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The Impact of the Crisis-induced Reduction in Air Pollution on Infant Mortality in India: A Policy Perspective

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The impact of the crisis-induced reduction in air pollution on infant mortality in India: A policy perspective[†]

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ABSTRACT

Credible estimates of the health effects associated with changes in air pollution exposure are of considerable importance for research and policy agendas, especially for developing countries. This paper estimates the impact of the sharp reduction in particulate air pollution driven by the Global Financial Crisis of 2008 on district-level infant mortality in India. Utilizing plausibly exogenous geographic variation in the crisis-induced changes in air quality and novel data from household surveys and satellite-based sources, I find that the infant mortality rate fell by 24% more in the most affected districts, implying 1338 fewer infant deaths than would have occurred in the absence of the crisis. Analysis of the mechanisms indicates that the $PM_{2.5}$ reductions affected infant mortality mainly through respiratory diseases and two biological mechanisms: in utero and post-birth $PM_{2.5}$ exposure. Back-of-the-envelope calculations suggest that the estimated decline in infant mortality translates into a three-year after crisis total of 312.5 million U.S. dollars. The resulting health benefits could be used as a benchmark for assessing the effectiveness of the policies designed to improve air quality in India.

Keywords: Air Pollution, Infant Mortality, Crisis, India
JEL Codes: Q53, I12, O13

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1 INTRODUCTION

Air pollution is a grave concern in the developing world, where it kills millions, leads to enormous costs, and constrains economic development (IHME, 2013a; Lim et al., 2013).[1] Despite this, many developing countries avoid committing themselves to the reduction of air pollution because of the natural fear that the economic costs of pollution abatement may outweigh the health benefits (Tanaka, 2015). Thus, measuring the benefits resulting from improvements in air quality has important policy implications. Such measures would allow the evaluation of potential regulations and ensure that their costs are justified. However, empirical studies estimating health benefits associated with reductions in air pollution in developing countries are still scarce (Arceo, Hanna, & Oliva, 2015; Tanaka, 2015; Heft-Neal et al., 2018).

This paper addresses this literature gap by quantifying the impact of air pollution reductions on infant mortality in India.[2] Specifically, it takes advantage of the economic slowdown caused by the Global Financial Crisis of 2008 and exploits the episode of synchronous decline in industrial production, reduction in air pollution, and improvement in infant mortality.[3] The specific questions this paper addresses are whether the crisis-induced reductions in air pollution caused a decline in infant mortality, and what the transmission channels are through which reductions in air pollution affect infants' health. I also examine the benefits of the decline in infant mortality resulting from the improvement in air quality.

India provides a compelling setting for this study for several reasons. First, two decades of industrialization and rapid economic growth led to severe air pollution in India. Of the 20 most polluted cities in the world, 13 are located in India, including the capital city Delhi, which is ranked as the most polluted (Greenstone et al., 2015; WHO, 2014d). India has the world's highest proportion of deaths caused by respiratory diseases (WHO, 2014e). The Global Burden of Disease ranks air pollution as the second leading health risk factor in India (IHME, 2013b). Second, despite the severity of the Global Financial Crisis, India escaped a full-scale recession and suffered instead from the delayed second-order effects that led to a temporary economic downturn. According to the Index of Industrial Production (IIP), the output of the integrated industrial sector in India hit a record low of -

[1] Of the 7 million annual deaths linked to air pollution, 5.9 million occurred in low and middle-income countries of South-East Asia and the Western Pacific (WHO, 2014a). Newborns and infants are particularly vulnerable to air pollution exposure. Around 6.3 million children under the age of five died in 2013, of which 70% and 41% were infants and newborns. About half of under-five deaths were concentrated in just five countries of Africa and South-East Asia, including India with a share of 21% (WHO, 2011, 2014b). The prime cause of these deaths is respiratory diseases attributable to air pollution (WHO, 2014c). The costs of premature mortality caused by the exposure to particulate matter and ozone in 2013 translated into $5.11 trillion and $225 billions of global losses in total welfare and forgone labor income. Developing countries, mostly in Africa, East and South Asia, incurred the record high losses equivalent to up to 9% of the country's GDP (WB, 2016). India's annual GDP growth was 6.6% in 2013 (WB, n.d.) implying that the pollution-related losses could have offset the whole year of the country's economic development. If no abatement policies are implemented, the number of premature deaths due to exposure to just such air pollutant as particulate matter (PM) will likely more than double, mostly because of an increasing number of deaths in China and India (OECD, 2012).

[2] Infant mortality is defined as the death of children under one year old.

[3] I exploit the economic slowdown caused by the Global Financial Crisis of 2008 rather than environmental regulations as a natural experiment. This is because environmental regulations in developing countries, even if they are designed similarly to those in the developed ones, often involve implementation problems that complicate the estimation of the effect of interest (Arceo et al., 2015; Duflo, Greenstone, Pande, & Ryan, 2013, 2018).

7.20% in March 2009, from about 20% of its pre-crisis level (MOSPI, n.d.). Also, the average contraction of trade was around 20% during the period from October 2008 to September 2009 (EAC, 2009; Kumar & Alex, 2009). Third, the contribution of the global financial turmoil to the reductions in air pollution in the U.S. and Europe is well documented (Davis et al., 2010; Castellanos & Boersma, 2012; Vrekoussis et al., 2013), but it is still understudied in the case of India. Taken together, the substantial drop in IIP and the dominant share of the manufacturing and energy sectors in the index (78% and 8%, MOSPI, n.d.) imply that the economic slowdown affected areas in India differentially, based on the pre-crisis industrial structure and industry-specific pollution intensities. As I demonstrate below, this led to substantial reductions in air pollution in some areas, but not in others. Indian districts with larger shares of the manufacturing, mining, construction, or energy sectors experienced a more substantial decline in air pollution than districts without these pollution-intensive sectors. Altogether, such a setting allows me to study the relationship between air pollution reductions and infant mortality at greater pollution concentration levels and to do so using a credible quasi-experimental approach.

To implement the analysis, I combine state-of-the-art satellite-based estimates for annual concentrations of fine particulate matter ($PM_{2.5}$)[4] with survey-based household information on nearly 2 million births and 150 thousand deaths and their determinants for 284 districts across 9 states during 2007-2011. I use a quasi-experimental difference-in-differences approach in an attempt to isolate the role of the reductions in $PM_{2.5}$ pollution from other confounding factors that affect infant mortality. This approach exploits both the timing of the crisis and its differential effect across districts, depending on their pre-crisis industrial specialization. However, using the crisis as a source of treatment variation exposes this approach to two conceptual challenges: unknown timing of the crisis-induced effects on air pollution and sorting the districts into treated and control groups. I overcome these challenges by exploiting such methodological innovations as a time-series econometric test for structural trend break and a spatial Hot Spot Analysis. Based on the results of these analyses, my preferred specification compares pre- vs. post- 2010 levels and trends in infant mortality rates between treated and control districts. The key identifying assumptions are that, conditional on district-specific trends, any pre- versus post-2010 changes in infant mortality rates caused by factors besides air pollution are the same for the treated and control districts, and that air pollution is the only factor differentially affecting the treated districts beginning in 2010.

Answering the first question, I find that the crisis-induced reductions in $PM_{2.5}$ pollution led to a statistically significant decline in district-level infant mortality rates. Regression coefficients indicate that the infant mortality rate in the treated districts fell by about 24% more than in the control districts

[4] The United States Environmental Protection Agency [U.S. EPA] defines particulate matter (PM) as "a complex mixture of extremely small particles and liquid droplets that get into the air" (U.S. EPA., n.d.a). Particulate air pollution can be categorized in a number of ways, including size and sources of emissions. Size is an important indicator of the particles' penetrating ability, which highlights the most probable region of the respiratory tract where inhaled particulates could be deposited. By this criterion, particulate air pollution can be broken down into total suspended particles with an aerodynamic diameter of less than 100 μm, coarse or inhalable (less than 10 μm in diameter), fine or respirable (smaller than 2.5 μm) and ultra fine (less than 0.1 μm). Particulate matter can be originated from anthropogenic (human-made) or natural sources. The former sources include industrial activity, transport exhaust, power generation, household heating, cooking and fuel combustion, while the latter add sea salt, dust, volcanic and fire ash (Van Donkelaar et al, 2010; Van Donkelaar et al., 2016).

between pre- and post-crisis periods. The estimates are robust to a variety of specifications and falsification tests. Addressing the second question, I examine the impact of the changes in $PM_{2.5}$ concentrations on the mortality of infants at different ages and from various diseases. My findings suggest that the $PM_{2.5}$ reductions affected infant mortality mainly through respiratory diseases and two biological mechanisms: in utero and post-birth $PM_{2.5}$ exposure. Finally, I use the quantified relationship to measure health benefits and monetary gains from the crisis-induced episode of $PM_{2.5}$ pollution reduction. My calculations suggest that 1338 infant lives were saved, implying a contribution of 11% to the overall decline in infant mortality during the post-crisis period and leading to monetary benefits of $312.5 million.

The research design used in this paper allows me to overcome or substantially mitigate some of the frequent empirical challenges of the endogeneity of air pollution exposure. First, the temporary nature of the economic crisis in India allows me to address one of the major causes of endogeneity – residential sorting.[5] In my research design, it is unlikely that households migrate in search of new employment or because of their preferences for better air quality in the short crisis time frame. The limited geographical mobility of infants and pregnant women also helps to alleviate this threat to identification. Second, since the reduction in air pollution concentrations was caused simultaneously by global and nationwide phenomena, unobserved behavioral changes within the treatment group that could also affect health and invalidate research design are unlikely in this paper's settings. In addition, the control group of districts accounts for any common responses to the crisis. Finally, it is also critical to control for other changes accompanying the crisis, including changes in per capita income and meteorological confounders. I address these challenging issues in the study.

The paper builds on economic and epidemiological literature that uses quasi-experimental designs to quantify the causal relationship between various health outcomes and reductions in air pollution. Prominent epidemiological studies by Pope (1989), Pope, Schwartz, and Ransom (1992), Ransom and Pope (1995), Parker, Mendola, and Woodruff (2008) exploit closure and reopening of a steel mill in the Utah Valley to show that improvements in air quality are associated with the decline in respiratory morbidity, mortality, and preterm births. Related studies from economic literature by Lavaine and Neidell (2013), Currie et al. (2013), and Hanna and Oliva (2015) also estimate the health effects from the variations induced by operational distortions of specific polluters, oil refineries or toxic plants, in both developed and developing countries. However, these studies rarely exploit recession as a source of exogenous variation, with the remarkable exception represented by Chay and Greenstone (2003b), who link changes in infant mortality to the reduction in total suspended particles (TSP) across U.S. counties caused by the U.S. 1981-1982 recession. [6] They show that a 1% reduction in TSP resulted in a 0.35-0.45% decline in infant deaths at the county level. Other quasi-experimental

[5] Residential sorting is the optimizing behavior of individuals choosing residential locations based on attributes, including air quality, that can lead to the non-random assignment of air pollution (Graff Zivin & Neidell, 2013; Currie et al., 2014).

[6] Sanders (2012) investigates the relationship between early-life exposure to air pollution and long-term outcomes (Currie et al. 2014). Similar to Chay and Greenstone (2003b), the author uses the U.S. 1981-1982 recession and the related decline in manufacturing employment as a source of variation to estimate the impact of the reduction in fetal TSP exposure on educational outcomes in Texas. Sanders (2012) finds that a one standard deviation decline in TSPs around the time of students' birth increases high school test performance by 6% of standard deviation.

studies focus on regulation-induced changes in air quality. This strand of literature benefits from the contribution by Chay and Greenstone (2003a) and Sanders, Barreca, and Neidell, (2020) for the U.S. and Luechinger (2014) for Germany. Until recently, much of what we knew from the literature came from the developed countries. However, as these countries are generally wealthier, have much lower mortality rates and air pollution concentrations, the transferability of estimates from developed to developing countries remained unvalidated in most cases. Currently, a growing literature provides causal evidence on the effects of environmental policies on infant mortality in developing countries: Greenstone and Hanna (2014) for India, Ebenstein et al. (2015) and Tanaka (2015) for China, and Cesur, Tekin, and Ulker (2016) for Turkey. My paper builds on the successful design of the previous studies and contributes to overcoming the scarcity of studies that link infant mortality and reductions in air pollution in developing countries, using a different quasi-experimental setting. Additionally, the paper estimates the health benefits of reducing air pollution, which could be used as a benchmark to assess potential policies designed to improve air quality.

2 DATA

To implement the analysis, I constructed a panel of district-by-year data on infant mortality, mortality-related controls, fine particulate matter, and confounding factors for 2007-2011. Raw data are from a variety of survey-based and satellite-based sources.

Mortality data

Data on infant births and deaths came from the Annual Health Survey (AHS) of India. The AHS is the first population-representative longitudinal demographic survey in India designed to collect health-related information at the district level, with the infant mortality rate taken as the decisive indicator for the sample size. The survey structure corresponds to the typical structure of demographic and health surveys (DHS) conducted in many low- and middle-income countries.

The AHS is a sub-national survey that covers 284 districts across 9 states from 2007 to 2011 (Figure 1). These districts are a particularly relevant study area. They represent nearly 50% of the overall population and account for 60% of all births and 70% of all infant deaths in the country. The AHS was conducted during 2010-2013 in three consecutive rounds and four schedules, specifically House-listing, Household, Woman, and Mortality. Each round recorded health-related information at the individual and household levels for 12 months before the survey was taken. A representative sample of 20694 Primary Sample Units, selected based on a uni-stage (two-stage in cases of larger rural villages) stratified simple random sample without replacement, covered around 20.6 million individuals and 4.3 million households (Census of India, n.d.). I downloaded the AHS data from the Health Management Information System, a digital initiative of the Ministry of Health & Family Welfare, Government of India (HMIS, n.d.).

Overall, my sample includes 1,883,456 individual births and 148,398 deaths. The outcome of interest for this study is the infant mortality rate (IMR), which is conventionally expressed as the number of

infant deaths per 1000 live births. I derived information on the number of deaths within one year of life from the Mortality schedule and further aggregated the number at the district-by-year level. As the numerator for infant mortality rate I used the total number of infant deaths due to all causes, within one day, within 28 days, between one and eleven months, and between eleven months and one year. I collected data on the total district-by-year number of live births for the denominator from the Woman schedule reporting the outcomes of pregnancies. I further disaggregated the total number of deaths using information on different symptoms of death pertaining to the deceased infants. I also use a perinatal or a stillbirth mortality rate as the outcome variable. This measure of mortality is computed as the number of stillbirths or fetal deaths per 1000 total live births and stillbirths combined. The average annual infant mortality rate for all causes is 87.4 per 1000 live births.

Mortality-related controls

The Mortality and Woman AHS schedules are the primary sources of the mortality-related controls. From the former schedule, I derived three groups of control variables: characteristics of the deceased infants, characteristics and habits of the infants' Heads of the households (HH)[7], and deceased infants' household characteristics. These variables include a percentage of male infants, share of infant deaths in rural areas and average birth order; the share of the male HHs, percentage of the HHs affiliated with social groups, including scheduled castes and scheduled tribes, HHs' educational qualification, religion and occupation, as well as the percentage of HHs smoking and drinking alcohol; the percentage of houses with filtered water, different sources of lightning, type of cooking fuel used, whether households cook inside the house and use open defecation as a toilet facility. The purpose of these controls is to capture the effects of either changes in indoor air pollution or potential sources of deadly infectious diseases, for example, malaria.

Some of the district-specific attributes and indicators of the utilization of medical services by mothers and infants were extracted from the Woman schedule. The controls from this survey are the average number of births and population, average age of mothers and percentage of those married. Indicators of the utilization of medical services by mothers and infants include percentage of mothers who did not receive any ante natal care during pregnancy, percentage of deliveries at the government medical facilities, share of newborns who did not receive any checkups after birth and percentage of babies who received any vaccination. These variables highlight the importance of the medical services in saving infant lives.

Pollution data

Satellite-derived data for the construction of the main variable of interest, the annual district-level average $PM_{2.5}$ concentrations, were obtained from the Atmospheric Composition Analysis Group (ACAG) at Dalhousie University. The data represent global gridded datasets of annual bias-corrected average surface $PM_{2.5}$ concentrations at 0.01º x 0.01º spatial resolution (1 x 1 km at the equator)

[7] I use characteristics and habits of the deceased infants' Heads of the households as a proxy for parental characteristics.

estimated by combining Aerosol Optical Depth[8] retrievals from multiple satellite sources (MODIS, MISR, SeaWIFS) with simulations in the GEOS-Chem chemical transport model, subsequently calibrated against ground-based monitor data using geographically weighted regressions (Van Donkelaar et al., 2016; ACAG, 2016). AOD-based $PM_{2.5}$ estimates are widely considered as a good proxy of air pollution over India (Dey et al., 2012). I downloaded ArcGIS-compatible files with dust and sea-salt removed estimates, which allowed me to focus on anthropogenic, human-made particulate air pollution. $PM_{2.5}$ concentrations were calculated by taking averages of annual mean concentrations at all grid points within districts' administrative boundaries overlying the ACAG gridded $PM_{2.5}$ data using the ArcGIS platform. I downloaded shapefiles with districts' boundaries for such computations from the Global administrative areas [GADM] (2015) spatial database.[9] The average annual $PM_{2.5}$ concentration in my sample during the study period is 54.4 $\mu g/m^3$.

Economic data

Controlling for cross-districts differences in income changes during the crisis is important to mitigate potential confounding bias. However, official district-level data on income per capita do not exist. I thus constructed a proxy for this confounder using satellite-derived nighttime lights imagery. Evidence suggests that nighttime lights expressed in the form of a natural logarithm adequately explain GDP at the district-level for India (Chaturvedi, Ghosh, & Bhandari, 2011; Bhandari & Roychowdhury, 2011). I obtained nighttime lights satellite images from the repository at the National Geophysical Data Center (NGDC) of the National Oceanic and Atmospheric Administration (NOAA). These images were captured by the Operational Linescan System sensor onboard the Defense Meteorological Satellite Program satellites. The values of the pixels from the stable lights data show brightness in Digital Numbers and are cleaned from ephemeral lights from fires, gas flares and other similar events (NGDC, n.d.). Using ArcGIS, I first sum all lit pixels within the GADM districts' boundaries for each year as suggested by Lowe (2014). Then, relating the sums obtained to the district-level population and taking log-transformation, I derived a measure of the natural logarithm of the district-level GDP per capita.

District-level population data were retrieved from the world's gridded population count dataset for 2000, 2005, 2010 and 2015, obtained from the Center for International Earth Sciences Information Network (CIESIN) at Columbia University. The population count grids are consistent with national Censuses and population registers and contain estimates of the number of persons per grid cell (CIESIN, 2016). To construct a district-by-year population, I summed the number of persons in the cells within the overlaid GADM districts' boundaries. For missing years, the population was imputed by linear interpolation. I also use the CIESIN's population data to weight regressions and compute population-weighted dimensions of the variables.

[8] Aerosol Optical Depth measures the amount of sunlight absorbed, reflected, and scattered by the particles suspended in the air. Satellite observations of AOD make it possible to estimate surface $PM_{2.5}$ concentrations at granular spatial resolution and with comprehensive geographical and temporal coverage.

[9] I adjusted districts' borders in the GADM shapefiles so that they correspond to the districts' administrative boundaries as they were in 2001. As a reference, I used maps of the AHS districts downloaded from the Census of India website (Census of India, n.d.)

Weather data

As atmospheric conditions influence both air pollution and health, meteorological covariates are also potential confounders in my study. Addressing this concern, I control for temperature, precipitation, wind direction and speed. I use gridded datasets of average monthly temperature and precipitation from the Climatic Research Unit (CRU) at the University of East Anglia (Harris at al., 2014; CRU, 2017). The raw monthly means gridded data for u-wind (west-east), v-wind (south-north) vectors and wind speed were obtained from the NOAA's NCEP/NCAR Reanalysis 1 (Kalnay et al.,1996). By analogy to air pollution, I processed raw data in the ArcGIS to construct annual average air temperature, precipitation, wind directions and speed at the district level.

Descriptive statistics and data insights

Table 1 presents descriptive statistics for the districts from Figure 1. The table shows that the reduction in district-level $PM_{2.5}$ pollution during 2009-2011 is visibly larger than the changes in the majority of other variables during the same period.

Figure 2 illustrates the evolution of the district-level annual mean concentration of $PM_{2.5}$ in the study area for 1998-2015. Two observations deserve closer attention. First, air quality has been deteriorating continuously during the last two decades. The $PM_{2.5}$ level increased from an average of 43 $\mu g/m^3$ in 1999 to more than 60 $\mu g/m^3$ in 2015, a change of almost 40%. The worsening of air quality during this period could obviously be associated with rapid economic growth during the pre-crisis wave of globalization, accompanied by industrialization and urbanization, as well as a fast-growing population and deterioration of the natural environment (CPCB, 2014). Second, the figure documents two episodes of abrupt reduction in $PM_{2.5}$ concentrations, 2005-2006 and 2009-2012, followed by the comparably sharp reversals of the trends. The timing of the first episode is somewhat unfortunate for this study as it is close in time to the period of interest. In the next section, I conduct a formal test to ensure that my findings are not related to this period.

Improvement in air quality during the 2009-2012 episode is the focus of my study. The $PM_{2.5}$ curve does show a change in its trend around the alleged outbreak of the Global Financial Crisis. After reaching its record high maximum in 2008 at 58 $\mu g/m^3$, fine particulate air pollution fell by almost 9 $\mu g/m^3$, slightly above 15%, making improvement in air quality during this episode the largest for the entire 1998-2015 interval. This downward trend in $PM_{2.5}$ pollution was offset by the steep reversal during 2013-2015, when average $PM_{2.5}$ concentrations reached a record high of 60.25 $\mu g/m^3$, representing an increase of about 23%. This period coincides with the accelerating recovery of the Indian economy and its transit from volatile to stable real GDP growth (IMF, 2016).

Figure 3 compares kernel density estimates of the annual mean $PM_{2.5}$ distributions across the districts for 2008, 2012 and 2015, representing pre-crisis, crisis and post-crisis year-end points. Panel A demonstrates that the entire distribution shifted substantially to the left in 2012 compared to 2008. In contrast, Panel B documents a shift of the distribution to the right again in 2015. Panel A of Table 2 provides summary statistics for these changes. It demonstrates that the 2009-2012 improvement episode was remarkable in several aspects. While the mean $PM_{2.5}$ level declined by more than 15%,

the tenth percentile of the distribution as well as observed minimums remained unchanged. However, the drop in the ninetieth percentile was particularly noteworthy with a decrease of about 14 $\mu g/m^3$, more than 17%. The shift in observed maximums by almost 44 $\mu g/m^3$, representing 36%, is especially striking. During the post-crisis period, the sharp reversal of the improvement trend led to a substantial deterioration in air quality that was comparable to the pre-crisis period.

Taken together, Figure 3 and Panel A of Table 2 support the initial hypothesis that districts with high pre-crisis levels of air pollution likely experienced more substantial improvement in air quality than districts with initially low pollution concentrations. Figure 4 provides an overview of the spatio-temporal distributions of annual mean $PM_{2.5}$ concentrations across the study area for 2008, 2012 and 2015, which visually support this conclusion. Panel B of Table 2 relates changes in $PM_{2.5}$ concentrations from Figure 4 to population exposure, providing suggestive evidence that improvements in infant mortality could be more pronounced in districts with high pre-crisis levels of air pollution.

Figure 5 illustrates the evolution of the district-level annual means of $PM_{2.5}$ air pollution and the infant mortality rate during 2007-2011. The infant mortality rate followed a similar pattern to that of air pollution. The IMR increased to achieve its highest rate by 2009. Then, during the following two years, 2010-2011, the infant mortality rate decreased sharply from about 112 to 65 deaths per 1000 live births, an unprecedented 42%, and supposedly continued this path till the end of the time frame of the crisis in 2012. Further, Table 1 indicates that while the number of infant deaths from all causes declined substantially after 2009, the number of births remained almost unchanged. This implies that the decline in the IMR was likely driven by the substantial reduction in the number of deaths during the period, which I relate to the crisis-induced decline in air pollution. As analysis of different death categories suggests, the dynamics observed in the total number of deaths was caused mainly by the reduction in early neonatal and postneonatal mortality.

Both data series presented in Figure 5 provide visual evidence of structural breaks, marked by the dashed lines, and reversals in upward trends, beginning after 2008 and 2009, respectively for air pollution and mortality. Albeit with a time lag, both breaks correspond well to the time frame of the crisis, cautiously suggesting the presence of a direct relationship within the crisis-pollution-mortality nexus.

3 EMPIRICAL STRATEGY

This section introduces the empirical strategy that I use to answer the first research question. Specifically, in an attempt to isolate the causal relationship between the crisis-induced reductions in $PM_{2.5}$ and the infant mortality rate, I use a quasi-experimental difference-in-differences (DID) technique.

Standard model

The standard DID model in a two-way fixed effect regression framework is as follows:

$$\log (IMR)_{dt} = \alpha + \delta_1(Treated_d \cdot Post_t) + \beta_1 W_{dt} + \beta_2 X_{dt} + \mu_d + \gamma_t + \varepsilon_{dt} \qquad (1)$$

where $\log (IMR)_{dt}$ denotes a natural logarithm of the infant mortality rate[10] in district d and year t. $Treated_d$ is an indicator variable for whether district d belongs to the treatment group; $Post_t$ is an indicator variable for the years after a specific year τ_0, indicating a post-crisis time period. I delve into the more precise definition of the latter two variables further below. W_{dt} is a set of district-level meteorological covariates; X_{dt} is a set of observable time- and/or district-varying controls for a set of covariates in the mortality-pollution nexus. μ_d are district fixed effects that capture time-invariant heterogeneity between treated and control districts; γ_t are year fixed effects controlling for the year-specific common shocks for both types of districts; ε_{dt} are idiosyncratic error term, robust and clustered at the district level to account for serial correlation between districts over time (Bertrand, Duflo, & Mullainathan, 2004; Wooldridge, 2003). To account for differences in the size of the districts, equation (1) is weighted by the district-level population.

The coefficient of interest, δ_1, captures the difference between the districts from the treatment and control groups in changes in $\log (IMR)_{dt}$ before and after the crisis-induced decline in $PM_{2.5}$ pollution. If the crisis-induced reductions in air pollution contributed to a more substantial decline in infant mortality in the districts from the treatment group than those from the control group, $\widehat{\delta_1}$ will be negative. The interpretation of the coefficient would be that the crisis-induced reductions in $PM_{2.5}$ pollution are associated with a $100 \cdot (e^{\widehat{\delta_1}} - 1)$ percent lower infant mortality rate in the treated districts than in the control districts between pre- and post-crisis periods.

Using the crisis as a natural experiment exposes this empirical strategy to two conceptual challenges: unknown timing of the crisis-induced effects on air pollution (variable $Post_t$), and sorting of the districts into the treatment and control groups (variable $Treated_d$).

Timing of the crisis-induced effects

To address the first challenge, I associate the timing of the crisis-induced effects on air pollution with the break in the upward trend of $PM_{2.5}$ concentrations that occurred in a particular year. Then, this year can be considered as the year of critical changes in air pollution caused by the crisis and can be used to divide the whole period of interest into pre- and post- crisis intervals. Even though Figure 2 provides visual support that $PM_{2.5}$ pollution does indeed show a trend break around 2008[11], the timing of the effects of the crisis on air pollution requires more credible justification.

Therefore, I perform a time-series econometric test for a structural trend break, specifically supremum

[10] The reason for modeling infant mortality rate in a log-form is as follows. I hypothesize that the crisis-induced changes in air pollution could have had proportional effects on infant mortality. Specifically, districts with initially higher mortality rates could experience a larger decline in the level of mortality, due to changes in air pollution concentrations, than the districts with an initially lower rate. Using proportional changes also facilitates between-districts comparisons.

[11] I assume that this year can be considered as the first year when the crisis could potentially affect air pollution.

Wald and likelihood-ratio (LR) tests designed for cases when the breakpoints are unknown (Andrews, 1993, 2003; Hansen, 1997). The idea is to determine a statistically significant trend break in the aggregated average $PM_{2.5}$ pollution time series and check whether it corresponds to the initial point of the global financial crisis around 2008. Finding a statistically significant break in proximity to the alleged starting point of the crisis would suggest that the crisis might have had an impact on the level of particulate air pollution. Exploiting supremum tests for the purpose of finding structural breaks in time series was shown to be a reliable in contexts similar to that of this study and was adopted by economists in a number of papers (Piehl et al., 2003; Jayachandran, Lleras-Muney, & Smith, 2010; Greenstone & Hanna, 2014).[12]

I test for the structural break in $PM_{2.5}$ pollution time series in the year of the possible breakpoint, τ, using a model similar to Jayachandran et al. (2010):

$$\Delta PM_{t,t-1} = \alpha + \delta_0 D_t(\tau) + \varepsilon_t \quad (2)$$

where $\Delta PM_{t,t-1}$ is the first difference in the $PM_{2.5}$ pollution time series[13]; $D_t(\tau)$ is an indicator variable equal to zero for the years before τ and equal to one for those after τ; ε_t - robust standard errors.

Formally, sup Wald and LR tests are applied sequentially to test for constancy in the coefficients from the regression of model (2) with τ taking on each year within the interval of possible trend breaks, a test window, and calculate the *W*- and *F*-statistic associated with the null hypothesis of no trend break, $\delta_0 = 0$, for each tested year. The test window is shorter than the whole time series. For the test not to be misleading, it should have enough data points before and after the test window to estimate regressions before and after the breakpoint (Andrews, 1993; Piehl et al., 2003; Jayachandran et al., 2010; Greenstone & Hanna, 2014). I test for the single possible break in an eight-year test window, including a range of years in the 2004-2011 interval. Given quite a short time series, this is the maximal length of test window I could allow; it corresponds to a symmetric trimming of the pollution time series by 25%[14]. The test then selects the maximal among the resulting test statistics to define the best possible breakpoint, τ_0, and returns the associated *p*-value to gauge the significance of the detected break. Since the test statistics do not converge to any known distribution, the reported *p*-values are calculated by the method introduced in Hansen (1997).

[12] Several reasons make application of this technique in our research attractive. Firstly, both tests are robust to heteroscedasticity and overcome limitations inherent in the traditional Chow test that assumes homoscedasticity. Secondly, as Piehl et al. (2003) summarize, the intuition of sup Wald and LR tests is appropriate in the program evaluation context, the purpose similar to our aims in that the effect of the crisis can be treated in a way similar to the effect of a policy intervention. Finally, formal testing improves earlier attempts undertaken by Chay and Greenstone (1999, 2003b), Sanders (2012), Tanaka (2015) to overcome the same difficulties in the similar settings.

[13] The reason for using the first difference of the dependent variable is that by doing so I achieve stationarity of air pollution time series. To be valid, supremum tests require data to be stationary (Andrews, 1993; Piehl et al., 2003), a condition that my time series does not satisfy. Both Augmented Dickey-Fuller and Phillips-Perron tests fail to reject the null hypothesis of nonstationarity; however, they do reject the null in the case of the first-differenced series.

[14] For comparison, a common approach suggested by Andrews (1993) is to trim 15% from both ends. However, it is common to select a trimming percentage up to 49%.

Figure 6 and Table 3 present the results of the tests for structural break on an unknown year.[15] Both supremum tests identify structural breaks within the 2009-2012 air quality improvement episode, thus associating them with the respective reversal of the upward trend in $PM_{2.5}$. Sup Wald reports 2010 as a year of statistically significant break, while sup LR selects 2009 as a break year, although insignificant[16]. Panel A of Table 3 shows that whenever 2010 is included in the test window, the maximal *W*-statistics are concentrated at this year, and the null hypothesis can be rejected at the 1 percent level. When tested by the sup LR, the same applies to 2009 except that neither of the *F*-statistics is significant. As another specification test, in addition to different lengths of test window and trimming percentages, I test for possible trend breaks in the parameters after estimation of the log form of the model (2). The results are robust to different ranges of possible break years, trimming, or log-level model specifications. Panel B of Table 3 shows that neither of the years within the 2005-2006 interval, or the years of the largest pre-crisis drop in $PM_{2.5}$, are trend break years. This finding relaxes my previous concern about the possible confounding role of these years in my results. Thus, I consider 2010 in further analysis as the time of the effects of the crisis and on air pollution, τ_0, and the most important year when the crisis could affect air pollution in the sample districts.

Selection of the treatment and control groups

Addressing the second challenge, I designate districts with large improvements in air quality during the 2009-2012 improvement episode, those most impacted by the crisis, to the group of the treated districts, while districts with small or no changes, unaffected or least affected, are designated to the control group. I use several approaches that, nevertheless, lead to a very similar result.[17]

Panel B of Figure 7 demonstrates a geographical distribution of the district-wise changes in average $PM_{2.5}$ during 2009-2012. For comparison, Panel A illustrates the spatial variation in the pre-crisis levels of $PM_{2.5}$ pollution. The following observations are noteworthy. First, the two maps correlate very well visually. Thus, the levels of average $PM_{2.5}$ before the crisis could potentially be a good predictor for the effects of the crisis-induced reduction in air pollution. Second, in contrast to my expectations, some of the districts experienced worsening of air quality. Independent of the sign of the changes, these districts should be taken into account similarly to those with reductions in air pollution. Third, the variation in the magnitude of the crisis-induced changes in the $PM_{2.5}$ levels varied substantially across districts with a reduction or increase in air pollution. These changes are significantly larger in the former group and vary from zero to a substantial 45 $\mu g/m^3$ or almost 10 $\mu g/m^3$ on average. In the latter group, the maximum and average values of the increase in the level of fine particulate pollution are slightly above 8 $\mu g/m^3$ and 4 $\mu g/m^3$, respectively. Finally, it may well

[15] I also perform a Chow-type test for structural trend break on a known year (Chow, 1960). Using the same model and data, I construct a heteroscedasticity robust Wald statistic to test the null hypothesis of no trend break for each year within the same test window, separately. Thus, I pretend I know that each year from 2004 to 2011 might be a breakpoint. The test works similarly to supremum tests except that it is not conducted sequentially and the limiting distribution of the test-statistic is known. Conducted together, both tests complement each other.

[16] It is worth noting that the statistical insignificance of the latter breakpoint could potentially be caused by the relatively low statistical power of the test due to short pollution time series.

[17] Chay and Greenstone (1999, 2003b) divided U.S. counties into three groups with large, medium and small changes. These groups include quartiles of the counties with the largest reduction (upper quartile, >75%), smallest reduction (lower quartiles, <25%) and all other counties (combined second and third quartiles, between 25% and 75%).

be that the districts are spatially clustered depending on the magnitude of the changes. This is especially relevant to the districts with a larger reduction or increase in air pollution.

In view of the latter observation, I experiment with a Getis-Ord Hot Spot Analysis (Getis & Ord, 1992; Ord & Getis, 1995), applying this technique to $PM_{2.5}$ pollution data to sort districts into treatment and control groups. The Getis-Ord Hot Spot Analysis (HSA) is in essence a test for spatial dependence[18], designed to assess the extent of clustering between units based on their attributes, and to draw inference about its statistical significance.

Putting HSA in context, it is highly probable that the highly-polluted districts are surrounded by other similarly polluted districts. Moreover, air pollution in the latter could originate either from the districts' own sources or transported from outside. Such a scenario is quite possible given the ability of air pollution to travel across regions. In this case, even districts without pollution-intensive industries would likely demonstrate some degree of spatial association with heavily-polluted neighbors. More importantly, such districts could also experience the effects of the crisis related to a decrease or increase in pollution levels in nearby districts. In contrast, districts without polluting sectors, or districts located farther from the neighbors that have such sectors, might not exhibit any spatial association based on pollution-related attributes, and might not experience any impact of the crisis on air quality. Apart from the identification of spatial clusters in crisis-induced changes in air pollution, the HSA also provides a means to assess whether such a pattern of spatial dependence is statistically significant. Applying HSA, I am interested in identifying spatial clusters of districts with unusually large and statistically significant changes in $PM_{2.5}$ concentrations during the 2009-2012 improvement episode, relative to the pre-crisis 2008 $PM_{2.5}$ pollution levels. Therefore, HSA output allows me to assign districts within statistically significant clusters into the treatment group, while districts outside such clusters are assigned into the control group.

Technically, HSA boils down to the testing of the null hypothesis of "no spatial dependence". The null implies that the assignment of the input attribute values to the particular districts does not depend on spatial location; the value of the attribute itself is all that matters. The alternative hypothesis focuses instead on the cases where districts with large and small attribute values are systematically surrounded by other districts with respectively large and small values. Rejection of the null hypothesis would imply the presence of statistically significant spatial clusters of similar attribute values (Anselin, 1992). Statistically significant spatial clusters of high values are referred to as hot spots, while cluster of low values are referred to as cold spots. I implement HSA using ArcGIS's Getis-Ord G_i^* tool.

Panels C and D of Figure 8 provide visual representation of the HSA's results. Panel C shows the spatial distribution of the HSA input values – crisis-induced changes in mean $PM_{2.5}$ concentrations during 2008-2012 normalized by the pre-crisis 2008 concentrations. Panel D shows the resulting HSA output with hot- and cold-spot districts depicted in red and blue colors. There is a striking correspondence between the hot spots and the districts that experienced a statistically significant

[18] In spatial statistics, the notion of spatial dependence, reflecting the tighter relationship between near rather than distant units, means that the similar values of some attribute or characteristic for one unit will likely also occur in neighboring units, leading to the formation of spatial clusters (Anselin, 1992).

increase in air pollution, and also between the cold spots and the districts that experienced a statistically significant reduction in air pollution. The remaining districts, depicted in beige, are those in which relative changes in particulate air pollution are not statistically significant, implying that changes could likely occur by random chance or that these districts would experience such changes in the absence of the crisis. For the rest of the paper, I consider districts in hot and cold spots as treated districts with worsened and improved air quality, while those depicted in beige as control districts.

Identifying assumptions

The key identification assumption for equation (1) can be formulated in terms of the idiosyncratic error term, for $t = 1, 2, \ldots, T$:

$$E(\varepsilon_{dt} \mid \mu_d, \gamma_t, Treated_d \cdot Post_t, W_{dt}, X_{dt}) = 0 \quad (3)$$

In the DID context, this assumption is known as a parallel or common trends assumption, implying that, irrespective of the levels, comparison groups should have equally-sloped trajectories in the pretreatment outcomes of interest. Then, the unobserved average trend in the outcome variable of the treatment group in the absence of treatment should be equal to the observed trend of the control group. Further, the treatment is assumed to be the only process that induces deviations from the common trends between the comparison groups[19]. This assumption implies that districts from the control group provide valid counterfactual changes in infant mortality for the districts from the treatment groups in the absence of crisis-induced changes in air pollution.

One possible reason for violation of the assumption in equation (3) is the presence of time-varying unobservables as an additional source of heterogeneity, causing districts' individual trajectories in infant mortality to diverge from the parallel trends. As the baseline model in equation (1) controls only for time-constant unobservables, it would likely fail to produce unbiased estimates of the effect of interest. To overcome this concern, I extend the baseline specification to allow for heterogeneous trends by including district-specific slopes in equation (1):

$$\log (IMR)_{dt} = \alpha + \delta_1(Treated_d \cdot Post_t) + \beta_1 W_{dt} + \beta_2 X_{dt} + \mu_d + \gamma_t + \lambda_d t + \xi_{dt} \quad (4)$$

where $\lambda_d t$ is a time-varying unobserved heterogeneity that allows the possibility for each district to have differential trends through the distinct values of λ_d. Technically, the latter term represents time-invariant, either observed or unobserved, effects interacted with time to produce district-specific trajectories of outcomes. t is a continuous year variable centered on 2010 and normalized so that it equals zero in this year.

Based on the results of the trend break and Hot Spot analyses, the DID model in equation (4) compares pre- versus post-2010 levels and trends in infant mortality rates between the treated and control districts. The key identifying assumption is that, conditional on district-specific trends, any pre-

[19] This latter assumption is often referred to as a common shocks assumption (Dimick & Ryan, 2014; Kreif et al., 2015).

versus post-2010 changes in infant mortality rates caused by factors besides air pollution are the same for the affected and control districts, and that air pollution is the only factor differentially affecting the treated districts, beginning in 2010.

Taking into account the fact that the impact of the crisis could accelerate and decelerate over time, I further extend equation (4) to the following specification:

$$\log(IMR)_{dt} = \alpha + \delta_1(Treated_d \cdot Post_t) + \delta_2(Treated_d \cdot Post_t \cdot t) + \\ + \beta_1 W_{dt} + \beta_2 X_{dt} + \mu_d + \gamma_t + \lambda_d t + \xi_{dt} \quad (5)$$

where $Treated_d \cdot Post_t \cdot t$ allows for the effects of the crisis to evolve over time. Equation (5) is a trend-break model allowing a change in the slope after 2010. The statistical question of interest is whether $\widehat{\delta_1}$ and $\widehat{\delta_2}$ are jointly statistically significant after the trend-adjustment. The following concern should be taken into account while interpreting the estimation results. On the one hand, equation (5) introduces a dynamic structure that is consistent with the visual evidence from Figure 9, showing that the decline in infant mortality in the treated districts does not look like a one-time drop. On the other hand, the short length of our panel data set, especially the number of the post-crisis years, might mean that there could be limited statistical power to estimate a model with changes in slope. Therefore, the model in equation (4) might be more preferable.

I estimate models in equations (4) and (5) using an estimation method based on within transformation of data known as detrending. I prefer this approach because it is more efficient than others, especially in cases with relatively short and unbalanced panel datasets similar to mine (Brüderl & Ludwig, 2015). The idea of detrending is to subtract time-varying estimates of the individual-specific trends from the original variables. Applying this estimation approach essentially boils down to the following four-step procedure. First, for each district I estimate the regression of the form $\log(IMR)_{dt} = \mu_d + \lambda_d t + \zeta_{dt}$ to obtain predicted values of the outcome variable $\log \widehat{(IMR)}_{dt} = \hat{\mu}_d + \hat{\lambda}_d t$. Time-varying predicted values, $\log \widehat{(IMR)}_{dt}$, represent expected district-specific trends. Second, I subtract values predicted in step (1) from the original values of outcome to obtain the detrended dependent variables $\log \widetilde{(IMR)}_{dt} = \log(IMR)_{dt} - \log \widehat{(IMR)}_{dt}$. After this step, the only variation left in the dependent variable is the variation around the district-specific trend. Third, I apply steps (1) and (2) to detrend all explanatory variables $\tilde{x}_{jdt} = x_{jdt} - \hat{x}_{jdt}$ for any variable x_j. Detrending all variables of the model means that the estimation of the causal effect of interest is based solely on within around-trend variation. Finally, I run regressions on the detrended variables.

To further validate the DID identifying assumption of the model in equation (4), I formally address two violations common in the literature (Tanaka, 2015). First, the existence of a systematic difference in the pre-crisis trends in infant mortality rates. Second, the orthogonality of the impact of the crisis on other factors affecting the dependent variable in the post-crisis period.

To address the first concern, I examine the pre-crisis trends graphically. Figure 8 depicts the evolution of the average infant mortality rates across comparison groups, adjusted for the district-specific linear

trends and some basic characteristics of the deceased infants. The figure provides graphical evidence that trends in infant mortality rates are almost parallel in the pre-crisis period between control and treated districts with reduction in air pollution. However, adjustment for the district-specific trends fails to improve the presentation for the treated districts with an increase in air pollution. The parallel trends assumption is apparently violated in the case of these districts. Visual examination also provides evidence of the trend break for the treatment group right after the onset of the crisis.

To address the second concern, I follow Altonji, Elder, and Taber (2005) and examine whether the impact of the crisis has any association with changes in observable characteristics. I first successively regress my empirical model with every observable characteristic as the dependent variable. Then, I check whether the coefficients on the interaction term, $\widehat{\delta_1}$, are statistically significant. Although this is not a formal test for exclusion restrictions, the absence of statistically significant association with observable characteristics would suggest that there should not be a correlation with unobservable variables either (Altonji et al., 2005). Table 4 presents results for both types of treated districts. Although some of the point estimates are statistically significant, the vast majority show no evidence of the systematic difference in trends between districts from the treated and control groups. This is especially true for the group of districts with improvement in air quality, for which most of the coefficients are small or close to zero. It is noteworthy that, the impact of the crisis-induced reductions in $PM_{2.5}$ pollution is not associated with important determinants of infant mortality, including mother's age, household amenities and proxied parental characteristics. Although significant, the coefficients on the meteorological confounders are quite small.

Overall, the results provide suggestive evidence that the changes in $PM_{2.5}$ pollution attributable to the global financial crisis is orthogonal to other factors affecting the dependent variable in the post-crisis period. Therefore, the selected empirical strategy is unlikely to be biased due to changes in unobservable covariates. Additional falsification tests and robustness checks will further support this conclusion.

4 RESULTS

I first present baseline estimates of the impact of the crisis-induced changes in $PM_{2.5}$ on the infant mortality rate at the district level. I then perform sensitivity analysis to ensure that the proposed empirical strategy provides unbiased estimates. Finally, I perform a number of falsification tests and robustness checks to support the validity of the main findings.

Baseline results

Table 5 presents baseline results of the regression analysis by reporting the key estimates resulting from fitting equations (4) and (5). The dependent variable is the infant mortality rate for all causes of deaths. For both types of treated districts, columns (1) report the estimate of coefficient δ_1 after the estimation of equation (4), which tests for the effects of the crisis-induced changes in $PM_{2.5}$ on the infant mortality rate after adjustment for district fixed effects, year fixed effects and differential

trends. The second columns report the results from the equation (5) allowing for both level and slope changes during the post-crisis period. All regressions are run on the variables detrended as described in previous section.

The coefficients in both columns for the treated districts with reduction in $PM_{2.5}$ pollution suggest that these districts experienced a statistically significant decline in all-cause infant mortality after 2010. Moreover, column (2) provides evidence of a negative and statistically significant change in the slope of the infant mortality rate after 2010. Therefore, regression analysis confirms the visual impression that reduction in air pollution that occurred during the post-crisis period was strongly associated with a decline in infant mortality. In contrast, the regression coefficients for the treated districts with increase in $PM_{2.5}$ pollution captured by the variable $Treated \cdot Post$ are positive, small and insignificant. Thus, there is little evidence of the impact of the crisis-induced increase in air pollution on infant mortality. However, similarly to the districts with a decline in air pollution, the infant mortality rate in districts with worsened air quality demonstrates a negative and statistically significant change in slope after 2010. For both types of districts and across both specifications, the coefficients on $Treated \cdot Post$ are insensitive to the inclusion of the variable allowing change in the slope, $Treated \cdot Post \cdot t$.[20]

I use the resulting coefficients to assess the magnitude of the crisis-induced changes in $PM_{2.5}$ pollution on infant mortality. For that purpose, I focus on treated districts with improved air quality, which demonstrate a significant decline in infant mortality rates. The coefficient in column (1) indicates that the infant mortality rate in this group of treated districts fell by about 23% ($100 \cdot (e^{\hat{\delta}_1} - 1)$) more than in the group of control districts between the pre- and post-crisis period. The estimated decline is associated with 4.9 fewer infant deaths per 1000 live births.[21] Coefficients in column (2) from the model that allows for changes in the level and slope show an even larger effect of about 28%. I computed the total effect from equation (5) as $\hat{\delta}_1 + 0.5 \cdot \hat{\delta}_2$, where the factor of 0.5 is equal to the average value of the continuous year variable t for two post 2010 years ((0+1)/2; t is set to be equal to zero in 2010). A 28% decline translates into a total of 6.09 fewer infant deaths per 1000 live births.

Sensitivity analysis

Table 5 provides estimates of the baseline effect of interest without control variables. To address the concern that changes in the dependent variable may be explained by changes in the observable time-varying characteristics that potentially correlated with the impact of $PM_{2.5}$ pollution changes attributable to the effect of the crisis, I perform a sensitivity analysis. Table 6 reports results for both types of districts. Every pair of columns represents estimates from fitting equations (4) and (5).

[20] These findings are consistent with my expectations and much evidence from the rigorous analysis of the graphs and data in the preceding sections.

[21] My baseline results are quite similar to those reported in Tanaka (2015) who estimated the impact of the 1998 "Two Control Zones" environmental regulation on infant mortality in China, i.e. in similar pollution-mortality settings. The author found that a TZP status is associated with 3.3 fewer infant deaths per 1000 live births and a 20% reduction in infant mortality in the post-reform period.

First, I included confounders to the baseline specification, namely a natural logarithm of the district-level GDP per capita and meteorological covariates. In all specifications, the coefficient on GDP per capita is close to zero, not statistically significant and does not fluctuate much across specifications, apparently not affecting the point estimates on either $Treated \cdot Post$ and $Treated \cdot Post \cdot t$. This relaxes the concern about the income channel through which the crisis could also have affected infant mortality.

Columns (3) and (4) control for the average district-level temperature, precipitation, wind directions and speed. Inclusion of these factors makes the estimates larger but preserves their sign and significance. The wind-related controls dominate with the larger and significant coefficients. The coefficient on the west-east wind is the most important in terms of the magnitude. In contrast to previous specifications, the $Treated \cdot Post \cdot t$ coefficient drops to almost zero and becomes insignificant.

Columns (5) and (6) control for characteristics of the deceased infants. None of the coefficients on these variables are statistically significant. The inclusion of these controls does not have any effect on the coefficients of interest. Columns (7) and (8) control for characteristics and habits of the Heads of households. The coefficients of the main interest remained virtually unchanged. I also observe small, negative and statistically significant coefficients on the share of the Heads belonging to the Sikh and Buddhist religions, as well as the share of the Heads who are alcohol drinkers. The picture is very much the same with the next specification, columns (9) and (10), including the deceased infants' household characteristics, although the coefficients of interest become slightly smaller. However, the resulting coefficients on these control variables are close to zero and insignificant.

Columns (11) and (12) report coefficients from the regressions controlling for the district-specific indicators of the utilization of the medical services by mothers and infants. The main effect remains robust and statistically significant. As in the previous specification, the coefficient on $Treated \cdot Post \cdot t$ remains small and insignificant. This makes equation (4), with a combination of W_{dt} and X_{dt} vectors as specified in column (11), my preferable specification.

The coefficient in column (11) indicates that the infant mortality rate in the group of treated districts with improved air quality fell by about 24% more than in the group of the control districts between pre- and post-crisis periods. The estimated decline is associated with a total of 5.226 fewer infant deaths per 1000 live births. Combining changes in particulate air pollution with the estimated changes in infant mortality, I obtain an implied elasticity equal to 0.35.[22] The elasticity is within the range of elasticities reported in economic studies of the effects of air pollution on infant mortality in developed and developing countries. Chay and Greenstone (2003b) calculated elasticities for the effects of TSP equal to 0.35-0.5 in the U.S., while Arceo et al. (2015) find elasticity of 0.415 for exposure to PM_{10} in Mexico. The marginal effects of SO_2 found in Tanaka (2015) translates into an implied elasticity of 0.9 in China; Knittel, Miller, and Sanders (2016) provide evidence of a 1.827 elasticity for the impact of automobile air pollution on infant health in the U.S.; Currie and Schmieder (2009) report elasticity for chronic effects of toxic chemicals in a range of 1.82-6.49 in the U.S. On the lower end

[22] In the context of the paper, implied elasticity is a ratio of percentage changes in the infant mortality rate to the same period percentage changes in air pollution.

of the elasticities reported in economic studies are the estimates for the effects of SO_2, in the range of 0.07-0.13 in Germany from Luechinger (2014), and of 0.04-0.09 for acute effects of CO in the U.S., from Currie and Neidell (2005).

Finally, sensitivity analysis also supports the previous conclusion concerning the effects of the crisis-induced changes in $PM_{2.5}$ pollution on the infant mortality rate in the group of districts with worsened air quality. The main coefficient of interest remained close to zero and insignificant despite differences in specifications. In contrast to the analysis of the treated group of districts with improved air quality, the slope coefficients from the regressions fitting equation (5) become close to zero and insignificant only after controlling for district characteristics and utilization of medical services. Coefficients on the west-east wind and its speed are large, comparable in magnitude, and highly statistically significant, but have different signs. Therefore, the sensitivity analysis fails to capture any sign of the effects that documented an increase in $PM_{2.5}$ pollution attributable to the crisis could have on infant mortality in these districts, which is in line with the baseline results.

Experimentation with different specifications in this section provides evidence that the magnitude, sign and statistical significance of the coefficient of interest are insensitive to the inclusion of the control variables. This supports the credibility of my research design and estimates. Although there is always room for non-causal explanations between the variables of interest in non-experimental studies, the results of the sensitivity analysis do not directly contradict the causal nature of the relationship between the crisis-induced changes in air pollution and district-level infant mortality.

Falsification tests and robustness checks

Conditional on the results of Altonji et al. (2005) test and sensitivity analysis, equation (4) is likely to produce valid estimates of the crisis-induced reduction in $PM_{2.5}$ pollution on infant mortality in the Indian districts. Nevertheless, I provide further support for this conclusion by conducting a number of falsification tests and robustness checks.

For the first falsification test, I replace the dependent variable with another outcome variable that is not affected by the crisis-induced changes in air pollution. One of the most plausible candidates is infant mortality due to external causes of deaths, which include deaths caused by accidents and homicides that are not associated with air pollution. However, AHS contains disaggregated information only on internal causes of deaths. Nevertheless, I could select an internal disease that is potentially not associated with particulate air pollution for the test. The most promising candidate for this role is diarrheal diseases. To the best of my knowledge, there is no evidence of obvious causal links between exposure to air pollution and infant mortality due to diarrhea/dysentery. Another reason to think that the choice of this disease for the falsification test is appropriate is the evidence that diarrheal diseases are the concurrent cause of death to respiratory infections and have a comparable share of infant fatalities in my study area (Bassani et al., 2010).

Therefore, I use the infant mortality rate due to diarrhea/dysentery as the alternative dependent variable to evaluate the internal validity of the previous estimates. As Table 7 indicates, regressions return statistically insignificant coefficients. The result also provides evidence that the crisis-driven

reduction in air pollution had no additional effect on infant mortality through other diseases. Therefore, my specifications are likely unbiased.

As another falsification test, I re-estimate the model using observations only from the pre-crisis period where the effects of the crisis-induced reduction in air pollution could not exist. For this purpose, I assign 2008 as a placebo trend break point and thus consider 2007 as the pre-crisis period, and 2008 as the post-crisis period. Specifically, the variable $Post_t$ became equal to 1 for the year 2008, not after the formally identified τ_0. Thus, I use a classic two-periods model. The results are in Table 8. The table reports point estimates after fitting equation (4) with different combinations of W_{dt} and X_{dt} vectors for the districts from both treated groups with improved (Group 1) and worsened (Group 2) air quality. Since estimated coefficients using a pre-crisis sample and placebo trend break point are statistically insignificant and close to zero, my specifications can be considered as likely unbiased.

Further, I apply the model equation (4) to the alternative control group. For this purpose, I limit this group to the districts selected, based on the common support propensity score that restricts the sample to the districts that have similar observable characteristics to the districts from the treatment group of districts with improved air quality. I first computed the common support propensity score of being in the treatment group using available characteristics of the districts with significant crisis-induced air pollution reductions. Then, I constructed an alternative control group, including only those districts from the initially identified control group that are matched based on the propensity score. Finally, I re-estimated the model with this alternative control group. Table 9 reports the results of this falsification test. Since the sign, magnitude and the order of statistical significance of the estimated coefficient on the main effect of interest are not substantially different from that in the main analysis, I again concluded that my model is likely unbiased.

Performing robustness checks, I address concern that there may still be unobserved factors affecting infant mortality due to the differential response of air pollution concentrations within the similar geographic regions. To control for this issue, I include in the preferable specification additional National Sample Survey (NSS) region*year fixed effects. NSS regions do not represent administrative units but rather collections of districts grouped based on similar agro-climatic conditions. Thus, this specification identifies the effect of interest using variation in crisis-induced changes in $PM_{2.5}$ pollution within the NSS regions with similar characteristics. Thus, any potentially possible changes caused by any differences are purged at the level of NSS regions. This exercise does not affect either the sign or magnitude of the estimated effect.

Further, I use a number of other specifications to re-estimate the most preferable model in the analysis. First, I use different weighting schemes to check how sensitive the model is to these changes. The results show that the point estimate of the main effect of interest did not change in response to not weighting at all, and reacted by a not substantial reduction in the magnitude on the weighting by the number of births. Second, I cluster standard errors at the state and NSS regions levels, as well as at the state*year and region*year levels. The estimated effect of interest remained robust to these alternative specifications. Third, I run a regression with one dependent variable expressed in level rather than in the log-form to see that the coefficient on *Treated* · *Post* had the same sign and significance level. Table 9 summarizes results.

Complementing the robustness checks above, the Appendix provides two additional tests. The first is based on the model in equation (4) and focuses on alternative options for sorting sample districts into treatment and control groups. Table A1.1 compares the resulting estimates. Each column of the table corresponds to one of the eight options, which are intuitively illustrated by the graphs in Figure A1. The sign, magnitude and order of statistical significance of the estimated coefficients on *Treated · Post* remain similar between each other and to the coefficient estimated using my preferable specification.

The second additional test checks whether the main finding would remain robust to different estimation strategies, namely the detrending used in this paper, conditioning on the explanatory variables and district-specific trends, and a method combining matching on pre-crisis explanatory variables and trends with subsequent difference-in-differences. The results are presented in Table A1.2, which indicates that all specifications return estimated coefficients of interest that are not substantially different from each other, thus confirming the credibility of the main analysis.

Overall, conditional on the results of the falsification tests and robustness checks, I conclude that the main findings of the paper justify the causal impact of the crisis-induced reduction in air pollution on the infant mortality rate in the sample of the selected Indian districts.

5 PATHOPHYSIOLOGICAL MECHANISM

Addressing the second research question, I examine the impact of the changes in $PM_{2.5}$ concentrations on the mortality of infants at different ages and from various diseases. The analysis based on the specifications in columns (11) and (12) from Table 6 focus solely on the treated group of the districts with improved air quality.

Table 10 presents the estimated effects of the reductions in $PM_{2.5}$ pollution on the infant mortality rate within 1 day, 28 days, between 28 days and 11 months, within 11 months and between 11 and 12 months of life. The second category is also known as the neonatal infant mortality rate, which in turn is broken down into early and late neonatal mortality rates corresponding to the deaths occurring within 0-7 and 8-27 days from births, respectively. The third category is usually referred to as a postneonatal mortality rate. Separate analysis of these categories is performed purposefully. The large and statistically significant estimate in the neonatal period would likely suggest that particulate air pollution affects infant mortality through the adverse effects on fetal development, via in utero exposure to $PM_{2.5}$. Newborns whose mothers were exposed to high $PM_{2.5}$ concentrations during pregnancy have a higher probability of dying in the neonatal than the postneonatal period. In contrast, a large and statistically significant effect in the postneonatal period would highlight the importance of post-birth $PM_{2.5}$ exposure in the biological mechanism through which air pollution affects infants directly. However, the exact biological channels are not yet well-studied (Chay & Greenstone, 2003b; Tanaka, 2015).

The estimates reveal that both biological mechanisms are important in explaining the overall effect found above. However, the response of infant mortality during the postneonatal period (column (5))

is substantially larger than in the neonatal period (column (4)). The coefficients suggest that the neonatal IMR fell by 21% more in the districts with improved air quality, with a corresponding elasticity of 0.31, which is lower than the implied elasticity for infant mortality of 0.35. In contrast, postneonatal mortality shows a decline of almost 32% with the implied elasticity of 0.45, which is significantly larger than the elasticity for both overall infant and neonatal mortality. I estimate that the contribution of the reduction in $PM_{2.5}$ to the overall decline in neonatal mortality and postneonatal mortality is equal to 9% and 15%, respectively. Therefore, the effect of particulate air pollution on infant mortality is not larger in the neonatal period and is more likely disproportionally associated with the probability of dying during the postneonatal period.

Several aspects of the biological mechanism are worth noting. First, there is no effect on infant deaths within one day of birth. Although negative, the point estimates are small and statistically insignificant. Second, disaggregation of the overall neonatal mortality into early and late neonatal periods, presented in columns (2)-(3), reveals the important regularity of the biological mechanism. The response of neonatal mortality to the reduction in particulate air pollution is completely driven by the decline in infant mortality during the early neonatal period. Therefore, I cannot rule out the channel of in utero $PM_{2.5}$ exposure. Third, point estimates on the deaths of infants aged between eleven and twelve months (column (7)) are large and negative, but insignificant, additionally highlighting the importance of the postneonatal mortality and post-birth $PM_{2.5}$ exposure as the channel through which air pollution affects infant mortality. Finally, as presented in column (6), the estimated effects of the reduction in $PM_{2.5}$ concentrations on infant deaths within eleven months are identical to those I found for the all-cause infant mortality and thus support my main findings.

These findings are in contrast to conclusions made in Chay and Greenstone (1999, 2003b) and Tanaka (2015) about the disproportionate effect of air pollution on infant mortality during the neonatal period. For the U.S., Chay and Greenstone (2003b) attributed 80% of the effect of the reduction in TSPs on infant mortality to the decline in neonatal mortality, of which 60-70% is driven by fewer infant deaths within one day of birth. For China, Tanaka (2015) found that 26% and 63% of the effect of the TCZ regulation on infant mortality occurred within one day of births and during the neonatal period. On the other hand, my estimates are in line with the statistics for the districts in the study area according to which the decline in the number of infant deaths during the post-crisis period was due to a decline in the early neonatal and postneonatal periods, with respective shares of 42% and 41%.

I next examine the effect of the crisis-induced changes in $PM_{2.5}$ pollution on infant mortality disaggregated by various diseases. Table 7 presents the results from the regressions with IMR due to fifteen causes of deaths as the dependent variable. Although the AHS does not provide exact codes for different symptoms, I used the tenth International Classification for Diseases (ICD-10) to identify all of them except the category "Other" as internal causes of deaths. Internal causes of deaths are defined as health-related, non-accidental causes in contrast to non-health related external causes such as accidence, injury, homicides, poisoning and other similar causes. Since the exact pathology of diseases caused by particulate air pollution is not well-known, some of the internal diseases could potentially be associated with air pollution. Particularly, chronic exposure of infants to high $PM_{2.5}$ concentrations is expected to result in deaths due to respiratory infections. In contrast, there could well be internal diseases without any association with particulate air pollution.

Table 7 provides evidence of a large, negative and statistically significant impact of the crisis-induced reduction in $PM_{2.5}$ pollution on mortality from respiratory infections (column (1)). The effect is associated with 24% fewer infant deaths in the districts with improved air quality, translated into implied elasticity of 0.34. The calculations suggest that the crisis-induced reduction in particulate air pollution is associated with 15% of the overall improvement in infant mortality due to respiratory infections. The magnitude of the estimated effect is comparable to the impact on the all-cause infant mortality. Additionally, this finding supports the conclusion about the dominance of the post-birth $PM_{2.5}$ exposure channel and more frequent incidence of infants' deaths during the postneonatal period. In the case of the states from the study area, the prevalence of pneumonia or respiratory infections as the causes of children deaths directly attributable to air pollution is indeed much higher in the postneonatal period than in the neonatal period (Bassani et al., 2010). Moreover, my findings are consistent with evidence that respiratory diseases less probably cause infant death during the neonatal period since newborns spend the most of their time indoors, but are the major cause of death for infants in the postneonatal period (Woodruff, Grillo, & Schoendorf, 1997; Bobak & Leon, 1999; Woodruff, Parker, & Schoendorf, 2006). Thus, disproportional association of the infants' deaths due to respiratory infections in the postneonatal period is justified.

It is notable that for the majority of other cases representing quite a broad range of diseases, the estimated effect of the crisis-induced reduction in particulate air pollution is small and not statistically significant. The exceptions are convulsions and two types of fever, with jaundice and convulsions. Although large and statistically significant, the coefficients on these diseases are sensitive to the inclusion of additional variables, particularly the second variable of interest that allows changes in the slopes. While there are no obvious causal links between air pollution and infant mortality due to these diseases, Clay, Lewis, and Severnini (2015), using the 1918 influenza pandemic in the U.S. as a natural experiment, provide rare evidence that air pollution could adversely affect the susceptibility of infants to infectious disease. This is consistent with my findings for fever.

Overall, my findings suggest that the crisis-induced reductions in $PM_{2.5}$ pollution affect infant mortality through two biological mechanisms, particularly the adverse impact on fetal development and infants' early-life exposure. However, the estimates indicate that the former mechanism is not the primary channel as infants' deaths are more likely to occur during the postneonatal period. Moreover, the results indicate that the effects of $PM_{2.5}$ pollution on infant mortality are specific for respiratory infections and might be related to some of the infectious diseases. Nevertheless, the results should be interpreted with caution since the model designed for all-cause infant mortality could not capture effectively all underlying factors affecting some of the diseases, as an indicator of model fit suggests.

6 POLICY PERSPECTIVE: HEALTH BENEFITS

Finally, I use the quantified relationship to measure health benefits from the crisis-induced episode of air quality improvement. Moving the analysis to a policy perspective, this section demonstrates

how the resulting estimates can be applied to measure the effectiveness of the potential policies designed to improve air quality.

For this purpose, I first use estimated coefficients to assess the contribution of the crisis-induced reduction in $PM_{2.5}$ pollution to the overall improvement in infant mortality. Then I calculate the number of infant lives saved by the improvement in air quality. Further, using available life-years and life-expectancy metrics, I convert the number of infant lives saved into the number of infant life years saved. Finally, I use available estimates of the value of a statistical life to monetize potential gains from the crisis-induced improvement in air quality.

The average decline in district-level infant mortality in the group with the reduction in $PM_{2.5}$ concentrations fell by about 59 infant deaths per 1000 live births during the post-crisis period. In terms of marginal effects, my computations imply that a decline in $PM_{2.5}$ by 1 $\mu g/m^3$ results in about 1.09 fewer infant deaths per 1000 live births. Dividing the product of the implied marginal effects with respective average reduction in $PM_{2.5}$ levels of 5.75 $\mu g/m^3$, I find that an 11% overall decline in the infant mortality rate during the period of interest occurred due to improvement in air quality. Interestingly, had all districts in the study area experienced the same reduction in air pollution as the treated districts, the contribution would be of the same magnitude. For comparison, Jayachandran et al. (2010) show that the introduction of sulfa drugs, a groundbreaking medical innovation in 1930s in the U.S., resulted in a 17-32% decline in pneumonia mortality, 24-36% decline in maternal mortality, and 52-65% decline in scarlet fever mortality during 1937-1943. Among more recent economic studies, Luechinger (2014) finds that 25-44% of the infant mortality decrease in Germany in 1985-2003 was associated with the reduction in SO_2 concentrations. Therefore, although with a little lower magnitude, the contribution of improvement in air quality to the overall decline in infant mortality rates in the sample of Indian districts during the period of interest is comparable.

Knowing that the average district-level decline in $PM_{2.5}$ concentrations during the period of interest is 5.75 $\mu g/m^3$, and that the number of live births in the treated districts with reduction in $PM_{2.5}$ pollution is 214,173 out of 759,425 for the whole sample in the post-crisis period, I apply implied marginal effects to calculate the number of saved infant lives. The calculation suggests that the crisis-induced reduction in air pollution resulted in 1338 infant lives saved in the treated districts. This number is lower but still comparable with that in Chay and Greenstone (2003b), where the authors claim 2500 fewer infants died during the U.S. economic recession in 1980-1982. Assuming that there could be an environmental regulation that would have the equivalent impact for all sample districts, the number of infant lives saved by such an improvement in air quality could reach 3589.

Having calculated the number of infant lives saved, I convert it into the number of infant life years saved. For that purpose, I use official life tables published on the web page of the Ministry of Home Affairs' Office of the Registrar General and Census Commissioner of India. A life table states the probabilities of survival and life expectancies of the hypothetical group or cohort at different ages (Census of India, 2016). Particularly, a Sample Registration System's life table for the 2009-2013 period shows that the average life expectancy for individuals within one year of life is 67.5 years. Multiplying the number of infant lives saved by this life expectancy, I obtain a gain in life years saved of 90,319.1 for the treated districts and 242,282.02 for the whole sample of districts.

Finally, however impressive the estimated benefits from the improvement in air quality are, they would be pointless without an opportunity to compare them with the costs of environmental regulation. Therefore, all gains need to be monetized. For that purpose, estimates of the value of a statistical life are usually used.[23] As there is no standard concept for the value of a human life in economics, the authors typically use different measures varying from $1.7 million ($2000) in Ashenfelter and Greenstone (2004) to $6.7 million ($2000) in Viscusi and Aldy (2003) and the U.S. EPA (n.d.b) estimate of $7.4 million ($2006). I use the value of a statistical life estimated specifically for India by Madheswaran (2007), who finds it equals to 15 million INR or $233,619.

I monetize the estimated number of infants lives saved of 1338 for the treated districts and 3589 for the whole sample during 2010-2011 to obtain monetary values of health benefits in the range of $313 million and $839 million, respectively. Knowing that the average number of the households surveyed in the treated districts and in the whole sample is 1,081,727 and 4,280,315, an annual average per-household monetized benefit from the estimated reduction in $PM_{2.5}$ pollution is in the range of $289 for the treated districts, and $196 for the whole sample. For comparison, Luechinger (2014) reports that annual monetized benefit from the environmental regulation aimed at the reduction of SO_2 concentrations in West Germany in the year 1989/1990 varies from $50 to $343 per household.

It is worth mentioning that the overall health benefits of the crisis-induced reduction in air pollution could be underestimated in this study. My research design does not account for the effects of the decline in the concentrations of other air pollutants, as well as the crisis-driven impact on morbidity or labor productivity of the older cohorts of the Indian population. Nevertheless, the resulting monetary values of health benefits can be used as a benchmark against which the costs of the current or potential policies aimed at improving air quality can be compared. Thus, my estimates could be of considerable interest for policymakers aimed at finding the optimal balance between the costs and benefits of air pollution regulation in the specific context of early-life health in developing countries.

7 CONCLUSION

This study has attempted to isolate the causal relationship between the reductions in $PM_{2.5}$ pollution presumably caused by the Global Financial Crisis of 2008 and decline in infant mortality in India using a quasi-experimental difference-in-differences research design.

Combining state-of-the-art satellite-based estimates with household survey-based information for 284 districts across 9 states during 2007-2011, I find that the infant mortality rate fell by 24% more in the most affected districts, implying 1338 fewer infants deaths than would have occurred in the absence of the crisis. The analysis of the pathophysiological mechanism indicates that the effect of interest is strongest in the postneonatal period, specific for respiratory infections and might be related to infectious diseases. The findings also highlight the importance of two biological mechanisms: in utero

[23] In a statistical sense, the value of a statistical life is the cost of reducing the average number of deaths by one. Conducting a cost-benefit analysis of environmental policies in practice, the U.S. EPA, for example, estimates how much people are willing to pay for a marginal reduction in the risk of dying from the pollution-related adverse health conditions and refers to such estimates as the values of a statistical life (U.S. EPA, n.d.b).

and post-birth $PM_{2.5}$ exposure. The estimates are within the range reported in other economic studies and appear to be robust to a variety of specifications and falsification tests, prompting the belief that the relationship between crisis-induced reduction in particulate air pollution and decline in infant mortality is causal in nature.

Moving the analysis further into the policy perspective, I demonstrate how the resulting estimates of the health effects attributable to the crisis-induced reduction in $PM_{2.5}$ pollution could be applied to measure the effectiveness of the current and potential policies aimed at controlling air quality in India. For that purpose, I measured actual gains from improving air quality in the Indian districts during the crisis time-frame. The resulting gains comprise a number of infant lives saved, the corresponding increase in life expectancy at birth and monetary values of the improvements obtained.

Back-of-the-envelope calculations suggest that the estimated decline in infant mortality translates into a three-year after crisis total of $312.5 million. The resulting health benefits attributable to the crisis-induced reduction in air pollution can be used as a benchmark to assess the effectiveness of potential policies designed to improve air quality in the selected Indian districts.

Therefore, this study addresses more precisely the needs of policymakers aimed at finding the optimal balance between the costs and benefits of air pollution reduction in the specific context of developing countries.

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FIGURES

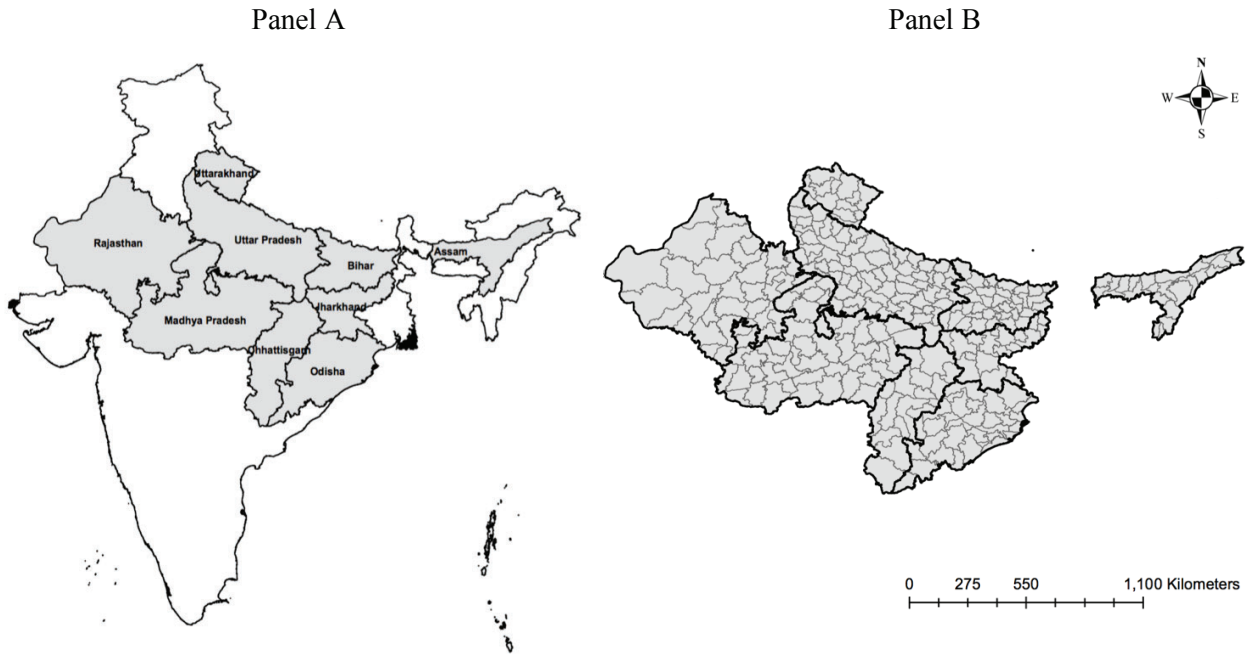


Figure 1. Study area

Notes: The figure demonstrates the 284 districts (as per Census 2001) across 9 states in India covered by the Annual Health Survey. These districts are a particularly relevant study area. They represent nearly 50% of the overall population and account for 60% of all births and 70% of all infant deaths in the country.

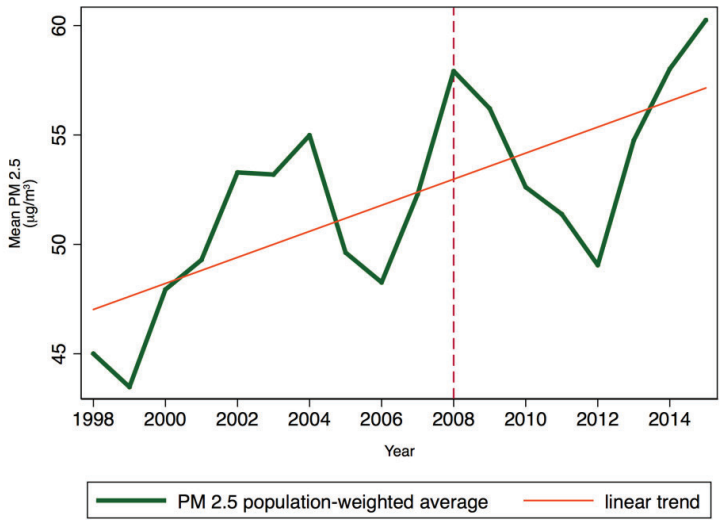


Figure 2. $PM_{2.5}$ concentrations in study area, 1998-2005

Notes: The figure shows the evolution of the district-level annual mean $PM_{2.5}$ levels in the study area for 1998-2015. Two observations emerge. First, air quality has been continuously deteriorating during the last two decades. Second, the figure documents two episodes of abrupt reduction in $PM_{2.5}$ concentrations, 2005-2006 and 2009-2012, followed by comparably sharp reversals of the trends. Air quality improvement during the 2009-2012 episode is the focus of my study.

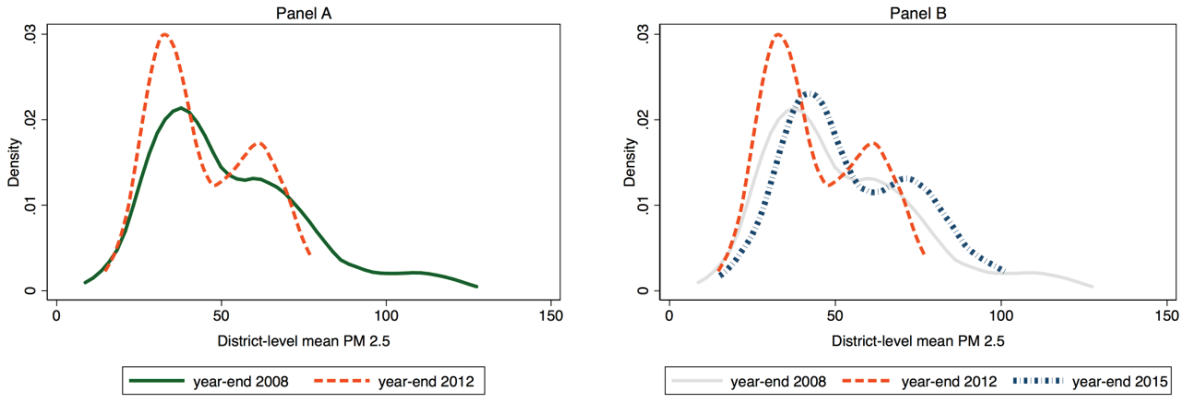


Figure 3. Kernel density graphs of air quality

Notes: The figure compares kernel density estimates of the annual mean $PM_{2.5}$ distributions across the districts in the study area for 2008, 2012 and 2015, representing pre-crisis, crisis and post-crisis cut-off points. Panel A demonstrates that the entire distribution shifted substantially to the left in 2012 compared to 2008. In contrast, Panel B documents the shift of the distribution to the right again in 2015.

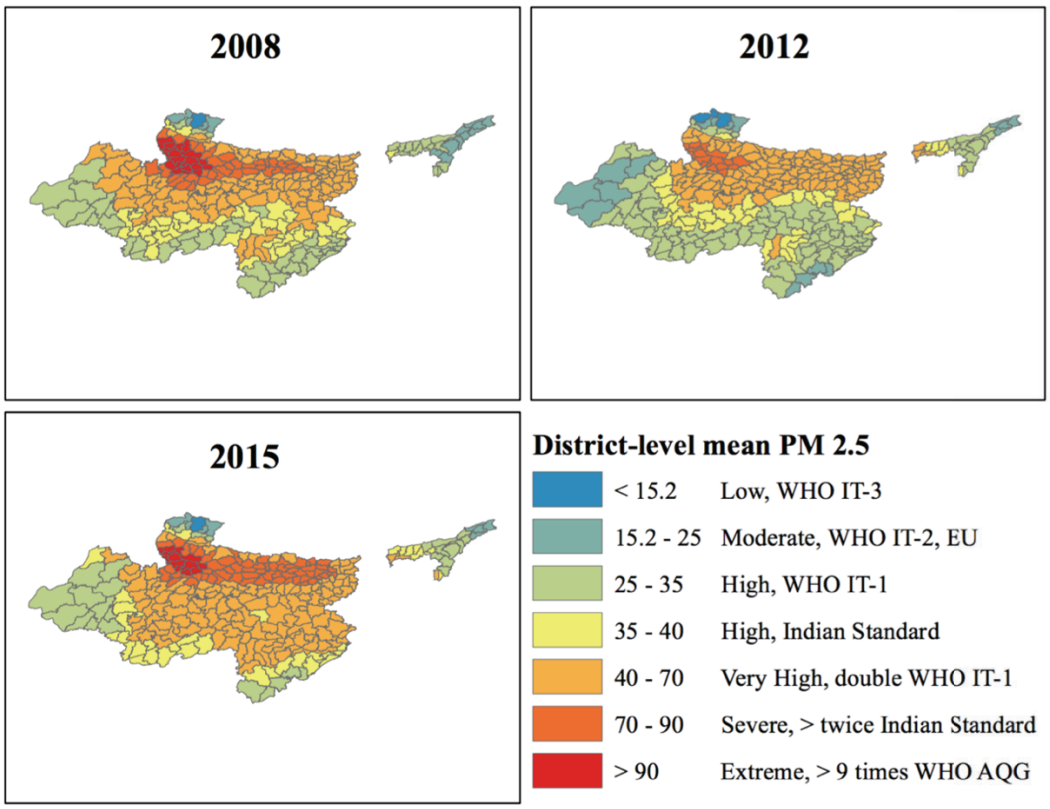


Figure 4. Spatio-temporal distribution of district-level annual mean $PM_{2.5}$

Notes: The figure depicts spatio-temporal distribution of district-level annual mean $PM_{2.5}$ concentrations in the study area for 2008, 2012 and 2015, representing pre-crisis, crisis and post-crisis cut-off points. The districts are classified into six categories using air quality thresholds adopted by the WHO, EU and Indian environmental agencies (similar to Chowdhury & Dey, 2016). I define "Low", "Moderate", "High", "Very High", "Severe" and "Extreme" categories in a way that their upper limits correspond to one of the standards. The limits for the first two categories are set to meet the WHO interim targets 3 (IT-3) and 2 (I-2), equal to 15 and 25 $\mu g/m^3$ respectively. The latter threshold also corresponds to the European Environmental Agency target value for European countries. The upper limit of 35 $\mu g/m^3$ in the lower "High" category is the WHO IT-1, while the limit in the upper "High" category is equivalent to the Indian National Ambient Air Quality Standard of 40 $\mu g/m^3$, the least stringent of the standards. The limits of the remaining categories are designed to highlight extremely high levels of air pollution in India. The "Very High" category corresponds to the $PM_{2.5}$ concentration equivalent to the double of the least demanding WHO IT-1, "Severe" pollution exceeds twice the Indian Standard and is nine times the WHO air quality guideline value of 10 $\mu g/m^3$, which is excluded from our classification. The last "Extreme" category comprises the remaining concentrations of fine particulate pollution exceeding 90 $\mu g/m^3$. More details about air quality standards and guidelines are in Panel B of Table 1, WHO (2006a), EEA (2014) and CPCB (2009).

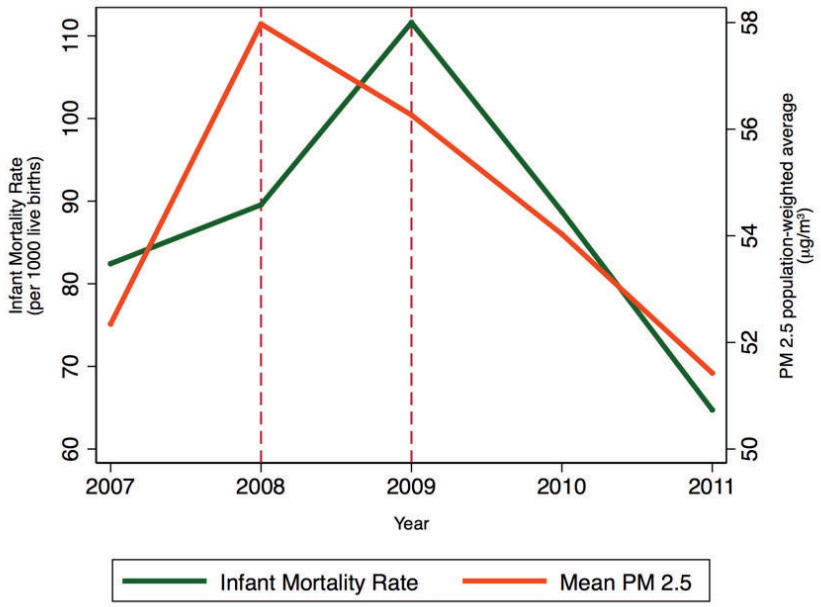


Figure 5. Trends in mean $PM_{2.5}$ concentrations and Infant mortality rates, study area, 2007-2011

Notes: The figure shows the evolution of the district-level annual means of $PM_{2.5}$ air pollution and the infant mortality rate during 2007-2011. The infant mortality rate followed a similar pattern to that followed by air pollution. Both data series provide visual evidence of structural breaks marked by the dashed lines and reversals in upward trends started after 2008 and 2009, respectively for air pollution and mortality. Although with a time lag, both breaks correspond well to the crisis' time frame, cautiously suggesting the presence of a direct relationship within the crisis-pollution-mortality nexus.

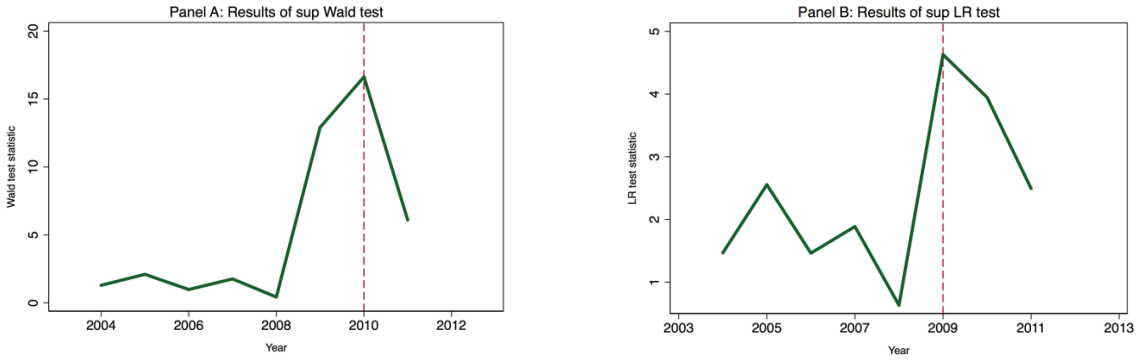


Figure 6. W- and F-statistics from sup Wald and sup LR tests for trend break (Stata output)

Notes: The figure presents the results of the time-series econometric test for structural trend break, specifically supremum Wald and likelihood-ratio (LR) tests designed for the cases when the breakpoints are unknown (Andrews, 1993, 2003; Hansen, 1997). Both supremum tests identify structural breaks within the 2009-2012 air quality improvement episode, thus associating them with the respective reversal of the upward trend in $PM_{2.5}$.

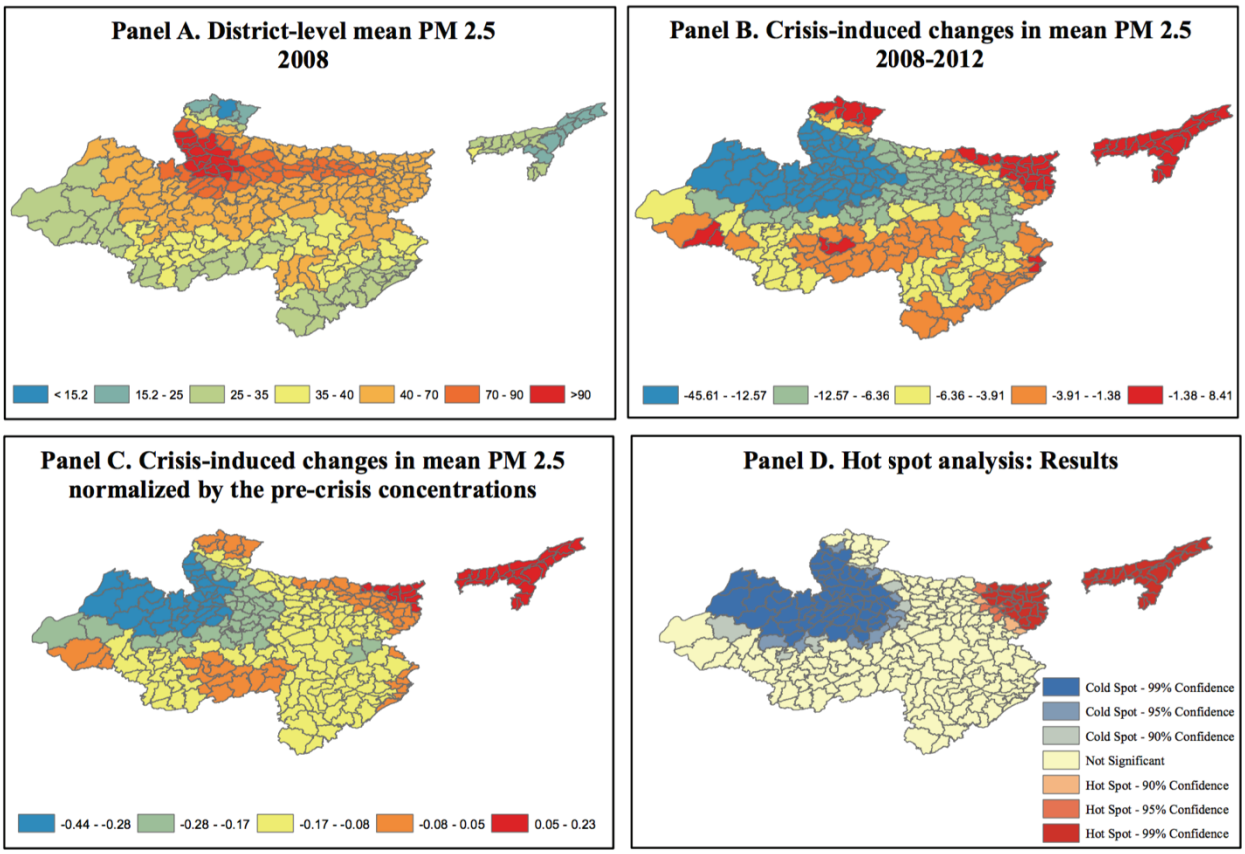


Figure 7. Spatial relationship between pre-crisis $PM_{2.5}$ and crisis-induced changes

Notes: Panel A illustrates spatial variation in the pre-crisis levels of $PM_{2.5}$ pollution. Panel B demonstrates a geographical distribution of the district-wise changes in average $PM_{2.5}$ during 2009-2012. Panels C and D provide visual representation of the Hot Spot Analysis results. Panel C shows spatial distribution of the HSA input values – crisis-induced changes in mean $PM_{2.5}$ concentrations during 2008-2012 normalized by the pre-crisis 2008 concentrations. Panel D shows the resulting HSA output with hot spot and cold spot districts depicted in red and blue. There is a striking correspondence between the hot spots and the districts that experienced statistically significant increase in air pollution, and the cold spots and the districts that experienced statistically significant reduction in air pollution. The remaining districts, depicted in beige, are the ones in which relative changes in particulate air pollution are not statistically significant, implying that they could likely happen by random chance or that these districts would experience such changes in the absence of the crisis. I consider districts belonging to the hot and cold spots as treated districts with worsened and improved air quality, respectively, while districts depicted in beige are control districts.

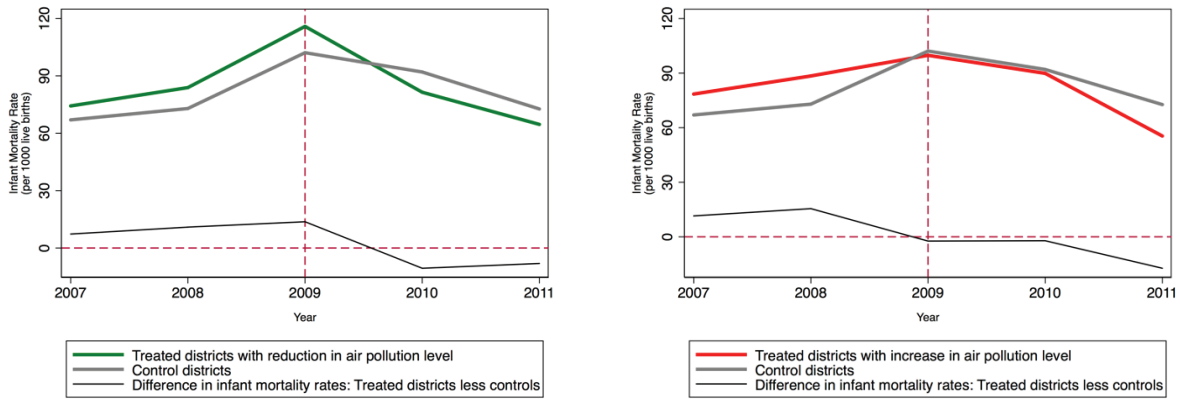


Figure 8. Visual examination of the parallel trend assumption

Notes: The figure depicts the evolution of the trends in infant mortality rates across comparison groups adjusted for the district-specific linear trends and some basic characteristics of the deceased infants. The dashed vertical line indicates the time of the effects of the crisis on air pollution started between years 2009-2010. The thin black line represents the difference in infant mortality rates between treatment and control groups of districts, allowing a rough comparison of the relative pre- and post-crisis trends.

TABLES

Table 1. Descriptive statistics

	2007	2008	2009	2010	2011
Panel A: District-specific characteristics					
Number of districts	283	283	283	257	281
Population in sample	605872070	617397169	628922276	598644463	648405359
Total number of live births	414075	373871	336085	335528	423897
Total number of infant deaths (all causes):	32310	31718	33515	25058	25797
early neonatal	14781	14271	15065	11199	11797
late neonatal	4897	4747	4976	4034	4088
postneonatal	9659	9486	10111	7488	6984
Infant Mortality Rates (all causes)	82.43	89.56	111.61	88.74	64.73
early neonatal	36.43	38.59	47.72	38.14	29.99
late neonatal	13.86	14.65	18.80	16.38	9.97
postneonatal	24.72	27.11	33.86	25.97	17.49
Mean district-level air pollution ($PM_{2.5}$)	52.35	57.97	56.27	54.03	51.43
Average age of mothers	28.21	27.35	26.72	26.41	26.40
% of Married mothers	98.92	99.09	99.24	99.46	99.40
Panel B: Deceased infants characteristics					
% of Male infants	50.97	51.21	50.13	50.93	50.40
% of Infant deaths in rural areas	86.86	87.89	87.43	87.56	85.84
Average birth order	2.75	2.79	2.76	2.52	2.41
Panel C: Head of the household characteristics					
% of Male Head of the household	90.09	89.91	89.80	86.27	84.50
% of Heads from Scheduled Castes	21.58	22.15	22.06	22.74	22.39
% of Heads from Scheduled Tribes	12.40	12.54	12.82	11.69	12.50
% of Illiterate Heads	40.70	40.16	40.88	39.81	40.04
% of Hindu Heads	82.59	82.47	82.77	82.70	82.84
% of Muslim Heads	15.82	15.86	15.46	14.60	14.64
% of Unemployed Heads	8.56	8.05	8.53	10.55	11.57
% of Smoking Heads	29.44	28.61	29.47	26.89	27.10
% of Alcohol-drinking Heads	24.98	24.52	24.40	22.13	22.30
Panel D: Deceased infants household characteristics					
% of Houses with filtered water	16.45	16.68	16.95	16.75	16.48
% of Houses with electrical lightning	40.32	40.08	40.17	38.39	37.15
% of Houses with kerosene lightning	58.09	58.13	58.16	60.05	61.10
% of Households cooking on firewood	54.57	54.45	54.52	52.79	55.24
% of Households cooking on cow dung cake	22.34	22.62	22.10	23.98	22.55
% of Households cooking on coal/charcoal	1.54	1.50	1.63	1.50	1.35
% of Households cooking on electricity	0.08	0.07	0.07	0.05	0.06
% of Households cooking inside	81.09	81.65	81.02	82.66	81.46
% of Households without toilet	74.24	74.12	74.01	74.42	75.17
Panel E: Medical services utilization					
% of Mothers with no ante natal care	17.12	15.04	13.51	1.61	1.67
% of Deliveries at government facilities	34.27	40.03	43.77	46.09	49.95
% of Newborns with no after births checkups	29.94	26.10	23.82	18.36	13.91
% of Vaccinated babies	93.29	94.13	94.54	92.64	95.22
Panel F: Meteorological covariates					
Mean district-level air temperature (p/a)	25.71	25.55	26.37	26.38	25.59
Mean district-level precipitation (p/a)	87.93	95.30	73.06	76.81	108.68

Notes: The table presents descriptive statistics for the districts from Figure 1. The table shows that the reduction in district-level $PM_{2.5}$ pollution during 2009-2011 is visibly larger compared to the changes in the majority of other variables during the same period.

Table 2. Summary statistics of changes in $PM_{2.5}$ and population at the crisis' cut-off points

Panel A: Summary statistics of changes in annual mean PM 2.5

	a) 2008	b) 2012	c) 2015
Mean	51.91	44.45	54.37
Standard deviation	22.25	15.54	19.43
Min	15.12	14.64	15.16
Max	120.92	77.32	101.61
10th percentile	28.93	27.43	32.54
90th percentile	80.44	66.54	80.81
Observations	284	284	284

Panel B: Classification of the PM 2.5 levels and population exposure

a) 2008

Annual PM2.5	Category	# of districts	% of districts	# of exposed population	% of exposed population	Exposed % > Indian standard	Comments
< 15.2	low	1	0.35	425428	0.07		WHO IT-III
15.2-25	moderate	14	4.93	11568477	1.87	28.59	WHO IT-II, EU AQS
25-35	high	63	22.18	102100000	16.5		WHO IT-I
35-40	high	38	13.38	62735096	10.14		Indian standard
40-70	very high	114	40.14	279800000	45.22		2*WHO IT-I
70-90	severe	35	12.32	106600000	17.23	71.41	>2*Indian standard
>90	extreme	19	6.7	55488672	8.97		>9*WHO AQG

b) 2012

Annual PM2.5	Category	# of districts	% of districts	# of exposed population	% of exposed population	Exposed % > Indian standard	Comments
< 15.2	low	3	1.05	1380248	0.21		WHO IT-III
15.2-25	moderate	17	5.99	24321796	3.64	39.97	WHO IT-II, EU AQS
25-35	high	91	32.04	159300000	23.87		WHO IT-I
35-40	high	37	13.03	81711800	12.24		Indian standard
40-70	very high	123	43.31	361700000	54.19		2*WHO IT-I
70-90	severe	13	4.58	38989920	5.85	60.03	>2*Indian standard
>90	extreme	0	0	0	0		>9*WHO AQG

c) 2015

Annual PM2.5	Category	# of districts	% of districts	# of exposed population	% of exposed population	Exposed % > Indian standard	Comments
< 15.2	low	1	0.35	441956	0.06		WHO IT-III
15.2-25	moderate	9	3.17	9059676	1.28	18.97	WHO IT-II, EU AQS
25-35	high	31	10.92	56973744	8.07		WHO IT-I
35-40	high	35	12.32	67430456	9.55		Indian standard
40-70	very high	139	48.94	318200000	45.08		2*WHO IT-I
70-90	severe	56	19.72	212000000	30.04	81.03	>2*Indian standard
>90	extreme	13	4.58	41676652	5.92		>9*WHO AQG

Notes: The table accompanies Figure 3 by providing summary statistics corresponding to changes in $PM_{2.5}$ and population exposure to $PM_{2.5}$ pollution across districts for 2008, 2012 and 2015, representing pre-crisis, crisis and post-crisis cut-off points. Panel A documents changes in $PM_{2.5}$ concentrations, while Panel B relates these changes to population exposure. Categorization of districts due to $PM_{2.5}$ concentrations corresponds to those explained in the notes to Figure 4. Taken together, Figure 3 and Panel A of Table 2 support the hypothesis that districts with high pre-crisis levels of air pollution likely experienced a more significant improvement in air quality than districts with initially low pollution concentrations. Figure 4 and Panel B Table 2 provide suggestive evidence that improvements in infant mortality could be more pronounced in districts with high pre-crisis levels of air pollution.

Table 3. Structural trend break analysis

Panel A: sup Wald and LR tests, unknown breaks, study area's aggregated time series

a) Sup Wald

Test window	Trimming, %	Year of break	W-statistic
2004-2011	25	2010***	16.63
2005-2010	30-35	2010***	16.63
2006-2009	40	2009***	12.92
2007-2008	45	2007	1.76

b) Sup LR

Test window	Trimming, %	Year of break	F-statistic
2004-2011	25	2009	4.63
2005-2010	30-35	2009	4.63
2006-2009	40	2009	4.63
2007-2008	45	2007	1.89

Panel B: Wald and LR tests, known year of break, study area's aggregated time series

a) Wald

Tested Year	W-statistic	Significance
2004	1.29	-
2005	2.09	-
2006	0.98	-
2007	1.76	-
2008	0.42	-
2009	12.92	***
2010	16.63	***
2011	6.1	***

b) LR

Tested Year	F-statistic	Significance
2004	1.47	-
2005	2.56	-
2006	1.46	-
2007	1.89	-
2008	0.63	-
2009	4.63	*
2010	3.95	-
2011	2.49	-

*** p<0.01, ** p<0.05, * p<0.1

Notes: The table shows the results of the time-series econometric test for structural trend break, supremum Wald and likelihood-ratio (LR) tests, designed for cases when the breakpoints are unknown (Andrews, 1993, 2003; Hansen, 1997). Both supremum tests identify structural breaks within the 2009-2012 air quality improvement episode, thus associating them with the respective reversal of the upward trend in $PM_{2.5}$. Panel A shows that whenever the year 2010 is included in the test window, the maximal W-statistics are concentrated at this year, and the null hypothesis of no trend break can be rejected at the 1 percent level. When tested by the sup LR, the same is relevant for the year 2009 except that neither of the F-statistics is significant. Panel B uses analogous tests for structural trend break but designed for cases when it is pretended that the year of trend break is known. It shows that neither of the years within the 2005-2006 interval, the years of the largest pre-crisis drop in $PM_{2.5}$, are trend break years. This relaxes a concern about the possible confounding role of these years in the results.

Table 4. Results of the test suggested by Altonji, Elder, and Taber (2005)

Panel A. Districts with improved air quality

Dependent variable	% Boys	% Rural Deaths	avg. Order of Birth	% Male hh Heads	% Sch. Casts Heads	% Sch. Tribes Heads	% Hindu Heads	% Muslim Heads
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treated · Post	-0.55	-0.10	0.02	-0.89	-0.03	-0.75	-0.17	0.27
	(1.50)	(1.33)	(0.10)	(1.12)	(1.83)	(0.82)	(1.08)	(1.03)
Dependent variable	% Christian Heads	% Sikh Heads	% Buddhist Heads	% Illiterate Heads	% Literate w/o Ed.	% Lit. w/ Mid. Ed.	% Lit. w/ Sec. Ed.	% Lit. w/ Grad. Ed.
	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
Treated · Post	0.04	-0.09	0.11	1.34	1.34	-0.47	-1.40	-0.86
	(0.14)	(0.16)	(0.07)	(1.71)	(1.05)	(1.26)	(1.35)	(0.59)
Dependent variable	% Cultivator Heads	% Agri. Wage	% Non-Agri. Wage	% Self-Employed	% Reg. Salaried	% Did not work	% Pens. & Other	% Smoking Heads
	(17)	(18)	(19)	(20)	(21)	(22)	(23)	(24)
Treated · Post	2.02	-1.88	-4.60**	0.87	0.74	1.29	-0.26	-2.07
	(1.44)	(1.34)	(1.93)	(1.12)	(0.89)	(1.30)	(1.23)	(1.69)
Dependent variable	% Alcohol Drinkers	% HH w/ Filt. Water	% HH w/ Electricity	% HH w/ Kerosene	% HH w/ Solar	% HH w/ Oils	% HH w/ Any Other	% HH w/ Firewood
	(25)	(26)	(27)	(28)	(29)	(30)	(31)	(32)
Treated · Post	-0.84	-0.47	1.45	-1.49	0.23	-0.08	-0.07	0.70
	(1.52)	(0.80)	(2.37)	(2.51)	(0.31)	(0.11)	(0.21)	(1.40)
Dependent variable	% HH w/ Cr. Residue	% HH w/ Cow Dung	% HH w/ Coal	% HH w/ Kerosene	% HH w/ LPG/PNG	% HH w/ Electricity	% HH No Cooking	% HH Cook Inside
	(33)	(34)	(35)	(36)	(37)	(38)	(39)	(40)
Treated · Post	-0.33	0.73	0.37	0.05	-1.54	-0.07	0.06	1.50
	(0.67)	(1.30)	(0.27)	(0.19)	(1.26)	(0.05)	(0.24)	(1.14)
Dependent variable	% HH w/o Toilet	ln(GDP per capita)	Num. of Births	Num. Popuation	avg. Age of Mothers	% Married Mothers	% No ANC Received	% Gov. Deliveries
	(41)	(42)	(43)	(44)	(45)	(46)	(47)	(48)
Treated · Post	-0.16	-0.03	110.03	32,257.84	-0.13	-0.05	-7.24***	1.93
	(1.43)	(0.07)	(86.60)	(31,695.21)	(0.17)	(0.11)	(1.91)	(2.14)
Dependent variable	% Babies No CheckUp	% Babies w/ Vaccine	avg. p/a Air Temp	avg. p/a Precipitation	avg. U-Wind Direct.	avg. V-Wind Direct.	avg. Wind Speed	
	(49)	(50)	(51)	(52)	(53)	(54)	(55)	
Treated · Post	-5.03*	2.58***	0.14***	1.82	-0.12***	0.11***	-0.16***	
	(2.62)	(0.80)	(0.02)	(3.70)	(0.02)	(0.03)	(0.03)	
District FE	Y	Y	Y	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y	Y	Y	Y

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

Notes: The table further validates the DID identifying assumption of the model in equation (4). The test suggested by Altonji, Elder, and Taber (2005) examines whether the impact of the crisis has any association with changes in observable characteristics. Although this is not a formal test for exclusion restrictions, the absence of a statistically significant association with observable characteristics would suggest that there should not be a correlation with unobservable variables either (Altonji et al., 2005). I first successively regress my empirical model with every observable characteristic as a dependent variable. Then, I check whether the coefficients on the interaction term, $\widehat{\delta_1}$, are statistically significant. Table 4 presents results for both types of treated districts in Panel A and Panel B, respectively. Heteroskedasticity-robust standard errors clustered at the district level are shown in parentheses.

Panel B. Districts with worsened air quality

Dependent variable	% Boys	% Rural Deaths	avg. Order of Birth	% Male hh Heads	% Sch. Casts Heads	% Sch. Tribes Heads	% Hindu Heads	% Muslim Heads
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treated · Post	-2.39	0.14	-0.21*	-2.05	1.72	-1.49	-3.39	-1.03
	(2.04)	(1.19)	(0.12)	(1.39)	(1.32)	(1.02)	(2.54)	(2.02)
Dependent variable	% Christian Heads	% Sikh Heads	% Buddhist Heads	% Illiterate Heads	% Literate w/o Ed.	% Lit. w/ Mid. Ed.	% Lit. w/ Sec. Ed.	% Lit. w/ Grad. Ed.
	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
Treated · Post	4.70	-0.02	0.05	2.91	0.28	-1.34	-1.37	-0.53
	(3.93)	(0.08)	(0.06)	(2.37)	(1.13)	(1.92)	(1.28)	(0.68)
Dependent variable	% Cultivator Heads	% Agri. Wage	% Non-Agri. Wage	% Self-Employed	% Reg. Salaried	% Did not work	% Pens. & Other	% Smoking Heads
	(17)	(18)	(19)	(20)	(21)	(22)	(23)	(24)
Treated · Post	-0.20	1.96	-3.09	1.30	-0.55	0.95	1.63*	0.19
	(1.85)	(1.68)	(1.98)	(1.07)	(0.89)	(1.36)	(0.88)	(1.87)
Dependent variable	% Alcohol Drinkers	% HH w/ Filt. Water	% HH w/ Electricity	% HH w/ Kerosene	% HH w/ Solar	% HH w/ Oils	% HH w/ Any Other	% HH w/ Firewood
	(25)	(26)	(27)	(28)	(29)	(30)	(31)	(32)
Treated · Post	-2.04	0.52	1.17	-1.17	0.32	0.03	-0.29	0.80
	(1.66)	(1.11)	(2.02)	(2.06)	(0.33)	(0.16)	(0.47)	(1.73)
Dependent variable	% HH w/ Cr. Residue	% HH w/ Cow Dung	% HH w/ Coal	% HH w/ Kerosene	% HH w/ LPG/PNG	% HH w/ Electricity	% HH No Cooking	% HH Cook Inside
	(33)	(34)	(35)	(36)	(37)	(38)	(39)	(40)
Treated · Post	-1.05	0.43	-0.21	-0.06	0.32	-0.08	-0.24	0.16
	(1.44)	(1.22)	(0.48)	(0.18)	(0.83)	(0.07)	(0.31)	(1.46)
Dependent variable	% HH w/o Toilet	ln(GDP per capita)	Num. of Births	Num. Popuation	avg. Age of Mothers	% Married Mothers	% No ANC Received	% Gov. Deliveries
	(41)	(42)	(43)	(44)	(45)	(46)	(47)	(48)
Treated · Post	-1.70	0.02	-54.86	-209.69*	0.02	-0.02	-4.10***	0.81
	(1.55)	(0.09)	(187.42)	(119.82)	(0.11)	(0.10)	(1.50)	(1.31)
Dependent variable	% Babies No CheckUp	% Babies w/ Vaccine	avg. p/a Air Temp	avg. p/a Precipitation	avg. U-Wind Direct.	avg. V-Wind Direct.	avg. Wind Speed	
	(49)	(50)	(51)	(52)	(53)	(54)	(55)	
Treated · Post	-0.12	-0.49	0.11***	32.66***	0.30***	-0.01	0.13***	
	(1.84)	(1.12)	(0.02)	(5.25)	(0.02)	(0.03)	(0.03)	
District FE	Y	Y	Y	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y	Y	Y	Y

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

Table 5. Effects of the changes in particulate air pollution on infant mortality

Dependent variable = ln(Infant Mortality Rate)	Districts with improved air quality		Districts with worsened air quality	
	(1)	(2)	(1)	(2)
	Detrended district-level data, all causes of death, 2007-2011			
Treated · Post	-0.26***	-0.25***	0.08	0.09
	(0.08)	(0.08)	(0.13)	(0.13)
Treated · Post · t		-0.17**		-0.24***
		(0.07)		(0.08)
Observations	1,115	1,115	1,007	1,007
R-squared	0.37	0.38	0.27	0.29
District FE	YES	YES	YES	YES
Year FE	YES	YES	YES	YES
District-specific trends	YES	YES	YES	YES

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

Notes: The table presents baseline results of the regression analysis by reporting the key estimates from fitting equations (4) and (5). The dependent variable is the infant mortality rate for all causes of deaths. For both types of treated districts, columns (1) report the estimate of coefficient δ_1 after the estimation of equation (4), which tests for the effects of the crisis-induced changes in $PM_{2.5}$ on the infant mortality rate after adjustment for district fixed effects, year fixed effects and differential trends. The second columns report the results from equation (5) allowing for both level and slope changes during the post-crisis period. All regressions are run on the variables detrended as described in section 3 of the paper. Standard errors clustered at the district level are shown in parentheses.

Table 6. Sensitivity analysis

Dependent variable = ln(Infant Mortality Rate)	Districts with improved air quality											
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Treated · Post	-0.26***	-0.25***	-0.31***	-0.30***	-0.31***	-0.30***	-0.32***	-0.32***	-0.31***	-0.31***	-0.28***	-0.29***
	(0.08)	(0.08)	(0.09)	(0.09)	(0.09)	(0.09)	(0.08)	(0.09)	(0.09)	(0.09)	(0.07)	(0.07)
Treated · Post · t		-0.17**		-0.04		-0.04		-0.04		-0.03		0.02
		(0.07)		(0.07)		(0.07)		(0.07)		(0.07)		(0.05)
ln(GDP per capita)	-0.03	-0.03	0.06	0.06	0.06	0.06	0.04	0.04	0.03	0.03	-0.02	-0.02
	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.04)	(0.04)
Observations	1,115	1,115	1,115	1,115	1,115	1,115	1,110	1,110	1,110	1,110	1,110	1,110
R-squared	0.37	0.38	0.43	0.43	0.43	0.43	0.47	0.47	0.48	0.48	0.73	0.73
Dependent variable = ln(Infant Mortality Rate)	Districts with worsened air quality											
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Treated · Post	0.08	0.09	-0.07	-0.03	-0.08	-0.03	-0.14	-0.10	-0.16	-0.12	0.03	0.03
	(0.13)	(0.13)	(0.15)	(0.16)	(0.15)	(0.16)	(0.15)	(0.16)	(0.15)	(0.16)	(0.08)	(0.08)
Treated · Post · t		-0.23***		-0.32***		-0.34***		-0.30***		-0.29***		-0.03
		(0.08)		(0.09)		(0.10)		(0.10)		(0.10)		(0.08)
ln(GDP per capita)	0.09	0.07	0.15**	0.13**	0.15**	0.13**	0.11*	0.10*	0.11*	0.10	0.01	0.01
	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.04)	(0.04)
Observations	1,007	1,007	1,007	1,007	1,005	1,005	1,001	1,001	1,001	1,001	1,001	1,001
R-squared	0.27	0.29	0.31	0.32	0.31	0.32	0.36	0.38	0.38	0.39	0.68	0.68
Income per capita	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Meteorology	-	-	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Infant	-	-	-	-	Y	Y	Y	Y	Y	Y	Y	Y
Head of household	-	-	-	-	-	-	Y	Y	Y	Y	Y	Y
Household	-	-	-	-	-	-	-	-	Y	Y	Y	Y
District & Med. System	-	-	-	-	-	-	-	-	-	-	Y	Y

*** p<0.01, ** p<0.05, * p<0.1

Notes: The table addresses concern that changes in the dependent variable may be explained by changes in the observable time-varying characteristics that potentially correlated with the impact of $PM_{2.5}$ pollution changes attributable to the effect of the crisis. For that purpose, I perform a sensitivity analysis. The table reports results for both types of districts. Every pair of columns represents estimates from fitting equations (4) and (5). All regressions include district FE, year FE, district-specific trends. Heteroskedasticity-robust standard errors clustered at the district level are shown in parentheses.

Table 7. Falsification test / Pathophysiological mechanism: Causes of deaths

Dependent variable = ln of Infant Mortality Rate due to	Respiratory diseases	Diarrhea / Dysentery	Congenital / Birth Defects	Preterm Birth Low Weight	Convulsions
	(1)	(2)	(3)	(4)	(5)
Tretaed · Post	-0.441*	-0.399	-0.024	0.161	-0.592**
	(0.260)	(0.263)	(0.310)	(0.314)	(0.276)
Observations	1,110	1,110	1,110	1,110	1,110
R-squared	0.779	0.700	0.725	0.745	0.749

Dependent variable = ln of Infant Mortality Rate due to	Hypothermia	Fever with Jaundice	Asphyxia	Bleeding (umbilicus)	Infections
	(6)	(7)	(8)	(9)	(10)
Tretaed · Post	-0.058	-0.566**	-0.160	0.036	-0.184
	(0.287)	(0.265)	(0.301)	(0.224)	(0.282)
Observations	1,110	1,110	1,110	1,110	1,110
R-squared	0.789	0.701	0.772	0.679	0.826

Dependent variable = ln of Infant Mortality Rate due to	Birth Injuries	Jaundice	Fever with Convulsions	Fever with Rash	Other
	(11)	(12)	(13)	(14)	(15)
Tretaed · Post	-0.170	-0.133	-0.482*	-0.397	-0.085
	(0.285)	(0.288)	(0.251)	(0.252)	(0.154)
Observations	1,110	1,110	1,110	1,110	1,110
R-squared	0.680	0.766	0.785	0.761	0.869

*** p<0.01, ** p<0.05, * p<0.1

Notes: The table shows the results from one of the falsification test and the results from examination of the channels through which air pollution affects infant mortality. For the falsification test, I replace the dependent variable in equation (4) with another outcome variable that is not affected by the crisis-induced changes in air pollution. Specifically, I use the infant mortality rate due to diarrhea/dysentery as the alternative dependent variable to evaluate the internal validity of the previous estimates. As expected, regressions return statistically insignificant coefficients. For examination of the channels, the table reports the effect of the crisis-induced changes in $PM_{2.5}$ pollution on infant mortality disaggregated by various diseases. The results indicate that the effects of $PM_{2.5}$ pollution on infant mortality are specific for respiratory infections and might be related to some of the infectious diseases. The analysis is based on the specifications in columns (11) and (12) from Table 6 and focuses on the treated group of the districts with improved air quality. Each column presents results from the regressions with IMR due to fifteen causes of deaths as the dependent variable. All regressions include district FE, year FE, district-specific trends; Controls: income per capita, meteorology characteristics of the deceased infants, head of household, household, medical services utilization, other. Heteroskedasticity-robust standard errors clustered at the district level are shown in parentheses

Table 8. Falsification tests: Pre-crisis data sample, placebo $Post_t$= 2008

Dependent variable = ln(Infant Mortality Rate)	(1) Group 1	(2) Group 2	(3) Group 1	(4) Group 2	(5) Group 1	(6) Group 2	(7) Group 1	(8) Group 2	(9) Group 1	(10) Group 2	(11) Group 1	(12) Group 2
Treated · Post	0.03	0.03	0.11	-0.12	0.10	-0.13	0.07	-0.09	0.08	-0.11	0.08	-0.11
	(0.06)	(0.09)	(0.08)	(0.10)	(0.08)	(0.10)	(0.08)	(0.11)	(0.09)	(0.10)	(0.08)	(0.08)
ln(GDP per capita)	-0.00	-0.11	0.00	-0.16	-0.00	-0.17	-0.01	-0.16	0.01	-0.18	-0.03	-0.13
	(0.12)	(0.15)	(0.13)	(0.16)	(0.12)	(0.15)	(0.13)	(0.16)	(0.13)	(0.15)	(0.12)	(0.12)
Observations	452	414	452	414	452	414	450	412	450	412	450	412
R-squared	0.41	0.37	0.47	0.44	0.48	0.48	0.56	0.54	0.62	0.60	0.73	0.74
District FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
District-specific trends	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Controls:	.	.	.	.	.	.	.	.	.	.	.	.
Income per capita	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Meteorology	-	-	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Infant	-	-	-	-	Y	Y	Y	Y	Y	Y	Y	Y
Head of household	-	-	-	-	-	-	Y	Y	Y	Y	Y	Y
Household	-	-	-	-	-	-	-	-	Y	Y	Y	Y
District & Med. System	-	-	-	-	-	-	-	-	-	-	Y	Y

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

Notes: The table shows the results of another falsification test. I re-estimate the model using observations only from the pre-crisis period when the effects of the crisis-induced reduction in air pollution could not exist. I assign 2008 as a placebo trend break point and thus consider 2007 as the pre-crisis period, and 2008 as the post-crisis period. The table reports point estimates after fitting equation (4) with different combinations of W_{dt} and X_{dt} vectors for the districts from both treated groups with improved (Group 1) and worsened (Group 2) air quality. As expected, the regressions return statistically insignificant coefficients. Heteroskedasticity-robust standard errors clustered at the district level are shown in parentheses.

Table 9. Additional falsification and robustness checks

Specifications	Coeff. on *Treated · Post*
Falsification Tests	
Not detrended pre-crisis observations	0.09
	(0.07)
Common support alternative control	-0.28***
	(0.07)
Robustness Checks	
NSS regions*year FE	-0.28***
	(0.07)
State*year FE	-0.28***
	(0.07)
No weighting	-0.28***
	(0.06)
Weighting by number of births	-0.25***
	(0.06)
Cluster at NSS regions level	-0.28***
	(0.06)
Controls:	.
Income per capita	Y
Meteorology	Y
Infant	Y
Head of household	Y
Household	Y
District & Med. System	Y

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

Notes: The table provides results of the additional falsification and robustness checks of my main finding. See the text for explanations.

Table 10. Pathophysiological mechanism: Timing of infants' deaths

Dependent variable = ln(Infant Mortality Rate)	within 1 day	early neonatal	late neonatal	neonatal (28 days)
	(1)	(2)	(3)	(4)
Treated · Post	-0.10	-0.22***	-0.13	-0.24***
	(0.12)	(0.08)	(0.14)	(0.07)
Observations	1,110	1,110	1,110	1,110
R-squared	0.48	0.63	0.35	0.69

Dependent variable = ln(Infant Mortality Rate)	postneonatal (1-11 months)	within 11 months	between 11-12 months	stillbirth
	(5)	(6)	(7)	(8)
Treated · Post	-0.38***	-0.30***	-0.21	0.17
	(0.09)	(0.07)	(0.14)	(0.17)
Observations	1,110	1,110	1,110	1,110
R-squared	0.63	0.73	0.41	0.20

*** p<0.01, ** p<0.05, * p<0.1

Notes: The table shows the results from examination of the channels through which air pollution affects infant mortality. It presents the estimated effects of the reductions in $PM_{2.5}$ pollution on the infant mortality rate within 1 day, 28 days, between 28 days and 11 months, within 11 months and between 11 and 12 months of life. The second category is also known as the neonatal infant mortality rate, which in turn is broken down into early and late neonatal mortality rates corresponding to the deaths occurring within 0-7 and 8-27 days from birth, respectively. The third category is usually referred to as a postneonatal mortality rate. Separate analysis of these categories is performed purposefully. The large and statistically significant estimate in the neonatal period would likely suggest that particulate air pollution affects infant mortality through the adverse effects on fetal development, via in utero exposure to $PM_{2.5}$. Newborns whose mothers where exposed to high $PM_{2.5}$ concentrations during pregnancy have a higher probability of dying in the neonatal than the postneonatal period. In contrast, a large and statistically significant effect in the postneonatal period would highlight the importance of post-birth $PM_{2.5}$ exposure in the biological mechanism through which air pollution affects infants directly. The analysis is based on the specifications in columns (11) and (12) from Table 6 and focuses on the treated group of the districts with improved air quality. All regressions include district FE, year FE, district-specific trends; Controls: income per capita, meteorology characteristics of the deceased infants, head of household, household, medical services utilization, other. Heteroskedasticity-robust standard errors clustered at the district level are shown in parentheses

APPENDIX

A1. Additional robustness checks

Table A1.1. Alternative sorting of districts into treatment and control groups (T&C)

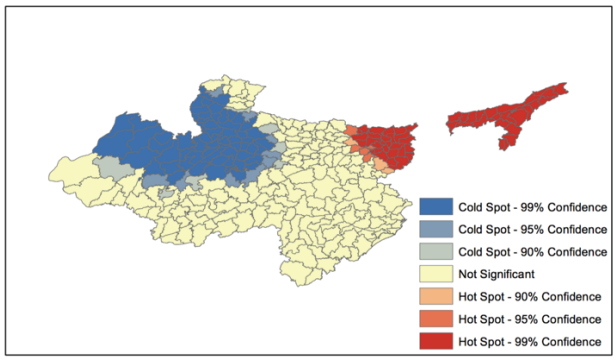
Dependent variable = ln(Infant Mortality Rate)	Hot Spot Analysis Reported results	Chay&Greenstone Simple change	Chay&Greenstone % change	Hot Spot Analysis Balanced panel	Hot Spot Analysis Adjacent T&C dist.	Hot Spot Analysis 10% drop 1	Hot Spot Analysis 10% drop 2	Hot Spot Analysis Drop adjasent T&C
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treated · Post	-0.283***	-0.305***	-0.287***	-0.270***	-0.323**	-0.293***	-0.282***	-0.257***
	(0.068)	(0.099)	(0.093)	(0.070)	(0.125)	(0.068)	(0.069)	(0.078)
Observations	1,110	550	553	1,040	257	1,095	1,095	853
R-squared	0.728	0.794	0.779	0.737	0.859	0.727	0.727	0.722
District FE	Y	Y	Y	Y	Y	Y	Y	Y
Year FE	Y	Y	Y	Y	Y	Y	Y	Y
District-specific trends	Y	Y	Y	Y	Y	Y	Y	Y
Controls:	.	.	.	.	.	.	.	.
Income per capita	Y	Y	Y	Y	Y	Y	Y	Y
Meteorology	Y	Y	Y	Y	Y	Y	Y	Y
Infant	Y	Y	Y	Y	Y	Y	Y	Y
Head of household	Y	Y	Y	Y	Y	Y	Y	Y
Household	Y	Y	Y	Y	Y	Y	Y	Y
District & Med. System	Y	Y	Y	Y	Y	Y	Y	Y

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

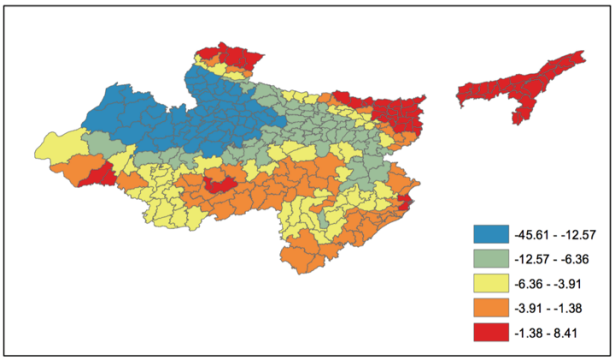
Notes: The table compares resulting estimates based on the model in equation (4) from the robustness checks that focus on alternative options for sorting districts into treatment and control groups. Each column of the table corresponds to one of the eight regressions, which are intuitively illustrated by the graphs in Figure A1 below. The sign, magnitude and order of statistical significance of the coefficients on Treated · Post remain similar between each other and to the coefficient estimated using my preferable specification. T&C refers to the treated and control districts. Heteroskedasticity-robust standard errors clustered at the district level are shown in parentheses.

Figure A1. Location of the treated and control districts: Illustrations to columns of the Table A1.1

Panel A. The location of the treated and control districts as in Column (1) of Table A1.1: Hot Spot Analysis (HSA)



Panel B. The location of the treated and control districts as in Column (2) of Table A1.1: 2008-2012 simple changes in $PM_{2.5}$ concentrations as in Chay and Greenstone (2003b)



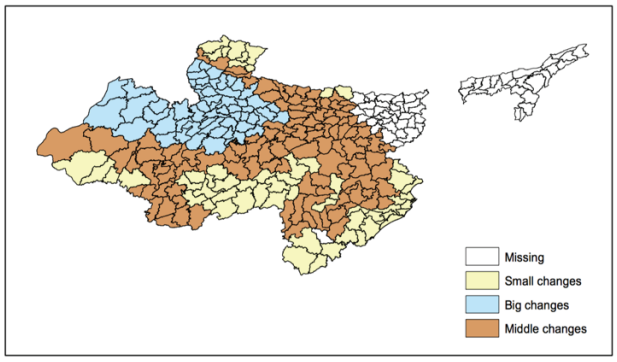
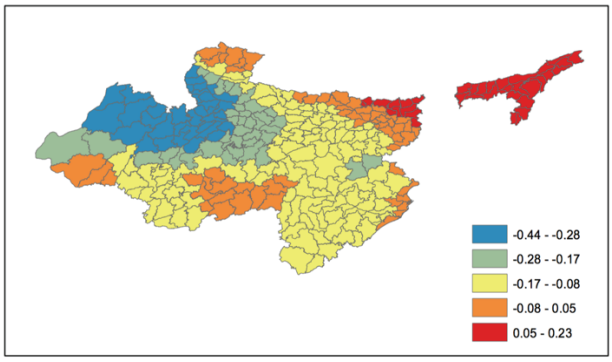
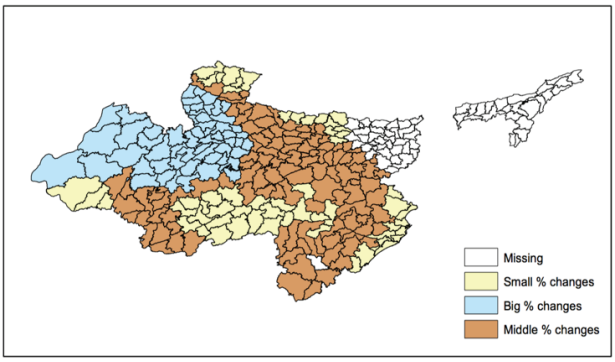


Figure A1 (continued). Location of the treated and control districts

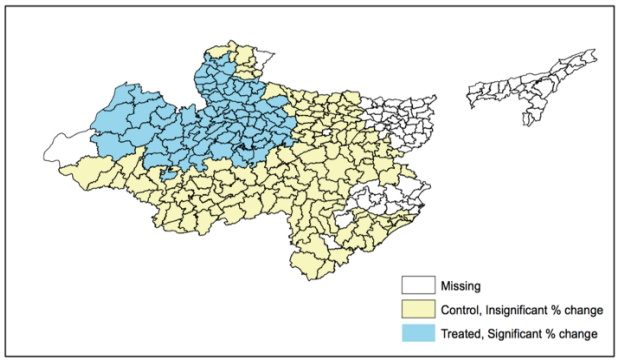
Panel C. The location of the treated and control districts as in Column (3) of Table A1.1: as in Chay and Greenstone (2003b) but alternatively based on 2008-2012 % changes in $PM_{2.5}$

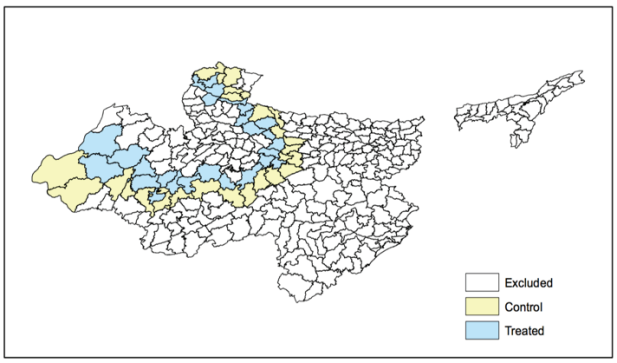




Panel D. The location of the treated and control districts as in Column (4) of Table A1.1: HSA, balanced panel

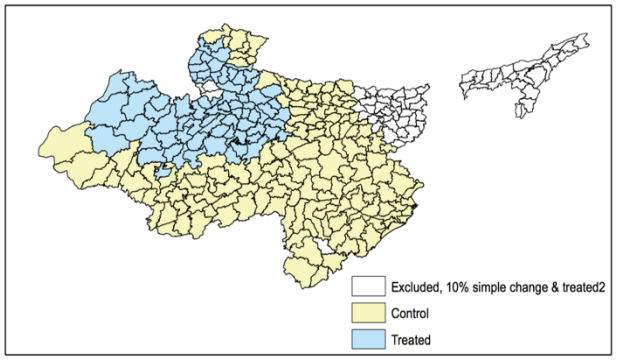
Panel E. The location of the treated and control districts as in Column (5) of Table A1.1: HSA, adjacent districts

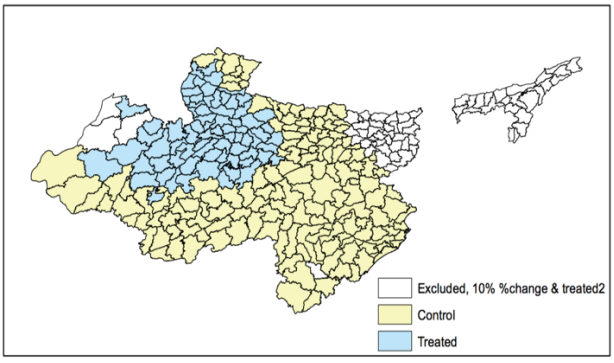




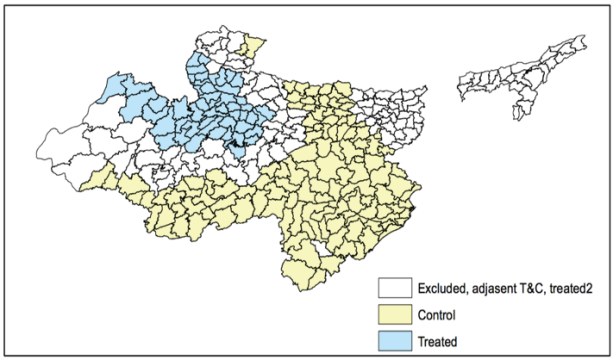
Panel F. The location of the treated and control districts as in Column (6) of Table A1.1: HSA, after drop of 10% of the most affected districts based on $PM_{2.5}$ change

Panel G. The location of the treated and control districts as in Column (7) of Table A1.1: HSA, after drop of 10% of the most affected districts based on % change in $PM_{2.5}$





Panel H. The location of the treated and control districts as in Column (8) of Table A1.1: HSA, after drop of the adjacent districts



Notes: The figure illustrates a column-wise location of the treated and control districts for the Table A1.1.

Table A1.2. Alternative estimation strategies

Dependent variable = ln(Infant Mortality Rate)	De-trended	Conditioning on Xs & districts' trends	Matching & DiD on pre-crisis Xs & districts' trends	Matching & DiD on pre-crisis Xs & districts' trends
	(1)	(2)	(3)	(4)
Treated · Post	-0.283***	-0.267***	-0.309***	-0.318***
	(0.068)	(0.092)	(0.114)	(0.086)
Observations	1,110	1,110	877	877
R-squared	0.728	0.891	0.887	0.701
District FE	Y	Y	Y	Y
Year FE	Y	Y	Y	Y
District-specific trends	Y	Y	Y	Y
Controls:	.	.	.	.
Income per capita	Y	Y	Y	Y
Meteorology	Y	Y	Y	Y
Infant	Y	Y	Y	Y
Head of household	Y	Y	Y	Y
Household	Y	Y	Y	Y
District & Med. System	Y	Y	Y	Y

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

Notes: The table compares resulting estimates based on the model in equation (4) from another set of robustness checks that focus on alternative estimation strategies. The test checks whether the main finding would remain robust to different estimation strategies, namely detrending, conditioning on the explanatory variables and district-specific trends, and the method combining matching on pre-crisis explanatory variables and trends with subsequent difference-in-differences. Column (1) shows the coefficient on Treated · Post estimated using my preferable specification based on detrending. Column (2) shows the same coefficient estimated using my preferable specification but based on the conditioning on explanatory variables and district-specific trends. Columns (3) and (4) show the results from the regressions that combine matching on pre-crisis explanatory variables and district-specific trends with specifications in columns (2) and (1), respectively. Matching is performed as a 1-to-1, nearest-neighbor, without replacement and with common support. In other words, the results in columns (3) and (4) are the estimated coefficients on Treated · Post obtained from the regressions like in columns (2) and (1) but on matched sample. The results indicate that all specifications return estimated coefficients of interest that are not substantially different from each other, thus confirming the credibility of the main analysis. Heteroskedasticity-robust standard errors clustered at the district level are shown in parentheses.

Abstrakt

Věrohodné odhady dopadů změn ve znečištění ovzduší na zdraví jsou důležité pro výzkum a formování enviromentálních politik, obzvláště pak v rozvojových zemích. Tento článek odhaduje vliv náhlé redukce znečištění ovzduší na kojeneckou mortalitu, která byla způsobena Velkou recesí v roce 2008. Změny jsou pozorovány v Indii na úrovni správních oblastí. S využitím možné exogenní geografické variace ve změnách kvality ovzduší způsobených krizí, nového datového souboru dotazníkového šetření mezi domácnostmi a satelitních dat zjišťuji, že kojenecká úmrtnost poklesla v nejvíce zasažených oblastech o 24 %. To představuje o 1338 méně úmrtí oproti situaci, že by krize nenastala. Analýza mechanismu naznačuje, že redukce $PM_{2.5}$ ovlivňuje kojeneckou úmrtnost zejména skrz respirační choroby a dva odlišné biologické mechanismy: vystavení se $PM_{2.5}$ před a po narození. Hrubé výpočty naznačují, že odhadovaný pokles v kojenecké úmrtnosti představuje v prvních třech letech po krizi zisk přibližně 312.5 milionů amerických dolarů. Výsledné zdravotní zlepšení by mohlo být použito jako benchmark pro vyhodnocování efektivity politik určených ke zlepšení kvality ovzduší v Indii.

Klíčová slova: znečištění vzduchu, kojenecká úmrtnost, krize, Indie

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