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Can environmental policy reduce infant mortality?

Evidence from the Ganga pollution cases

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Introduction

Environmental economics and development economics are unified by the persistent puzzle of poor air and water quality in developing countries (Greenstone and Jack 2015). A growing literature provides evidence that pollution imposes a significant health burden (e.g., Jayachandran 2008; Ebenstein 2012; and Brainerd and Menon 2014) and yet, corresponding estimates of willingness-to-pay for environmental quality are surprisingly low (Kremer et al. 2011) and policies aimed at improving environmental quality have not reliably done so (Greenstone and Hanna 2014; Field, Glennerster, and Hussam 2011). While high levels of air and water pollution may be due in part to high marginal utility of consumption (Hanna and Oliva 2015a) and high marginal costs of pollution abatement (Davis 2008), they are also likely driven by rent-seeking behavior (Duflo et al. 2013) and market failures (Jalan and Somanathan 2008), hence defining the scope of government regulation.

India provides a compelling setting in which to study developing-country environmental health and policy. The World Health Organization (2004) estimates that over 3 in every 1,000 Indian children under five in 2004 died because of water pollution. As of 2013, only 21 percent of the estimated daily sewage load in Indian cities could be treated (Central Pollution Control Board 2016), and the gap between load and treatment capacity is expanding (*Daily Mail* 2015). At the same time, the flagship policy for addressing water pollution in India – the National River Conservation Plan (NRCP), established in 1986 and still active today – has failed to improve water quality (Greenstone and Hanna 2014).

We investigate a landmark decision in India's environmental regulatory history: the Supreme Court case *M.C. Mehta vs. Union of India*, subsequently bifurcated and known as the "Ganga Pollution Cases". These cases represent India's first-ever environmental public-interest litigation, and their unprecedented ruling – which mandated pollution cleanup by the tanning industry concentrated along the Ganga River in Kanpur, Uttar Pradesh – marked the rise of environmental activism by the Indian judiciary (Mehta 2009). Exploiting the quasi-random incidence of the litigation, we find that the ruling is strongly associated with reductions in both river pollution and neonatal (one-month) mortality.

Our positive finding is an important data point because, in developing countries, there is very little evidence of environmental policies other than piped water provision being successful (Ravallion and Jalan 2003; Gamper-Rabindran, Khan, and Timmins 2010). However, having identified a reduced-form policy impact on health, a question remains: how did the Supreme Court ruling work? In general, pollution policy may affect health directly through improved environmental quality, but it may also do so through increased awareness and avoidance (Kremer et al. 2011; Graff-Zivin, Neidell, and Schlenker 2011) or follow-on action by communities and governments, among other channels. Reduced-form estimates of policy impacts aggregate the effects of all such channels.

We seek to show how a lack of data on all possible mechanisms, such as is common in quasi-experimental analysis, need not prevent the researcher from learning how a policy has worked. To do so in our specific context, we leverage a second source of variation in water quality that is orthogonal to the first: upstream river pollution. This instrument helps us identify the direct impact of water pollution on neonatal mortality – one particular channel through which the policy may have worked. Intuition suggests that if pollution is the *only* channel, then two statements should hold empirically: there should be no residual effect of policy on mortality after appropriately controlling for pollution and the policy should be a valid instrument for pollution.

Empirical tests of these two statements are therefore equivalent to tests of the null hypothesis that the policy affects mortality only through a reduction in pollution. They are also representative of a more general method of understanding policy impacts, in which outcomes of a policy change are compared to the outcomes of orthogonal changes in structural parameters (Angelucci and Attanasio 2013). We derive our version of this strategy from a basic theory of pollution, policy, and health, and we then use Two-Stage Least Squares (2SLS) to carry it out. In particular, our overidentification test shows that the judicial ruling is a valid instrument for river pollution alongside the upstream pollution instrument. In other words, we fail to reject the null hypothesis that the policy improved health only through pollution reductions.

Though the primary purpose of our instrumental variables (IV) strategy is to shed light on policy mechanisms, the strategy also provides an estimate of the water pollution-mortality dose response function. This is important in its own right: it demonstrates that exceedance of India's water quality standards, as measured by the general indicator Biochemical Oxygen Demand, significantly raises the risk of neonatal mortality. Furthermore, our reduced-form link between *upstream* pollution and *downstream* mortality indicates that such health consequences persist in downstream communities. Our research thus underscores the large social costs of continually poor environmental quality, as well as the negative spatial externality that is inherent to river pollution.

The remainder of this paper is organized as follows. Section 1 describes the Ganga Pollution Cases amidst the more general context of pollution and related policy in India. Section 2 introduces a simple model of pollution, policy, and health, which generates our estimating equations and statistical tests of the policy's impacts. Section 3 describes the various sources of data, while Section 4 provides our empirical results. Section 5 places our contribution within the environmental policy literature, especially with regard to the puzzle of persistently poor environmental quality in the face of large social costs of pollution. Section 6 concludes.

1 The Ganga Pollution Cases

In the aftermath of several decades of population and industrial growth, India's rivers are heavily polluted – particularly in urban areas (Murty and Kumar 2011). Monitoring of bioechmical oxygen demand (BOD – a broad-based measure of organic water pollution; see Section 3) carried out by India's Central Pollution Control Board (CPCB) suggests that water at approximately 40 percent of all sampling stations did not meet the government's standard of acceptability for bathing (BOD<3 mg/l; CPCB 2011).[1] The CPCB (2015) further observes that, as of 2013, Indian cities only have the capacity to treat approximately 30 percent of their daily sewage burden.

There have been efforts to improve Indian water quality, but there is little evidence that they have been successful. The most salient government effort to reduce river pollution is the National River Conservation Plan (NRCP), a national, top-down program targeting domestic pollution into India's surface waters. NRCP began in 1985 as the Ganga Action Plan but has expanded over 30 years to now cover 190 towns along 41 rivers across India. Its goal since 1987 has been to restore the Ganga River to the "Bathing Class" standard, as defined by India's "Designated Best Use" classification system. The primary lever for achieving this goal has been the "interception, diversion, and treatment" of sewage (Government of India 2003); to that end, 4,842 million-liters per day of sewage treatment capacity have been created since its inception (Ministry of Environment, Forests and Climate Change, Government of India, 2014:211). Nonetheless popular media and non-governmental organizations have panned NRCP for reasons such as poor inter-agency cooperation, funding imbalances across sites, and an inability to keep pace with growing sewage loads (Suresh 2007). Confirming public belief, Greenstone and Hanna (2014) find no discernible impact of NRCP on water quality levels.

The executive branch, however, is not the only source of environmental regulation in India; the Indian judiciary has, through the years, developed a reputation for environmental activism (Singh 2014). Article 21 of the Indian Constitution provides citizens with the "Right to Life", and much jurisprudence in recent years has centered on the protection of this constitutional right. This paper examines the first instance of Supreme Court involvement in issues of river pollution in India. The story begins in the pilgrimage city of Haridwar along the Ganga River; a matchstick tossed by a smoker resulted in the river catching on fire for more than 30 hours due to a toxic layer of chemicals produced by a pharmaceutical firm (Mehta 2009). In response to this event, environmental lawyer and social activist M.C. Mehta filed a writ petition in the Supreme Court of India charging that government authorities had not taken effective steps to prevent environmental pollution in the Ganga's waters.

The scale of the case, the whole 2,500-km stretch of the river, proved to be intractable. The court requested that Mr. Mehta narrow his focus; he chose the city of Kanpur (Supreme Court of India,

[1] These estimates are obtained from the CPCB website, http:cpcb.nic.inwater.php, accessed on July 8th, 2016.

1985). Kanpur is a city of 2.9 million people lying directly on the Ganga River in Uttar Pradesh State (see Figure 1). For more than 100 years, it has been a major center for India's tannery industry. Of the 400 tanneries currently located in Kanpur (*The Hindu* 2016), most are located in the neighborhood of Jajmau, which lies on the southern bank of the Ganga River. Leather processing is a highly polluting industry; the procedures for washing, liming, fleshing, tanning, splitting, and finishing involve a large number of chemicals (Cheremisinoff 2001). Tannery effluent is generally characterized by its strong color (reddish dull brown), high BOD, high pH, and high concentration of dissolved solids, as well as highly toxic chromium ions. In Jajmau, that effluent is routinely discharged from the tanneries directly into the river, rendering both river water and groundwater unfit for drinking, irrigation, and general consumption (Beg and Ali 2008, Tewari, Dubey, and Singh 2012). Mehta selected Kanpur despite not having been born in or lived in Kanpur. In interviews he granted our research team in June 2014, he explained that "[Kanpur] was in the middle of the Ganga basin, the reddish color of the pollution made the pollution highly salient, and the city seemed representative of many other cities in the Ganga Basin." The court subsequently split the petition into two parts. The first dealt with the tanneries of Kanpur and the second with the city government. These are now respectively called *Mehta I* and *Mehta II* in legislative digests, and are together known as the "Ganga Pollution Cases" – the foundational water pollution litigation in the Indian court system. In October 1987, the Court invoked the Water Act and Environment (Protection) Act as well as Article 21 of the Indian Constitution to rule in Mr. Mehta's favor and order the tanneries of Jajmau to clean their wastewater within six months or shut down entirely. This was followed by a January 1988 judgment that required the Kanpur local municipal bodies to take several immediate measures to control water pollution: the relocation of 80,000 cattle housed in dairies or the safe removal of animal waste from these locations; the cleaning of the city's sewers; the building of larger sewer systems; the construction of public latrines; and an immediate ban on the disposal of corpses into the river. The court also required all schools to devote one hour each week to environmental education and awareness.

Of the 87 tanneries named in Mr. Mehta's petition, approximately 20 were shut down and at least 60 established primary treatment plants (PTPs). Moreover, several "crash programs" were implemented in 1987 and 1988 to clean drains, expand the number of handpumps, and build latrines to improve sanitation systems in Jajmau (Alley 2002). Subsequent litigation in the Supreme Court over the past 25 years, and indeed many academic researchers of pollution in Kanpur, have argued that these projects were a failure and that newly established technologies were not appropriately maintained or used (Alley 2002, Singh 2006, Greenstone and Hanna 2014).

2 Pollution, Environmental Policy, and Health

We are interested in whether or not the 1987 Supreme Court decision affected environmental quality or health outcomes. As in the existing literature on environmental policy impacts (Greenstone and Hanna 2014; Spears 2012), we observe the incidence of a policy as well as some measures of target outcomes. We argue that the timing and geographic incidence of the Supreme Court decision was exogenous with respect to pollution and health: environmental public interest litigation had no prior precedent in India, and the selection of Kanpur was arbitrarily made by Mr. Mehta when asked to reduce the scale of his original petition. We therefore use difference-in-differences (DiD) regression – comparing outcomes in Kanpur to those elsewhere in the Ganga Basin, before and after the verdict – to estimate the impacts of *Mehta vs. Union of India* on water pollution and infant mortality, respectively. These estimates are denoted 'DiD' in Figure 3, a schematic diagram of our empirical strategy.

Our DiD estimates give a reduced-form impact of the ruling on health outcomes. But they cannot tell us about specific mechanisms of impact. As Figure 3 shows, policy may affect health through multiple channels: pollution levels and behavioral change are intuitive examples. To investigate these mechanisms, we isolate quasi-random variation in pollution, which allows us to identify the direct impact of pollution on mortality. Pollution is, in general, related to industrialization and urbanization – which themselves tend to improve health – so an Ordinary Least Squares regression of health outcomes on pollution levels is liable to be biased towards zero. To resolve this issue, we construct an instrument using upstream pollution levels, described at length in Section 3, and estimate the pollution-mortality dose-response function using Two Stage Least Squares ('IV' in Figure 3).

Figure 3 then shows how our estimated relationships line up. The ratio of the policy's health impact (the top 'DiD') to its pollution impact (bottom 'DiD') is equivalent to a 2SLS estimate of the pollution-health relationship using the policy as an instrument. In turn, we can compare that estimate to a 2SLS estimate using upstream pollution as the instrument. If they are different, then we can reject the null hypothesis that pollution is the only mechanism at work. Our econometric model in the subsections below builds up statistical tests of that hypothesis.

2.1 Reduced-form impact

To assess the impact of the Supreme Court ruling on welfare, we specify a simple reduced-form model of mortality:

$$Mortality_{idt} = a + bT_{dt} + X_{idt}\gamma + e_{idt} \tag{1}$$

$Mortality_{idt}$ is a dummy variable indicating whether a child i, born in district d, in year-month t, died within the first month of life. T_{dt} captures the incidence of policy – the *Mehta vs. Union of India*

court decision in our case – and takes a value of one in Kanpur after October 1987 and a value of zero otherwise. X_{idt} is a vector of individual, location-by-time characteristics, which includes district and year-month fixed effects.

The crux of our identification strategy is that $Cov\left(T_{dt}, e_{idt}\right) = 0$ – i.e., that the policy variable is uncorrelated with all unobserved predictors of neonatal mortality. Mehta's choice of Kanpur was primarily motivated by its central location and the salience of pollutants coming from its tanneries (Mehta 2014). Thus, Kanpur was *not* chosen on the basis of temporal trends in pollution, health, or citizen involvement. In fact, there is no evidence of any local movement to reduce pollution in the city in the mid-1980s (Jaiswal 2014).

2.2 Mechanisms

If the zero-covariance assumption above holds, b represents the net causal effect of policy on neonatal mortality, aggregated across all channels of impact. To gauge the *relative* importance of the various channels, we parsimoniously model the structural determinants of neonatal mortality rates as follows:

$$Mortality_{idt} = \alpha + \beta Poll_{dt} + \tilde{X}_{idt}\gamma + (Z_{idt}\delta + \varepsilon_{idt}) \tag{2}$$

Here, $Poll_{dt}$ is district-monthly average river pollution and $\tilde{X}_{idt}$ is a vector of observable mortality risk factors. We partition the space of *unobserved* risk factors into two: Z_{idt} and ε_{idt}. The former is a vector of all factors that are also correlated with environmental policy T_{dt}. These include, but are not restricted to, individual awareness about river water contamination, changes in factor prices stemming from the implementation of environmental policy T_{dt}, or any type of private or public interventions that might have been triggered by T_{dt}. The latter captures the other risk factors of infant mortality and is, by construction, such that $Cov\left(T_{dt}, \varepsilon_{idt} \mid \tilde{X}_{idt}\right) = 0$.

Our next step is to parameterize the relationship between policy and the the structural determinants of health (i.e., the right-hand side of equation [2]). We assume that Z_{idt} and $Poll_{dt}$ respond to environmental policy and other determinants in linear fashion. That is,

$$Z_{idt} = \alpha^1 + \beta^1 T_{dt} + \tilde{X}_{idt}\gamma^1 + \varepsilon^1_{idt} \tag{3}$$

and

$$Poll_{dt} = \alpha^2 + \beta^2 T_{dt} + \tilde{X}_{idt}\gamma^2 + \varepsilon^2_{idt} \tag{4}$$

We can then rewrite equation (2) by substituting for both Z_{idt} and $Poll_{dt}$, so as to decompose the

policy's effect on mortality into the different channels of impact:

$$\begin{aligned} Mortality_{idt} &= \left[\alpha + \beta\alpha^2 + \alpha^1\delta\right] + \left[\beta\beta^2 + \beta^1\delta\right] T_{dt} \\ &\quad + \tilde{X}_{idt}\left[\beta\gamma^2 + \gamma + \gamma_1\delta\right] + \left[\beta\varepsilon^2_{idt} + \varepsilon^2_{idt}\delta + \varepsilon_{idt}\right] \end{aligned} \tag{5}$$

The total impact of environmental policy T_{dt} on infant mortality (equal to b in reduced form) is here given by $\left[\beta\beta^2 + \beta^1\delta\right]$. The first term ($\beta\beta^2$) measures the contribution of the pollution channel, and the second ($\beta^1\delta$) aggregates all other channels; the challenge is to estimate each of these terms separately.

We estimate β^2 from equation (4). The remaining components of the policy-mortality relationship are obtained from equation (2), by substituting the right-hand side of equation (3) in for the unobservable Z_{idt}. This yields

$$Mortality_{idt} = \left[\alpha + \alpha^1\delta\right] + \beta Poll_{dt} + \tilde{X}_{idt}\left[\gamma + \gamma^1\delta\right] + \left[\beta^1\delta\right] T_{dt} + \left(\varepsilon^1_{idt}\delta + \varepsilon_{idt}\right) \tag{6}$$

which contains both β and the product $\beta^1\delta$.

However, equation (6) cannot be estimated without bias using OLS. This is because pollution is not random; in general, it is correlated with other factors affecting mortality, such as urbanization (which brings access to health care facilities and education) and economic productivity (which raises incomes). We address this endogeneity problem by revisiting equation (4) and positing that measured pollution levels in district d and year-month t are additionally driven by pollution *upstream* of district d in that time period, denoted $Poll^{-1}_{dt}$. By writing $\varepsilon^2_{idt} = \tilde{\varepsilon}^2_{it} + \eta^2 Poll^{-1}_{dt}$, we obtain

$$Poll_{dt} = \alpha^2 + \beta^2 T_{dt} + \tilde{X}_{idt}\gamma^2 + \eta^2 Poll^{-1}_{dt} + \tilde{\varepsilon}^2_{it}, \tag{7}$$

If upstream pollution affects downstream infant mortality rates only through its persistence as the river flows – that is, $Cov\left(Poll^{-1}_{dt}, Z_{idt}\delta + \varepsilon_{idt}|\tilde{X}_{idt}\right) = 0$ – then we can estimate equation (6) using two-stage least squares (2SLS). This, in turn, gives unbiased estimates of both β and $\beta^1\delta$. We can then test $H_0 : \beta^1\delta = 0$.

Note that under this null hypothesis, equation (6) can be rewritten

$$Mortality_{idt} = \left[\alpha + \alpha^1\delta\right] + \beta Poll_{dt} + \tilde{X}_{idt}\left[\gamma + \gamma^1 \cdot \delta\right] + \left(\varepsilon^1_{idt}\delta + \varepsilon_{idt}\right) \tag{8}$$

so that T_{dt} is additionally excluded from second-stage equation (8) and becomes another valid instrument for $Poll_{dt}$. One test of H_0 is therefore an over-identification test that assesses the orthogonality condition for T_{dt}, as part of the larger set of instruments $\left\{T_{dt}, Poll^{-1}_{dt}\right\}$. To implement such a test, we

construct a *C-statistic* (see, e.g., Eichenbaum, Hansen, and Singleton 1988), also known as a difference-in-Sargan test statistic. This statistic is equal to the difference of the two Sargan-Hansen *J-statistics* obtained from the regression using both T_{dt} and $Poll_{dt}^{-1}$ as instruments on the one hand and the regression using only $Poll_{dt}^{-1}$ on the other hand.

3 Data

3.1 Pollution data

Our main source of data is a subset of the universe of data collected under India's national water quality monitoring program, culled from a combination of CPCB online and print records. These data were originally gathered and used by Greenstone and Hanna (2014). We limit our analysis to the years 1986-2004, because our most recent infant mortality data are from 2004 and our earliest pollution data are from 1986. In most of our analysis, we further restrict our sample to the geographic region encompassed by the Ganga River Basin, depicted in Figure 1. We make this second restriction because of the singularity of this area in the context of our analysis. The Ganga River Basin is not only a much more densely populated region than anywhere else in India; it is also a region in which water issues have received special government attention. As an example, the National River Conservation Plan (NRCP) focused exclusively on the Ganga River Basin (including the Ganga, Yamuna, Damodar, Gomti, and Mahananda Rivers) throughout its first 10 years (1986-1995). Extending the analysis beyond the Ganga and its tributaries might then confound the effect of the Supreme Court ruling with the effect of broad scrutiny in the Ganga Basin during this time period.

The aforementioned sample restrictions produce a set of 101 unique pollution monitors situated along 29 rivers within the Ganga Basin. Over the 19 year sample time period, this set provides 13,466 monitor-month observations of water quality. As many as 46 different measures of water quality are recorded at these monitoring stations, but only a few measures are consistently recorded over the whole sample timeframe. To mitigate measurement errors and missing values, we construct moving averages of the data over a four-month window at the district level.

For our analysis, we choose to focus primarily on BOD. This common, broad-based indicator of water pollution measures the amount of dissolved oxygen needed by water-borne, aerobic organisms to break down organic material present (at a certain temperature, over a specific time period). Its units are milligrams of oxygen consumed per liter (mg/l). Reduction of BOD is the primary goal of waste treatment plants in general (Brown and Caldwell 2001), but BOD is a particularly good choice for pollution measurement in the setting of Kanpur. Pollution from the tanning process primarily comes from two sources: the animal hides themselves, and the chemicals used to tan them. Both of

these sources contain large amounts of organic matter, and this results in abnormally high BOD levels in tannery effluent. According to the United Nations Industrial Development Organization (UNIDO 2011), effluent discharge into surface water typically is required to have BOD below 30-40 mg/l, while the typical BOD in *raw* tannery effluent is approximately 2,000.[2]

To support the evidence provided by BOD, we also consider four other pollutants that shed light on the impacts of the Kanpur Supreme Court verdicts: calcium, sulfate, chloride, and fecal coliforms (FCOLI). Calcium is the key component of lime, which is a standard ingredient used in the removal of hair and flesh and the splitting of the hide into its two primary layers. Sulfate and chloride ions, meanwhile, are the main components of the TDS produced in tanning. FCOLI is a measure of domestic (as opposed to industrial) pollution, which is the major focus of the National River Conservation Plan.

Together, calcium and sulfate are a robustness check on our primary BOD-based analyses: if the policy truly reduced pollution, and if what we are capturing in our difference-in-difference analysis is indeed that policy impact, then we should find a reduction in these pollutants after policy implementation. Chloride and FCOLI, on the other hand, provide falsification checks. The former is present in high numbers in tannery effluent but is so soluble in water that it is not affected by standard tannery waste treatment (UNIDO 2011). The latter should not be significantly affected by the rulings, since it is not produced in large quantities by tanning.

Our econometric model and identification strategy rely heavily on the measurement of *upstream* pollution values. Such measurement requires information on the precise location of pollution monitors. Unfortunately, latitude and longitude of monitors are incomplete and unreliable in our dataset. To circumvent this problem, we manually map each monitor according to the administrative descriptors provided (state, town, river) and an accompanying string description of location (e.g., "Sabarmati at Ahmedabad at V.N. Bridge"). With our monitors mapped, we trace the path of all rivers in our sample, from origin to last monitor downstream, and measure distances between all pairs of neighboring monitors.

Many water quality monitors in our dataset have more than one possible upstream counterpart. There is no single 'correct' distance at which to measure upstream pollution. When two monitors are far from each other, upstream pollution will be a weaker instrumental variable, due to pollution decay. On the other hand, close monitors are more likely to be subject to common weather or economic shocks such as rainfall, which create a spurious correlation. We therefore adopt a variety of definitions of "upstream", and check the robustness of our results to the choice of a specific instrumental variable. To assign an upstream counterpart to a given pollution monitor, we use the following algorithm:

[2]Total suspended solids (TSS), and total dissolved solids (TDS) are potential alternatives to BOD in analysis, but the first of these is not recorded in large numbers in our data, and the second does not provide adequate coverage of our policy pre-period. Chromium, perhaps the highest-profile pollutant in the tanning process, was not widely measured by the CPCB as of 2004.

first, we follow the river upstream until it reaches a new district; then, we locate the nearest monitor along the river that falls within a distance range (in km) of $[X, Y]$ from the original monitor, where $X \in \{0, 20, 50, 75, 100\}$ and $Y \in \{200, 300\}$. When a river splits upstream of a given monitor, so that there is an upstream monitor on each of two tributaries, we take the average of these monitors as our upstream measure. When there is no upstream monitor of any kind to be found, we use the river's origin as an upstream location (subject to the distance-range requirement) and assign the sample-wide minimum value of pollution as our upstream measure.

3.2 Health data

For our study of the health burden imposed by river pollution in India, we choose infant mortality as our key health outcome. This choice follows those of many others in the literature (e.g., Greenstone and Hanna 2014 and Brainerd and Menon 2014) and is motivated by science, policy, and statistical considerations. Research in epidemiology has shown that infants are highly susceptible to pollution (Fewtrell and Bartram 2001). Even *in utero* exposure can have long-term impacts on individual welfare, through channels such as birth weight, cognitive development and susceptibility to diseases (Currie 2008 and Currie and Almond 2011). The vulnerability of infants to water pollution is of particular policy interest in India, where infant death rates have been high relative to the global average (World Development Indicators, 2016).[3] While infant mortality is clearly an incomplete measure of the health costs imposed by water pollution, it nonetheless represents a very large loss of life in the Indian context.

Furthermore, the use of infant mortality as an outcome of interest conveys at least two significant statistical advantages. The first is general to infant health outcomes, as noted by Chay and Greenstone (2003): newborns do not have a long history of prior exposure to pollution, so the link between water quality and their health is immediate, and an analysis of pollution levels during the first year of life nearly fully captures lifetime exposure (in direct contrast with studies of adult, or even under-5, mortality). The second pertains to statistical power: complete birth histories are available in certain Indian demographic surveys, so we can construct long pseudo-panels of infant survival status.[4] Panel variation in infant mortality allows us to include detailed temporal and cross-sectional fixed effects in regression analysis, removing some of the concern we have about omitted variable bias.

Our infant health data come from the Reproductive and Child Health II (RCH-2) module of the District-Level Household Survey II (DLHS-2), a national demographic survey conducted in two phases from 2002 to 2005. In the RCH-2 module, mothers report age and survival for all of their children;

[3] Indian infant mortality rate, measured in terms of deaths per thousand children, in the years 1990, 2000 and 2010 was 88, 66, and 46. The world average was 63, 51 and 38 respectively (World Development Indicators, 2016). The data was accessed at http://data.worldbank.org/indicator/SP.DYN.IMRT.IN on July 30, 2016.

[4] Variables such as diarrhea incidence and low birth weight, on the other hand, are only available cross-sectionally from the time of survey.

from these birth histories, we create a panel of child-month mortality in the first month ('neonatal') and in the first year ('infant'). Starting with a raw total of 1,393,431 births from 1967 through 2004, we match each birth to pollution data from the district in which the birth took place.[5] This restricts our sample to 161,139 births. Further limiting our analysis to the Ganga Basin produces a sample of 33,932 children spanning 50 districts and 9 states.

3.3 Other data

We include several types of variables as controls in many of our regression analyses. The main body of these controls is composed of cross-sectional survey answers about birth, mother, and child characteristics taken from RCH-2. A pair of climate controls are created using monthly, gridded rainfall averages from the University of Delaware and air temperature averages from the Indian Meteorological Institute; we use these gridded averages to interpolate rainfall and temperature values at each monitor-month. Finally, we add variables that measure Common Effluent Treatment Plant (CETP) capacity and the incidence of NRCP.

4 Empirical Results

We begin our analysis with a brief statistical description of the key variables measuring mortality, pollution, and policy. We then move on to an exploration of policy impacts, using a difference-in-differences framework. We estimate equation (1) in order to identify the infant mortality impacts of the Supreme Court verdict; and we estimate equation (4) to identify the pollution impacts. After establishing these impacts, we then use instrumental variables to investigate the channels of the policy-health linkage. We estimate equation (6) via both Ordinary Least Squares (OLS) and Two-Stage Least Squares (2SLS) with our upstream instrument; we compare the latter specification to a specification using both upstream pollution and the policy as instruments − which corresponds to 2SLS estimation of equation (8).

4.1 Summary statistics

Table 1 reports summary statistics for the key variables used in our regression analyses. With respect to health, we focus on neonatal (i.e., one-month) mortality because 95 percent of infant (i.e., one-year) mortality occurs in the first month of life. The adverse effect of pollution beyond the first month is thus more likely to be captured by morbidity, which we do not observe over time. With this focus on the first month of life, we first observe that, from 1986-2004, the national average of district-level neonatal

[5] In DLHS-2 as well as all other potential data sources of which we are aware, child location is only available down to the district level.

mortality indicates that 5 percent of a district's newborns die before the end of the first month of life. From the right panel of Table 1, that neonatal mortality rate is even higher in the Ganga Basin at 6 percent. This could potentially be explained by higher pollution in the Ganga Basin, greater use of polluted water, or higher poverty, among many other patterns.

Statistics for BOD and FCOLI indeed corroborate the claim that pollution in the Ganga Basin is greater than elsewhere in India. District-level BOD averages 3.68 across the whole of India but 4.17 within the Ganga Basin. Looking at the "bathing class" cutoff used by the government of India, i.e., BOD<3 mg/l, the data indicate that 40 percent of districts in the Ganga basin are considered to be too polluted to be of the bathing class as opposed to 33 percent nationally. This trend is equally apparent from data on FCOLI, which is specifically a domestic pollution metric.

A third set of variables in Table 1 pertains to the Supreme Court verdict. The mean values of 1[Born after 10/1987] and 1[Kanpur] highlight the fact that, regardless of which sample is used, the overwhelming majority of observations come from after the date of the verdict and from outside of Kanpur, respectively. The indicator for incidence of the National River Conservation Plan (NRCP), meanwhile, shows that the Ganga Basin is the predominant focus of government water pollution policy: 23 percent of observations are covered by NRCP in the "All India" sample, while 52 percent are covered in the Ganga Basin sample. The stark differences in baseline infant mortality, pollution, and policy between the two samples underlies our focus on the Ganga Basin in our preferred specifications, while keeping the all-India sample for robustness analyses.

Finally, Table 2 displays for each of six different upstream distance ranges, the number of qualifying monitors and their corresponding summary statistics. As the upper-bound distance increases, the number of monitors that can be matched with an upstream monitor increases accordingly. Conversely, when the lower-bound distance increases, the reverse holds. The average distance between a monitor and its upstream analog varies between 98 and 170 km.

4.2 The impact of *Mehta vs. Union of India* on infant health

Determining whether the Supreme Court verdict improved water quality and health in the Kanpur area is important because there is no clear precedent for successful water pollution policy in India. Table 3 focuses in particular on health outcomes, detailing results from the estimation of equation (1). Each column corresponds to a different specification, with respect to geographic coverage, time period, and parameterization of the policy variable. All regressions estimate the mortality impact of being in Kanpur after the ruling was announced.

Columns 1-3 depict the results of using our primary sample, which consists of districts in the Ganga basin only, while varying the post-policy time period. The point estimates in these columns imply that

the magnitude of the mortality reduction is about 3 percentage points, on a baseline of 6 percent (which comes from Table 1). In column 4, the full 1986-2005 time period is used, but the policy variable is broken into short-run, medium-run, and long-run terms; the results suggest the possibility of attenuation in the mortality impact over the long run. Meanwhile, our All-India estimate in column 5 exhibits the same relationship as the Ganga Basin result in column 1.

While Table 3 provides evidence that neonatal mortality dropped in Kanpur in the aftermath of the verdict, one may wonder whether it additionally had an impact on mortality *downstream* of Kanpur. To assess this possibility, we include in column 6 a dummy variable for being born in Allahabad – lying approximately 200 km beyond Kanpur at the confluence of the Ganga and Yamuna Rivers – after the ruling. The result indicates no persistence at this particular location. However, Allahabad is far enough way from Kanpur (~200 km) that changes in Kanpur may not affect it. Furthermore, the Yamuna River joins the Ganga at Allahabad and contributes 58.5 percent of the combined river flow at that confluence (Singh 2007).

4.3 The impact of *Mehta vs. Union of India* on river pollution

The Ganga Pollution Cases therefore seem to have had a beneficial effect on infant health. Have they done so by reducing river pollution? Table 4 tests equation 4, a necessary condition for this to be the case. The upper panel of Table 4 reports regression results with our primary pollutant, BOD, while the lower panel leverages other relevant pollutants to further evaluate the policy's impact.

The BOD results displayed in columns 1-6 uniformly imply a significant drop in pollution. Given the choice of BOD as a dummy variable for exceeding the "Bathing Class" threshold, the estimated magnitude is a 40-60 percentage-point drop in the likelihood of exceedance, from a 100 percent probability before the Supreme Court ruling. Columns 2, 3, and 4 all suggest that the impact on BOD did not lessen over time. Column 6 however indicates *growth* in downstream pollution after the ruling. As discussed previously, however, the measurement of pollution downstream of Kanpur is taken 200 km away, just after a major river, the Yamuna, joins and dilutes the Ganga.

In columns 7 and 8, we use the logarithm of the BOD level as our outcome variable instead of the "Bathing Class" dummy. With that specification however, we fail to detect an impact of the Supreme Court decision in contrast with earlier results, for either of the time periods considered. A closer look at the difference between columns 7-8 and say column 1 can be seen in Figure 2. This figure presents the residuals from regressing the logarithm of BOD on district and year-month fixed effects. The top panel plots the distribution of the residuals before and after the ruling for treatment districts, i.e. districts of Kanpur, while the bottom panel shows the same curve for control districts, where control districts are required to also be in the Ganga basin. While the graphs visually suggest that pollution

did decrease in treatment districts while remained roughly unchanged in control districts, the impact seems unclear at the lower tail of the distribution. This largely explains why we might be able to detect an impact with a dummy variable when we fail to do so with a continuous measure.

As discussed in Section 3, while BOD could be an appropriate pollutant to study in the context of tanning, so are several others for which we have data. Both a reduction in tanning volume and an improvement in effluent treatment should also cause drops in calcium and sulfate concentrations. On the other hand, changes made by the tanneries should not affect levels of fecal coliforms − which is a domestic pollutant − nor should they affect chloride concentration, chloride not being affected by standard tannery waste treatment (UNIDO 2011). Thus, pollutants such as FCOLI and chloride are candidates for placebo tests. The lower panel in Table 4 indeed shows that the measured effect of the Supreme Court decision on each of these four pollutants is consistent with theory. Calcium and sulfate levels drop in Kanpur after the verdict date, though only the drop in sulphates is statistically significant at the 1 percent level. FCOLI and chloride levels are unaffected by the verdict. The combined evidence suggests that the *Mehta vs. Union of India* verdict both reduced neonatal mortality and reduced pollution into the Ganga River.[6] Whether pollution reduction was the channel of impact, let alone the main one, is a question to which we now turn.

4.4 Mechanisms of policy impact

The pollution reduction documented in Table 4 may or may not fully explain the avoided loss of life identified in Table 3. For instance, avoidance behavior by households, with respect to water treatment and/or usage, could have improved health outcomes independently of water quality changes, as the Supreme Court decision might have increased overall awareness. The reduced form impact of environmental regulation on health, such as we estimate in Table 3, thus does not allow further understanding and quantifying of possible mechanisms.

Policy research in the environment-development literature does not generally attempt to disentangle the relative importance of the pollution channel from that of other possible channels (e.g., Chay and Greenstone 2003 and Watson 2006). An econometric examination of some mechanism of impact requires a second instrument to identify the contribution of that mechanism separately from the whole policy impact. In our context, we construct this second instrument from upstream pollution. As discussed in Section 2, pollution upstream is plausibly uncorrelated with downstream determinants of neonatal mortality and yet reliably flows downstream to have an impact on survival. As a valid instrument, it calibrates the dose-response function of water pollution and neonatal mortality in India. Essentially, we use this function as a yardstick for the policy's impact on health.

[6] We note that our results in Tables 3 and 4 are robust to the upstream sample restriction employed in later tables as well. These results are available upon request.

The first step of this instrumental variables (IV) strategy consists of an estimation of the first stage, the results of which are displayed in Table 5. Each set of three numbers in this table corresponds to a different upstream definition: the top number is the estimated coefficient η^2 from equation (7), the middle number is its standard error, and the bottom number is the sample size using the listed upstream definition.

As indicated in Table 5, the first stage is both strong and robust to choices about time-period, geographic sample, and upstream definition. Upstream pollution predicts downstream pollution in both the Ganga Basin (columnn 1-3) and the All-India sample (columns 4-6). It also maintains its predictive power over the medium (columns 2 and 5) and long run (columns 3 and 6). Finally, varying the lower and upper bound for eligible upstream pollution monitors (by row, in Table 5) has little effect on magnitude or significance. Our preferred range is $[75, 200]$ because it is the most conservative from among those with consistently strong first stages (point estimates with the 100-km lower bound retain the flavor of the other results but are not across-the-board statistically significant). As the lower bound rises, the risk of off-river spatial correlation drops. As the upper bound falls, pollution measured *up*stream is increasingly likely to reach the area of measurement *down*stream.

The next step of our IV strategy is an analysis of the impact of upstream pollution on downstream *mortality* – the reduced-form effect. Such an analysis provides a first check on whether our IV strategy will successfully identify the dose-response function. Moreover, it facilitates a falsification test using neighboring, *non*-upstream pollution monitors. Our strategy implicitly assumes that pollution is predictive of mortality in neighboring states only through river flow; if that assumptions holds, then non-upstream neighbors should not have any predictive power.

Table 6 shows that pollution at upstream locations does indeed predict mortality in downstream locations. The implied effect of exceeding an upstream BOD of 3 mg/l is a 2-3 percentage-point rise in neonatal mortality rates from a baseline of 11 percent. This effect is significant when we use our preferred range of $[75, 200]$ km (column 1), as well as with all other permutations involving the 200-km upper bound (columns 2-4). Only when we extend the range to 300 km (column 5) does the point estimate lose its precision.

What makes this pattern even more striking is the fact that, when upstream neighbors are replaced with *non*-upstream neighbors, the statistical relationship disappears. Columns 6 and 7 show that fact using the Ganga sample and the full national sample, respectively, with quantitatively small and statistically insignificant point estimates. When we include non-neighbors for the entire country, we get a statistically significant negative coefficient. While this is puzzling and incongruous with our hypothesis, we note that the magnitude of the coefficient is very small and close to zero. This result reduces our concern that the link we estimate between upstream pollution and downstream health is

being driven by simple spatial correlation that is not mediated by rivers.

Since the Wald estimator of pollution's impact on mortality is the ratio of the reduced-form impact and the first-stage relationship, our results in Tables 5 and 6 suggest that 2SLS can identify our desired dose-response function. That function, in turn, makes possible testing some mechanisms at work. Table 7 thus puts the results of three different strategies for estimating the function side-by-side: OLS in column 1; 2SLS using upstream pollution as the instrument; and 2SLS *additionally* using the policy itself as an instrument: if the impact of the Supreme Court decision on neonatal mortality only goes through pollution, then our policy variable can be excluded from the second stage as a valid instrument for pollution.

The OLS point estimate on pollution, shown in column 1, is a statistical zero; this is consistent with the notion that OLS is biased downwards by the positive correlation between pollution and other factors that are beneficial to health. In stark contrast, columns 2-5 reveal a strong positive relationship between pollution and mortality after instrumenting for the former. The implication is that a deterioration of water quality from "bathing class" standard to below that standard is associated with, on average, a double-digit rise in the likelihood of neonatal mortality (for example, a short-run estimate of 14.4 percentage points in column 2, and a long-run estimate of 11.1 in column 4).

To understand the relative importance of the pollution channel in the policy's health impact, we must compare columns 2-5 with the corresponding columns in the lower panel respectively. The latter columns detail the results of 2SLS with the policy dummy as an additional instrument. Inspection of the differences in point estimates provides a visual test of mechanisms, while the p-value for the C-statistic provides the statistical test. Point estimates of very similar magnitudes suggest that the policy's impact on neonatal mortality does not significantly differ from the mortality effect predicted by the policy's pollution reduction – i.e., that pollution fully explains the policy's health impact. Point estimates of very *different* magnitudes suggest the opposite – that the policy's health impact does not match up with expectations based on the policy's pollution effect, and that some other channel(s) must be at work.

From our test results, we fail to reject the null hypothesis of pollution fully explaining the Supreme Court ruling's impact on neonatal mortality. While the 1-IV and 2-IV point estimates in Table 7 are not of exactly the same magnitude, the p-value for the test statistic ranges from 0.35 to 0.63 – too high to reject at conventional significance thresholds. This finding is underscored by the point estimates on the policy dummy. In column 1, where the impact of pollution on health is not captured in isolation, the policy coefficient is significant; but in columns 2-5, when the upstream instrument accurately accounts for the pollution impact, the policy coefficient loses that significance. In other words, the policy is not predictive of health impacts after partialling out the pollution channel.

Table 8 shows that the results of our test of mechanisms are robust to changes in upstream definition, geographic area, and religious identity. Just as in Table 6, the choice of upstream definition does not affect the size of the estimated mortality impact (columns 1-3). Expanding geographic coverage to the entire nation reduces the size the mortality impact, while restricting to only Hindu households increases it.[7] Across all variations, however, the overidentification test implies failure to reject the null hypothesis.

5 Discussion

The fact that the Ganga Pollution Cases have produced sizable environmental and health benefits is important in its own right: the outcomes of the cases represent the first documented success in India's regulation of water quality. In addition, our findings are differentiated by the type of pollution studied. Prior work has focused on pollution from either domestic (Field, Glennerster, and Hussam 2011) or agricultural (Brainerd and Menon 2014) sources. Our own work links mortality to industrial pollution for the first time, and it more generally establishes the association between health and a new pollutant – biochemical oxygen demand. Finally, we have developed a novel, and yet replicable, methodology for producing an unbiased estimate of the water pollution-health dose-response function by leveraging the flow of rivers.

Our reduced-form impact of improved environmental quality on health mirrors the impacts measured by Ebenstein (2012) and Brainerd and Menon (2014) and indicates that the costs of river pollution are significant.[8] The latter authors' findings are particularly consistent with our own results, in that they, too, specifically show neonatal mortality to be affected by water pollution. Given widespread breastfeeding practices in India, these results suggest that alternative modes of contamination other than river water drinking are at work (Cifuentes et al. 2000).

In spite of the harms of pollution, environmental quality remains low in many developing countries. Air quality in Delhi, India, for example, averaged 128 μg/m^3 of fine particulate matter (PM2.5) in 2015 – ten times more than in Washington D.C. (*Washington Post* 2016) and significantly *worse* than in 2010. In that same five-year period, the number of classified 'polluted river stretches' in India doubled from 150 to 302, and the gap between sewage load and sewage treatment capacity expanded (*Daily Mail* 2015). Why, if pollution is so costly, does it persist at high levels?

One explanation is a high marginal utility of consumption relative to the cost of abatement (see

[7] The difference between the Hindu-only point estimate and our main point estimate, which includes Muslims among other minority religions, is statistically significant and consistent with the observation that rivers are more likely to be used by Hindus for ritual purposes.

[8] Ebenstein (2012) shows that decreases in Chinese river water quality are associated with rises in adult deaths due to stomach cancer: a one-grade deterioration (from a six-grade scale) predicts a 10 percent rise in stomach-cancer mortality. Brainerd and Menon (2014), meanwhile, find that a 10 percent rise in agricultural pollution into India's rivers is associated with a 6 percent increase in neonatal mortality.

Greenstone and Jack 2015), which could make the private costs of pollution reduction greater than the private benefits. In support of this hypothesis, Hanna and Oliva (2015a) show that when Indian households are randomly made wealthier (through a cash and livestock transfer), they choose to consume more energy but not cleaner energy. A second explanation is a high marginal cost of improving environmental quality. For instance, Davis (2008) and Field, Glennerster, and Hussam (2011) identify environmental regulations whose designs had unintended consequences for air quality in Mexico and infant health in Bangladesh, respectively. Greenstone and Hanna (2014), meanwhile, find that some Indian pollution policies have worked (i.e., those pertaining to air quality) and others (such as the water policies encompassed by NRCP) have not. They cautiously argue that observed differences in policy efficacy are explained by greater demand for air quality than for water quality, which they suggest using a variety of qualitative and quantitative strategies. In our context, Kanpur itself had BOD levels that placed it at the 63rd percentile of districts in the Ganga Basin prior to the Supreme Court ruling. Higher pollution levels may in principle be associated with larger willingness-to-pay for environmental quality, but our qualitative investigations suggest that demand for water quality was quite low in Kanpur in the 1980s: there was no local movement to improve water quality at the time of Mehta's writ petition, but rather there *was* significant concern about the economic impacts of regulation in a city that relied heavily on the tanning industry for jobs.[9]

Alternatively, the fact that environmental quality is a public good causes private under-investment in pollution control. For both air and water pollution, which travel significantly after their initial production due to wind and river flow, respectively, the negative externality affects not just the community in which polluting takes place but also those communities downwind and downstream. In the case of rivers, it has long been known that free-riding results in heightened pollution levels upstream of administrative borders (Oates 2001; Sigman 2005; Lipscomb and Mobarak 2007). Our empirical investigation indeed documents the existence of spatial spillovers: the first-stage of our IV analysis shows that pollution persists more than 100 km downstream of its measurement. We further link this spatial externality directly to health outcomes, by showing (through our reduced-form estimation in Table 6) that pollution actually imparts a health burden on downstream communities. River pollution is thus a collective action problem requiring central government regulation and/or inter-jurisdictional bargaining.

As has already been noted, India's primary central-government-led effort to improve water quality

[9]We learned these facts and general history of Kanpur through a comprehensive qualitative research study in Kanpur in the winter of 2014. The study consisted of interviews of a large number of stakeholders and informants in the Ganga Pollution Cases. These included government officials, owners of tanneries, two NGOs, several journalists, three professors at the Indian Institute of Technology, an operator of a Common Effluent Treatment Plant (CETP), elderly rickshaw pullers who worked in Jajmau in the 1980s, and taxi-drivers. We examined official documents from the time that the Ganga Action Plan was implemented and progress reports in later years. For background on citizen activism, we are grateful to the Ecofriends organization, which was established in Kanpur in 1993.

(NRCP) has not been successful. One feature that distinguishes the Ganga Pollution Cases from NRCP is that the environmental policy produced by the former emanated from the judicial rather than the executive branch. Article 21 of the Indian Constitution indeed provides citizens with the "Right to Life"; *Mehta vs. Union of India* represents a watershed moment in Indian legal and environmental history, because it was the first time that this constitutional 'lever' was used to drive environmental policy through the Indian Judiciary. Plausibly, decisions from the judiciary differ from the ones enacted by the executive in that they mandate agents to take specific – and verifiable– actions rather than design complex investment and incentive schemes that are arguably more difficult to implement (Davis 2008). Furthermore, the set of stakeholders empowered to monitor the execution of a judicial decision, which includes citizens, might reduce the scope for firms to cheat the government's monitoring system (Duflo et al. 2013). Further understanding what made the Ganga Pollution Cases successful might therefore shed light on what institutional arrangements led to successful policy implementation.

6 Conclusion

This paper provides empirical evidence that the 1987 Supreme Court decision in *Mehta vs. Union of India*, which primarily targeted the tanning industry in Kanpur district, induced a drop in both surface water pollution and neonatal mortality. Our investigation of the mechanisms of policy impact suggests that pollution is the major channel of the identified mortality effect. In deriving and conducting our tests of this pollution channel, we show how information about the different potential mechanisms can be backed out from analysis even when data on all possible mechanisms are not available.

We believe our analysis represents an important contribution to the broader puzzle of continually poor environmental quality in developing countries. First, we have identified a precedent for successful water pollution policy in India. Though a formal analysis of the reasons for these two policies' starkly different outcomes is beyond the scope of this paper, the available evidence suggests that differential costs of environmental quality improvements – rather than differential demand – provide the most plausible explanation.

In addition, we have demonstrated that river pollution has a real, adverse impact on infant health in India. This is important because existing research predominantly focuses on air quality rather than water quality, and demand for the latter appears correspondingly lower than demand for the former. Our results highlight the susceptibility of newborns in their first month of life, the harms of industrial pollution, and the association between mortality and the broad-based measure of biochemical oxygen demand. Methodologically, we show how the unidirectional flow of rivers can be leveraged for unbiased estimation of the water pollution-health dose-response function.

Finally, we have shown that the ultimate incidence of the costs of pollution is not limited to the origin of that pollution. Rather, water pollution flows downstream to other communities living along rivers, reducing not just water quality but also the likelihood of infant survival. This finding highlights the spatial externality inherent to pollution and underscores the need for inter-jurisdictional bargaining. All in all, we provide several pieces of evidence that high marginal costs of pollution control and widespread market failures in developing countries are key to explaining the puzzle of poor environmental quality.

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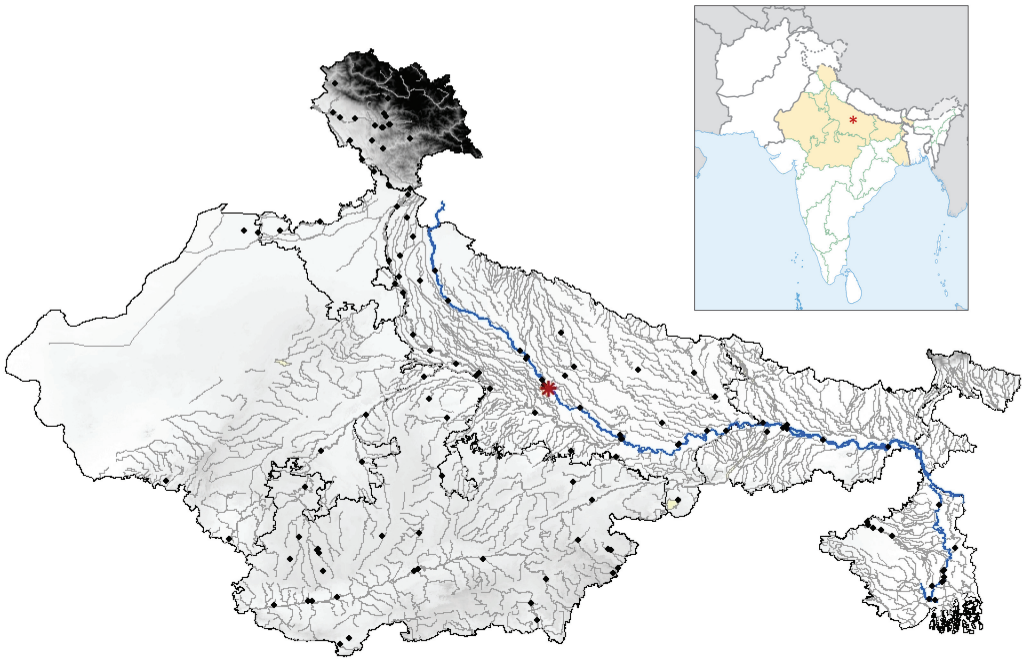
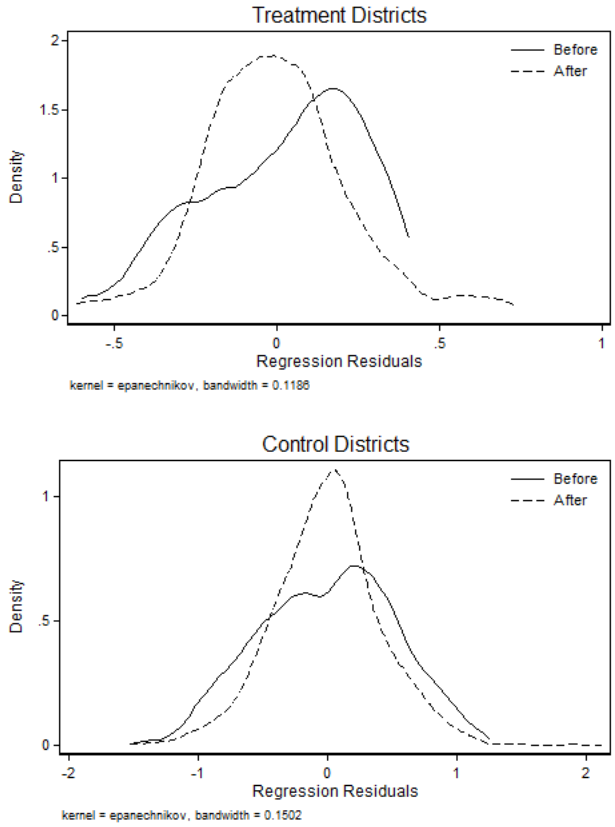
Figure 1: Locus of the Study



Notes: The asterisk marks the city of Kanpur. Black dots indicate pollution monitors within the Ganga Basin, and the blue line highlights the Ganga River itself.

Figure 2: Biological Oxygen Demand (BOD)



Notes: These graphs present residual values of biological-oxygen-demand (BOD) from a regression of log BOD on district and year-month fixed effects. The sample is the Ganga basin for the years 1986–2005.

Figure 3: Schematic Diagram of Empirical Strategy

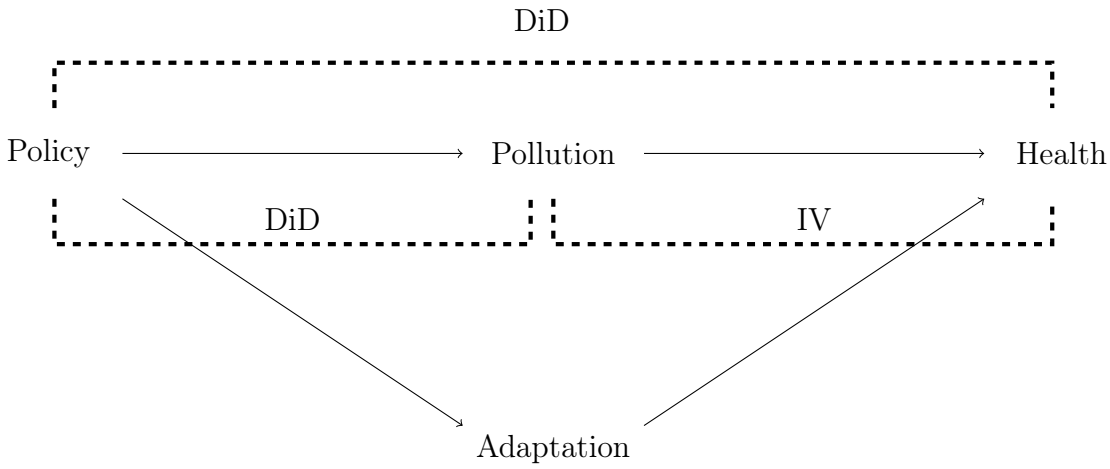


Table 1: Summary Statistics for All Variables

	All India					Ganga Basin Only				
	N	Mean	St. Dev.	Min	Max	N	Mean	St. Dev.	Min	Max
1[Child died in the first month of life]	228,352	0.05	0.22	0	1	68,577	0.06	0.24	0	1
BOD (mg/l)	24,879	3.68	5.50	0	100	5,632	4.17	5.51	0	61
1[BOD > 3]	24,879	0.33	0.47	0	1	5,632	0.40	0.49	0	1
FCOLI (organisms/100 ml)	19,465	22,340.85	102850.31	1	2,100,000	4,271	52,431.46	156759.46	4	2,100,000
1[FCOLI > 5,000]	19,465	0.16	0.36	0	1	4,271	0.40	0.49	0	1
Calcium	23,096	75.43	68.04	1	898	4,924	87.36	60.66	1	600
1[Calcium > median]	23,096	0.47	0.50	0	1	4,924	0.63	0.48	0	1
Sulfates	22,033	43.79	106.05	1	1,700	4,700	27.44	47.12	1	663
1[Sulfates > median]	22,033	0.50	0.50	0	1	4,700	0.48	0.50	0	1
Chlorides	23,009	55.56	230.21	1	7,340	5,004	39.71	72.77	1	1,256
1[Chlorides > median]	25,349	0.54	0.50	0	1	5,785	0.47	0.50	0	1
1[District of birth is Kanpur]	228,352	0.01	0.12	0	1	68,577	0.05	0.21	0	1
1[Child was born after the verdict]	228,352	0.97	0.16	0	1	68,577	0.96	0.19	0	1
1[Kanpur] X 1[Post-Verdict]	228,352	0.01	0.11	0	1	68,577	0.04	0.20	0	1
1[Mother is Hindu]	228,352	0.83	0.38	0	1	68,577	0.81	0.39	0	1
1[Scheduled Caste/Scheduled Tribe]	228,352	0.31	0.46	0	1	68,577	0.25	0.43	0	1
Age of mother (years) at time of interview	228,352	30.15	6.08	15	44	68,577	30.25	6.36	15	44
1[Mother is literate]	228,352	0.48	0.50	0	1	68,577	0.35	0.48	0	1
CETP capacity (MLD)	228,352	0.49	3.91	0	55	68,577	0.10	0.80	0	24
Air temperature (C)	228,301	25.70	4.59	8	36	68,577	25.57	5.51	10	36
Precipitation (mm)	228,352	104.90	149.98	0	1,469	68,577	93.04	127.01	0	817
1[National River Conservation Plan]	228,352	0.23	0.42	0	1	68,577	0.52	0.50	0	1

Notes: In the left panel, the sample is all of India; in the right, it is the Ganga Basin only.

Table 2: Sample Composition and Upstream Definition

	Ganga Basin Only		All India	
	Number of Monitors	Upstream Distance (km)	Number of Monitors	Upstream Distance (km)
[0, 200]	75	98	337	89
[20, 200]	74	101	338	92
[50, 200]	67	113	313	108
[75, 200]	53	130	277	124
[100, 200]	49	145	257	140
[75, 300]	76	170	329	143

Notes: The window [X,Y] defines the range, in km, of distances at which a pollution monitor lying upstream of some monitor m qualifies as its upstream match. That match is also conditional on the upstream monitor lying in a different district than monitor m. Tabulated numbers count the monitors in the sample that have upstream matches, as well as the average distance upstream of those matches. In the left panel, the sample is the Ganga Basin only; in the right, it is all of India

Table 3: *Mehta vs. Union of India* and Infant Mortality

	Dependent variable: 1[Child died in first month of life]					
	(1)	(2)	(3)	(4)	(5)	(6)
1[Kanpur] X 1[Post-Verdict]	-0.027***	-0.033***	-0.027***		-0.042***	-0.026***
	(0.009)	(0.009)	(0.009)		(0.005)	(0.008)
1[Kanpur] X 1[10/1987 <= t <= 12/1994]				-0.030***		
				(0.008)		
1[Kanpur] X 1[1/1995 <= t <= 12/1999]				-0.036***		
				(0.010)		
1[Kanpur] X 1[1/2000 <= t]				-0.003		
				(0.010)		
1[Downstream of Kanpur] X 1[Post-Verdict]						0.003
						(0.012)
Pre-ruling treatment area mortality	0.110	0.110	0.110	0.110	0.110	0.110
Geographic coverage	Ganga	Ganga	Ganga	Ganga	India	Ganga
Last sample year	1994	1999	2004	2004	1994	1994
Adjusted R-Squared	0.015	0.012	0.010	0.010	0.015	0.015
N	20,499	47,810	68,577	68,577	83,440	20,499

Notes: The dependent variable in all regressions is a binary variable equaling one if a child died in the first month of life. An observation is a child-month. All regressions include a set of controls (CETP capacity, air temperature, total precipitation, and NRCP dummy) and district and year-month fixed effects. Standard errors are clustered at the district level in parentheses. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent levels, respectively.

Table 4: *Mehta vs. Union of India* and Pollution

Panel A. Biochemical Oxygen Demand	BOD						Ln(BOD)	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
1[Kanpur] X 1[Post-Verdict]	-0.528***	-0.447***	-0.410***		-0.600***	-0.384***	0.083	-0.075
	(0.058)	(0.039)	(0.048)		(0.032)	(0.089)	(0.109)	(0.139)
1[Kanpur] X 1[10/1987 <= t <= 12/1994]				-0.415***				
				(0.145)				
1[Kanpur] X 1[1/1995 <= t <= 12/1999]				-0.400***				
				(0.080)				
1[Kanpur] X 1[1/2000 <= t]				-0.416***				
				(0.123)				
1[Downstream of Kanpur] X 1[Post-Verdict]						0.263***		
						(0.092)		
Geographic coverage	Ganga	Ganga	Ganga	Ganga	India	Ganga	Ganga	Ganga
Last sample year	1994	1999	2004	2004	1994	1994	1994	2004
Adjusted R-Squared	0.615	0.537	0.548	0.548	0.597	0.624	0.784	0.718
N	1,901	3,911	6,042	6,042	9,826	1,901	1,901	6,042

Panel B. Non-BOD Pollutants	Calcium	Sulfates	FCOLI	Chlorides
	(1)	(2)	(3)	(4)
1[Kanpur] X 1[Post-Verdict]	-0.103	-0.505***	-0.044	-0.058
	(0.066)	(0.046)	(0.129)	(0.057)
Geographic coverage	Ganga	Ganga	Ganga	Ganga
Last sample year	1994	1994	1994	1994
Adjusted R-Squared	0.636	0.377	0.560	0.638
N	1,913	1,749	1,363	1,971

Notes: The dependent variable is listed above each column number. An observation is an average at the district-month level. All regressions include a set of controls (CETP capacity, air temperature, total precipitation, and NRCP dummy) and district and year-month fixed effects. Standard errors are clustered at the district level in parentheses. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent levels, respectively.

Table 5: First-Stage Results

	Dependent variable: 1[BOD>3 mg/l]					
	Ganga Basin Only			All India		
	(1)	(2)	(3)	(4)	(5)	(6)
[0, 200]	0.130*	0.130*	0.148*	0.236***	0.198***	0.260***
	(0.055)	(0.050)	(0.058)	(0.032)	(0.026)	(0.030)
N	1,350	2,746	4,033	8,023	14,894	20,258
[20, 200]	0.167*	0.156*	0.188**	0.237***	0.209***	0.277***
	(0.061)	(0.060)	(0.067)	(0.033)	(0.030)	(0.031)
N	1,332	2,671	3,906	8,183	15,097	20,474
[50, 200]	0.198**	0.192**	0.226**	0.272***	0.231***	0.278***
	(0.064)	(0.064)	(0.070)	(0.036)	(0.032)	(0.035)
N	1,157	2,366	3,496	7,876	14,472	19,555
[75, 200]	0.199**	0.175*	0.177*	0.298***	0.212***	0.253***
	(0.068)	(0.068)	(0.073)	(0.038)	(0.036)	(0.036)
N	964	1,943	2,870	7,500	13,538	18,101
[100, 200]	0.228*	0.121	0.157	0.260***	0.177***	0.224***
	(0.096)	(0.086)	(0.084)	(0.041)	(0.036)	(0.041)
N	844	1,672	2,437	6,698	12,170	16,309
[75, 300]	0.189*	0.145**	0.161*	0.280***	0.190***	0.228***
	(0.073)	(0.052)	(0.061)	(0.034)	(0.031)	(0.032)
N	1,452	3,055	4,458	9,163	16,862	22,615
Last sample year	1994	1999	2004	1994	1999	2004

Notes: Each trio of coefficient, standard error and N corresponds to a different regression. All regressions estimate Equation 6 by 2SLS, using upstream BOD as an instrument for its downstream analog. Reported point estimates correspond to the coefficient on the instrument, 1[Upstream BOD>3 mg/l], in the first stage. The window [X,Y] defines the range, in km, of distances at which a pollution monitor lying upstream of some monitor m qualifies as its upstream match. An observation is a district-month. All regressions include a set of controls (CETP capacity, air temperature, total precipitation, and NRCP dummy) and district and year-month fixed effects. Standard errors are clustered at the district level in parentheses. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent levels, respectively.

Table 6: Reduced-Form Results

	Dependent variable: 1[BOD>3 mg/l]							
	Upstream Districts					Placebo Districts		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
1[US BOD>3]	0.031***	0.022***	0.030**	0.019**	0.007	-0.002	0.007	-0.009*
	(0.009)	(0.007)	(0.014)	(0.009)	(0.006)	(0.010)	(0.004)	(0.005)
Upstream definition	[75,200]	[0,200]	[100,200]	[75,200]	[75,200]	N/A	N/A	[75,200]
Geographic coverage	Ganga	Ganga	Ganga	Ganga	India	Ganga	Ganga	India
Last sample year	1994	1994	1994	2004	1994	1994	2004	1994
Adjusted R-Squared	0.015	0.016	0.016	0.009	0.016	0.016	0.012	0.021
N	10,066	13,608	8,979	33,391	61,735	2,466	16,446	19,597

Notes: Columns 1-5 present reduced-form estimates of the association between upstream pollution levels and a binary dependent variable equaling one if a child died in the first month of life. Columns 6-8 show results from a placebo test, in which the upstream district for each observation is replaced by a different, neighboring district that is not upstream. In each column, sample selection is characterized by its listed 'Upstream definition', 'Geographic coverage', and 'Last sample year' ("N/A" indicates no sample restriction based on upstream distance). In all regressions, an observation is a child-month, and the dependent variable is a binary variable equaling one if a child died in the first month of life. All regressions additionally include a set of controls (religion of the household head, caste of the household head, mother's age, mother's literacy, local CETP capacity, air temperature, total precipitation, and NRCP) as well as district and year-month fixed effects. Standard errors are clustered at the district level in parentheses. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent, respectively.

Table 7: *Mehta vs. Union of India* and Infant Mortality: Channels of Impact

Panel A. OLS and 1-IV 2SLS	OLS	1-IV			
	(1)	(2)	(3)	(4)	(5)
1[BOD>3]	-0.007	0.144**	0.126**	0.111***	0.121***
	(0.011)	(0.061)	(0.054)	(0.034)	(0.040)
1[Kanpur] X 1[Post-Verdict]	-0.038**	0.050	0.018	0.009	
	(0.014)	(0.043)	(0.028)	(0.019)	
1[Kanpur] X 1[10/1987 <= t <= 12/1994]					0.024
					(0.033)
1[Kanpur] X 1[1/1995 <= t <= 12/1999]					-0.007
					(0.020)
1[Kanpur] X 1[1/2000 <= t]					0.027
					(0.024)
Panel B. 2-IV 2SLS		2-IV			
		(1)	(2)	(3)	(4)
1[BOD>3]		0.090***	0.100***	0.102***	0.107***
		(0.020)	(0.032)	(0.027)	(0.025)
C-statistic (P-value)		0.348	0.525	0.634	0.469
Last sample year	1994	1994	1999	2004	2004
N	9,706	9,603	21,703	32,561	32,561

Notes: The dependent variable in all regressions is a binary variable equaling one if a child died in the first month of life. An observation is a child-month. Column 1 shows results from OLS regression. Columns 2-5 show second-stage results of 2SLS; in Panel A, the endogenous variable (BOD > 3) is instrumented using its upstream analog, while in Panel B, both upstream pollution and the policy variable (1[Kanpur] X 1[Post]) are used as instruments. In each column, the two panels use the exact same sample – the Ganga Basin up to the year indicated by 'Last Sample Year' – and thus have the same 'N'. The P-value for the C-statistic tests the null hypothesis that the endogenous variable is overidentified. All regressions include set of controls (religion of the household head, caste of the household head, mother's age, mother's literacy, local CETP capacity, air temperature, total precipitation, and NRCP) as well as as district and year-month fixed effects. Standard errors are clustered at the district level in parentheses. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent, respectively.

Table 8: Robustness Checks on IV Regressions

Panel A. 1-IV 2SLS	Alternative Upstream Definition			All India	Hindus Only
	(1)	(2)	(3)	(4)	(5)
1[BOD>3]	0.154**	0.110**	0.137	0.028	0.174**
	(0.062)	(0.044)	(0.085)	(0.020)	(0.083)
1[Kanpur] X 1[Post-Verdict]	0.054	0.035	-0.001	-0.031**	0.039
	(0.042)	(0.034)	(0.043)	(0.015)	(0.059)
Panel B. 2-IV 2SLS	(1)	(2)	(3)	(4)	(5)
1[BOD>3]	0.096***	0.074***	0.139***	0.035*	0.124***
	(0.027)	(0.018)	(0.034)	(0.019)	(0.026)
C-statistic (P-value)	0.259	0.393	0.978	0.214	0.536
Upstream Definition	[20,200]	[50,200]	[100,200]	[75,200]	[75,200]
Geographic Coverage	Ganga	Ganga	Ganga	India	Ganga
Last sample year	1994	1994	1994	1994	1994
N	13,309	11,573	8,509	60,511	7,680

Notes: The dependent variable in all regressions is a binary variable equaling one if a child died in the first month of life. An observation is a child-month. All results shown are from the second stage of 2SLS instrumental variables regressions; in Panel A, the endogenous variable (BOD $>$ 3) is instrumented using its upstream analog, while in Panel B, both upstream pollution and the policy variable (1[Kanpur] X 1[Post]) are used as instruments. In each column, the two panels use the exact same sample − stipulated by the column header as well as the rows labeled 'Upstream Definition', 'Geographic Coverage', and 'Last Sample Year' − and thus have the same 'N'. The P-value for the C-statistic tests the null hypothesis that the endogenous variable is overidentified. All regressions include set of controls (religion of the household head, caste of the household head, mother's age, mother's literacy, local CETP capacity, air temperature, total precipitation, and NRCP) as well as as district and year-month fixed effects. Standard errors are clustered at the district level in parentheses. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent, respectively.

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