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Can environmental policy reduce infant mortality? Evidence from the Ganga Pollution Cases☆

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ABSTRACT

In many developing countries, environmental quality remains low and policies to improve it have been inconsistently effective. We conduct a case study of environmental policy in India, focusing on unprecedented Supreme Court rulings that targeted industrial pollution in the Ganga River. In a difference-in-differences framework, we find that the rulings precipitated reductions in river pollution and one-month infant mortality, both of which persist for more than a decade. We then estimate a pollution-mortality dose-response function across twenty-nine rivers in the Ganga Basin, instrumenting for pollution with its *upstream* counterpart. The estimation reveals a significant external health burden of river pollution, not just in the district of measurement, but also on downstream communities. It further provides suggestive evidence that reducing pollution was an important driver behind declines in infant mortality observed after the rulings.

1. 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; Brainerd and Menon, 2014), 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 et al., 2011). While high levels of air and water pollution may be due in part to high marginal utility of consumption (Hanna and Oliva, 2015b) 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 estimates that over three in every 1000 Indian children under five years old in 2004 died because of water pollution (WHO, 2004). India's Central Pollution Control Board reports that sewage treatment capacity amounts to only 21 percent of the estimated daily sewage load in Indian cities (CPCB, 2017a), 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 rulings – which mandated pollution cleanup by the tanning industry concentrated along the Ganga River in

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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 (as measured by biochemical oxygen demand [BOD]) 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 et al., 2010). Our reduced-form evidence highlights a rare instance of successful policy: judicial-led mandates primarily targeting industrial pollution. Our results suggest that the rulings improved several measures of water quality – BOD as well as calcium, sulfur, and chloride concentrations – and that mortality impacts occur primarily in the first month of life. Furthermore, the rulings' impacts on pollution and health persist over time for at least 10 years as well as downstream into neighboring districts.

In the second phase of our analysis, we quantify the external infant health costs of river pollution in general and investigate the mechanisms of the Ganga Pollution Cases' impacts. To do so, we estimate a pollution-health dose-response function in the Ganga River Basin. Since pollution is potentially endogenous, we instrument for it using its *upstream* counterpart. This identification strategy has a strong logic: the decision to pollute upstream is orthogonal to downstream health inputs, but pollution reliably flows downstream nonetheless. The persistence of downstream impacts of the Supreme Court rulings, as well as recent research leveraging the upstream-downstream geographic relationship (Garg et al., 2016), provide further support for our instrumental variables (IV) strategy.

We first find that exceedance of India's water quality standards raises the risk of neonatal mortality by 10–14 percentage points, on average, in the IV framework; the analogous ordinary least squares (OLS) regression yields a statistical zero. Since our IV point estimates are identified by variation in upstream water quality, they also show that the mortality burden of river pollution persists in downstream districts. Reduced-form regression of downstream neonatal mortality on upstream water quality indicate that the downstream mortality effect is approximately twenty percent of the main, in-district effect. Prior research documents the flow of pollution into downstream water sources (e.g., Lipscomb and Mobarak, 2017), but we believe we are the first to connect such spillovers to economically-meaningful health impacts. Our work thus underscores the large social costs of continually poor environmental quality, both at the point of measurement and in nearby areas linked by surface water flow.

Next, we leverage the IV strategy to investigate the mechanisms at 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 et al., 2011). Reduced-form estimates of policy impacts aggregate the effects of all such channels. Our finding of simultaneous reductions in river pollution and neonatal mortality in the Kanpur region is thus a necessary, but not sufficient, condition for pollution being the primary channel of the verdicts' health impact.

Intuition suggests that if reduced water contamination is the *only* channel of mortality reductions, then two statements should hold empirically: there is no residual effect of policy on mortality after appropriately controlling for pollution; and the policy is a valid instrument for pollution. Testing the degree to which these statements hold is equivalent to testing the null hypothesis that the policy's mortality impacts are fully explained by its pollution impacts. Empirically, we formulate this as an overidentification test with two instruments for pollution: upstream pollution and the policy itself. This strategy is 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 (e.g., Angelucci and Attanasio, 2013).

Armed with a plausibly exogenous source of pollution variation, we confirm that the two aforementioned statements hold empirically. First, the policy variable is no longer a significant predictor of mortality once pollution is included alongside it as an explanatory variable. Second, our overidentification tests fail to reject the null hypothesis that the rulings are a valid instrument for river pollution – i.e., that water quality improvements are the only channel through which the verdicts affected neonatal mortality.

The remainder of this paper is organized as follows. Section 2 describes the Ganga Pollution Cases amidst the more general context of pollution and related policy in India. Section 3 describes the various sources of data, while Sections 4 and 5 present our empirical strategy and results, respectively. Section 6 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 7 concludes.

2. 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 biochemical oxygen demand (BOD) — a broad-based measure of organic water pollution— carried out by India's Central Pollution Control Board (CPCB) shows that, as of 2011, average readings at approximately 37 percent of all sampling stations did not meet the government's standard of acceptability for bathing set at 3 mg/l (CPCB, 2017b). Waste from domestic consumption, industry, and agriculture all contribute to widespread water quality problems in surface and groundwater resources (Murty and Kumar 2011).

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 federal 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 standard for outdoor bathing, as defined by India's "Designated Best Use" classification system.[1] The primary lever for achieving this goal has been the "interception, diversion, and treatment" of sewage (Madhya Pradesh Pollution Control Board, 2017). To that end, 4704 million-liters per day of sewage treatment capacity have been created since its inception (Ministry of Environment and Forests, 2013). 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 et al., 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. The Ganga is India's longest river; in the Hindu religion, it is revered as a goddess. Upwards of 100,000 industrial operations

[1] India's official water quality criteria have five tiers (A-E), each of which includes an acceptable range for multiple water quality measures, including BOD, dissolved oxygen, total coliforms, and pH. Outdoor bathing is tier B; tier A corresponds to untreated (but disinfected) drinking water.

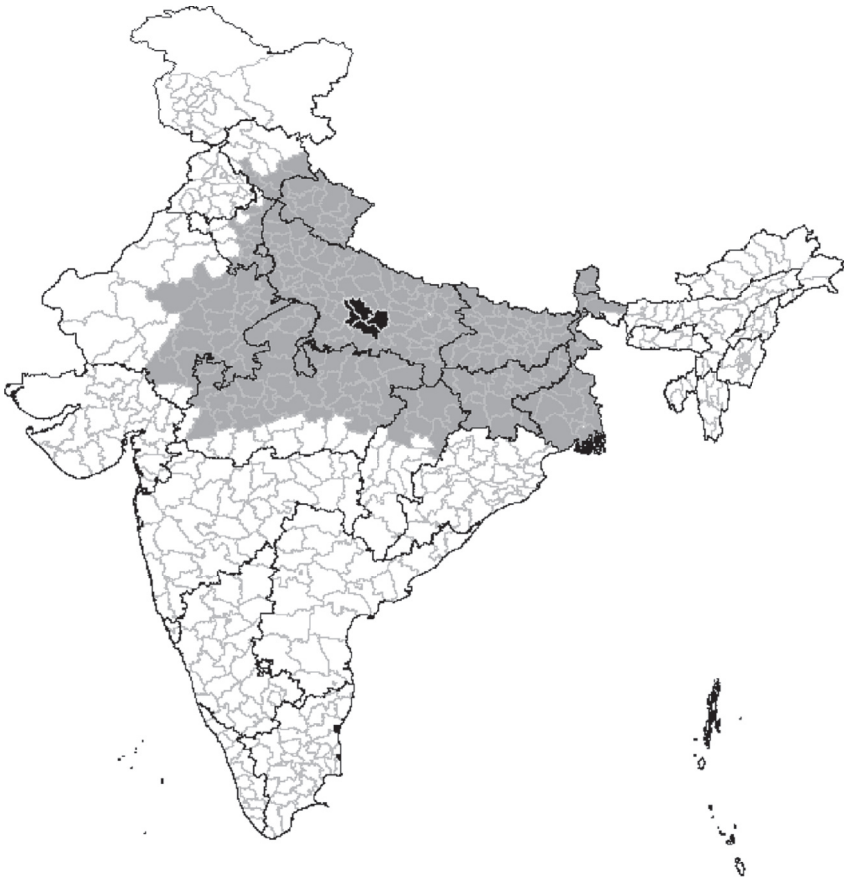


Fig. 1. A district map of India.

Notes: Light gray lines denote district borders, while black lines denote state borders. The gray shaded area is the Ganga Basin (240 districts), while the black shaded area is our group of four "treatment" districts (Kanpur, Unnao, Fatehpur, and Rae Bareli).

(Mehta, 2009) and 330 million people (CPCB, 2009) reside in its basin (see Fig. 1). In 1984, a matchstick tossed into the river by a smoker in Haridwar resulted in the river catching on fire for more than 30 h, 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 2500-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, 1985).

Kanpur is a city of 2.9 million people lying directly on the Ganga in the State of Uttar Pradesh). 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 concentrated in the neighborhood of Jajmau, which lay directly 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 characterized by large amounts of organic material – which, when deposited in rivers, depletes dissolved oxygen levels and thus the overall health of the watershed – and heavy metals like chromium, which is a documented carcinogen (Stout et al., 2009). It is also known for its conspicuous reddish-brown color (Durai and Rajasimman, 2011).

Mehta selected Kanpur despite not having been born in or lived in Kanpur. In interviews granted to 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 1 h 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 initiatives were undertaken 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, 2007).

3. Data

To assess the relationship between policy, water quality, and health in Kanpur, we collect and combine three types of data: infant mortality, river pollution, and other variables to be used as controls. In most of our analysis, we restrict our sample geographic area to the Ganga Basin, which is defined as the area that drains the Ganga and all of its tributaries. We make this restriction because of the singularity of this area in the context of our analysis. The Ganga Basin is not only a much more densely populated region than anywhere else in India, but also a region in which water issues have received special government attention: the National River Conservation Plan (NRCP) focused exclusively on the Ganga Basin (including the Ganga, Yamuna, Damodar, Gomti, and Mahananda Rivers) throughout its first 10 years (1986–1995). Extending the analysis beyond this region might confound the effect of the Supreme Court ruling with the effect of broad scrutiny in the Ganga Basin during this time period.

3.1. Health data

We choose infant mortality as our primary health outcome. This choice conveys at least two significant statistical advantages. First, as noted by Chay and Greenstone (2003), infant survival is a short-term measure; other measures of health stock, captured later in life, necessarily reflect the effect of many more health shocks experienced in an individual's lifetime. Second, complete birth histories are available in certain Indian demographic surveys, so we can construct long pseudo-panels of infant survival to maximize statistical power. In contrast, morbidity variables such as diarrhea incidence and low birth weight are only available cross-sectionally from the time of survey; too few of such surveys have been completed for us to construct a meaningful panel. Panel variation in infant mortality allows us to include detailed temporal and cross-sectional fixed effects in regression analyses, which account for time-varying shocks to national infant health and time-invariant characteristics of specific areas, respectively.

We focus primarily on deaths within the first month of life, i.e., neonatal mortality, although we also test for a policy impact on one-*year* mortality. The first month is the most vulnerable period of an infant's life (United Nations Children's Fund, 2015). Globally, neonatal mortality accounts for over half of all infant deaths (World Development Indicators, 2017); in our own data from India, its share of under-one mortality is 70 percent. Furthermore, several existing studies of pollution – in both water and air – and child health show that the largest impacts occur within one month of birth (Chay and Greenstone, 2003; Gluckman et al., 2008; Currie and Almond, 2011; and Brainerd and Menon, 2014). While neonatal mortality is not a complete measure of the health costs imposed by water pollution, it nonetheless represents a very large loss of life, especially in the Indian context.

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; from these birth histories, we create a panel of child-month mortality in the first year. The full national dataset spans the years 1967–2004. The first four years of this range includes 2, 0, 0, and 6 reported births nationally; starting in 1971, the reported birth count rises roughly monotonically from 42 towards its peak of 80,961 in 1998. Because of the lack of data from 1967 to 1970, we drop these years from the sample. This leaves us with with 1,393,330 birth observations from the years 1971–2004, 647,865 of which are to mothers living within the Ganga Basin (240 districts) at the time of survey.

3.2. Pollution data

To measure water quality, we use river pollution data collected under the auspices of India's National Water Quality Monitoring Program (NWMP). These data were originally gathered by Greenstone and Hanna (2014), from a combination of CPCB online and print records. The dataset spans the whole of India and runs from 1986 to 2004; an observation is a pollution monitor-month.

The national dataset contains information from 470 monitors situated along 162 rivers in 28 states. Excluding all areas outside the Ganga Basin leaves us with 101 unique pollution monitors along 29 rivers, in 62 districts representing 10 states; these are mapped in Fig. 2. Over our 19-year period of observation, 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 entire sample time frame. The data are noisy, suggesting significant measurement error, and the panel is imbalanced. To mitigate these problems, we collapse monitor-level observations to the district level and construct moving averages of the data over a four-month window. These steps produce 8725 district-month observations in the Ganga Basin with at least one non-missing value from our pollution measures of interest.

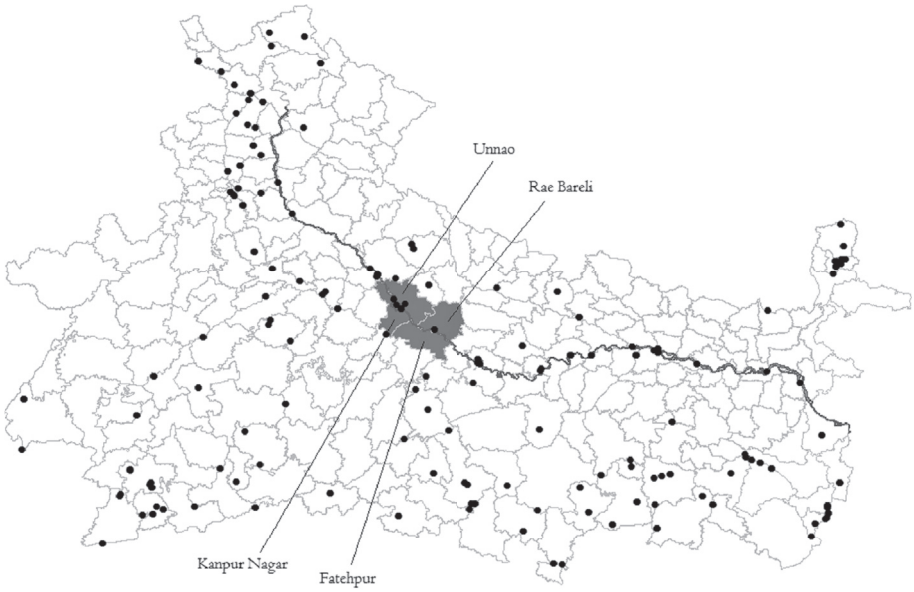
Our main indicator of river quality is BOD. This common, broad-based indicator of water pollution captures the amount of dissolved oxygen needed by water-borne, aerobic organisms to break down organic material present at a certain temperature (usually 20 °C) and over a specific time period (usually five days). 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, resulting in abnormally high BOD levels in tannery effluent. According to the United Nations Industrial Development Organization (UNIDO), 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 2000 (UNIDO, 2011).

Use of a broad-based indicator of pollution raises the issue that it is likely affected by a variety of factors, not just the activities of tanneries. During the Supreme Court hearings, lawyers representing the tanneries argued that other firms, a negligent Kanpur municipality, and other negligent municipalities were contributing to the pollution around the tanneries (Mehta, 2009: 78–82). This argument, however, was eventually dismissed in light of a report from the CPCB which demonstrated that the specific pollutant readings in the river were not even theoretically capable of being generated by any entity other than the cluster of tanneries (Mehta, 2009: 83–84). Field reports of Jajmau report no other major industry in the area (Alley, 2002).

In most of our analysis, we parameterize BOD as a dummy variable equaling one if average BOD in a district-month exceeds the national standard of 3 mg/l for bathing in surface water.[2] This 3-mg/l threshold is also a stated goal of the National River Conservation Plan, and is consistent with the critical values used by regulatory agencies in other countries (APHA, 1992). It is also consistent with the standards of proper measurement. According to the International Standards Organization (2003), the limit of detection for BOD is 3 mg/l; that is, BOD readings below three cannot be statistically distinguished from zero.

[2] In addition to this BOD standard, suitability for outdoor bathing requires a total coliforms concentration of less than 5 per ml, pH between 6.5 and 8.5, and dissolved oxygen levels exceeding 5 mg/l.

Fig. 2. Pollution monitors in the Ganga basin.



Notes: Black dots indicate the location of water pollution monitors. The meandering dark gray line is the Ganga River; all monitors not on the Ganga are situated along one of its tributaries (not shown). The gray shaded area delineates the four "treatment" districts (Kanpur, Unnao, Fatehpur, and Rae Bareli).

These and other water quality standards in general highlight the non-linear nature of the pollution–health relationship that has been documented in previous economics research on pollution and health (Chay and Greenstone, 2003; Ebenstein, 2012). For comparison, we also use the natural logarithm of BOD in certain analyses.

We also consider four other pollutants that shed additional light on the impacts of the Kanpur Supreme Court verdicts: calcium, sulfates, chlorides, 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 total dissolved solids (TDS) produced in tanning.[3] All three of these pollutants are measured in milligrams per liter (mg/l). Fecal coliforms (FCOLI) are an oft-used measure of domestic (as opposed to industrial) pollution, which was a major focus of the second verdict in the Ganga Pollution Cases. It is measured as the "most probable number" of coliform organisms per 100 mL of water (MPN/100 ml, reported in thousands). Other theoretically-relevant pollutants in our context are not recorded consistently in our time period.[4]

3.2.1. Assignment of upstream pollution

Part of our analytical strategy relies on the measurement of pollution *upstream* of a given location. 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 along the river between all pairs of neighboring monitors.

How far upstream should upstream pollution be measured? Two issues arise when answering this question. First, many water quality monitors in our dataset have more than one possible upstream counterpart. Second, there is no single "correct" distance at which to measure upstream pollution. The larger this distance, the weaker the upstream instrument will be as a result of pollution decay. On the other hand, monitors that are closer together are more likely to be subject to common shocks such as rainfall, which would create spurious correlation.

We thus adopt a variety of definitions of "upstream", and check the robustness of our results to the definition used. To assign an upstream counterpart to a given pollution monitor, we use the following protocol: 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 unweighted average pollution reading of these monitors as our upstream measure.[5] 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] Chloride ions are highly soluble in water, so their concentration is insensitive to standard tannery waste treatment (UNIDO, 2011). However, surface-water chloride levels may respond to other policy impacts on the tanning industry, such as reduction or cessation of tannery operations.

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

[5] We are unable to weight tributaries by their contribution to downstream water flow, due to a lack of data on volumetric flow rates at specific locations.

3.3. Other data

We include several types of variables as controls in many of our regression analyses. The main body of these controls consists of cross-sectional birth, mother, and child characteristics taken from RCH-2. A pair of climate controls are created using monthly, gridded rainfall totals from the University of Delaware and air temperature averages from the Indian Meteorological Institute; we use these gridded averages to interpolate monthly rainfall and temperature values at each district (in mortality-only regressions) or monitor (in regressions involving pollution). Finally, we add time-varying measures of common effluent treatment plant (CETP) capacity and the incidence of the NRCP, which capture the non-uniform intensity of environmental cleanup and policy efforts within the Ganga Basin.[6]

4. Empirical strategy

Our aim is to determine whether or not the 1987 Supreme Court decision affected environmental quality or health outcomes. Our analysis has two broad parts. In the first part, we use difference-in-differences (DD) – comparing outcomes in the affected area to those elsewhere in the Ganga Basin, before and after the verdict – to estimate the impacts of *Mehta vs. Union of India* on infant mortality and river pollution. In the second, we employ instrumental variables (IV) to identify the direct relationship between river pollution and neonatal mortality. The IV strategy serves two purposes: first, it quantifies an important component of the external damages of river pollution; and second, it sheds some light on the mechanisms through which environmental regulation affects health.

4.1. Difference-in-differences

We specify the reduced-form impact of the Supreme Court verdict on neonatal mortality as follows:

$$Mortality_{idt} = \alpha_1 + \alpha_2 T_{dt} + X_{idt}\eta + e_{idt} \quad (1)$$

where $Mortality_{idt}$ 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 decisions in our case – and takes a value of one in affected — or "treated"— districts after October 1987 and zero otherwise. X_{idt} is a vector of individual, location-by-time characteristics, which includes district and year-month fixed effects. The verdict's impact on river pollution is specified analogously:

$$Pollution_{dt} = \beta_1 + \beta_2 T_{dt} + X_{idt}\theta + \epsilon_{idt} \quad (2)$$

where $Pollution_{dt}$ is a dummy for BOD greater than 3 mg/l in district d and year-month t.

From the pool of all Ganga Basin districts, we define our "treatment group" to comprise four districts: Kanpur Nagar, Unnao, Fatehpur, and Rae Bareli. This definition is justified by two facts. First, these are the four districts whose water quality is most likely to be affected by the verdict. Kanpur Nagar is the physical locus of the verdict; Unnao shares the same stretch of the Ganga River with Kanpur Nagar; and Fatehpur and Rae Bareli are the first districts downstream of these two, with borders approximately 28 and 44 km from the Jajmau tannery cluster, respectively (in fact, the Jajmau tanneries are very near to Kanpur Nagar district's downstream border, so that the majority of Kanpur's population is not directly exposed to tannery pollution). Second, Unnao city contains its own cluster of tanneries, so inclusion of this district in the treatment group yields impacts that are net of any tanning output "leakage". Nonetheless, we also test the robustness of our results to the exclusion of one or more non-Kanpur districts from the treatment group.[7]

The crux of our identification strategy is the assumption that $Cov(T_{dt}, \epsilon_{idt}) = 0$ – i.e., that the policy variable is uncorrelated with all unobserved predictors of neonatal mortality (and of pollution). We argue that this is a plausible assumption because environmental public interest litigation had no prior precedent in India, and because M.C. Mehta's choice of Kanpur was motivated primarily by its central location and the salience of pollutants coming from its tanneries (M.C. Mehta, personal communication, 12/16/2014). Thus, Kanpur was not *a priori* 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, 2007).

To visually inspect the credibility of our identification assumption, we plot annual averages of both neonatal mortality and exceedance of the BOD threshold in the treatment and control groups over time; the annual averages smooth out otherwise noisy monthly time series. We then fit equations (1) and (2) to produce point estimates of policy-induced mean-shifts in these outcomes. In all cases, mortality regressions are run with child-month observations and pollution regressions are run with district-month observations.

4.2. Instrumental variables

Estimation of equation (1) yields the reduced-form impact of policy on neonatal mortality, but it provides no information on the mechanisms of that impact. Theoretically, the Supreme Court decision could have had an impact on mortality through three channels: (i) reduced water contamination, (ii) reduced *exposure* to contaminated water, and (iii) increased ability to cope with the consequences of pollution. In our context, it is likely that, in addition to reducing water contamination, the Supreme Court verdicts also drew attention to the river pollution problem; this could have driven households to avoid exposure through water-source switching or filtration, and to seek information on how to treat the consequences of contamination. Policy research in the environment-development literature frequently does not attempt to disentangle the relative importance of the reduced-contamination channel from that of other possible channels (e.g., Greenstone and Hanna, 2014; Watson, 2006).

In principle, knowledge of river pollution's direct impact on mortality can improve policy design. First, in quantifying the health burden imposed by pollution, we gain a sense of the potential for health improvements through policies that reduce pollution. Second, we can use this "dose-response function" to translate estimated policy impacts on pollution (from equation (2) into a prediction of policy impacts on mortality *exclusively* through the reduced-contamination channel. If that prediction differs significantly from our reduced-form estimates of policy impacts on mortality (from equation (1)), it is evidence that the policy operates at least in part through other channels. Note, moreover, that the reduced-contamination channel captures both the direct impact of the Supreme Court decision and any follow-on contamination-reducing policies communities could have undertaken in the aftermath of the verdicts.

Our primary obstacle in estimating a pollution-mortality dose-response function is the endogeneity of pollution. In general, pollution 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). To solve this problem, we instrument for pollution in a given location-time with its *upstream*

[6] We do not have any other information about the placement, utilization or other operational details of this CETP, or others like it, in India. All the results in this paper however, are robust to the exclusion of these policy controls.

[7] There is no pollution monitor in Fatehpur district, so the treatment group contains only three districts in regressions involving pollution data.

Table 1
Summary statistics for all variables.

	Treatment (Kanpur region)					Control (Rest of Ganga Basin)				
	N	Mean	St. Dev.	Min	Max	N	Mean	St. Dev.	Min	Max
Panel A. Child-level variables										
1[Child died in the first month of life]	8175	0.08	0.27	0	1	639,690	0.06	0.25	0	1
1[Child died in the first year of life]	8175	0.11	0.32	0	1	639,690	0.09	0.29	0	1
1[Child died in the first year of life | survived first month]	7517	0.04	0.19	0	1	598,254	0.03	0.18	0	1
1[Child was born after the verdict]	8175	0.81	0.40	0	1	639,690	0.83	0.38	0	1
1[Mother is Hindu]	8175	0.80	0.40	0	1	639,690	0.84	0.37	0	1
1[Scheduled Caste/Scheduled Tribe]	8175	0.29	0.45	0	1	639,690	0.27	0.44	0	1
Age of mother (years) at time of interview	8175	33.19	6.68	15	44	639,690	32.58	6.71	15	44
1[Mother is literate]	8175	0.35	0.48	0	1	639,690	0.33	0.47	0	1
Panel B. District-level variables										
Ln(BOD)	383	1.07	0.27	0	2	8048	0.94	1.06	−2	5
1[BOD > 3]	383	0.34	0.48	0	1	8048	0.39	0.49	0	1
1[FCOLI > 50]	285	0.59	0.49	0	1	6147	0.34	0.47	0	1
1[Calc > median]	380	0.36	0.48	0	1	6394	0.51	0.50	0	1
1[Sulfates > median]	294	0.50	0.50	0	1	6275	0.49	0.50	0	1
1[Chlorine > median]	386	0.59	0.49	0	1	6842	0.49	0.50	0	1
Air temperature (degrees C)	1095	25.47	5.95	11.92	35.47	84,740	24.78	5.71	8.1	36.11
Monthly precipitation (mm)	1095	71.32	110.76	0	706.26	84,740	89.77	134.55	0	1467.02
1[National River Conservation Plan]	398	0.56	0.50	0	1	8337	0.43	0.50	0	1
Common Effluent Treatment Plant capacity (MLD)	398	0.00	0.00	0	0	8337	0.12	1.29	0	24

Notes: In Panel A, an observation is a child. In Panel B, it is a district-month. The sample marked 'Treatment' consists of observations from Kanpur, Unnao, Fatehpur, and Rae Bareli districts. The sample marked 'Control' consists of observations from all other districts in the Ganga Basin.

counterpart, as defined in Section 3. The logic of this instrument leverages the unidirectional flow of rivers: upstream pollution reliably flows downstream (subject to some decay function of distance) to impart a negative health impact, and yet the determinants of this upstream pollution are plausibly orthogonal to downstream determinants of health. Our implicit assumption is that river flow is distinct from movements in economic, political and demographic variables, which likely take longer to diffuse from upstream to downstream.

We start by estimating the reduced-form analog of our IV strategy:

$$Mortality_{idt} = \gamma_1 + \gamma_2 T_{dt} + \gamma_3 Pollution^{u}_{dt} + X_{idt}\phi + \epsilon_{idt}, \quad (3)$$

where $Pollution^{u}_{dt}$ is the pollution reading upstream of district d in year t. The primary parameter of interest in equation (3) is γ_3, the impact of upstream pollution on downstream mortality. The point estimate of γ_3 gives us a first check on whether our IV strategy will successfully identify the dose-response function; it also provides a causal estimate of the downstream external damages created by pollution, hence extending the work of Lipscomb and Mobarak (2017) and Sigman (2002, 2005) on the presence of pollution spillovers. Moreover, we can test the credibility of our instrument with a falsification test: for every district in our pollution sample, we replace our first-choice "upstream district" with the nearest off-river district available in our data and re-estimate equation (3). Statistical insignificance of γ_3 in the falsification check is a necessary condition of instrument validity.

We then estimate our full two stage least squares (2SLS) model using, as a first stage,

$$Pollution_{dt} = \gamma'_1 + \gamma'_2 T_{dt} + \gamma'_3 Pollution^{u}_{dt} + X_{idt}\phi' + \epsilon'_{idt} \quad (4)$$

and, as a second stage,

$$Mortality_{idt} = \gamma_1 + \gamma_2 T_{dt} + \gamma_3 \widehat{Pollution}_{dt} + X_{idt}\phi + \epsilon_{idt}. \quad (5)$$

Coefficient γ_3 in equation (5) now provides the estimated impact of river pollution on neonatal mortality in the average district. This point estimate provides a reference point for the magnitude of pollution's burden on infant health. Combining this with the spillover mortality impact in the next district downstream identified from equation (3), we have a summary estimate of the external infant mortality cost imposed by river pollution.

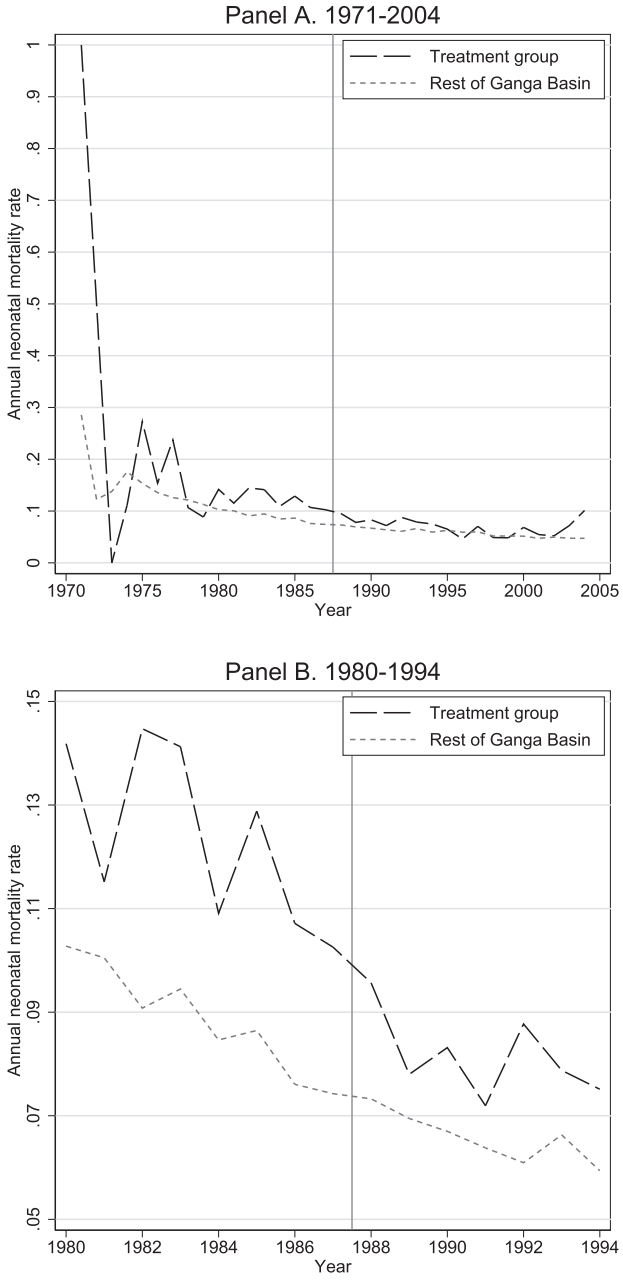
Equations (4) and (5) also provide the basis for testing the mechanisms at work. We compare the estimated mortality burden of pollution with a single instrument (upstream pollution) to the estimated burden with two instruments (adding the Supreme Court verdict). If the policy affects health through non-pollution channels, then an overidentification test should reject the null hypothesis that the policy instrument is a valid instrument for pollution. This null will also be rejected if the effect of pollution on mortality is heterogeneous across (e.g.) locations or pollutants, so that each of the two instruments measures a different local average treatment effect. *Failure* to reject the null, on the other hand, provides suggestive evidence that pollution is the primary channel of policy-induced reductions in neonatal mortality. The overidentification test uses a C-statistic (see, e.g., Eichenbaum et al., 1988), also known as a difference-in-Sargan test statistic. It is equal to the difference of the two Sargan-Hansen J-statistics obtained from the regression using both T_{dt} and $Pollution^{u}_{dt}$ as instruments on the one hand and the regression using only $Pollution^{u}_{dt}$ on the other hand.[8]

5. Results

We report summary statistics for each analysis variable in Table 1, comparing the treatment group to the control group. The top panel tabulates statistics from variables observed at the child level. The bottom panel, meanwhile, describes those variables that we measure at the district level.

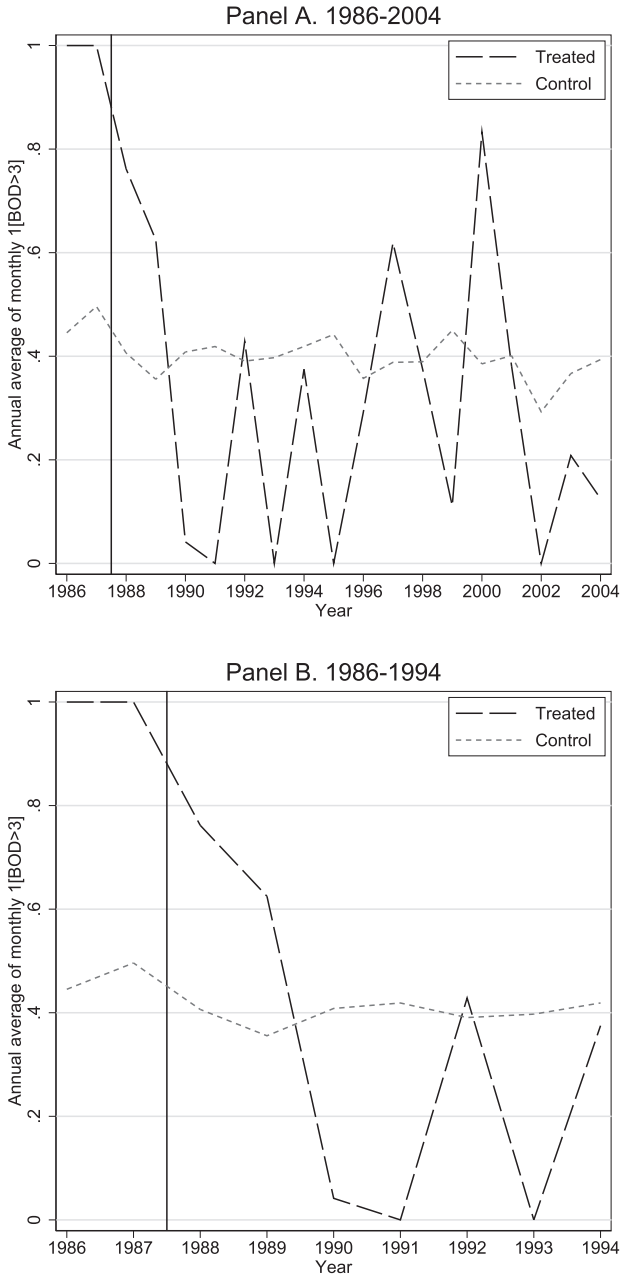
We highlight several noteworthy observations from Table 1. District-level neonatal and infant mortality rates over our observed time period are somewhat larger (8 percent and 11 percent, respectively) in the treatment group than in the control group (6 percent and 9 percent, respectively). These Ganga Basin rates are, in turn, larger than the analogous national rates of 5.3 percent and 7.6 percent in our data,

[8] In Appendix B, we formally derive this test from a sparse model of pollution, policy, and health.



Notes: Both panels plot, separately for the treatment group and control group, annual averages of monthly neonatal mortality rates. Panel A displays averages from the full sample period of 1971-2000. Panel B focuses on the shorter time period of 1980-1994.

Fig. 3. Neonatal mortality: Graphical difference-in-differences.



Notes: Both panels plot, separately for the treatment group and control group, annual averages of the monthly value of 1[BOD>3]. Panel A displays averages from the full period of available data, 1986-2004. Panel B focuses on the shorter time period of 1986-1994.

Fig. 4. Biochemical oxygen demand: Graphical difference-in-differences.

respectively.[9] Meanwhile, average log-BOD is higher in the treatment group than in the control group (1.07 vs. 0.94), but the relationship flips when we summarize the frequency of exceeding the national standard of BOD < 3 mg/l for "bathing class" water (0.34 in the treatment group vs. 0.39 in the control group). Moreover, there is no consistent relationship between treatment and control across all pollutants considered: high sulfate and chloride levels are more frequently observed in the Kanpur region, but high calcium levels are *less* frequently observed there, and high FCOLI levels are about equally likely to occur in each group. While Table 1 reveals several cross-sectional differences between the treatment and control, the inclusion of district fixed-effects controls for these differences.

[9] According to the World Development Indicators (2017), the national infant mortality rate was 14.6 in 1971 and 5.78 in 2004; our measured sample-wide rate of 7.6 is closer to the latter number because births recorded in DLHS-2 are predominantly from more recent years.

Table 2
Mehta vs. Union of India and Infant Mortality.

	(1) IM	(2) NM	(3) Cond. IM	(4) IM	(5) NM	(6) Cond. IM
1[Kanpur] X 1[Post-Verdict]	−0.005***	−0.024***	−0.002	−0.004***	−0.020***	−0.001
	(0.001)	(0.006)	(0.001)	(0.001)	(0.005)	(0.001)
Dependent variable mean	0.008	0.065	0.003	0.010	0.073	0.004
Sample time frame	1971–2004	1971–2004	1971–2004	1980–1994	1980–1994	1980–1994
Adj. R-Squared	0.003	0.014	0.001	0.003	0.017	0.001
N	7,215,872	647,861	6,568,008	3,440,687	326,430	3,114,257

Notes: All results are based on estimation of equation (1). An observation is a child-month. In all columns, the dependent variable is a dummy for mortality. In columns 1 and 4, the sample includes all observations from the first twelve months of life and corresponds to infant mortality (IM). In columns 2 and 5, the sample includes only observations from the first month, corresponding to neonatal mortality (NM). Columns 3 and 6 include observations from months 2–12, which captures infant mortality conditional on neonatal survival (Cond. IM). All regressions include a set of controls (religion of the household head, caste of the household head, mother's age, mother's literacy, CETP capacity, air temperature, total precipitation, and NRCP dummy) and district and year-month fixed effects. Observations are weighted by the product of survey sampling weight and inverse size of the relevant age cohort (in months). Standard errors are clustered at the district level in parentheses. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent levels, respectively.

5.1. Difference-in-differences: a graphical representation

We begin our analysis by graphically inspecting our difference-in-differences (DD) assumption of parallel trends in the treatment and control group. The results are depicted in Figs. 3 and 4. In both figures, data points are annual averages of monthly observations in either the treatment group or the control group. Pre-ruling summary statistics for treatment and control are provided in Appendix Table A1.

Fig. 3 contains two panels; the top panel shows neonatal mortality averages from the full period, while the bottom panel uses data within a smaller, symmetric 15-year window (1980–1994) surrounding the Supreme Court verdicts. In both panels, the vertical distance between the treatment and control group means is smaller after the verdicts than before them. The smaller scale of Panel B allows for a closer look, which shows parallel decreasing pre-trends, a roughly-constant slope in the control group over the full period, and a drop in treatment-group mortality in the aftermath of the verdicts.

Fig. 4 provides analogous results for river pollution, using a dummy for exceedance of the bathing quality standard (BOD > 3) as the dependent variable. Since this specification requires pollution data, the full time period available to us is 1986–2004. The top panel utilizes the full period, while the bottom panel restricts the estimation sample to 1986–1994 – a "short-run" post-period. Here, the outcome variable is somewhat noisy in the treatment group. In the short pre-period available to us for pollution, however, treatment- and control-group trends are parallel. Meanwhile, the likelihood of BOD exceeding the 3-mg/l cutoff drops significantly for the treatment group in the post-period, indicating a large DD estimate of the impact of the ruling on water quality.

While in principle it is possible to compare our treatment group to a single nearby (but not downstream) control district, inherent noise in our infant mortality and pollution data makes such a comparison imprecise. We illustrate this in Appendix Fig. 1 by plotting average mortality (Panel A) and pollution (Panel B) in the treatment group and in a single district (Kannauj) just upstream of Kanpur Nagar.[10] These graphs document a large time-series variance in outcomes of interest. Using the entirety of the Ganga Basin in our control group allows us to smooth our counterfactual significantly.[11]

5.2. The impact of Mehta vs. Union of India on infant health

To obtain a point estimate of the Supreme Court verdicts' impact on infant health, we estimate equation (1). Table 2 provides our first set of results from this estimation. In columns 1–3, we use the full 1971–2004 period, while in columns 4–6, we use the shorter 1980–1994 period. Within each of these triplets, the first column uses one-year (infant) mortality as the dependent variable, the second uses one-month (neonatal) mortality, and the third uses one-year mortality *conditional on survival past the first month*. In all columns, we multiply RCH-2 sampling weights by the inverse size of the relevant age cohort and use these as weights – for example, all observations from children at an age of one month are assigned a weight equal to (1/# of observations with age = one month). This weighting makes possible the translation of our point estimates, which come from monthly observations, into aggregate changes in mortality risk across the whole full first year of an infant's life.[12]

The remainder of the columns in Table 2 separate out these one-year mortality results into impacts in the first month of life vs. in months two through twelve. Given a baseline neonatal mortality risk of 12.1 percent in the treatment group prior to the verdicts, columns 2 and 5 indicate large impacts, with statistically significant point estimates of −0.024 (or 19.5 percent) and −0.020 (or 16.2 percent), respectively. Meanwhile, the estimated mortality impact in months two through twelve is a much smaller and statistically insignificant −0.001 in both column 3 and column 6. Together, Tables 1 and 2 reveal that most infant mortality events are neonatal and that the environmental policy in question here is associated with large-magnitude reductions in neonatal mortality – on the order of 16–20 percentage points. The adverse effect of pollution beyond the first month is more likely to be captured by morbidity, which we do not observe over time. We thus focus exclusively on neonatal mortality for the remainder of this paper, touching on possible mechanisms of exposure in Section 6.

We explore the timing and geography of the verdicts' impacts on neonatal health in Table 3, using the same child-month mortality panel as in Table 2. Columns 1–3 show results from neonatal mortality regressions using post-periods of different lengths, while column 4 shows results from simultaneous estimation of short-, medium-, and long-run policy impacts. Column 5 tests the robustness of the main effect to the use of all of India as a control group. All estimates are statistically significant at the five-percent level or lower. Magnitudes range from 2 to 3.5 percentage points in these columns, which imply a 16–29 percent

[10] The district of Kannauj is immediately upstream of Kanpur Nagar. The two Kannauj pollution monitors in our data lay approximately 93 km upstream of the tanneries.

[11] We also test the robustness of the graphical DD strategy to the definition of the treatment group, by graphing treatment- and control-group average outcomes after redefining the treatment group to only include Kanpur Nagar district itself. The results are depicted in Appendix Fig. A2; they do not change in any qualitatively meaningful way.

[12] For example, column 1 indicates that, after the verdicts, the probability of infant death in a given month drops by 0.005 in the Kanpur region. This translates to a 28.9 percent reduction in the aggregate risk of infant mortality. The significance of point estimates in Table 2 is unaffected by choice of weights.

Table 3
Timing and scope of neonatal mortality impacts.

	Dependent variable: 1[Child died in first month of life]						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
1[Kanpur] X 1[Post-Verdict]	−0.026***	−0.027***	−0.020***		−0.026***		−0.020***
	(0.006)	(0.005)	(0.005)		(0.005)		(0.005)
1[Kanpur] X 1[10/1987 < t < 12/1994]				−0.020***			
				(0.005)			
1[Kanpur] X 1[1/1995 < t < 12/1999]				−0.035***			
				(0.006)			
1[Kanpur] X 1[1/2000 < t < < 12/2004]				−0.024**			
				(0.010)			
Kanpur X 1[Some *in utero* treatment]						0.016	
						(0.015)	
Kanpur X 1[Full *in utero* treatment]						−0.029***	
						(0.010)	
1[Near Downstream] X 1[Post-Verdict]							−0.032***
							(0.005)
1[Intermediate Downstream] X 1[Post-Verdict]							0.000
							(0.007)
1[Far Downstream] X 1[Post-Verdict]							−0.004
							(0.007)
Dependent variable mean	0.064	0.067	0.073	0.064	0.059	0.073	0.073
Geographic coverage	Ganga	Ganga	Ganga	Ganga	India	Ganga	Ganga
Sample time frame	1980–2004	1980–1999	1980–1994	1980–2004	1980–1994	1980–1994	1980–1994
Adj. R-Squared	0.013	0.014	0.017	0.013	0.021	0.017	0.017
N	633,530	509,490	326,430	633,530	720,640	326,430	326,430

Notes: All results are based on estimation of equation (1). An observation is a child-month. The dependent variable in all columns is a binary variable equaling one if a child died in the first month of life. All regressions include a set of controls (religion of the household head, caste of the household head, mother's age, mother's literacy, CETP capacity, air temperature, total precipitation, and NRCP dummy) and district and year-month fixed effects. Observations are weighted by survey sampling weights. 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	1[BOD > 3]						Ln(BOD)	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
1[Kanpur] X 1[Post-Verdict]	−0.534***	−0.568***	−0.607***		−0.696***	−0.637***	−0.236*	−0.108
	(0.058)	(0.050)	(0.055)		(0.028)	(0.052)	(0.125)	(0.116)
1[Kanpur] X 1[10/1987 < t < 12/1994]				−0.492***				
				(0.133)				
1[Kanpur] X 1[1/1995 < t < 12/1999]				−0.548***				
				(0.079)				
1[Kanpur] X 1[1/2000 < t]				−0.578***				
				(0.133)				
1[Near Downstream X 1[Post-Verdict]						−0.655***		
						(0.051)		
1[Intermediate Downstream X 1[Post-Verdict]						−0.249		
						(0.229)		
1[Far Downstream X 1[Post-Verdict]						0.060		
						(0.058)		
Dependent variable mean	0.392	0.408	0.416	0.392	0.286	0.416	0.947	0.889
Geographic coverage	Ganga	Ganga	Ganga	Ganga	India	Ganga	Ganga	Ganga
Sample Time Frame	1986–2004	1986–2000	1986–1994	1986–2004	1986–1994	1986–1994	1986–2004	1986–1994
Adj. R-Squared	0.563	0.559	0.594	0.563	0.553	0.608	0.781	0.800
N	8431	5530	2883	8431	12,521	2883	8431	2883

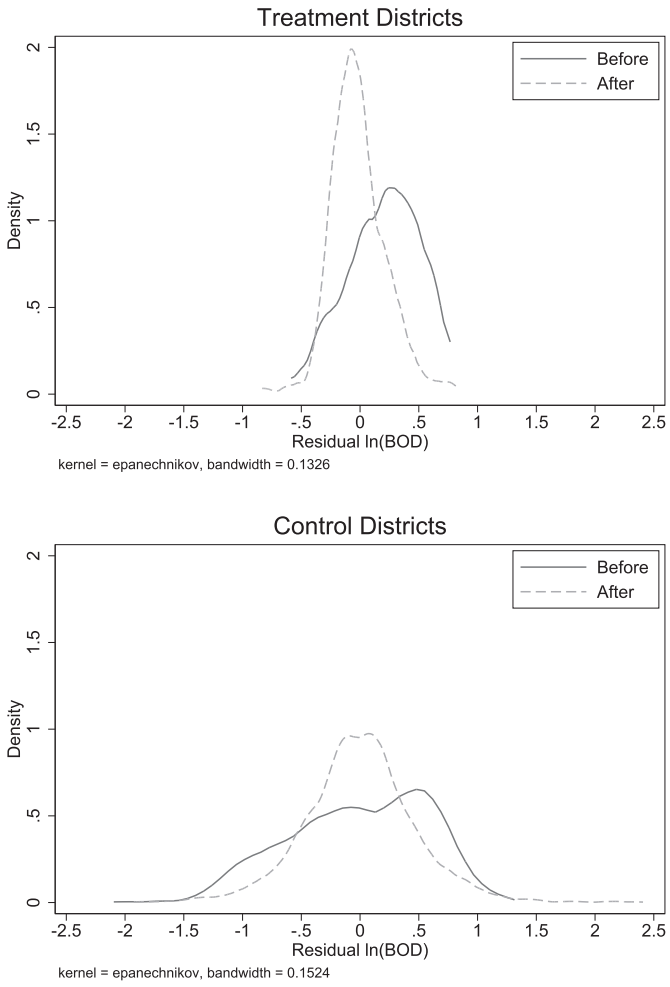
Panel B. Non-BOD Pollutants	Calcium	Sulfates	Chlorides	FCOLI
	(1)	(2)	(3)	(4)
1[Kanpur] X 1[Post-Verdict]	−0.178**	−0.444***	−0.149**	−0.010
	(0.074)	(0.048)	(0.056)	(0.101)
Dependent variable mean	0.631	0.551	0.458	0.447
Geographic coverage	Ganga	Ganga	Ganga	Ganga
Sample Time Frame	1986–1994	1986–1994	1986–1994	1986–1994
Adj. R-Squared	0.595	0.412	0.650	0.531
N	2616	2542	3018	2003

Notes: All results are based on estimation of equation (2). An observation is a district-month. The dependent variable is listed above each column number. 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.

drop in neonatal mortality rates as a result of the rulings. Pairwise F-tests reject the null hypothesis that the 1995–1999 (medium-run) coefficient is equal to either of the other two period-specific coefficients (p-value < 0.01).

In column 6, we investigate the extent to which the mortality reduction is driven by reduced *in utero* exposure to pollution. To do so, we categorize treatment-group births by their degree of *in utero* exposure. *All* children born after the Supreme Court verdicts experience lower post-natal exposure. However, children born in the first nine months after the Supreme Court verdicts (November 1987 to July 1988) experience only partial *in utero* reduction in exposure to contaminated water, while children born after July 1988 fully benefit from the effect of the verdicts. We define dummies for these two groups of children in the treatment region and estimate impacts for each group simultaneously. The predictive effect of fully benefiting from the verdicts while *in utero* is a statistically-significant 2.9 percentage-point drop in neonatal mortality risk. Meanwhile, for children only partially "treated" by the Supreme Court rulings while *in utero*, we find a positive though statistically *in*significant effect on mortality risk. This result is consistent with much existing evidence that links mother's health to the risks of neonatal mortality (Gluckman et al., 2008; Currie and Almond, 2011). We note, however, that the difference between being partially and fully affected by the policy *in utero* and other temporal factors cannot be disentangled. That is, other changes in the treatment region after July 1988 could also explain the result – for instance, relatively weaker policy effectiveness in the initial year following the verdicts.

In column 7, we test whether neonatal mortality impacts of regulation persist downstream of our defined treatment group. We define three downstream groups. 'Near Downstream' denotes the nearest group downstream and includes Pratapgarh, Kaushambi, and Allahabad districts. 'Intermediate Downstream' is the next nearest group and consists of Sant Ravidas Nagar, Mirzapur, and Varanasi districts. 'Far Downstream' collects the remaining 11 Indian districts through which the Ganga River travels beyond Varanasi. We estimate coefficients on the interaction between dummies for each of these groups and the post-ruling dummy. Only the 'Near Downstream' coefficient is statistically significant. In fact, it is significantly larger than the main treatment-group coefficient (−0.32 vs. −0.20), which may be explained by the location of Kanpur's tanneries very near to the downstream district border. Meanwhile, the lack of impacts beyond Allahabad is not surprising, because pollution decay and other inputs to water quality should attenuate the effect the rulings as we move our focus downstream.



Notes: Both panels show kernel densities of residuals from a regression of ln(BOD) on CETP capacity, air temperature, total precipitation, and NRCP dummy and district and year-month fixed effects. Panel A shows residuals from treatment districts, while Panel B shows residuals from control districts. Both panels plot separate densities for before vs. after the Supreme Court verdicts.

Fig. 5. Biological oxygen demand (BOD).

5.3. 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. Did they do so by reducing river pollution? One necessary condition for this explanation to hold is that the verdict is associated with reduced pollution levels. We test this condition in our reduced-form DD framework, estimating equation (2). Panel A of Table 4 reports regression results with our primary pollutant, BOD, while Panel B shows results for other relevant pollutants.

The BOD results displayed in columns 1–6 uniformly imply a significant drop in pollution. Our baseline estimate, shown in column 1 and corresponding to the full time-period of available pollution data, implies a 53.4 percentage-point drop in the likelihood of exceeding the bathing class BOD standard after the Supreme Court rulings. Prior to the ruling, the likelihood of exceedance in treated districts was 100 percent. The drop in exceedance is consistently observed throughout the post-verdict period, as shown in columns 2–4. It is also robust to an expansion of the control group to include all of India (column 5). Meanwhile, downstream impacts in column 6 mirror those from Table 3, column 7: the nearest group of districts downstream of the treatment group is associated with a large and significant average drop in exceedance of the water quality standard, and the point estimate monotonically drops in the next two groups downstream. The evidence thus implies that the rulings' impacts on both water quality and neonatal mortality persist some distance downstream of the cases' focus.

In columns 7 and 8, we use the logarithm of the BOD level as our outcome variable instead of the bathing class dummy. With this specification of BOD, and over the full time period (column 7), the rulings are associated with a marginally significant drop in log-BOD, but the association is smaller and insignificant in the short run. To investigate the divergent results between a dummy variable and the continuous logarithm form, we graphically examine the distribution of the logarithm of BOD by time and treatment status. Fig. 5 provides kernel densities of the residuals from regressing log- BOD on weather controls and district and year-month fixed effects. Panel A suggests that the verdicts are associated with significant, yet non-uniform, drops in BOD levels; the central peak shifts downwards, but the left tail is relatively unchanged. In contrast, Panel B illustrates a symmetric tightening of the log-BOD distribution around the mean. Put together, the two kernel density plots paint a picture of treatment-group reductions in BOD that are relatively concentrated in the middle to upper half of the distribution.

Table 5
Impact of upstream pollution on downstream mortality.

	Dependent variable: 1[Died in first month]						
	OLS	Upstream Districts				Placebo Districts	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
1[BOD > 3]	−0.007						
	(0.010)						
1[US BOD > 3]		0.030***	0.020**	0.022***	0.030**	−0.006	0.006
		(0.010)	(0.009)	(0.007)	(0.014)	(0.009)	(0.004)
Dependent variable mean	0.067	0.067	0.058	0.067	0.066	0.071	0.057
Upstream definition	[75,200]	[75,200]	[75,200]	[0,200]	[100,200]	N/A	N/A
Last sample year	1986–1994	1986–1994	1986–2004	1986–1994	1986–1994	1986–1994	1986–2004
Adj. R-Squared	0.015	0.016	0.010	0.016	0.016	0.012	0.011
N	9603	9603	32,560	13,608	8979	2626	16,891

Notes: An observation is a child-month. The dependent variable in all regressions is a binary variable equaling one if a child died in the first month of life. Column 1 shows the results from OLS estimation of mortality on pollution. Columns 2–5 show results from estimation of equation (3). Columns 6 and 7 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, the sample is defined by its listed 'Upstream definition', 'Geographic coverage', and 'Last sample year' ("N/A" indicates no sample restriction based on upstream distance). 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. Observations are weighted by survey sampling weights. Standard errors are clustered at the district level in parentheses. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent, respectively.

Panel B of Table 4 provides estimates of the impact of other pollutants that are relevant to the tanning industry and the Supreme Court verdicts. Calcium, sulfate, and chloride ions are all by-products of the tanning process; all drop significantly in the aftermath of the rulings (columns 1, 2, and 3). FCOLI, meanwhile, is a strong indicator of domestic pollution and thus should be observed to drop if the second of the two Supreme Court verdicts was successful. We find no evidence of such a drop (column 4).

5.4. Mechanisms of policy transmission

Having identified a strong link between the Ganga Pollution Cases and the local health of both infants (Tables 2 and 3) and rivers (Table 4), we now turn to the questions of how water pollution affects infant health and what that means for Indian environmental policy. Our strategy for answering these questions relies on an estimation of a dose-response function for water pollution and neonatal mortality. We use the merged sample of water pollution and neonatal mortality in this phase of analysis, which limits us to the 1986–2004 time period. To utilize our IV, we must further restrict our geographic focus to only those districts with non-missing upstream pollution measurements. Appendix Table A2 shows how the pool of available pollution monitors and their distance from upstream counterparts varies with our definition of upstream (which is detailed in Section 3). Appendix Table A3 summarizes the merged sample using our preferred upstream definition of [75 km, 200 km].[13] Finally, Appendix Table A4 reproduces the main results of our DiD analysis with the merged sample.

Table 5 establishes a causal relationship between pollution and infant mortality. Column 1 shows the OLS relationship between BOD pollution (i.e., exceedance of the bathing-class standard) and neonatal mortality. It is a statistical zero, which may reflect the upward bias that would result from positive correlations between pollution, urbanization, and economic activity. Columns 2–5 replace the key independent variable with its upstream counterpart – the reduced-form analog of 2SLS with upstream BOD as the instrument. The specifications in these columns vary only by either sample time period or the definition of upstream. In stark contrast to the OLS result, the coefficient on upstream water quality is positive and significant across all four specifications. The interpretation of these coefficients is that exceeding the BOD standard for bathing-class water quality upstream increases the downstream likelihood of neonatal mortality by 2–3 percentage points – a significant spatial externality.

Our empirical strategy hinges on the assumption that upstream river pollution is exogenous once we condition on weather, policy, and district and year-month fixed effects. Implicit in this assumption is that these controls fully capture the endogenous location of households. To bolster this argument, we employ a placebo test that provides additional evidence supporting our exclusion restriction. In columns 6 and 7, each upstream district is replaced with the nearest off-river neighbor with non-missing pollution data. If the resulting 'falsified' upstream pollution variable remains a significant predictor of downstream mortality, then we are likely capturing spurious, spatial correlation between a given district and its neighbors due to the regional nature of many health and environmental shocks. This, however, is not the case: the falsified upstream pollution variable loses both its magnitude and significance, thereby suggesting that our true upstream variable is isolating quasi-random variation in pollution that originates upstream and yet flows downstream to other districts.

The next step is to estimate the dose-response function and use it to test the importance of the pollution mechanism. The results of this step are documented in Table 6, which details the first and second stages of our 2SLS estimation and the associated over-identification tests. In columns 1–4, we employ a single instrument – upstream pollution, as before. Once again, the first three columns pertain to short-, medium-, and long-run periods of observation, while the fourth uses the full time period but parameterizes the policy variable as three period-specific dummies. The first-stage is strong in all four columns.[14] In the second stage, all specifications yield statistically significant point estimates of the pollution coefficient (in Panel B), which is approximately four to six times larger in magnitude than its upstream counterpart in Table 5. The implication is that surpassing the BOD standard of 3 mg/l raises the risk of neonatal mortality by 10–15 percentage points.

The first four columns of Table 6 also provide some initial evidence on policy mechanisms. In these columns, the point estimates of the treatment coefficient in Panel B reveal whether the verdicts remain predictive of mortality impacts once pollution enters into the equation directly. In all cases, these point estimates are statistically insignificant, which strongly suggests that the verdicts acted on health primarily

[13] The 75-km lower bound minimizes the risk of off-river spatial correlation, while the 200-km upper bound excludes locations that are so far upstream as to not physically affect downstream pollution levels. We test the robustness of the lower bound in columns 4 and 5 of Table 5 as well as in columns 1–3 and 5–7 of Table A5.

[14] The first stage is also very strong in IV models that replace BOD with sulfates, chlorides, or calcium. These results are available upon request.

Table 6
Instrumental variables impact of pollution on mortality.

	1-IV				2-IV			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A. 2SLS first stage								
1[US BOD>3]	0.211***	0.167**	0.180**	0.172**	0.211***	0.167**	0.180**	0.172**
	(0.064)	(0.065)	(0.073)	(0.072)	(0.064)	(0.065)	(0.073)	(0.072)
1[Kanpur] X 1[Post-Verdict]	−0.571***	−0.462***	−0.389***		−0.571***	−0.462***	−0.389***	
	(0.068)	(0.058)	(0.075)		(0.068)	(0.058)	(0.075)	
1[Kanpur] X 1[10/1987 < t < 12/1994]				−0.446***				−0.446***
				(0.143)				(0.143)
1[Kanpur] X 1[1/1995 < t < 12/1999]				−0.353***				−0.353***
				(0.102)				(0.102)
1[Kanpur] X 1[1/2000 < t]				−0.316*				−0.316*
				(0.189)				(0.189)
Panel B. 2SLS second stage								
1[BOD > 3]	0.144**	0.126**	0.111***	0.121***	0.090***	0.100***	0.102***	0.107***
	(0.061)	(0.054)	(0.034)	(0.040)	(0.020)	(0.032)	(0.027)	(0.025)
1[Kanpur] X 1[Post-Verdict]	0.050	0.018	0.009					
	(0.043)	(0.028)	(0.019)					
Dependent variable mean	0.067	0.61	0.058	0.058	0.067	0.061	0.058	0.058
F-statistic (p-value)	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
C-statistic (p-value)					0.348	0.525	0.634	0.469
Sample time period	1986–1994	1986–1999	1986–2004	1986–2004	1986–1994	1986–1999	1986–2004	1986–2004
N	9603	21,703	32,561	32,561	9603	21,703	32,561	32,561

Notes: An observation is a child-month. The dependent variable in all regressions is a binary variable equaling one if a child died in the first month of life. All columns display results from Two-Stage Least Squares estimation of equations (4) and (5); panel A details 2nd-stage results and panel B details 1st-stage results. In columns 1–4, the endogenous variable (BOD > 3) is instrumented using its upstream analog ('1-IV'), while in columns 5–8, both upstream pollution and the policy variable ("1[Kanpur] X 1[Post-Verdict]") are used as instruments ('2-IV'). In 2-IV columns, the listed p-value corresponds to a C-statistic, which tests the null hypothesis that the endogenous variable is overidentified. All regressions 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. Observations are weighted by survey sampling weights. Standard errors are clustered at the district level in parentheses. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent, respectively.

through the pollution channel.[15] We further test for the role of pollution in policy impacts by re-estimating the 2SLS model in columns 5–8 with the same parameters as in columns 1–4, respectively, except with two instruments instead of one. A rejection of the null (i.e., that the policy instrument is valid) is evidence of either (a) multiple channels of policy impact or (b) different local average treatment effects being captured by the two instruments. A failure to reject, on the other hand, does not formally rule out the existence of other mechanisms, but it does support the notion that reductions in pollution were central to the observed declines in mortality.

The results of the over-identification tests are tabulated as *p*-values for the associated *C*-statistic in columns 5–8. These *p*-values are statistical measures of the difference between the dose-response coefficients from 1-IV and 2-IV specifications. The naked-eye version of the test is therefore a pairwise comparison of coefficients on 1[BOD > 3] between columns 1 and 5, 2 and 6, 3 and 7, and 4 and 8. In all cases, the 2-IV coefficient is smaller, but some of the deviation is a natural result of statistical noise.[16] The *p*-value measures the significance of the deviation, and in columns 5–8 it runs from 0.348 to 0.634. That is, in all cases we fail to reject the null hypothesis that pollution fully explains the verdicts' health impact. Appendix Table A5 shows that this finding is robust to the use of different definitions of upstream pollution and extending the control group to the entire remainder of India.

6. Discussion

Our estimates of the Ganga Pollution Cases' sizable environmental and health impacts are important because they represent the first documented success in India's regulation of water quality. In addition, our findings are differentiated by the type of pollution studied. We provide the first quasi-experimental evidence of the association between biochemical oxygen demand and health. Prior work has focused on pollution from either domestic (Field et al., 2011) or agricultural (Brainerd and Menon, 2014) sources. Our own work links mortality to industrial pollution for the first time.[17] In addition, we have described and implemented a novel, replicable methodology for producing an unbiased estimate of the water pollution-health dose-response function leveraging the flow of rivers.

Our reduced-form estimate of the relationship between environmental quality and health mirrors those of Ebenstein (2012) and Brainerd and Menon (2014) and indicates that the costs of river pollution are significant.[18] The latter authors' findings are particularly consistent with our own results, in that they, too, specifically show neonatal mortality

[15] The three period-specific treatment coefficients are omitted from Panel B, column 4 for table conciseness but are similarly small and insignificant.

[16] One back-of-the-envelope method of interpreting our results is to compare the product of our 'policy-on-pollution' and 'pollution-on-health' point estimates with our 'policy-on-health' point estimate. The relevant results must hold constant the time-period (1986–2004), geography (Ganga Basin) and upstream range (here, we choose [75,200]). The reduced form results for this specific sample are in Table A3 (column 1 and column 4) and Table A4 (column 3) respectively. The product of the two coefficients from the reduced form coefficients is 0.111 * (−0.381) = −0.042. This is larger than, but still in the range of, the policy-mortality coefficient of −0.034. We stress, however, that the overidentification test is the statistically correct way to interpret our policy mechanism results.

[17] Our identified BOD impacts could, in principle, capture reductions in domestic pollution, but the absence of impacts on FCOLI – a more direct measure of domestic pollution – makes that explanation unlikely.

[18] 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.

to be affected by water pollution in India. Given widespread breastfeeding practices in India, these results suggest that alternative modes of contamination other than drinking river water – such as bathing and person-to-person transmission – are at work (Cifuentes et al., 2000). Alternatively, pollution exposure *in utero* may explain our neonatal mortality estimates. While we have limited ability to investigate the timing of pollution exposure, our policy analysis suggests that infants whose gestation periods fall fully after the verdicts have a better probability of survival.

In spite of the harms of pollution, environmental quality remains low in many developing countries. Air quality in Delhi, India, for example, averaged 128 $\mu g/m^3$ 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 (see Greenstone and Jack, 2015), which could make the private opportunity cost of pollution reduction greater than the private benefit. 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. Relatedly, Greenstone and Hanna (2014) argue that, in India, air pollution policy has been more effective than water pollution policy because of relatively greater demand for air quality than for water quality. In our context, Kanpur Nagar district had BOD levels that placed it in the 63rd percentile of Ganga Basin water quality prior to the Supreme Court rulings. 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 there *was* significant concern about the economic impacts of regulation in a city that relied heavily on the tanning industry for jobs.[19]

High marginal costs of abatement and political economic distortions are two other possible explanations for poor environmental quality in the developing world. Davis (2008) and Field et al. (2011), for instance, identify environmental regulations whose designs had unintended consequences for air quality in Mexico and infant health in Bangladesh, respectively. Sigman (2002, 2005) and Lipscomb and Mobarak (2017), meanwhile, document greater water pollution upstream of administrative borders, where even a public agent may not fully internalize the social cost of poor water quality. Our IV analysis underscores this problem by documenting the existence of spatial spillovers in river pollution. It also connects this spatial externality directly to health outcomes for the first time, by showing that upstream pollution imparts a mortality burden on downstream communities. River pollution is thus a collective action problem requiring central government regulation and/or inter-jurisdictional bargaining.

Why, then, did the Ganga Pollution Cases succeed where other Indian government action – such as the ambitious National River Conservation Plan (NRCP) – failed? One feature that distinguishes the verdicts from the NRCP is that the environmental policy produced by the former emanated from the judicial branch rather than the executive. Article 21 of the Indian Constitution provides citizens with the "Right to Life"; *Mehta vs. Union of India* is a watershed moment in Indian legal and environmental history because it was the first instance in which this constitutional 'lever' was used to drive environmental policy through the Indian Judiciary. It is plausible that decisions from the judiciary differ from those made by the executive in that they mandate agents to take specific – and verifiable – actions, rather than designing 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 of what made the Ganga Pollution Cases successful might therefore shed light on what types of institutional arrangements lead to successful policy implementation.

7. Conclusion

This paper provides empirical evidence that the Supreme Court decisions in *Mehta vs. Union of India*, which primarily targeted the tanning industry in Kanpur district, induced a drop in both river pollution and neonatal mortality. Our investigation of the mechanisms of policy indicates that the observed drop in pollution may have played an important role in explaining the identified mortality effect. In deriving and conducting our tests of this pollution channel, we show how information about different potential mechanisms can be backed out from analysis even when data on all possible mechanisms are not available.

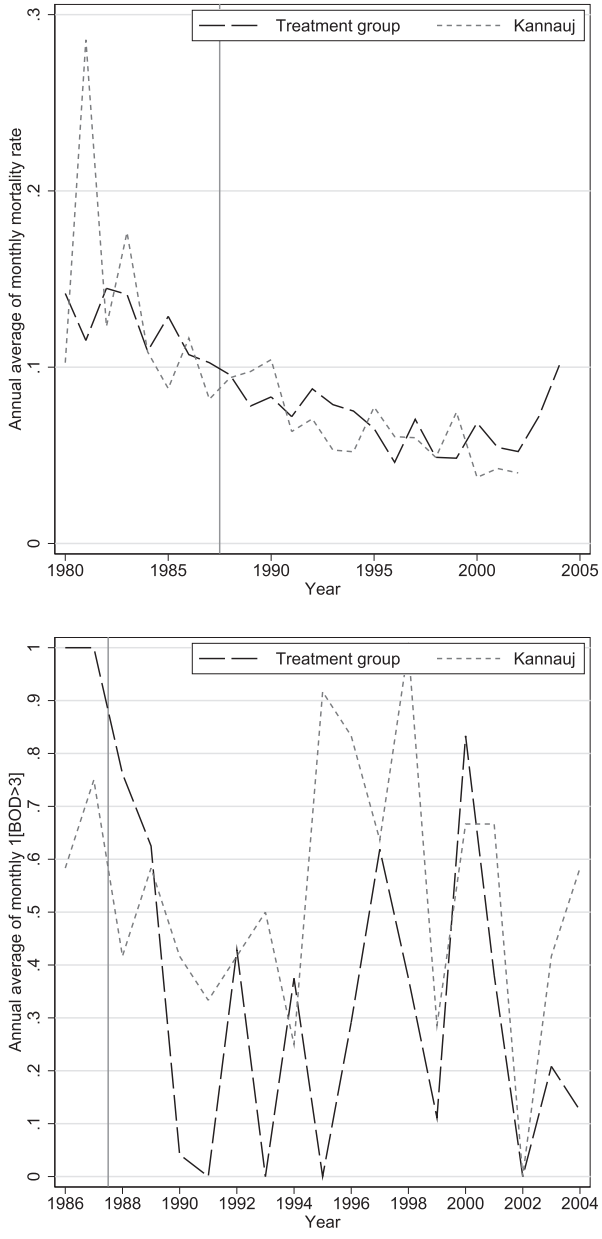
We believe our analysis represents an important contribution to the broader puzzle of continually poor environmental quality in developing countries. First, we have identified a precedent for successful water pollution policy in India. While one data point cannot identify the keys to broadly effective environmental policy, the attributes of the Ganga Pollution Cases and their context – including (but not limited to) judicial backing, industrial focus, and water quality improvements rather than behavior change – may inform the design of successful policy in an area where it has previously proven elusive.

Second, 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 insight into the challenges of designing effective environmental policy in the context of a developing country.

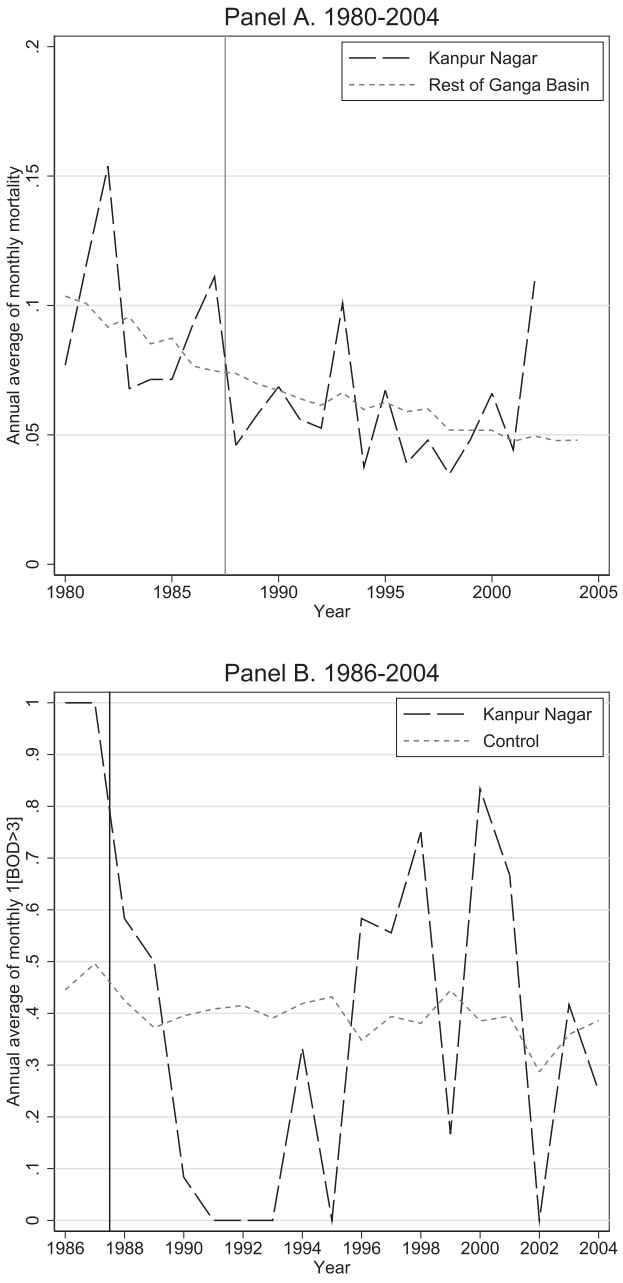
[19] We conducted a qualitative research study in Kanpur in the winter of 2014. The study consisted of interviews of a large number of stakeholders and informants in the Ganga Pollution Cases. These included government officials, owners of tanneries, two non-governmental organizations, several journalists, 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.

A. Appendix tables and figures



Notes: Panel A plots annual average mortality; Panel B plots annual average BOD exceedance (1[BOD>3]). Both panels plot averages in the treatment group and control group separately. The treatment group is comprised of Kanpur Nagar, Unnao, Fatehpur, and Rae Bareli districts. The control group includes only a single upstream district, Kannauj.

Fig. A1. Time trends in the treatment group vs. Kannauj district.



Notes: Panel A plots annual average mortality; Panel B plots annual average BOD exceedance (1[BOD>3]). Both panels plot averages in the treatment group and control group separately. The treatment group is Kanpur Nagar district only. The control group is the rest of the Ganga Basin.

Fig. A2. Time trends in Kanpur Nagar district vs. the control group.

Table A1
Pre-Ruling Summary Statistics for All Variables.

	Treatment (Kanpur region)					Control (Rest of Ganga Basin)				
	N	Mean	St. Dev.	Min	Max	N	Mean	St. Dev.	Min	Max
Panel A. Child-level variables										
1[Child died in the first month of life]	1821	0.12	0.32	0	1	125,931	0.09	0.29	0	1
1[Child died in the first year of life]	1821	0.17	0.38	0	1	125,931	0.13	0.34	0	1
1[Child died in the first year of life | survived first month]	1582	0.06	0.24	0	1	112,623	0.04	0.21	0	1
1[Mother is Hindu]	1821	0.79	0.40	0	1	125,931	0.85	0.36	0	1
1[Scheduled Caste/Scheduled Tribe]	1821	0.25	0.43	0	1	125,931	0.26	0.44	0	1
Age of mother (years) at time of interview	1821	39.54	3.34	28	44	125,931	39.55	3.26	21	44
1[Mother is literate]	1821	0.33	0.47	0	1	125,931	0.30	0.46	0	1
Panel B. District-level variables										
Ln(BOD)	22	1.66	0.31	1	2	468	1.09	0.85	−1	3
1[BOD > 3]	22	1.00	0.00	1	1	468	0.46	0.50	0	1
1[FCOLI > 50]	22	1.00	0.00	1	1	411	0.51	0.50	0	1
1[Calc > median]	22	0.86	0.35	0	1	367	0.47	0.50	0	1
1[Sulfates > median]	22	0.91	0.29	0	1	404	0.47	0.50	0	1
1[Chlorine > median]	22	0.50	0.51	0	1	435	0.50	0.50	0	1
Air temperature (degrees C)	22	26.27	5.90	16	35	471	26.33	5.24	11	36
Monthly precipitation (mm)	22	53.11	75.98	0	291	471	95.52	135.10	0	753
1[National River Conservation Plan]	22	1.00	0.00	1	1	471	0.42	0.49	0	1
Common Effluent Treatment Plant capacity (MLD)	22	0.00	0.00	0	0	471	0.00	0.00	0	0

Notes: All statistics are based on pre-Supreme Court verdicts data. In Panel A, an observation is a child. In Panel B, it is a district-month. The sample marked 'Treatment' consists of observations from Kanpur, Unnao, Fatehpur, and Rae Bareli districts. The sample marked 'Control' consists of observations from all other districts in the Ganga Basin.

Table A2
Sample Composition and Upstream Definition.

	Ganga Basin Only		All India	
	Number of Monitors	Upstream Distance (km)	Number of Monitors	Upstream Distance (km)
[0, 200]	77	96	343	87
[20, 200]	70	102	333	92
[50, 200]	68	114	315	108
[75, 200]	54	128	278	124
[100, 200]	48	146	255	141
[75, 300]	78	177	328	149

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 A3
Summary Statistics for Variables in the Merged Mortality-Pollution Sample.

	Treatment (Kanpur region)					Control (Rest of Ganga Basin)				
	N	Mean	St. Dev.	Min	Max	N	Mean	St. Dev.	Min	Max
1[Child died in the first month of life]	2962	0.06	0.24	0	1	29,599	0.06	0.23	0	1
1[BOD>3]	2962	0.38	0.48	0	1	29,599	0.41	0.49	0	1
Upstream 1[BOD>3]	2962	0.92	0.28	0	1	29,599	0.36	0.48	0	1
1[Child was born after the verdict]	2962	0.95	0.21	0	1	29,599	0.96	0.20	0	1
1[Mother is Hindu]	2962	0.73	0.45	0	1	29,599	0.80	0.40	0	1
1[Scheduled Caste/Scheduled Tribe]	2962	0.26	0.44	0	1	29,599	0.27	0.44	0	1
Age of mother (years) at time of interview	2962	31.27	6.11	16	44	29,599	30.15	6.35	15	44
1[Mother is literate]	2962	0.43	0.49	0	1	29,599	0.37	0.48	0	1
Monthly mean of daily mean airtemp (C)	2962	25.93	5.80	14	35	29,599	25.45	5.69	10	36
Interpolated Precipitation (mm)	2962	73.21	98.83	0	406	29,599	85.46	121.67	0	742
1[National River Conservation Plan]	2962	0.53	0.50	0	1	29,599	0.49	0.50	0	1
Common Effluent Treatment Plant capacity (MLD)	2962	0.00	0.00	0	0	29,599	0.23	1.18	0	6

Notes: The sample includes all child-months with non-missing values of the variables listed, which are those required for IV estimation. The upstream buffer used is [75 km, 200 km]. "N" lists the number of child-month observations. The sample marked 'Treatment' consists of observations from Kanpur, Unnao, and Rae Bareli districts. The sample marked 'Control' consists of observations from all other districts in the Ganga Basin.

Table A4
Policy Impact Regressions with Merged Mortality-Pollution Sample.

	NM	NM	NM	1[BOD > 3]	1[BOD > 3]	1[BOD > 3]
	(1)	(2)	(3)	(4)	(5)	(6)
1[Kanpur] X 1[Post-Verdict]	−0.033**	−0.040***	−0.034**	−0.381***	−0.455***	−0.587***
	(0.013)	(0.013)	(0.014)	(0.076)	(0.059)	(0.071)
Dependent variable mean	0.058	0.061	0.067	0.408	0.391	0.367
Sample Time Frame	1986–2004	1986–1999	1986–1994	1986–2004	1986–1999	1986–1994
Adj. R-Squared	0.009	0.013	0.015	0.681	0.646	0.686
N	32,560	21,703	9603	32,560	21,703	9603

Notes: The sample includes all child-months with non-missing values for neonatal survival, BOD, and upstream BOD (with 'upstream' defined as being in the range [75 km, 200 km]). Columns 1–3 correspond to estimation of equation (1), with a binary dependent variable equaling one if a child died in the first month of life. Columns 4–6 correspond to estimation of equation (2), with a binary dependent variable equaling one if district-average BOD exceeds 3 mg/l in a given month. All regressions include a set of controls (religion of the household head, caste of the household head, mother's age, mother's literacy, CETP capacity, air temperature, total precipitation, and NRCP dummy) and district and year-month fixed effects. Observations are wThe ed by survey sampling weights. Standard errors are clustered at the district level in parentheses. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent levels, respectively.

Table A5
Robustness Checks on Instrumental Variables Regressions.

	1-IV				2-IV			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A. 2SLS first stage								
1[US BOD > 3]	0.169***	0.213***	0.207**	0.275***	0.169***	0.213***	0.207**	0.275***
	(0.063)	(0.062)	(0.093)	(0.039)	(0.063)	(0.062)	(0.093)	(0.039)
1[Kanpur] X 1[Post-Verdict]	−0.561***	−0.604***	−0.459***	−0.674***	−0.561***	−0.604***	−0.459***	−0.674***
	(0.072)	(0.061)	(0.092)	(0.039)	(0.072)	(0.061)	(0.092)	(0.039)
Panel B. 2SLS second stage								
1[BOD>3]	0.154**	0.110**	0.137	0.028	0.096***	0.074***	0.139***	0.035*
	(0.062)	(0.044)	(0.085)	(0.020)	(0.027)	(0.018)	(0.034)	(0.019)
1[Kanpur] X 1[Post-Verdict]	0.054	0.035	−0.001	−0.031**				
	(0.042)	(0.034)	(0.043)	(0.015)				
Dependent variable mean	0.065	0.066	0.067	0.053	0.065	0.066	0.067	0.053
F-statistic (p-value)	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
C-statistic (p-value)					0.259	0.393	0.978	0.214
Upstream Range (km)	[20,200]	[50,200]	[100,200]	[75,200]	[20,200]	[50,200]	[50,200]	[75,200]
Geographic coverage	Ganga	Ganga	Ganga	India	Ganga	Ganga	Ganga	India
N	13,309	11,573	8509	60,511	13,309	11,573	8509	60,511

Notes: An observation is a child-month. The dependent variable in all regressions is a binary variable equaling one if a child died in the first month of life. All columns display results from Two-Stage Least Squares estimation of equations (4) and (5); panel A details 2nd-stage results and panel B details 1st-stage results. In columns 1–4, the endogenous variable (BOD > 3) is instrumented using its upstream analog ('1-IV'), while in columns 5–8, both upstream pollution and the policy variable ("1[Kanpur] X 1[Post-Verdict]") are used as instruments ('2-IV'). In 2-IV columns, the listed P-value corresponds to a C-statistic, which tests the null hypothesis that the endogenous variable is overidentified. All regressions 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. Observations are weighted by survey sampling weights. Standard errors are clustered at the district level in parentheses. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent, respectively.

B. A model to test the mechanisms of impact

We derive a statistical test of the mechanisms of policy impact by leveraging two potential instruments for river pollution: (1) *upstream* river pollution, and (2) the policy itself. We begin by reprinting equation (1).

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

In this reduced-form model of infant mortality, identification rests on the assumption that $Cov(T_{dt}, e_{idt}) = 0$ – i.e., that the policy variable is uncorrelated with all unobserved predictors of neonatal mortality. If this zero-covariance assumption 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 mortality rates as follows:

$$Mortality_{idt} = \alpha + \beta Pollution_{dt} + \widetilde{X}_{idt}\gamma + (Z_{idt}\delta + \varepsilon_{idt}) \tag{A.2}$$

This equation is analogous to equation (5) in the main text, except that it partitions 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 $Cov\left(T_{dt}, \varepsilon_{idt} \mid \widetilde{X}_{idt}\right) = 0$.

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

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

and

$$Pollution_{dt} = \alpha^2 + \beta^2 T_{dt} + \widetilde{X}_{idt}\gamma^2 + \varepsilon^2_{idt} \tag{A.4}$$

We can then rewrite equation (A.2) by substituting for both Z_{idt} and $Pollution_{dt}$, so as to decompose the policy's effect on mortality into the different channels of impact:

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

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

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

which contains both β and the product $\beta^1\delta$. We estimate equation (A.6) using 2SLS, where we instrument for $Pollution_{dt}$ with its upstream analog, $Pollution^u_{dt}$. With unbiased estimates of both β and $\beta^1\delta$, we can then test $H_0 : \beta^1\delta = 0$, the null hypothesis that non-pollution channels of policy impact are inactive.

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

$$Mortality_{idt} = \left[\alpha + \alpha^1\delta\right] + \beta Pollution_{dt} + \widetilde{X}_{idt}\left[\gamma + \gamma^1 \cdot \delta\right] + \left(\varepsilon^1_{idt}\delta + \varepsilon_{idt}\right) \tag{A.7}$$

so that T_{dt} is additionally excluded from second-stage equation (A.7) and becomes another valid instrument for $Pollution_{dt}$. 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 $\left\{T_{dt}, Pollution^u_{dt}\right\}$. To implement such a test, we construct a *C-statistic* (see, e.g., Eichenbaum et al., 1988), which we describe further in the main text.

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