zhujiang River basin).

Precipitation affects surface water quality through two primary channels. First, rain directly dilutes the concentration of water pollutants, and thus improves water quality. Second, rain causes the river to flow faster, which carries away water pollutant quickly and makes the river less prone to eutrophication (Zhong et al., 2005). Arguably, rainfall doesn't cause cancer except through its impact on water pollution.[17]

3.4 Regression Results and Robustness Checks

3.4.1 Main Findings

Our main findings are summarized in Table 3.3. We focus on five measures of cancer mortality rate. "All Cancer" indicates the by age-group (5-year increment) all-cancer mortality rate for the elderly population (age>60), adjusted by age distribution.[18] We investigate digestive cancers and urinary cancers because they are directly related to water pollution. We didn't analyze respiratory cancers because proper controls on air quality are not available at the county level in China.

Digestive cancers include cancers in anus, colon, esophagus, liver, oral cavity, pancreas, pharynx, other oral\pharynx, rectum, salivary glands, small intestine, stomach, and tongue. Urinary cancers include cancers in bladder, kidney, renal pelvis, and ureter, etc. We specifically investigate how water pollution affects liver cancer and stomach cancer because the mortality rates for these two cancers are particularly high.

In Panel A of Table 3.3, we reported the regression results from the between estimator without any controls. Water pollution is statistically significantly associated with all cancer mortality rates at the conventional level. One degree deterioration of water quality is associated with an increase of 8.46 in all-cancer mortality rate for one age group (i.e. 60-65 years old). Since there are six age groups, a rough calculation shows that water quality degrading by one degree will cause 50 more people to die from cancers

[17] For details, please see Ebenstein (2012) and He (2012). Both studies have demonstrated that precipitation is a valid instrument for water quality in China.

[18] Without further clarification, all cancer mortality rates used in this paper are age-group specific age-adjusted death rate per 100,000 people. Weights for age adjustments are based on China's 2000 Census data. The results from crude mortality rate are similar and available upon request.

among the old per 100,000 people. The average cancer mortality among the old is 1157 per 100,000 people, thus one-degree deterioration of water quality will roughly increase cancer mortality rate by 4.3 percent.

We find similar relationship between water pollution and digestive cancer to that of Ebenstein's (2012) study. The effect of water pollution on digestive cancer is statistically significant at 1 percent level. As water quality deteriorates by one degree, the by age-group digestive cancer mortality rate increases by 3.9. By calculation, if water quality becomes one-degree worse, around 25 people will die from digestive cancers among the old, which is equivalent to 3.45 percent increase in digestive cancer mortality rate.

Water pollution also has significant effects on urinary cancers, liver cancer and stomach cancer. The estimated coefficients are 0.187, 1.08 and 1.085, respectively. Correspondingly, the elderly's urinary cancers mortality rate, liver cancer mortality rate, and stomach cancer mortality rate will increase by 4.3 percent, 3.2 percent, 2.2 percent, respectively.

In Panel B of Table 3.3, we report regression results with control variables. The R-square of each regression significantly increases. The estimated coefficients of water pollution slightly decease but are still statistically significant in all specifications.

We include a rural dummy to control for the rural-urban differences. Rural areas have higher cancer mortality rates than urban areas. GDP per capita is negatively associated with all-cancer mortality rate and is statistically significant at 10 percent level. The relationship is not significant for the liver cancer regression. Richer regions have statistically lower digestive, urinary, and stomach cancer mortality rates.

The share of agricultural and manufacture output value measures the structure of local economy. The share of agricultural output has mixed relationships with different cancers. It is negatively associated with all-cancer mortality rate but it is not statistically significant, negatively and significantly associated with digestive cancers and stomach cancer, and positively associated with liver cancer. The relationship between the share of manufacture output and the cancer mortality rate are positive. Thus the higher the share of manufacture output in GDP, the more likely people get cancers.

Government expenditure per capita is negatively associated with cancer mortality rates but the relationship is not statistically significant for stomach cancer. Investment per capita is positively associated with all-cancer death rate, digestive cancer death rate and urinary cancer death rate. In regions where the number of doctors per 10,000 people is high, cancer death rates tends to be high but the relationship is not statistically significant. In Table 3.4, we estimate the same set of regressions separately for males and females. The coefficients for males are always larger than that for females. For digestive cancer urinary cancer and stomach cancer, the coefficients for males are about twice larger as that for females. The results may suggest that males are more likely engaged in risky activities than females.

3.4.2 Robustness Checks

We run a wide array of regressions to check the robustness of our results. First, we run between estimator instrumental variable regressions, where precipitation is used as an

instrument. The results are summarized in Table 3.5. The positive associations between water pollution and cancer mortality rates are statistically significant at 1 percent level in all the regressions.

As water quality decreases by one degree, by age-group all-cancer mortality rate increase by 28.32. If we multiply this number by 6 (age groups) then divide it by the average cancer mortality rate 1157, we conclude that water quality becoming worse by one degree will increase all-cancer mortality rate among the old by roughly 14.7 percent. For digestive cancers, the estimated coefficient is 26.05, suggesting that water quality deteriorating by one level will raise digestive cancer mortality rate by roughly 21.6 percent. Similar calculations show that urinary cancer mortality rate will increase by 4.3 percent, liver cancer mortality rate will increase by 7.6 percent, and stomach cancer mortality rate will increase by 26.1 percent.

Compared to the results from OLS between estimators, the estimated coefficients of water pollution on cancer mortality rates are much larger for some groups in the IV models.

Second, we run regressions using a subsample, where we match water quality data and DSPS data only by river basins. Matching only by river basins reduces the measurement errors for each observation. However, due to data availability, the matching process leaves us with only about half of the observations. The results are presented in Table 3.6. The relationships between water pollution level and by age-group all-cancer mortality rates are still statistically significant. The estimated coefficient is also statistically significant in the digestive cancer regression and the liver cancer regression, but no longer significant in the urinary and stomach cancer.

We also compare our results with that using the 2004 water quality data from the World Bank (same dataset as Ebenstein, 2012). The 2004 water quality data has larger sample size and therefore provides substantial larger cross-sectional variations. However, it still suffers the problem that water quality in 2004 cannot represent the long run water quality in the monitoring sites. For robustness check, we crudely treat water quality from 2006-2009 the same as water quality data in 2004, and match the 2006-2009 DSPS data with water quality data in 2004 by distance. The findings are very similar, as reported in Table 3.7. We find that the estimates are a little bit smaller than that using our matched dataset.

To justify our migration hypothesis, we estimate the relationship between water pollution and by age-group cancer mortality rate using the younger adults (age: 20-50). The results are arranged in Table 3.8. We find that the relationship between water pollution and cancer mortality rates disappears for those younger people. Sometimes the estimated coefficients become negative. The only exception is that cancer mortality rate in urinary system is still significantly positively associated with water pollution.

Lastly, we run a falsification test. We estimate the relationship between water pollution and the mortality rate caused by all other diseases. We find that water pollution is still positively associated with the all-but-cancer morality rate for the elderly people, but the relationship is no longer statistically significant. For the younger adults group, the results are mixed, as shown in Table 3.9.

3.5 Conclusion

We estimate the effects of water pollution on cancer mortality rate among the elderly (Age>60) in China. We focus on the elderly people because their mobility is extremely restricted by the Household Registration System. Their life-time exposure to water pollution is more likely captured by our data in recent years.

We find that water pollution has large, significant, positive effects on by age-group all cancer mortality rate, digestive cancer mortality rate, urinary cancer mortality rate, liver and stomach cancer mortality rate. The results are robust to a variety of specifications, such as including more control variables, using IV estimator, focusing on a subsample, and using other water quality data. We also find that water pollution has no impact on cancer mortality rates for the younger adults (Age: 20-50), which may partially justify our argument that pollution exposure for the younger people cannot be accurately measured because they migrate. Besides, water pollution is not statistically significantly associated with all-but-cancer mortality rates.

Table 3.1 Age-Adjusted Death Rate 2006-2009

	All	Females	Males
All cancer	1157	737	1642
Digestive	724	472	1015
Urinary	26	14	40
Respiratory	373	219	551
Other	34	32	35
Liver	202	125	290
Stomach	300	198	417

Source: Chinese Death Surveillance Points System (DSPS). The sample includes 161 locations selected to represent Chinese population. The reported death rates are the average rates for the 161 locations: each entry in the table is age-adjusted death per 100,000 people per year. Note that all cancer mortality rate for women is much lower because breast cancer data is not included in the sample.

Table 3.2 Summary Statistics

Variable	Obs	Mean	Std.	Min	Max
Rural	620	0.59	0.49	0.00	1.00
GDP per capita (log)	612	9.85	0.90	7.60	11.99
Agricultural industry (%)	600	15.60	14.10	0.36	61.30
Manufacture industry (%)	600	45.34	14.81	11.31	88.76
Government expenditure per capita(log)	616	7.82	0.76	6.12	10.00
Investment per capita(log)	615	8.98	1.13	5.34	11.23
# of doctor per 100000	599	19.98	12.46	1.37	82.83
East	620	0.34	0.47	0.00	1.00
Middle	620	0.32	0.47	0.00	1.00
West	620	0.34	0.47	0.00	1.00

Table 3.3 OLS Regression (Water Basin Match Supplement with Distance Match)

	All Cancer	Digestive	Urinary	Liver	Stomach
Water Pollution	8.462***	3.945***	0.187***	1.080***	1.085**
	(1.555)	(0.998)	(0.0529)	(0.292)	(0.477)
Constant	88.47***	51.54***	1.764***	14.43***	23.03***
	(5.058)	(3.271)	(0.173)	(0.957)	(1.563)
Observations	3,744	3,672	3,672	3,672	3,672
R-squared	0.030	0.017	0.013	0.015	0.006
Number of panel ID	948	930	930	930	930
Water Pollution	6.375***	3.152***	0.164***	0.860***	1.004**
	(1.268)	(0.849)	(0.0516)	(0.232)	(0.421)
Rural	34.82***	27.06***	0.173	5.639***	10.41***
	(8.420)	(5.626)	(0.342)	(1.537)	(2.791)
GDP per capita (log)	-10.43*	-10.29***	-0.576**	1.007	-7.526***
	(5.499)	(3.929)	(0.239)	(1.073)	(1.949)
Agricultural industry (%)	-0.368	-0.671***	-0.00582	0.204***	-0.643***
	(0.252)	(0.178)	(0.0108)	(0.0487)	(0.0884)
Manufacture industry (%)	0.660***	0.358***	0.0104	0.106***	0.129*
	(0.179)	(0.134)	(0.00816)	(0.0367)	(0.0666)
Government expenditure per capita(log)	-10.56**	-3.418	-0.169	-3.515***	1.045
	(4.819)	(3.245)	(0.197)	(0.886)	(1.610)
Investment per capita(log)	9.817**	8.131***	0.476***	0.942	2.211*
	(3.913)	(2.666)	(0.162)	(0.728)	(1.322)
# of doctor per capita	0.353	-0.313	0.0420***	0.0653	-0.226**
	(0.308)	(0.221)	(0.0134)	(0.0603)	(0.109)
Regional dummy	Yes	Yes	Yes	Yes	Yes
Age group dummy	Yes	Yes	Yes	Yes	Yes
Observations	3,492	3,432	3,432	3,432	3,432
R-squared	0.428	0.362	0.217	0.442	0.309
Number of panel ID	918	900	900	900	900

The sample only includes senior Chinese citizens (>60 years old) to minimize the confounding effect of migration. Panel A represents the between estimator regressions of the death rate of a cause on the average water pollution level of the river basin in which the DSPS site is located. If the river basin in which the DSPS site is located does not have a water quality monitor site, we use the nearest water quality monitor site. Panel B adds control variables to the between estimator regressions. The water pollution level measure at each DSPS site reflects the average weekly water quality of the monitoring site in the same river basin. Standard errors are in parenthesis. Asterisks, ** and *, indicate the 1%, and 5% significance level, respectively.

Table 3.4 OLS Regression (Water Basin Match Supplement with Distance Match)

	Female		Male	
	Coef.	S.E.	Coef.	S.E.
All Cancer	5.240***	(0.951)	7.229***	(1.763)
Digestive	2.133***	(0.612)	4.152***	(1.209)
Urinary	0.111**	(0.0473)	0.219***	(0.0809)
Liver	0.710***	(0.185)	1.029***	(0.351)
Stomach	0.515*	(0.295)	1.498**	(0.625)

The sample only includes senior Chinese citizens (>60 years old) to minimize the confounding effect of migration. Each entry represents a between estimator of the death rate of a cause on the average water pollution level of the river basin in which the DSPS site is located. If the river basin in which the DSPS site is located does not have a water quality monitor site, we use the nearest water quality monitor site. All regressions include control variables. The water pollution level measure at each DSPS site reflects the average weekly water quality of the monitoring site in the same river basin. Standard errors are in parenthesis. Asterisks, ** and *, indicate the 1%, and 5% significance level, respectively.

Table 3.5 Instrumental Variable Regression

	All Cancer	Digestive	Urinary	Liver	Stomach
Water Pollution	28.32***	26.05***	0.884***	2.549***	13.09***
	(5.896)	(4.393)	(0.218)	(0.915)	(2.244)
Rural	30.57***	23.35***	0.0564	5.365***	8.455**
	(9.783)	(7.628)	(0.379)	(1.589)	(3.896)
GDP per capita (log)	-10.12	-9.924*	-0.565**	1.034	-7.334***
	(6.348)	(5.306)	(0.264)	(1.105)	(2.710)
Agricultural industry(%)	-0.829***	-1.129***	-0.0202	0.170***	-0.885***
	(0.314)	(0.255)	(0.0127)	(0.0532)	(0.130)
Manufacture industry(%)	0.198	-0.114	-0.00442	0.0714*	-0.120
	(0.239)	(0.201)	(0.0100)	(0.0419)	(0.103)
Government expenditure per capita(log)	-8.201	-2.403	-0.137	-3.440***	1.581
	(5.597)	(4.387)	(0.218)	(0.914)	(2.240)
Investment per capita(log)	8.387*	6.347*	0.420**	0.811	1.269
	(4.533)	(3.615)	(0.180)	(0.753)	(1.846)
# of doctor per capita	0.246	-0.397	0.0393***	0.0590	-0.271*
	(0.356)	(0.298)	(0.0148)	(0.0621)	(0.152)
Regional dummy	Yes	Yes	Yes	Yes	Yes
Age group dummy	Yes	Yes	Yes	Yes	Yes
Observations	3,492	3,432	3,432	3,432	3,432
Number of panel ID	918	900	900	900	900

The sample only includes senior Chinese citizens (>60 years old) to minimize the confounding effect of migration. Each entry represents an instrumental variable between estimator of the death rate of a cause on the average water pollution level of the river basin in which the DSPS site is located. If the river basin in which the DSPS site is located does not have a water quality monitor site, we use the nearest water quality monitor site. All regressions include control variables. The water pollution level measure at each DSPS site reflects the average weekly water quality of the monitoring site in the same river basin. Instrumental variable is the annual precipitation of the DSPS location. Standard errors are in parenthesis. Asterisks, ** and *, indicate the 1%, and 5% significance level, respectively.

Table 3.6 OLS Regression (Water Basin Match Only Without Distance Match Supplement)

	All Cancer	Digestive	Urinary	Liver	Stomach
Water Pollution	4.448***	3.459***	0.00835	0.639**	0.438
	(1.528)	(1.068)	(0.0613)	(0.309)	(0.521)
Rural	-14.37	-1.281	-0.464	-1.085	-4.026
	(10.22)	(7.145)	(0.410)	(2.068)	(3.485)
GDP per capita (log)	-8.076	-13.62***	-0.537**	2.462*	-10.98***
	(6.712)	(4.694)	(0.269)	(1.358)	(2.290)
Agricultural industry(%)	-0.455	-0.585**	-0.00320	0.301***	-0.618***
	(0.325)	(0.227)	(0.0130)	(0.0658)	(0.111)
Manufacture industry(%)	0.442*	0.481***	0.00856	0.105**	0.214***
	(0.242)	(0.169)	(0.00970)	(0.0490)	(0.0825)
Government expenditure per capita(log)	-3.784	-0.820	0.0189	-2.729*	4.654*
	(7.099)	(4.964)	(0.285)	(1.437)	(2.422)
Investment per capita(log)	-3.534	2.683	0.148	-2.002**	0.312
	(4.780)	(3.342)	(0.192)	(0.967)	(1.630)
# of doctor per capita	1.428***	0.304	0.0885***	0.229***	-0.0653
	(0.421)	(0.294)	(0.0169)	(0.0852)	(0.144)
Regional dummy	Yes	Yes	Yes	Yes	Yes
Age group dummy	Yes	Yes	Yes	Yes	Yes
	98.15***	52.04***	17.98***	19.01***	17.98***
Observations	2,004	2,004	2,004	2,004	2,004
R-squared	0.496	0.429	0.223	0.498	0.372
Number of panel ID	522	522	522	522	522

The sample only includes senior Chinese citizens (>60 years old) to minimize the confounding effect of migration. Each entry represents a between estimator of the death rate of a cause on the average water pollution level of the river basin in which the DSPS site is located. If the river basin in which the DSPS site is located does not have a water quality monitor site, the DSPS site is not used in the regression. All regressions include control variables. The water pollution level measure at each DSPS site reflects the average weekly water quality of the monitoring site in the same river basin. Standard errors are in parenthesis. Asterisks, ** and *, indicate the 1%, and 5% significance level, respectively.

Table 3.7 OLS Regression (Ebenstein(2012) Water Quality Match)

	All Cancer	Digestive	Urinary	Liver	Stomach
Water Pollution	4.994***	2.545***	0.137***	0.177	1.156***
	(1.042)	(0.700)	(0.0422)	(0.193)	(0.347)
Rural	36.93***	28.17***	0.312	6.256***	10.75***
	(8.320)	(5.562)	(0.335)	(1.533)	(2.753)
GDP per capita (log)	-11.25**	-10.69***	-0.673***	1.063	-7.694***
	(5.510)	(3.948)	(0.238)	(1.088)	(1.954)
Agricultural industry(%)	-0.230	-0.593***	0.00223	0.226***	-0.620***
	(0.250)	(0.177)	(0.0107)	(0.0488)	(0.0877)
Manufacture industry(%)	0.846***	0.452***	0.0206**	0.127***	0.156**
	(0.178)	(0.135)	(0.00811)	(0.0371)	(0.0666)
Government expenditure per capita(log)	-11.44**	-4.138	-0.153	-3.468***	0.752
	(4.814)	(3.245)	(0.196)	(0.894)	(1.606)
Investment per capita(log)	10.92***	8.726***	0.504***	1.031	2.537*
	(3.892)	(2.655)	(0.160)	(0.732)	(1.314)
# of doctor per capita	0.347	-0.282	0.0430***	0.0597	-0.206*
	(0.311)	(0.223)	(0.0135)	(0.0616)	(0.111)
Regional dummy	Yes	Yes	Yes	Yes	Yes
Age group dummy	Yes	Yes	Yes	Yes	Yes
Observations	3,516	3,456	3,456	3,456	3,456
R-squared	0.431	0.364	0.229	0.436	0.316
Number of panel ID	912	894	894	894	894

The sample only includes senior Chinese citizens (>60 years old) to minimize the confounding effect of migration. Each entry represents a between estimator of the death rate of a cause on the average water pollution level of the river basin in which the DSPS site is located. We use the water quality data in Ebensten (2002). Standard errors are in parenthesis. Asterisks, ** and *, indicate the 1%, and 5% significance level, respectively.

Table 3.8 OLS Regression (20-50 Years Old)

	All Cancer	Digestive	Urinary	Liver	Stomach
Water Pollution	0.0277	-0.0316	0.00736***	-0.0331	-0.0197
	(0.0647)	(0.0409)	(0.00263)	(0.0279)	(0.0150)
Rural	-0.473	0.0368	-0.0276	0.0631	-0.167*
	(0.429)	(0.271)	(0.0175)	(0.185)	(0.0997)
Agricultural population(%)	0.697	0.554	-0.0111	-0.134	0.687***
	(0.730)	(0.470)	(0.0303)	(0.321)	(0.173)
GDP per capita (log)	-0.323	-0.231	-0.00190	-0.0283	-0.163**
	(0.280)	(0.189)	(0.0122)	(0.129)	(0.0696)
Agricultural industry(%)	0.0269**	0.000569	0.000242	0.0149**	-0.0115***
	(0.0128)	(0.00858)	(0.000553)	(0.00586)	(0.00316)
Manufacture industry(%)	0.0181**	0.00271	0.000511	0.00285	-0.00192
	(0.00913)	(0.00647)	(0.000417)	(0.00441)	(0.00238)
Government expenditure per capita(log)	-0.569**	-0.312**	-0.00696	-0.463***	0.161***
	(0.246)	(0.156)	(0.0101)	(0.107)	(0.0575)
Investment per capita(log)	0.307	0.193	-0.00176	0.164*	0.0159
	(0.200)	(0.128)	(0.00827)	(0.0876)	(0.0472)
# of doctor per capita	0.0217	-0.000946	-0.000450	0.000143	-0.00535
	(0.0157)	(0.0106)	(0.000684)	(0.00725)	(0.00391)
Regional dummy	Yes	Yes	Yes	Yes	Yes
Age group dummy	Yes	Yes	Yes	Yes	Yes
Observations	3,492	3,432	3,432	3,432	3,432
R-squared	0.725	0.679	0.155	0.570	0.565
Number of panel ID	918	900	900	900	900

The sample only includes younger Chinese citizens (20-50 years old). Each entry represents a between estimator of the death rate of a cause on the average water pollution level of the river basin in which the DSPS site is located. If the river basin in which the DSPS site is located does not have a water quality monitor site, the DSPS site is not used in the regression. All regressions include control variables. The water pollution level measure at each DSPS site reflects the average weekly water quality of the monitoring site in the same river basin. Standard errors are in parenthesis. Asterisks, ** and *, indicate the 1%, and 5% significance level, respectively.

Table 3.9 Falsification Test for All Death except Cancer (OLS Regression)

	>60 Years Old			20-50 Years Old		
	All	Females	Males	All	Females	Males
Water Pollution	0.575	0.684	0.217	-0.0273	-0.103***	0.0473
	(0.430)	(0.509)	(0.501)	(0.0462)	(0.0396)	(0.0630)
Rural	-0.904	-0.0684	-2.642	-0.340*	-0.0423	-0.571**
	(1.864)	(2.209)	(2.172)	(0.200)	(0.172)	(0.273)
Agricultural population(%)	0.0850	0.0357	0.109	0.000541	-0.00416	0.00422
	(0.0853)	(0.101)	(0.0995)	(0.00917)	(0.00786)	(0.0125)
GDP per capita (log)	0.139**	0.0938	0.195***	0.00191	-0.000301	0.00250
	(0.0607)	(0.0720)	(0.0708)	(0.00652)	(0.00559)	(0.00890)
Agricultural industry(%)	4.532***	-4.494**	-4.823**	0.902***	0.684***	1.100***
	(1.633)	(1.936)	(1.904)	(0.176)	(0.151)	(0.239)
Manufacture industry(%)	0.960	0.928	1.042	-0.0255	-0.122	0.0397
	(1.326)	(1.572)	(1.546)	(0.143)	(0.122)	(0.194)
Government expenditure per capita(log)	0.0934	0.100	0.123	-0.00947	0.0277***	0.00723
	(0.104)	(0.124)	(0.121)	(0.0112)	(0.00961)	(0.0153)
Investment per capita(log)	37.28***	55.55***	22.20***	6.208***	-3.744***	8.576***
	(1.933)	(2.291)	(2.253)	(0.208)	(0.178)	(0.283)
# of doctor per capita	28.12***	47.24***	12.30***	5.907***	-3.509***	8.207***
	(1.933)	(2.291)	(2.253)	(0.208)	(0.178)	(0.283)
Regional dummy	Yes	Yes	Yes	Yes	Yes	Yes
Age group dummy	Yes	Yes	Yes	Yes	Yes	Yes
Observations	3,492	3,492	3,492	3,492	3,492	3,492
R-squared	0.447	0.504	0.296	0.632	0.479	0.635
Number of panel ID	918	918	918	918	918	918

Standard errors are in parenthesis. Asterisks, ** and *, indicate the 1%, and 5% significance level, respectively

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Appendix A: Types of Cancer

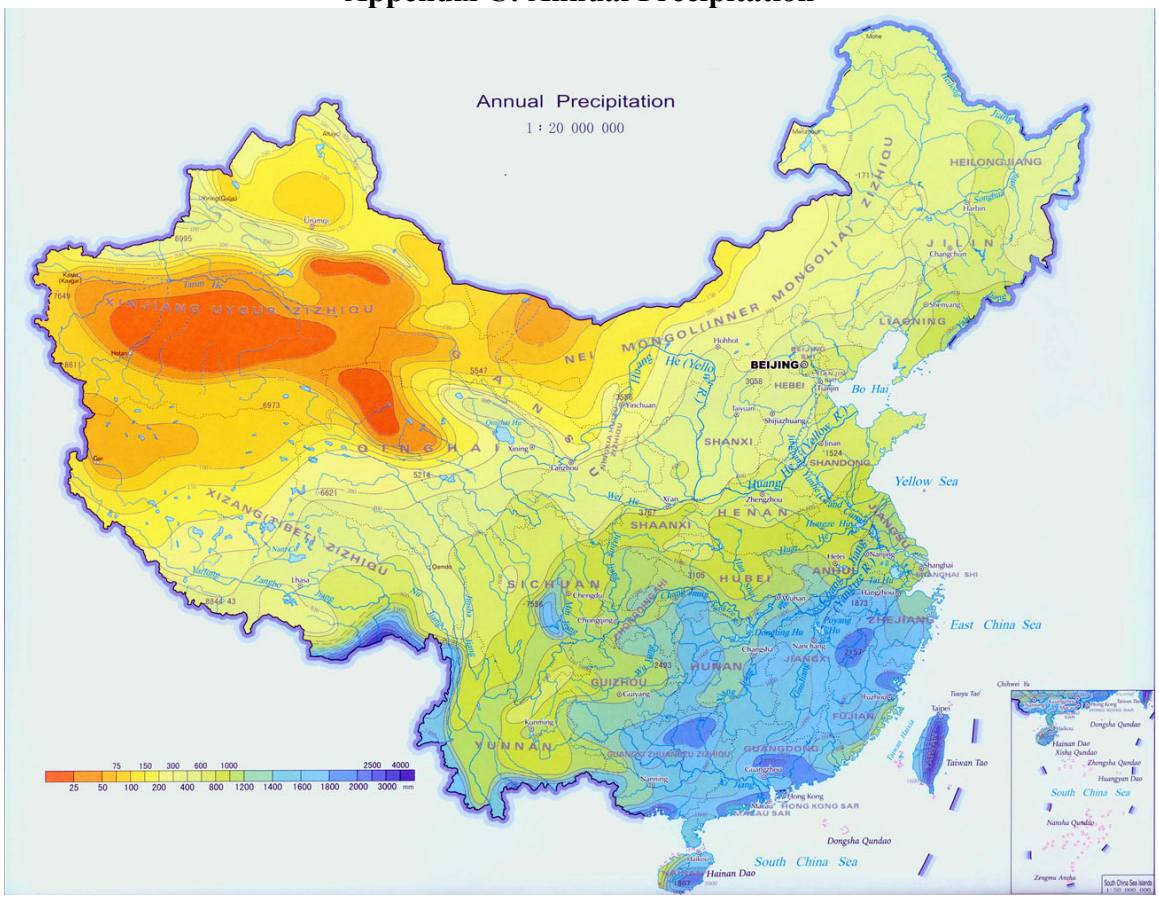
	Cancer Type
1	Bladder cancer
2	Nasopharyngeal cancer
3	Sinus and other cancer (Paranasal Sinus Cancer)
4	Tonsil cancer
5	Lip cancer
6	Gallbladder and other cancer
7	Liver cancer
8	Anal cancer
9	Bone cancer
10	Throat cancer
11	Laryngopharynx cancer
12	Colon cancer
13	Mouth cancer
14	Other oropharyngeal cancer
15	Other urinary organs cancer
16	Other thoracic cavity organs cancer
17	Tracheobronchial lung cancer
18	Tongue cancer
19	Kidney cancer
20	Pelvic cancer
21	Esophageal cancer
22	Ureteral cancer
23	Salivary gland cancer
24	Stomach cancer
25	Small intestine cancer
26	Pharyngeal (parts unknown) cancer
27	Pancreatic cancer
28	Rectal cancer

Appendix B: Water Quality Standard

The water quality classification system is based on Environmental Quality Standard GB3838-2002 in China.

Type I – Mainly applicable to the source of water bodies and national nature preserves.
Type II – Mainly applicable to class A water source protection area for centralized drinking water supply, sanctuaries for rare species of fish, and spawning grounds for fish and shrimps.
Type III – Mainly applicable to class B water source protection area for centralized drinking water supply, sanctuaries for common species of fish, and swimming zones.
Type IV – Mainly applicable to water bodies for general industrial water supply and recreational waters in which there is not direct human contact with the water.
Type V – Mainly applicable to water bodies for agricultural water supply and for general landscape requirements.
Type VI - Essentially useless.

Appendix C: Annual Precipitation



http://www.chinamaps.org/china/china-map-of-precipitation-annual.html