

The Lifesaving Benefits of Water Infrastructure: Quantifying the Mortality Impact of Abandoning Shallow Tubewells in Bangladesh

Nina Buchmann, Erica Field, Rachel Glennerster and Reshmaan Hussam*

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Abstract

We document the consequences of a public health campaign that led to the sudden abandonment of local water infrastructure by one-fifth of Bangladesh's population. Households that were encouraged to abandon shallow tubewells on account of arsenic contamination, which is quasi-randomly distributed in underground aquifers, experienced 28% higher child mortality and 47% higher elderly mortality post-campaign than those not motivated to shift, while there were no differences in mortality pre-trends. Verbal autopsy data reveal that the sudden mortality increases are driven by diarrheal disease. Changes in mortality increase with distance to alternative clean water infrastructure, suggesting that greater storage time is the culprit: those with an alternative source within 300 meters of their home experience no increase in mortality, but mortality rises as households are forced to walk further for arsenic-free water. Our results quantify the mortality benefits of local water infrastructure and underscore the importance of physical proximity, rather than mere access, to pathogen-free water sources.

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*Buchmann: Stanford University, Stanford CA 94305, United States, nina.buchmann@stanford.edu. Field: Duke University and NBER, Durham NC 27710, United States. Glennerster: University of Chicago and NBER, Chicago IL 60637, United States. Hussam: Harvard University and NBER, Boston MA 02163, United States. We thank Mahnaz Islam, Pronita Saxena, Rudmila Rahman, Akshay Dixit, Mehrab Ali, Shakil Ayan, Monia Tomasella, and Pulkit Aggarwal for superb fieldwork and research assistance, and Save the Children for enabling this study, and the National Science Foundation, and the National Institutes of Health for financial support. This project received IRB approval from MIT (no. 0612002074), IPA (no. 121.11June-007), and Harvard (no. 00000109). The authors do not have any conflicts of interest to disclose.

1 Introduction

Globally, diarrhea is the fifth most common cause of death among children under five and the eighth most common cause of death among adults over 70. High among the risk factors documented for diarrheal disease is unsafe water (Troeger et al., 2018). Yet the relationship between water infrastructure and mortality remains disputed, due primarily to the challenge of establishing plausible exogeneity in the presence, or use, of such infrastructure (Cutler and Miller, 2005; Jamison, 2018; Anderson et al., 2021; Kremer et al., 2022). The absence of a clear causal link between water infrastructure and mortality means that water infrastructure is *excluded* from lists of recommended interventions to reduce childhood mortality—let alone adult mortality—by the WHO and the influential Disease Control Priorities (Stenberg et al., 2021). Beyond the availability of clean water infrastructure alone, the appropriate density of clean water sources necessary to protect households from bacterial contamination, particularly for settings in which water sources are outside the home, remains an open question.

Bangladesh offers a unique opportunity to causally identify the impact of water infrastructure and its density on mortality. The surprise discovery in 1998 of naturally-leaching arsenic in groundwater across the country led a large number of households to rapidly abandon shallow tubewells in their backyards for more distant primary water sources (D G Kinniburgh, 2001). Specifically, a government-initiated campaign in 1999 tested millions of tubewells nationwide and encouraged households to abandon those that tested above 50ppb for arsenic (Ahmed et al., 2006). This public health campaign yielded one of the most dramatic changes in health behavior in recent history: by 2006, most households in endemic regions (80%) were aware of arsenic contamination, and in Barisal, where our study takes place, the fraction of households drinking from arsenic-contaminated tubewells dropped from 69% in 1999 to 1% in 2006 (Bangladesh Bureau of Statistics and Unicef, 2006; D G Kinniburgh, 2001).

The campaign raised widespread awareness about the possibility of arsenic contamination, however, the particular subset of households that was encouraged to abandon their backyard water source was effectively exogenous at the local level. Specifically, the geography of Bangladesh generates an unusual degree of micro-spatial variation in arsenic contamination: nearly all contaminated villages in Bangladesh contain pockets of arsenic-free groundwater alongside contaminated water, and these pockets are impossible to predict above ground, making them plausibly exogenous to the socioeconomic status of the households drawing water from these locations before the testing campaign (Smedley and Kinniburgh, 2001).

We exploit the local variation in arsenic exposure, paired with the time discontinuity in knowledge of arsenic contamination generated by the campaign, to examine the impact of shifting from backyard shallow tubewells to further away water infrastructure. Shallow tubewells, while vulnerable to arsenic contamination, are considered “the most appropriate technology in terms of microbiologically clean water” (Lokuge et al., 2004) in settings such as Bangladesh. Not only is water from shallow tubewells unlikely to be contaminated with bacteria at source; it also faces little risk of becoming contaminated at point of use since most households have

shallow tubewells within their compounds and therefore have little need to store water in the home.¹ It is for this reason that millions of shallow tubewells were initially constructed across Bangladesh in the 1970s and 1980s —as a prophylactic to water-borne diseases such as cholera and dysentery. (Prüss et al., 2002).

In this paper, we show that the sudden abandonment of shallow tubewells, which are in close proximity to homes, is associated with a substantial *rise* in mortality among both children and the elderly, which we argue is driven by increased distance to covered (and therefore pathogen-free) water sources. During the campaign, the vast majority of households had access to alternative safe water infrastructure in the form of deep tubewells. However, because deep tubewells are expensive to build, they are present in lower density and hence on average farther away. In our sample, the average minimum distance to the closest deep tubewell from the residence was 541 meters in 2000 instead of fewer than 50 meters to the closest shallow tubewell. As a result, drinking water collected from deep tubewells was typically stored in containers in the home, introducing the risk of contamination at point of use (Cocciolo et al., 2021; Goel et al., 2023). Evaluating the shift from backyard shallow tubewells, which are almost as convenient as piped water, to further away deep tubewells, which are also safe at source but require a commute and storage, therefore allows us to evaluate the mortality impacts of *proximity*, rather than merely access to safe water infrastructure, holding bacterial contamination at source constant.

To do so, we collect novel data on the location and arsenic content of the closest shallow tubewells of approximately 3,000 households randomly sampled from 162 villages in the Barisal district of Bangladesh, as well as the location and date of construction of deep tubewells in these communities. We use within-village spatial variation in whether households were required to abandon shallow wells due to arsenic along with time variation in knowledge of contamination generated by the public health campaign to create a continuous measure of exposure to drinking water drawn from more distant sources. Using annual data on child and elderly mortality before and after the campaign, we then compare mortality outcomes in households encouraged to switch water sources to those of their neighbors in the same village that were not encouraged to switch before and after the campaign. Given the quasi-random distribution of arsenic, our identification meets the strong parallel trends assumption for multi-valued exposure difference-in difference-estimation (Callaway et al., 2021): in the absence of the public health campaign, trends in mortality over time would have been the same for high- and low-concentration households.²

Our results indicate that child and elderly mortality rates were almost identical in households with arsenic-contaminated vs. uncontaminated shallow tubewells within villages before the arsenic campaign. However, mortality diverged sharply immediately afterwards. Post-2000, households encouraged to switch to further away water sources experienced a 28% increase in

¹Proximity of water source is also likely to increase the overall *amount* of drinking water consumed, further decreasing mortality from diarrheal disease.

²Formally, the difference in mortality between low- and high-concentration households would be the same for every number of years lived after the campaign.

child mortality and a 47% increase in elderly mortality relative to those with arsenic-free wells. Verbal autopsy data confirm that this divergence is driven by water-related deaths only and coincides with contaminated households switching to more remote water sources.

To further quantify the health costs of distance to water infrastructure, we compare households located equidistant from a deep tubewell but that either were or were not forced to switch to the deep tubewell due to quasi-random variation in arsenic. We find that mortality effects increase linearly with distance to source—which varies over time and across space—even within the radius of one kilometer: while those who can access a clean well within 300 meters of their home experience no adverse mortality impacts from abandoning their backyard shallow tubewells, each additional 100 meters to a clean alternative source raises both child and elderly mortality.

Our large estimated impacts raise the question of why families did not take greater compensating action to counter the increased exposure to pathogen-contaminated water. [Keskin et al. \(2017\)](#), for example, show mothers in households with arsenic-contaminated wells increase their duration of breastfeeding.³ However, the authors also find that mothers do *not* alter breastfeeding behavior if they have alternative clean water infrastructure within 1,000 meters, as is true for most households in our study context. This suggests that, while the risks of arsenic and surface water are highly salient, the risks of re-contamination of stored water may not be.⁴ Over time, however, we observe compensating behavior in the form of construction of more deep tubewells, especially in areas that had to abandon more shallow tubewells, which may be a reaction to the increase in mortality or the burden of time spent collecting water.

This study makes three contributions. First, we provide evidence of the large causal impacts of pathogen-free water infrastructure on child and elderly mortality. Importantly, we find these mortality impacts despite a context in which individuals frequently ingest pathogens through food and hands ([Kwong et al., 2020](#)), and access to diarrhea treatment is affordable and widespread. Existing economics literature that aims to disentangle the role of water infrastructure, public health reforms, and rising incomes in explaining the sharp declines in mortality in high- and middle-income countries ([Anderson et al., 2021](#); [Cutler and Miller, 2005](#); [Galiani et al., 2005](#); [Devoto et al., 2012](#)) finds widely varying results for the degree to which water infrastructure alone reduces mortality.⁵ This mixed evidence has policy implications:

³This may be a direct response to knowledge of arsenic or pathogen-contaminated alternative sources, or it may be a means of coping with the inconvenience of more distant alternative water sources.

⁴An alternative compensatory action households may undertake is to treat one's water at point-of-use with boiling or chlorination. However, less than 1% of our sample report treating their water at point-of-use. This may be for the same reason as the above: contamination at point-of-use may be less salient than unclean water sources, or it may be a matter of inconvenience/high costs to treating water: "Sometimes we drink rainwater, other times water from the pond," says Masuma Begum. "We boil it if we can but don't always have time. There is no deep tube well near our home, no piped water, no other options for us. My children are often sick and weak. We are too poor to invest in a well." ([Livani et al., 2021](#)).

⁵[Cutler and Miller \(2005\)](#) attribute half the mortality declines in US cities in the late 19th and early 20th century to improved water with water filtration reducing total mortality by 15% and infant mortality by 35%. [Anderson et al. \(2021\)](#) expand the sample and conclude that chlorination and filtration had no measurable impact on elderly mortality while filtration reduced infant mortality by 11%. They also find that water infrastructure reduces deaths from cholera and typhoid but these diseases are a relatively small proportion of overall mortality. Data from

the policy-influential Disease Control Priorities 3 (Jamison, 2018) concludes that there exists limited robust evidence on the impact of clean water on mortality. Water treatment (whether at point of use or source) is, therefore, *not included* in lists of recommended interventions to reduce childhood mortality by DCP-3 or the WHO (Stenberg et al., 2021).⁶ Our study provides new evidence that access to pathogen-free water significantly reduces not only infant and child mortality but also elderly mortality in contexts like that of Bangladesh.

Second, our results underscore the necessity of *proximity* to an improved water source for meaningful improvements in mortality. While there is broad consensus that on-premises water supply reduces diarrhea rates (Wolf et al., 2022), the majority of studies compare improved (treated) on-premises sources to untreated off-premises sources, making it hard to infer the importance of water infrastructure proximity alone.⁷ Existing evidence also focuses on diarrhea rates rather than mortality; and while there is a well-established causal chain from diarrhea to infant and child mortality, there is considerably less evidence on the mortality impacts of diarrheal pathogens on mortality in the elderly. Two studies (to our knowledge) have exogenous variation in distance to an improved source. Most closely related is Kremer et al. (2011), which randomizes the protection of springs in Kenya and finds that diarrhea reduction is correlated with distance to an improved spring, but lacks statistical power to detect impacts on infant mortality and does not collect elderly mortality data. Devoto et al. (2012) randomizes a household's access to piped water supply into the home relative to clean but outside-the-home water in Morocco and finds no impacts on health. However, the external water source in Devoto et al. (2012) was close to the home (at an average distance of 140 meters) and thus akin to household shallow tubewells in Bangladesh, suggesting that the health gains of proximity are largely achieved once water infrastructure is within roughly 100-300 meters.

Our study setting permits us to hold the pathogen-cleanliness of well sources constant—as shallow and deep tubewells are equally protected from pathogens—but vary distance to source, and uncovers substantial mortality gains for both children and adults from proximity to a well alone. This has direct relevance to institutions seeking to invest in water infrastructure, as determining the density of such infrastructure requires a careful assessment of the trade-off

middle-income countries is similarly mixed: Galiani et al. (2005) find child mortality fell 8% in Argentinean municipalities that privatized their water and sewage infrastructure and hypothesized part of this is due to increased water access.

⁶Kremer et al. (2022) suggest that this omission reflects the limited statistical power to detect child mortality effects in most RCTs. Combining results from multiple RCTs, Kremer et al. (2022) estimate that improving water quality reduces child mortality by approximately 30%. However, only one of their fifteen studies randomizes water sources (Kremer et al., 2011) and alone lacks statistical power to identify mortality effects. A similar exercise has not been carried out for elderly mortality.

⁷Distance to a clean water source could impact health through several mechanisms. Re-contamination of stored water from distant wells is a documented risk and the reason home chlorination is effective in reducing diarrhea and mortality (Kremer et al., 2022). Distance from water source has been shown to be positively correlated with fecal contamination (Goel et al., 2019; Cociolo et al., 2021; Goel et al., 2023) and bacterial infections such as trachoma and diarrheal disease (Esrey et al., 1991). Greater travel time may also lead to a reduction in the quantity of water consumed (Hoque et al., 1989), resulting in health costs for children facing dehydration from diarrheal disease. Esrey et al. (1991) in fact finds that the quantity of water used is a better predictor of child health than the quality of water used.

between the material costs of more construction and the health benefits of greater proximity.

Finally, our findings raise questions about how to shape public health efforts around arsenic and the use of potentially contaminated shallow tubewells in contexts such as Bangladesh. We examine a campaign conducted at the turn of this century, but the question of what constitutes safe public health recommendations for arsenic exposure remains relevant today: between 2021 and 2022, the government of Bangladesh tested the water of 5.4 million tubewells in 54 districts under the Arsenic Risk Reduction Project (ARRP), again marking high-concentration tubewells with red paint (Rahman, 2022). Notably, while we find little evidence of large mortality impacts of arsenic poisoning, other recent work has found cognitive impairment from arsenic contamination (Pitt et al., 2021). Policy recommendations must therefore weigh the health effects of reducing the distance to pathogen-free water against the health consequences of arsenic exposure. Our estimates suggest that, in terms of short-term mortality, the impact of increased exposure to fecal contamination from abandoning nearby water sources outweighs the mortality risk from increased arsenic exposure. We also find no evidence of mortality differences by long-term arsenic exposure among the elderly in our setting. This combination of evidence increases the case for permitting shallow tubewells as a legitimate drinking water source—at least for vulnerable populations—unless safe alternative sources, such as deep tubewells, are proximate (Larsen, 2016).

2 Background

In this section, we describe the state of water infrastructure in Bangladesh and the potential health risks of arsenic-contaminated water that motivated the abandonment of shallow tubewells in our study area.

2.1 Water infrastructure in Bangladesh

Due largely to its geographic vulnerability to flooding and high population density, Bangladesh has a high incidence of water-borne viral and parasitic infections, with six percent of children under five experiencing diarrhea in the last two weeks as of 2019 (Das et al., 2019). While diarrhea related deaths have fallen sharply since the 1970s, diarrhea remains among the top ten causes of death and disability in Bangladesh, not only for the young but across the population as a whole (Vos et al., 2020). To reduce the incidence of diarrhea and other water-borne diseases, an estimated 8.6 million shallow tubewells were constructed throughout the country from the 1970s to the 1990s, an effort funded by the Bangladeshi government, UNICEF, the World Bank, and other public and private organizations. This campaign succeeded in moving at least 94% of rural Bangladeshis from parasite-infected surface water to protected groundwater (Caldwell et al., 2003).

Efforts were halted and reversed in the late 1990s with the discovery of arsenic naturally

leaching into Bangladesh's groundwater, the water source from which shallow tubewells drew.⁸ In 1997, the WHO publicly declared groundwater arsenic contamination to be a "major public health issue," and the following year, the World Bank approved a \$32.4 million grant to address the emergency (Caldwell et al., 2003).

In 1998, the British Geological Society conducted a nationwide study measuring levels of arsenic contamination in shallow tubewells (D G Kinniburgh, 2001). Results indicated that 15% of the population was drinking water with more than 50 μ g (D G Kinniburgh, 2001) and was determined to be in "grave danger." Following government screening of all shallow tubewells in endemic regions, 1.4 million wells were found to be contaminated (above 50 μ g) and painted red, and 3.5 million were painted green to indicate they were safe (Ahmed et al., 2006). Households were encouraged to avoid drinking from red tubewells and switch to alternative sources (Jakariya, 2007). Public education campaigns raised awareness of arsenic (BMOH, 2004), and by 2004, an estimated 80% of the population was aware that arsenic might be a danger (relative to less than ten percent in the late 1990s), and 70% reported changing their primary water source to avoid arsenic (UNICEF, 2008).

However, households—particularly in the immediate wake of the campaign—had limited access to safe and feasible alternatives. Arsenic-free alternatives include piped water, dug wells, surface water, harvested rainwater, and deep tubewells, with the latter being the most commonly promoted alternative (Howard et al., 2006; Hug et al., 2011; Johnston et al., 2014; Edmunds et al., 2015; Iqbal et al., 2020).⁹ By 2007, 200,000 functional deep tubewells had been built nationwide, largely funded by donors and the government in response to the arsenic crisis (Ahmed et al., 2010). Estimated at \$850 to construct relative to \$100 for a shallow tubewell, such infrastructure was prohibitively expensive for the typical household to build themselves (Ravenscroft et al., 2014).

As such, safe alternatives as convenient as shallow tubewells were often scarce for those households who were told to abandon their wells because shallow tubewells are often built inside a family compound, and they require minimal water storage time relative to less proximate water sources. A study in the Araihasar District of Bangladesh found that those who abandoned shallow tubewells increased the time spent obtaining water by fifteen-fold (Madajewicz et al., 2007). And while water filtering and cleaning methods can (partly) address point-of-use contamination, survey data indicate that these have largely been abandoned in rural Bangladesh since the construction of shallow tubewells (Lokuge et al., 2004), with less than one percent of our study sample reporting use of such methods in 2007.

2.2 Health risks of arsenic contaminated water

The 1999 public health campaign intended to reduce arsenic exposure and improve health. Those who switched away from their backyard wells were therefore not only exposed to less

⁸See Online Appendix figure A.1 for the spatial distribution of arsenic-contaminated wells in Bangladesh.

⁹It is possible to remove arsenic from water, but at the time of the study, the technology was expensive and very rarely used.

convenient alternative sources; they had also been exposed to relatively higher amounts of arsenic *prior* to the campaign.

Our setting, in which households were only encouraged to abandon their tubewells if they tested above the $50\mu\text{g}$ arsenic cutoff, permits a clean way to rule out that the mortality effects we estimate are due to delayed effects of arsenic exposure: among households who abandon their wells, we observe no relationship between the level of arsenic contamination in their shallow tubewell and mortality outcomes (see Section 4.4). Observationally, we also document that households above and below the contamination cutoff follow identical mortality trends *prior* to the campaign but diverge immediately thereafter, whereas delayed effects of arsenic exposure should transpire continuously over time. Our results thus suggest that the short-term mortality effects of switching away from convenient pathogen-free sources, at the least, far outweigh those of prior arsenic exposure. To benchmark this finding against existing evidence on the health impacts of arsenic exposure, we briefly review the relevant literature.

Arsenic is a known carcinogen that has been shown in laboratory studies to cause or catalyze cancer of the lung and bladder (Kozul et al., 2009). Field studies have found a dose-response relationship between arsenic exposure through drinking water and skin lesions (Rahman et al., 2006, 2019), the main arsenic-induced skin cancer (Chen et al., 2006). These studies have resulted in broad scientific consensus that exposure to high levels of arsenic ($>100\mu\text{g}$) increases cancer-related deaths and morbidity in older adult populations. Recent studies also suggest that arsenic exposure is associated with higher cardiovascular, cerebrovascular, and respiratory mortality risk in young adults (Abdul et al., 2015; Moon et al., 2017; Rahman et al., 2019).

Due to the decades-long latency of most arsenic-related health problems, however, there remains considerable debate around the magnitude of the health impacts of arsenic in Bangladesh. A widely-cited 2010 epidemiological study following over 10,000 adults across villages in the Araihsazar District in Bangladesh estimates that approximately 20% of all deaths documented over nine years were attributable to arsenic, with mortality rates nearly 70% higher for those exposed to concentrations of over $150\mu\text{g}$ relative to those exposed to less than $10\mu\text{g}$ (Argos et al., 2010). An important shortcoming not addressed in the study, however, is the correlation between arsenic and socioeconomic status at the macro-spatial level. Specifically, arsenic concentrations in groundwater are not orthogonal to socioeconomic status at the macro-spatial level due to differences across underground aquifers—which cover large geographic areas—in mean levels of arsenic. As demonstrated in Madajewicz et al. (2007), such arsenic clustering means that uncontaminated wells in the 54 study villages in Araihsazar were concentrated in villages with significantly lower average income and assets.¹⁰ These differences in arsenic disappear when accounting for mean levels of village income, suggesting that the Argos et al.

¹⁰Van Geen et al. (2003) describe these spatial patterns in detail: “Most of the wells with the lowest As concentrations are located in the northwestern portion of the study area (Figure 4),” which appears to contain higher SES villages. According to Madajewicz et al. (2007), there is a potential “correlation between soil types and arsenic levels and, therefore, possibly between arsenic levels and incomes. However, this correlation would not be likely to appear within villages. The surrounding fields are fairly uniform geologically, while the dispersion of incomes and wealth within villages is large.”

(2010) estimates of mortality impacts of arsenic exposure are biased upwards.

In contrast to [Argos et al. \(2010\)](#), [Lokuge et al. \(2004\)](#), taking into account only “strong causal evidence” from existing studies, estimate that arsenic-related disease led to the loss of 174,174 disability-adjusted life years (DALYs) per year in 2001 (i.e., roughly at the time of the arsenic testing campaign), amounting to 0.3% of the total disease burden in Bangladesh. Diarrheal disease, in comparison, accounted for 7.2% to 12.1% of the total disease burden in 2001 ([Lokuge et al., 2004](#)). [Pitt et al. \(2021\)](#) exploit genetic variation in the body’s ability to break down arsenic to generate a comparison group within arsenic-exposed areas in Bangladesh. While the study does not find a measurable impact of arsenic on morbidity (it does not test mortality), it documents significant impacts on cognition and income.

3 Estimation strategy

In this section, we describe the five datasets we assemble and our estimating strategy to quantify the effects of the abandonment of local water infrastructure in Bangladesh.

3.1 Data sources

We capitalize on extensive household survey data collected by the authors in 2007, 2011, and 2016 (the data collection timeline is shown in appendix figure [A.1](#)). These data cover 3,160 households randomly sampled from 162 villages in two subdistricts of Barisal, one of the most heavily contaminated districts in the country, with over 60% of tubewells in the area contaminated in 1999 ([Smith et al., 2000](#)). The aquifer geography of Barisal also makes it especially amenable to deep tubewell construction, allowing for substantial variation in access to alternative water infrastructure: By 2004, 65% of impacted households were accessing a deep tubewell in their village, as compared to only 4% in the rest of the country ([NIPORT and Macro, 2005](#)). This resulted in high switching rates in response to the public health campaign: By 2004, only 9% of impacted households used contaminated water sources, as compared to 30% in the rest of the country ([NIPORT and Macro, 2005](#)). Barisal’s geospatial conditions thus make the district particularly suited to test the impact of proximity to clean water infrastructure rather than access alone. While households in other parts of the country often faced a choice between arsenic-contaminated water or surface water (with the latter highly prone to pathogen contamination at source), households in Barisal were most likely to face a choice between arsenic-contaminated water or alternative arsenic and pathogen-safe but less proximate water sources.

In 2007, we collected reproductive and child health outcomes for all children born to the household head and the age of death of the household head and partner.¹¹ In 2009, we suc-

¹¹The full household survey collected data from 9,155 households in three districts, only one of which (Barisal) is contaminated with arsenic ([Caldwell et al., 2006](#)). Because the purpose of the survey was to evaluate an adolescent girls program operating in the region, households were surveyed only if they included at least one adolescent girl. Households in our sample have older mothers, more children, longer birth intervals, and have spent more

cessfully revisited 3,138 (99%) of households in our sample. At this time, each household's closest shallow tubewell was tested for arsenic using a standard field testing kit.¹² In 2011, we revisited 3,090 of the households in our sample and administered an extensive module on the timing and cause of death of the children in our original sample as well as the parents of the household head and his spouse and all other children and adult members of the household that passed away between 2001 and 2011.¹³ In 2016, we revisited all villages in our sample and collected data on the location and timing of construction of all deep tubewells in each village. In total, we collected construction data on 4,442 tubewells. In addition, we collected data on the timing of the parents' death of the household head and partner.¹⁴

As we did not collect the timing of the birth of new children born between 2007 and 2011, our analysis sample includes all children born in the residence between 1980 and 2007 as well as all adults aged 50 years or older at the time of data collection or death.¹⁵ Our final sample encompasses 12,195 children and 2,422 adults (244,584 life years).

3.2 Identification strategy

Our identification strategy makes use of small-scale variability in arsenic concentrations in groundwater (Yu et al., 2003) that generates substantial within-village variation in exposure via well contamination: an estimated 88% of contaminated wells are located within 100 meters of an uncontaminated well (Van Geen et al., 2002). Local pockets of contamination (unlike macro-spatial contamination) are extremely hard to predict and do not appear to be correlated with observable features of the land. This small-scale variability in arsenic concentration, paired with the sharp time discontinuity in knowledge about arsenic contamination, precludes selection into treatment or years of exposure, allowing us to compare households residing close to one another who are and are not encouraged to abandon water sources at different points in their lives in a difference-in-difference (DID) estimation strategy (Callaway et al., 2021).

time in their current residence than similar households in the 2007 DHS (rural households in Barisal with at least one adolescent girl in 2007; see appendix table B.1). The households in our sample are also more likely to be Muslim and are slightly wealthier. Using data from the 2007 DHS only, we find few differences between individuals in rural Barisal and the rest of rural Bangladesh (Online Appendix table B.1), including no differences in child mortality.

¹²A research agency based in Dhaka, Data International, collected up to two samples per household using the Wagtech Digital Arsenator Arsenic kit, which is reliable in testing both As(III) and As(V) concentrations between 5-100 μ g (Sankararamakrishnana et al., 2008). Save the Children re-tested 5% of all tests performed by the testing facility and collected new samples for all tubewells for which the retests differed by more than 5% from the original tests.

¹³We merge data collected in 2007 and 2011 by household identifier, name, birth year, and birth order. Merging errors are balanced by arsenic contamination status. As enumerators were instructed to probe respondents about the timing of death in 2011, we use the timing of death collected in 2011 if available.

¹⁴As recall bias is likely to increase over time, we use the timing of death collected in 2011 if available.

¹⁵We drop 198 households because of missing or inconclusive arsenic measurements as well as 3,258 children (21%) that were born after 1980 but turned one before the household moved into the current residence. Results are qualitatively similar when including them. Our estimates do not converge for a subset of specifications if we exclude all adult life years lived in another residence because of insufficient data. However, results excluding all adult life years lived in another residence are similar and presented in the appendix for our main specifications.

Reflecting the nature of our data, we define a binary level of arsenic contamination (*treatment*) and a continuous level of exposure (*post*, i.e., years lived after the campaign). The binary level of arsenic contamination categorizes households as contaminated (or “high concentration”) if the concentration of arsenic in the shallow tubewell closest to the household is greater than $60\mu\text{g}$ when measured by our field team in 2009.¹⁶ The continuous level of post-campaign exposure exploits between-child variation in the share of life potentially exposed to pathogenically unsafe water in the short window below age five in which children are most vulnerable to diarrhea. We thus categorize children by the share of this critical window (out of one, two, or five years) that is lived following the abandonment of contaminated backyard wells. In contrast, because all adults in our sample live most of their lives before the public health campaign, there is no precise window of vulnerability to calculate a share of post-campaign exposure. Furthermore, arsenic has a cumulative effect that acts with an undetermined lag. Therefore, we exploit variation between adults and within an adult’s lifetime by testing whether, among adults age 50 or older, the probability of death rises more for those encouraged to abandon wells following the campaign.

Our inclusion of village fixed effects absorbs differences in mean characteristics between relatively exposed and relatively unexposed villages arising from potential correlations between the macro-spatial distribution of arsenic and village characteristics. To test the validity of our assumption that *within*-village variation in arsenic exposure is orthogonal to household characteristics, we present mean differences between low and high-concentration households for a host of time-invariant characteristics (panel I of appendix table B.2). All rows contain regression-controlled means that account for village fixed effects, as do t-statistics of the differences in means. None of the nineteen variables, including indicators of socioeconomic status, are significantly different across the two subsamples. An F-test of joint significance indicates the samples are balanced on observables.¹⁷ In contrast, the same exercise conducted *without* accounting for mean differences across villages shows a high degree of imbalance (Online Appendix table B.2), as was observed in the Arai hazar study area. This highlights the importance of our identification strategy of relying on within-village, rather than between-village, spatial variation in arsenic concentrations.

¹⁶We chose $60\mu\text{g}$ as the cutoff to reflect the $50\mu\text{g}$ WHO cutoff, taking into account an estimated 10% per decade increase in arsenic levels so that contaminated wells in our sample are those believed to have tested above $50\mu\text{g}$ in 1999. Relatively constant groundwater arsenic concentrations have been reported in several time series studies (Van Geen et al., 2002). Since our survey data on the history of shallow tubewell use indicate a tendency to underreport the use of highly contaminated wells, we deem “measured contamination” more conservative than reported contamination. Our results are similar, though noisier when using reported contamination (appendix table B.2 shows that reported concentration is highly correlated with measured concentration). In addition, there is evidence that the government testing underestimated well As concentrations using the Hach kit (van Geen et al., 2004).

¹⁷Similarly, none of the 19 variables significantly correlate with the continuous arsenic measure after controlling for village fixed effects.

3.3 Estimating equations

For infant and child mortality, we estimate the following difference-in-difference equation for child i born in village j in year t :

$$Y_{ijt} = \theta_j + t + \gamma HighCon_i + \delta Exposure_t + \beta(HighCon_i \times Exposure_t) + \epsilon_{ijt} \quad (1)$$

HighCon is an indicator variable equal to one if the child was born into a household exposed to arsenic above $60\mu\text{g}$ in 2009 and thus encouraged to switch water sources. *Exposure* denotes the fraction of a child’s life that he or she was potentially exposed to microbiologically unsafe water due to the household switching *away* from a shallow tubewell due to the testing campaign. Hence, for under-1 mortality, *Exposure* is an indicator variable equal to 1 if the child was born in or after 2000 and 0 if born before 2000. For under-2 mortality, *Exposure* takes a value of 1 if the child was born after 2000, 0.5 if born in 2000, and 0 before 2000.¹⁸ The estimates adjust for both village fixed effects θ_j and a continuous birth year time trend t . Robust standard errors are clustered at the village level. Although the nationwide campaign began in mid-1999, 2000 is our preferred cutoff since behavioral change is presumed to respond with a lag. However, our regression estimates are robust to using 1999 as a cutoff point (as well as 2002, as in Keskin et al. (2017)).

Our coefficient of interest is β , the coefficient on the interaction between high concentration and exposure. As our exposure levels are continuous, our identifying assumption is that no other events during the period differentially affect infant and child mortality rates for children exposed for 0 to 5 years in households that were (or were not) encouraged to stop using shallow tubewells (Callaway et al., 2021).¹⁹ The high variation in arsenic exposure across very small distances and the similarity across contaminated and uncontaminated households in baseline characteristics lend credibility to this assumption. However, to account for any differences in baseline characteristics that may contribute to time trends in mortality, we also estimate versions of equation 1 with birth year fixed effects, as well as a series of exogenous controls for individual’s sex and birth order, the mother’s age at birth, and mother’s and father’s education.²⁰

For elderly mortality, we estimate the following time-varying Cox-hazard model for adult i in village j and year t for all adult years age 50 or higher:

$$h_{ijt} = h_0(t)e^{\theta_j + y(i) + \gamma HighCon_i + \delta Post2000_t + \beta(HighCon_i \times Post2000_t) + \epsilon_{ijt}} \quad (2)$$

¹⁸The maximum number of years of exposure is the mortality age (of one, two, or five years) being measured. We drop all children for whom outcomes are censored, i.e., who would not yet have reached the age of one, two, or five by the time of surveying. However, results are almost identical when assuming that all alive children for which outcomes are censored would not have died under the age cutoff.

¹⁹We also show results using a binary exposure measure as well as using the imputation approach by Borusyak et al. (2022).

²⁰We also present results with an extended list of controls, including the mother’s age at first birth, years since the birth of the mother’s last child, the household income, the household land size, the number of rooms in the household, whether the house has electricity, the years the family has lived in the house, and the distance of the house to the village center in the appendix. Note, however, that these controls might vary with the campaign, making the more parsimonious specification our preferred specification.

where $h_0(t)$ is the baseline hazard function, $HighCon$ is an indicator variable equal to one if the household was exposed to arsenic above $60\mu\text{g}$ in 2009, and $Post - 2000$ is an indicator that takes a value of 1 for all life years after the campaign, i.e., after households were encouraged to switch water sources. The estimates again adjust for village fixed effects, θ_j , and a continuous birth year time trend, y . Robust standard errors are clustered at the village level.

Our coefficient of interest is β , the hazard of dying after 2000 in a high-concentration household relative to a low-concentration household. We also estimate a more data-intensive version of equation 2 with village-specific baseline hazards, birth-year fixed effects, and a series of exogenous controls for the individual’s sex and the education of the household head and his spouse.

4 Results

We first illustrate raw time trends of child and adult mortality across household types, and then present our regression results for mortality, both overall and disaggregated into water- and arsenic-related deaths. We then test for heterogeneity by distance to the closest alternative source of pathogen-free water.

4.1 Time trends

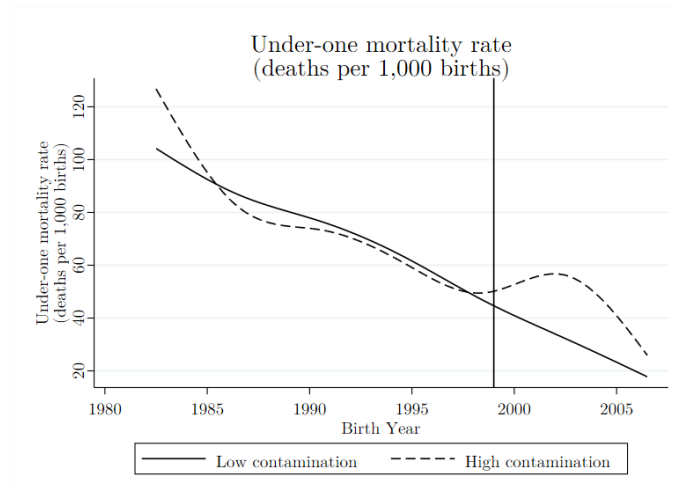
Trends in raw, unadjusted averages of under-one mortality in households with arsenic largely track those of households with non-arsenic wells until 1999, when they diverge sharply as infant mortality rises among individuals in households encouraged to abandon contaminated wells (figure 1). Mortality rates appear to move towards convergence beginning in 2003, likely in response to the remedial measures we discuss in section 4.3—namely, the ongoing construction of deep tubewells in Barisal. We find similar results for two-year and five-year mortality (appendix figure A.2).²¹

A parallel pattern is observed for mortality in older adults when plotting the mortality rates of all adults ages 50-59 in 2000 (born between 1941 and 1950) from 1980 onward (i.e., the mortality every year among all individuals still alive at the beginning of that year).²² While mortality rises over time for both high- and low-contamination adults as the cohort ages (following a classic exponential curve), the levels closely track one another from 1980 to 2000 despite years of differential accumulated arsenic consumption but diverge sharply immediately after the campaign (figure 2). As with childhood mortality, there is evidence that mortality begins to converge in later years with increased deep tubewell construction across the region.

²¹We observe a slight increase in child mortality even among low-contamination households for five-year mortality after 1999. This could be due to some mismeasurement of contamination status or low-contamination households switching water sources because of fear of arsenic. Note that low-contamination households switching water sources implies that our DiD strategy likely *underestimates* the true effect of abandoning shallow tubewells.

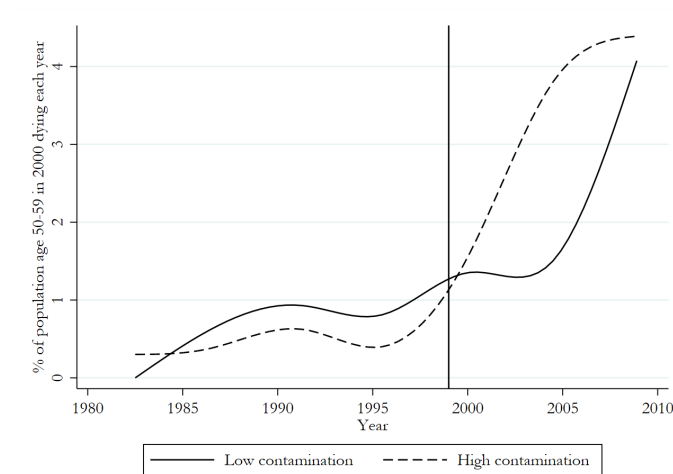
²²We do not observe any deaths after 2011 and exclude 2011 from the graph as the mortality outcomes are censored for households surveyed in 2011.

Figure 1: Under-1 mortality rate (0 – 1 yrs), four-year unadjusted averages



Notes: Data from our 2007 data collection and 2009 tubewell tests. “Under-one mortality rate” is deaths between 0 and 12 months of age per 1,000 births observed every four years, plotted as cubic splines for smoothness. High contamination households are defined as those with tubewells that contain arsenic contamination greater than $60\mu\text{g}$ according to field tests of the shallow tubewells closest to the residence.

Figure 2: Death rate among adults age 50-59 in 2000, unadjusted averages



Notes: Data from our 2007, 2011, and 2016/2017 data collections and 2009 tubewell tests. “Death rate” is the percentage of adults predicted to be 50-59 years old in 2000 (the vulnerable age range) and alive at the beginning of a year that dies in that year, plotted as cubic splines for smoothness. High contamination households are defined as those with tubewells that contain arsenic contamination greater than $60\mu\text{g}$ according to field tests of the shallow tubewells closest to the residence.

4.2 Impact of abandoning shallow tubewells on mortality: regression estimates

Consistent with figure 1, the coefficient estimates from equation 1 for infant, under-two, and under-five mortality indicate a substantial and statistically significant increase in mortality

after 2000 among individuals encouraged to use less proximate water infrastructure (table 1). Being born into a household that has been encouraged to abandon their shallow tubewell is associated with a 2.5 percentage point (36%) increase in the likelihood of death within one year (column 3), a 2.7 percentage point (31%) increase within two years (column 6), and a 3.9 percentage point (28%) increase within five years (column 9).²³

Table 1: Child mortality

| | Death < 12 months | | | Death < 24 months | | | Death < 60 months | | |
|------------------------------|--------------------|--------------------|--------------------|--------------------|--------------------|--------------------|--------------------|--------------------|--------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High con. | -0.001 (0.007) | -0.001 (0.007) | -0.002 (0.007) | 0.006 (0.008) | 0.006 (0.008) | 0.004 (0.008) | -0.002 (0.009) | -0.002 (0.009) | -0.006 (0.009) |
| Exposure | -0.001 (0.009) | | | 0.009 (0.011) | | | 0.036** (0.016) | | |
| High con. * Exposure | 0.024** (0.011) | 0.024** (0.011) | 0.025** (0.011) | 0.024** (0.011) | 0.024** (0.011) | 0.027** (0.011) | 0.033** (0.015) | 0.034** (0.015) | 0.039** (0.015) |
| Mean(Low con. & No exposure) | 0.069 | 0.069 | 0.069 | 0.088 | 0.088 | 0.088 | 0.138 | 0.138 | 0.138 |
| Observations | 11979 | 11979 | 11979 | 11755 | 11755 | 11755 | 10810 | 10810 | 10810 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ | | | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. We exclude all children that had not yet reached age 1 in columns (1)-(3), age 2 in columns (4)-(6), and age 5 in columns (7)-(9). High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than 60µg, according to field tests conducted in 2009. Exposure denotes the fraction of life that the child was potentially exposed to unsafe water (time lived after 2000). Controls include the child's sex and birth order, the mother's age at birth, and the mother's and father's education. All regressions were adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

We re-estimate equation 1 for six disease-specific causes of death that can be classified as either 1) bacterial ingestion (diarrhea and vomiting), 2) arsenic ingestion (spasms and pneumonia), and 3) not related to either water or arsenic (tetanus and accidents). The results on the cause of death presented in table 2 suggest that the mortality effects we document are driven by bacterial pathogens in water.

Table 2: Child mortality: child died < 60 months from ...

| | Water-related | | Arsenic-related | | Not water-/arsenic-related | |
|------------------------------|-------------------|--------------------|-------------------|-------------------|----------------------------|-------------------|
| | Diarrhea (1) | Vomiting (2) | Spasms (3) | Pneumonia (4) | Tetanus (5) | Accident (6) |
| High con. | -0.003 (0.005) | -0.006 (0.004) | -0.001 (0.004) | 0.001 (0.005) | 0.001 (0.003) | -0.000 (0.003) |
| High con. * Exposure | 0.011* (0.007) | 0.017** (0.007) | 0.004 (0.006) | -0.005 (0.009) | 0.001 (0.004) | 0.005 (0.005) |
| Mean(Low con. & No exposure) | 0.033 | 0.028 | 0.018 | 0.025 | 0.010 | 0.012 |
| Observations | 10533 | 10532 | 10534 | 10534 | 10534 | 10532 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Controls | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |

Notes: See notes to table 1.

²³The coefficient estimates are similar but noisier when including household fixed effects.

Consistent with figure 2, the coefficient estimates from equation 3 for adult mortality indicate that the hazard ratio of dying in households encouraged to switch water sources relative to those not encouraged to switch is 1.47 (table 3, column (3)): in other words, adults over 50 years of age in switching households are 47% more likely to die over the period 2000 to 2011 than their non-switching counterparts.²⁴

As with child mortality, we observe an increase in water-related deaths post-2000 (column (4)), with a hazard ratio of dying of 2.6 (+160%) over the period 2000 to 2011 among adults in households encouraged to switch water sources relative to households not encouraged to switch water sources. We document a small but statistically insignificant reduction in arsenic-related deaths after households abandon contaminated wells (column (5)).²⁵

Table 3: Elderly mortality: hazard of dying among adults age 50+ from...

| | Any | | | Water | Arsenic |
|---------------------------------|---------------------|---------------------|---------------------|---------------------|---------------------|
| | (1) | (2) | (3) | (4) | (5) |
| High con. | 0.853 (0.143) | 0.841 (0.154) | 0.838 (0.156) | 0.498 (0.301) | 1.251 (0.758) |
| Post-2000 | 3.681*** (0.612) | 2.947*** (0.484) | 2.928*** (0.476) | 7.193*** (3.407) | 7.480*** (3.842) |
| High con. * Post-2000 | 1.425** (0.239) | 1.444** (0.259) | 1.468** (0.270) | 2.600* (1.387) | 0.739 (0.415) |
| Mean(Low con. & After campaign) | 0.048 | 0.048 | 0.048 | 0.009 | 0.011 |
| Observations | 45,555 | 45,555 | 45,555 | 45,555 | 45,555 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | ✓ | ✓ |
| Controls | | | ✓ | ✓ | ✓ |

Notes: The table shows results from Cox proportional hazards models with Huber-White robust SEs clustered at the village level. Each observation is one person-year (each year of life from birth to death, or the year of data collection if still alive, in which case the data is censored for that person). The failure event is death. The sample includes all person-years age 50 or higher. High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than $60\mu\text{g}$, according to field tests conducted in 2009. Post-2000 is an indicator that is 1 in the years 2000 to 2017. Controls include the gender of the adult, as well as the education of the household head and his spouse. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

In sum, we find that switching to more inconvenient water sources reduced not only child but also adult life expectancy, a result consistent with evidence that pathogenically contaminated water puts sub-populations with weakened immune systems, such as children and the elderly, at higher risk of water-borne infectious diseases (Yoshikawa, 2000). The sudden increase in mortality when arsenic exposure declines seems inconsistent with arsenic driving our

²⁴Displayed hazard ratios are significantly different if the confidence interval does not include 1, meaning that the hazard of dying significantly differs by groups.

²⁵We have very few adult cases of tetanus-related deaths or accidents.

results. However, we cannot completely rule out the possibility that accumulated arsenic exposure made people more susceptible to death from heightened bacterial exposure. In other words, the *combination* of previous exposure to arsenic-contaminated water and new exposure to pathogenically contaminated water may have led to higher mortality among this subpopulation after switching water sources. However, three key patterns in our data are inconsistent with this hypothesis. First, infants born after 2000 were (by and large) unexposed to arsenic but still see higher mortality if born into households that had to switch water sources. Second, mortality rates among the elderly are independent of arsenic exposure before 2000, even though exposure to fecal pathogens was unlikely to be zero during this period. Third, mortality rates among the elderly after 2000 vary with deep tubewell distance (a pattern documented below), even though previous arsenic ingestion levels should be independent of one's distance to their nearest deep tubewell (as drinking water was almost exclusively drawn from shallow tubewells before 2000).

4.3 Heterogeneity by distance to alternative water sources

For those seeking to invest in water infrastructure, determining the optimal density of clean water sources to construct depends on the trade-off between the material costs of construction and the health benefits of proximity. Our results thus far suggest a significant mortality differential between having very local water infrastructure (i.e., within the home or compound) relative to more distant sources (i.e., within the village). In this section, we seek more precision on the relationship between distance to a pathogen-free water source and infant, child, and elderly mortality.

One concern in calculating such a gradient is that one's distance to a clean alternative source is not exogenous to household characteristics, as the location of deep tubewells is often at the discretion of local politically connected elites (Mobarak and Van Geen, 2019). A simple OLS estimate of mortality on the distance to a deep tubewell would therefore be biased upwards. Using a triple difference strategy, however, we can compare *changes* in mortality for those in high- relative to low-contamination households who are *equidistant* to alternative water sources. Our identifying assumption is that households with the same number of deep tubewells within a certain radius only experience a differential change in mortality due to having to shift away from their backyard tubewell due to differential arsenic concentration.

To perform this exercise, we use data we collected in 2016 on the GPS location and age of all deep tubewells in our study area. Summary statistics suggest the construction of deep tubewells was much faster in villages with high arsenic levels: a behavioral response to the reduction in usable water infrastructure which could explain the convergence of mortality rates towards the end of our data window (appendix tables B.3 and B.4). We use this data to calculate a variable ($\#Tubewells$) equal to the number of deep tubewells within a given radius of each household in each year (birth year for child mortality and life year for elderly mortality). We then estimate triple-difference versions of equations 1 and 2 by including $\#Tubewells$ and

its interaction with *Exposure*, *HighCon*, and their interaction. The coefficient on the triple interaction can be interpreted as the impact on child or adult mortality of having an additional deep tubewell within a given radius for those encouraged to abandon their shallow tubewells.

Having access to an additional deep tubewell within 300 meters of the residence reduces water-related child mortality by 2.5 percentage points for those required to abandon their tubewells (table 4, column (1)). This almost entirely offsets the 2.8 percentage point mortality impact of being encouraged to abandon the closest shallow tubewell.²⁶ As expected, the water-related mortality benefits of deep tubewells decrease linearly with distance (columns(2)-(8)). Accordingly, the combined effect of $(HighCon \times Exposure) + (HighCon \times Exposure \times \#Tubewells)$ is significantly different from zero for a tubewell distance of 1,000, but not 300 meters (though we note that the coefficients on 300 vs. 1,000 meters are not significantly different). Reassuringly, we find no significant coefficient on the triple interaction in either of our two placebo estimations (columns (11)-(12)): neither the likelihood of abandoning a shallow tubewell nor access to an additional deep tubewell alters arsenic or non-water-related death rates for children.

One may be concerned that household responses to the differential change in mortality by arsenic status after 2000 are endogenous (Lee et al., 1997); namely, wealthier and more health-conscious high-concentration households may be more likely to invest in deep tubewells within their family compound than poorer households. To address this, we consider a specification with household fixed effects (column (9)), comparing water-related child mortality for children within the same household born before and after 2000 for switchers vs non-switchers. Estimates remain unchanged, with strong and significant reductions in mortality *within* households from one additional deep tubewell and the value of an additional deep tubewell declining in distance (appendix table B.5).²⁷ Meanwhile, we find no within-household effects for non-water (appendix table B.6) or arsenic-related (appendix table B.7) deaths, suggesting that the within-household effects of access to deep tubewells are not driven by concurrent increases in time-varying household wealth. These patterns lend credibility to our interpretation that the increase in child mortality after 2000 is indeed driven by a reduction in proximity to sources of pathogen-free water among households that were encouraged to abandon shallow tubewells.

Consistent with our findings on child mortality, we also observe that the difference in the hazard of dying for adults above 50 years decreases monotonically with proximity to deep tubewells (table 5): hazard ratios on the triple difference coefficient grow closer to one with distance, implying that, as the distance grows, an additional deep tubewell generates smaller and smaller reductions in the risk of dying among those encouraged to switch.²⁸

²⁶The effect of being encouraged to abandon a shallow tubewell when there is one deep tubewell within 300 meters is the sum of the coefficients on $HighCon \times Exposure$ and $HighCon \times Exposure \times \#Tubewells$.

²⁷However, it should be noted again that the coefficients on 300 meters vs. 1,000 meters are not significantly different.

²⁸We have insufficient data on causes of death to do this analysis for water-related deaths only.

Table 4: Child mortality: Cause-specific mortality by exposure to deep tubewells

| Tubewells within (meters): | Water-related | | | | | | | | | Arsenic-related | Non-water-arsenic |
|------------------------------------|---------------------|----------------------|----------------------|----------------------|---------------------|---------------------|---------------------|---------------------|---------------------|-------------------|-------------------|
| | 300 | 400 | 500 | 600 | 700 | 800 | 900 | 1,000 | 500 | 500 | 500 |
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) | (11) |
| High con. | -0.007 (0.006) | -0.009 (0.006) | -0.009 (0.006) | -0.010 (0.006) | -0.009 (0.006) | -0.009 (0.006) | -0.010 (0.006) | -0.010 (0.007) | | -0.001 (0.006) | -0.000 (0.005) |
| High con. * Exposure | 0.028*** (0.010) | 0.029*** (0.011) | 0.028** (0.011) | 0.030*** (0.011) | 0.028** (0.012) | 0.027** (0.012) | 0.025** (0.012) | 0.025** (0.012) | 0.030** (0.013) | -0.004 (0.013) | 0.003 (0.008) |
| # Tubewells | -0.008 (0.005) | -0.005 (0.004) | -0.003 (0.003) | -0.003 (0.003) | -0.002 (0.002) | -0.002 (0.002) | -0.002 (0.002) | -0.002 (0.002) | -0.002 (0.005) | 0.003 (0.004) | 0.000 (0.002) |
| High con. * # Tubewells | 0.011* (0.006) | 0.012** (0.005) | 0.008** (0.003) | 0.007** (0.003) | 0.005** (0.002) | 0.004* (0.002) | 0.003* (0.002) | 0.003* (0.001) | 0.003 (0.005) | 0.001 (0.004) | 0.001 (0.003) |
| Exposure * # Tubewells | 0.021** (0.010) | 0.014** (0.006) | 0.008** (0.004) | 0.008*** (0.003) | 0.005** (0.002) | 0.004* (0.002) | 0.003* (0.002) | 0.003* (0.002) | 0.008* (0.005) | 0.002 (0.005) | 0.001 (0.003) |
| High con. * Exposure * # Tubewells | -0.025** (0.011) | -0.020*** (0.007) | -0.013*** (0.005) | -0.011*** (0.004) | -0.008** (0.003) | -0.006** (0.003) | -0.005** (0.002) | -0.004** (0.002) | -0.012** (0.005) | -0.001 (0.005) | 0.000 (0.004) |
| Mean(Low con. & No exposure) | 0.039 | 0.039 | 0.039 | 0.039 | 0.039 | 0.039 | 0.039 | 0.039 | 0.039 | 0.040 | 0.021 |
| Observations | 10533 | 10533 | 10533 | 10533 | 10533 | 10533 | 10533 | 10533 | 10298 | 10534 | 10534 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Controls | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Household FE | | | | | | | | | ✓ | | |

Notes: See notes to table 1. Child mortality is an indicator that is 1 if the child died < 60 months. # Tubewells is the number of deep tubewells within X meters of the residence in the year of the child's birth. Controls include the child's sex and birth order, the mother's age at birth, and the mother's and father's education. We also control for income and distance to the village center to ensure that distance to tubewells does not only proxy income or location. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table 5: Elderly mortality: hazard of dying among adults age 50+, by # of tubewells within X meters

| Tubewells within (meters): | Any | | | | | | | |
|-------------------------------------|---------------------|---------------------|---------------------|---------------------|---------------------|---------------------|---------------------|---------------------|
| | 300 | 400 | 500 | 600 | 700 | 800 | 900 | 1,000 |
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| High con. | 0.788 (0.167) | 0.854 (0.182) | 0.843 (0.182) | 0.850 (0.185) | 0.869 (0.190) | 0.902 (0.204) | 0.883 (0.200) | 0.877 (0.201) |
| Post-2000 | 2.415*** (0.420) | 2.375*** (0.435) | 2.351*** (0.425) | 2.230*** (0.406) | 2.248*** (0.406) | 2.216*** (0.423) | 2.267*** (0.440) | 2.408*** (0.489) |
| High con. * Post-2000 | 1.754*** (0.373) | 1.722** (0.378) | 1.830*** (0.402) | 1.885*** (0.422) | 1.783*** (0.393) | 1.634** (0.371) | 1.654** (0.374) | 1.596** (0.375) |
| # Tubewells | 0.671* (0.146) | 0.869 (0.123) | 0.935 (0.087) | 0.964 (0.064) | 0.976 (0.053) | 0.980 (0.047) | 0.980 (0.041) | 0.986 (0.040) |
| High con. * # Tubewells | 1.713** (0.414) | 1.174 (0.195) | 1.131 (0.125) | 1.090 (0.087) | 1.055 (0.073) | 1.025 (0.063) | 1.027 (0.052) | 1.029 (0.047) |
| Post-2000 * # Tubewells | 1.641** (0.354) | 1.238 (0.172) | 1.153 (0.107) | 1.123* (0.075) | 1.090 (0.060) | 1.070 (0.051) | 1.057 (0.043) | 1.040 (0.040) |
| High con. * Post-2000 * # Tubewells | 0.549** (0.132) | 0.798 (0.132) | 0.830* (0.092) | 0.861* (0.068) | 0.904 (0.062) | 0.946 (0.057) | 0.950 (0.047) | 0.962 (0.044) |
| Mean(Low con. & After campaign) | 0.048 | 0.048 | 0.048 | 0.048 | 0.048 | 0.048 | 0.048 | 0.048 |
| Observations | 42,123 | 42,123 | 42,123 | 42,123 | 42,123 | 42,123 | 42,123 | 42,123 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Controls | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |

Notes: The table shows results from Cox proportional hazard models with Huber-White robust SEs clustered at the village level. Each observation is one person-year (each year of life from birth to death, or the year of data collection if still alive, in which case the data is censored for that person). The failure event is death. The sample includes all person-years age 50 or higher. High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic greater than $60\mu\text{g}$ according to field tests conducted in 2009. Post-2000 is an indicator that is 1 in years 2000 to 2017. # Tubewells is the mean number of deep tubewells within X meters of the residence each year. Controls include the gender of the adult as well as the education of the household head and his spouse. We also control for income and distance to the village center in the tubewell regressions to ensure that distance to tubewells does not only proxy income or location. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

4.4 Robustness checks

Our estimates are robust to several alternative specifications, as detailed in the appendix. For infant and child mortality, using the extended set of controls (appendix table B.8) and replacing early life exposure with the number of years exposed (appendix table B.9) or a binary measure equal to 1 if the child was born in or after 2000 (appendix table B.10) as well as using the imputation approach for difference-in-difference designs by [Borusyak et al. \(2022\)](#) (appendix table B.11) produce very similar results. Using reported as opposed to measured contamination (appendix table B.12) or a $50\mu\text{g}$ cutoff (appendix table B.13) yields slightly noisier but very similar results, and using 1999 (the first year of the campaign) or 2002 (as in [Keskin et al. \(2017\)](#)) instead of 2000 as the switching date (appendix tables B.14 and B.15) produce very similar results. We also estimate the hazard of death among children in each life year for those encouraged and not encouraged to switch their water source using a Cox proportional-hazards model (our preferred model for estimating elderly mortality, appendix table B.16). Consistent with the difference-in-difference estimations, we find no difference between contaminated and uncontaminated households in the hazard of a child dying before 2000. However, *after* 2000, the hazard ratio of dying is 1.38 (column (3)) in high-contamination households, which implies that children in high-contamination households experienced a risk

of dying that is 1.38 times that of children in low-contamination households (or 38% greater) over the period 2000 to 2007.

To further test the assumption of parallel time trends between households who were and were not encouraged to switch, we exclude households with arsenic contamination below $60\mu\text{g}$ and estimate a placebo check in which we test whether an imaginary cutoff point of $100\mu\text{g}$ produces similar patterns within the subsample of households that we know were *all* encouraged to abandon shallow tubewells (appendix table B.17). If the level of arsenic contamination in groundwater is correlated with unobservable household characteristics that are giving rise to differential time trends in mortality, we should expect to see positive and significant point estimates on the interaction terms in both regressions. On the other hand, if we observe a significant difference-in-difference estimate only when the true, or programmatic, cutoff is used, we can deduce that the estimates reflect the causal effect of being encouraged to change water sources rather than time trends in unobservables correlated with underlying arsenic concentration. In line with our hypotheses, our placebo test shows no significant effect on mortality of arsenic levels above $100\mu\text{g}$ relative to those between 60 and $100\mu\text{g}$.

Finally, we explore several alternative explanations for the increased mortality among households encouraged to switch. First, to verify that the increase in mortality is not due to delayed effects of arsenic exposure, we test whether mortality is increasing in the *level* of arsenic contamination. We do not find any measurable effect of a continuous arsenic variable on mortality among households encouraged to switch water sources, providing strong evidence that the increase in mortality is driven by being above the cutoff value for switching rather than prior exposure to arsenic (appendix table B.18). Second, we consider whether alerting families to switch water sources may have led to a larger migration of health-conscious households out of properties contaminated with arsenic. However, we do not find any differences in migration, as measured by the number of years lived in the house, by contamination status (appendix table B.2).

For elderly mortality, we find smaller but significant effects when expanding the sample to adults age 40+ (appendix table B.19) and smaller and insignificant effects when expanding the sample to adults age 30+ (appendix table B.19), suggesting that the mortality increase from switching away from arsenic-contaminated wells to less convenient water sources was largest among the oldest and therefore most vulnerable subpopulations.

We also find slightly larger effects on elderly mortality in the data-intensive estimation with village-specific baseline hazards or when excluding all person-years before the individual moved into the current residence (appendix table B.20). The results are almost identical when restricting the sample to life years between 1980 and 2007, our analysis sample in the child mortality estimations (appendix table B.20). Finally, we estimate a linear probability model for adults similar to our child mortality analysis by interacting the high-concentration indicator with the fraction of one's vulnerable adult life (age 50-80) that one was potentially exposed to unsafe water (time lived after 2000). We find qualitatively similar results (appendix table B.21).

A placebo test shows that the hazard of dying did not diverge between high- and low-contamination households at an earlier point in time (appendix table B.22), indicating that differences in mortality risk post-2000 are not due to differential time trends in elderly mortality but indeed due to the change in water sources. We also do not find any measurable effect of the continuous arsenic variable on adult mortality among households encouraged to switch water sources, reinforcing that the increase in mortality is not due to a delayed arsenic effect (appendix table B.22).

5 Conclusion

We exploit the sudden abandonment of many shallow tubewells in Bangladesh to identify the protective effects of convenient pathogen-free water relative to more distant alternatives. Infant, child, and elderly mortality rose significantly among households encouraged to abandon nearby shallow tubewells and were thus forced to access less proximate alternative sources. We document these mortality effects, although the vast majority of households in our sample (81%) had an alternative pathogen-free source in the form of a deep tubewell within 1,000 meters of their home. Underscoring the importance of proximity, we find that having a deep tubewell within 300 meters (a 3-4 minute walk) almost entirely offsets the negative mortality effect of abandoning one's shallow tubewell, while those with wells further away experience increasing mortality costs to shifting away from their backyard well.

Our results strengthen the limited evidence that clean water infrastructure may generate significant mortality reductions among infants and children in vulnerable contexts. They provide new evidence of concomitant protective effects on the lifespan of the elderly. Beyond access alone, our results demonstrate that proximity to a clean water source is critical to achieving health gains, likely because of the risk of re-contamination of stored water collected from water sources located further away. We find relatively little evidence of large negative effects of arsenic consumption on mortality among infants or the elderly. However, this does not mean arsenic is not dangerous: this study was not designed to pick up arsenic-related health impacts, and recent studies have suggested that arsenic exposure impairs cognition and reduces later-life income.

The results of this study also imply that future public health interventions in arsenic-prone areas such as Bangladesh should reconsider efforts to convince households to abandon shallow tubewells when alternatives equally protected from water-borne pathogens are not readily available. More generally, our findings highlight the importance of prospectively accounting for competing health risks of likely alternatives when issuing recommendations to avoid unintended health consequences of behavior change. Since all members of a household tend to drink from the same water source, such policy recommendations should be especially sensitive to intra-household differences in the decision-making power of household members—namely children and the elderly relative to an adult head of household—who may face and internalize different potential risk trade-offs from alternative sources.

A Appendix figures

Figure A.1: Timeline of Data Collections

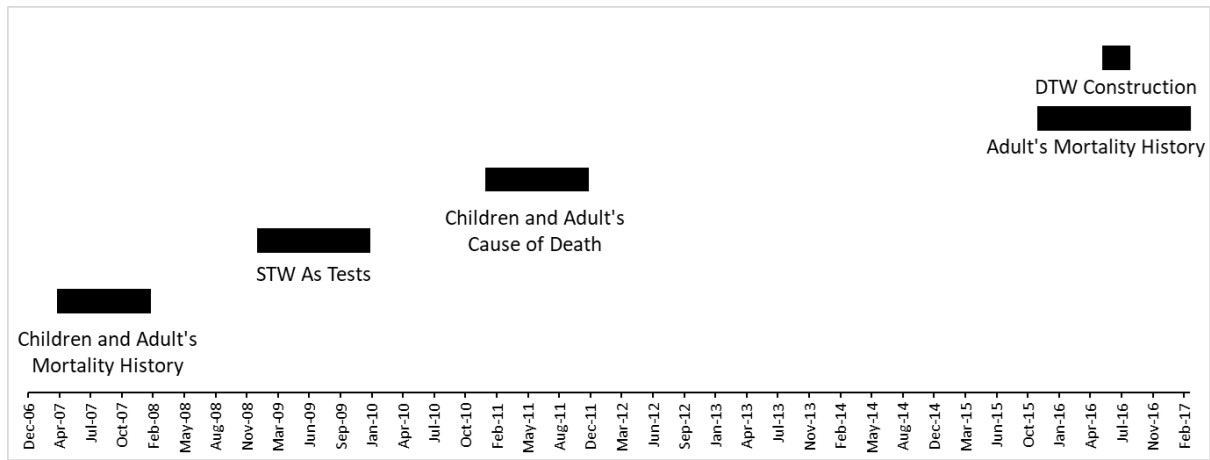
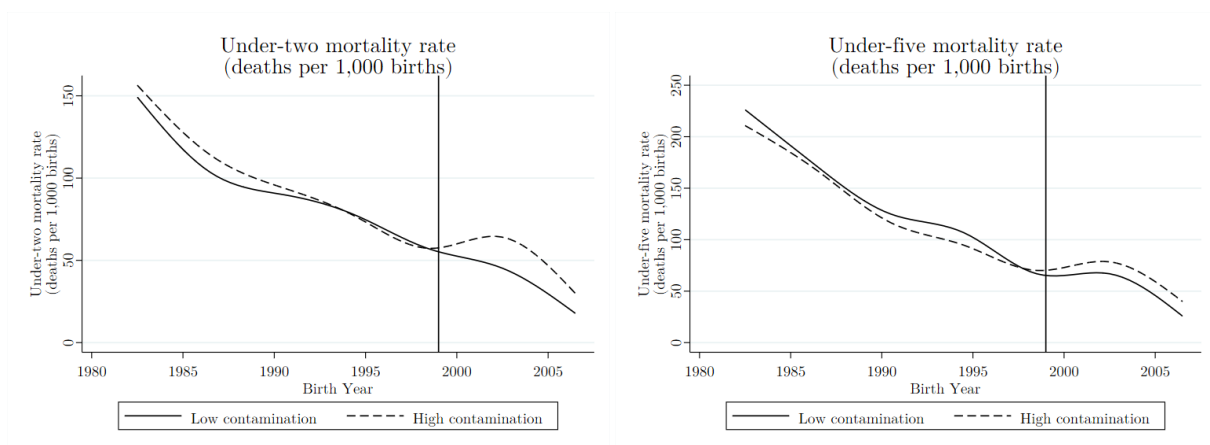


Figure A.2: Under-2 (0 – 2 yrs) and Under-5 (0 – 5 yrs) mortality rate, four-year unadjusted averages



Notes: Data from our 2007 data collection and 2009 tubewell tests. “Under-two mortality rate” (left panel) is deaths between 0 and 24 months of age, and “Under-five mortality rate” (right panel) is deaths between 0 and 60 months of age, per 1,000 births observed every four years, plotted as cubic splines for smoothness. High contamination households are defined as those with tubewells that contain arsenic contamination greater than $60\mu\text{g}$ according to field tests of the shallow tubewells closest to the residence. The figure shows the mean mortality rates across all high- and low-contamination households.

B Appendix tables

Table B.1: Sample means in the 2007 Kishoree Kontha survey data and the sample of rural households in Barisal in the 2007 DHS

Panel I: Exogenous Variables

| VARIABLE | Survey Data | | DHS Data | | Diff. | P-value |
|--|-------------|-------|----------|-----|-------|--------------|
| | Mean | N | Mean | N | | |
| Age of mother | 41.43 | 2,680 | 35.76 | 327 | 5.66 | 0.000 |
| Age of mother at earliest birth | 18.27 | 1,678 | 16.85 | 327 | 1.42 | 0.000 |
| Education of mother | 3.46 | 2,677 | 3.48 | 326 | -0.03 | 0.930 |
| Education of father | 4.59 | 2,622 | 4.67 | 326 | -0.08 | 0.824 |
| Land size (acres) | 0.81 | 2,690 | 0.82 | 327 | -0.01 | 0.977 |
| Number of rooms in house | 2.75 | 2,705 | 2.72 | 327 | 0.04 | 0.618 |
| Electricity | 0.39 | 2,710 | 0.28 | 310 | 0.11 | 0.040 |
| Muslim | 0.97 | 2,712 | 0.87 | 327 | 0.09 | 0.003 |
| Years lived in house | 25.66 | 2,713 | 23.00 | 310 | 2.66 | 0.000 |
| Head of household works in agriculture | 0.42 | 2,627 | 0.28 | 327 | 0.15 | 0.000 |
| Head of household works in business | 0.16 | 2,627 | 0.20 | 327 | -0.05 | 0.104 |
| F-test combined sample means | | | | | | 9.640 |

Panel II: Endogenous Variables

| VARIABLE | Survey Data | | DHS data | | Diff. | P-value |
|-------------------------------------|-------------|--------|----------|-------|-------|---------------|
| | Mean | N | Mean | N | | |
| Number of births | 6.80 | 12,185 | 5.11 | 1,130 | 1.68 | 0.000 |
| Mean birth interval | 4.62 | 12,155 | 3.10 | 1,130 | 1.51 | 0.000 |
| Fraction of deaths under 12 mos | 0.06 | 12,168 | 0.09 | 1,130 | -0.02 | 0.026 |
| Fraction of deaths under 24 mos | 0.08 | 12,152 | 0.11 | 1,129 | -0.03 | 0.019 |
| Fraction of deaths under 60 mos | 0.11 | 12,062 | 0.13 | 1,129 | -0.01 | 0.225 |
| F-test combined sample means | | | | | | 13.616 |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. Households with children born between 1980 and 2007 who did not turn one before the household was living at the current place of residence. The DHS data additionally excludes urban households and households with no female children between the age of 10 and 17 in 2007. *Fraction of deaths under 12, 24, and 60 mos* exclude children under the age cutoff in 2007 (i.e., children who were alive but below the respective age cutoffs), for whom mortality is censored.

Table B.2: Sample means by measured contamination

| Panel I: Exogenous Variables | | | | | | |
|--|--------------------|-------|-------------------|-------|-------|---------|
| VARIABLE | High Contamination | | Low Contamination | | Diff. | P-value |
| | Mean | N | Mean | N | | |
| Age of mother | 42.08 | 2,512 | 41.90 | 1,316 | 0.18 | 0.709 |
| Age of mother at earliest birth | 18.20 | 1,560 | 18.44 | 852 | -0.24 | 0.355 |
| Education of mother | 3.38 | 2,508 | 3.38 | 1,316 | 0.00 | 0.989 |
| Education of father | 4.57 | 2,462 | 4.60 | 1,293 | -0.02 | 0.912 |
| Solvency | 0.70 | 2,543 | 0.68 | 1,333 | 0.01 | 0.563 |
| Land size (arces) | 0.81 | 2,521 | 0.83 | 1,322 | -0.02 | 0.768 |
| Number of rooms in house | 2.77 | 2,537 | 2.81 | 1,331 | -0.04 | 0.547 |
| Electricity | 0.41 | 2,541 | 0.40 | 1,332 | 0.00 | 0.863 |
| Muslim | 0.97 | 2,542 | 0.96 | 1,333 | 0.01 | 0.195 |
| Fraction of children living in household | 0.73 | 2,515 | 0.73 | 1,318 | -0.00 | 0.747 |
| Respondent's age | 41.51 | 2,542 | 41.33 | 1,333 | 0.18 | 0.746 |
| Male respondent | 0.17 | 2,543 | 0.18 | 1,333 | -0.00 | 0.813 |
| Sufficiency of food per week | 0.93 | 2,543 | 0.92 | 1,333 | 0.01 | 0.645 |
| Outstanding loan | 0.54 | 2,534 | 0.54 | 1,332 | -0.00 | 0.925 |
| Years lived in house | 27.24 | 2,543 | 26.64 | 1,333 | 0.59 | 0.400 |
| Years lived in village | 31.30 | 2,044 | 30.99 | 1,074 | 0.30 | 0.695 |
| Mean monthly income of household (\$) | 57.51 | 2,543 | 55.71 | 1,333 | 1.80 | 0.540 |
| Head of household works in agriculture | 0.43 | 2,470 | 0.42 | 1,296 | 0.01 | 0.625 |
| Head of household works in business | 0.16 | 2,470 | 0.16 | 1,296 | -0.00 | 0.935 |
| F-test combined sample means | | | | | | 1.664 |

| Panel II: Endogenous Variables | | | | | | |
|-------------------------------------|--------------------|-------|-------------------|-------|-------|---------|
| VARIABLE | High Contamination | | Low Contamination | | Diff. | P-value |
| | Mean | N | Mean | N | | |
| Arsenic concentration (ppb) | 93.92 | 2,267 | 32.18 | 1,278 | 61.74 | 0.000 |
| High reported concentration | 0.87 | 2,543 | 0.44 | 1,333 | 0.43 | 0.000 |
| Number of births | 6.22 | 2,535 | 6.05 | 1,331 | 0.16 | 0.319 |
| Mean birth interval | 5.06 | 2,525 | 5.34 | 1,319 | -0.29 | 0.263 |
| Fraction of deaths under 12 mos | 0.06 | 2,525 | 0.05 | 1,325 | 0.01 | 0.120 |
| Fraction of deaths under 24 mos | 0.07 | 2,516 | 0.06 | 1,318 | 0.02 | 0.012 |
| Fraction of deaths under 60 mos | 0.10 | 2,479 | 0.08 | 1,280 | 0.01 | 0.049 |
| Deep tubewell \leq 500 meters | 0.54 | 2,418 | 0.52 | 1,247 | 0.02 | 0.146 |
| Number of drinking sources used | 1.59 | 2,543 | 1.64 | 1,333 | -0.05 | 0.389 |
| Closest well tested | 0.81 | 2,104 | 0.76 | 1,171 | 0.05 | 0.048 |
| Closest well painted | 0.70 | 2,301 | 0.62 | 1,267 | 0.08 | 0.010 |
| Value of house (\$) | 38.95 | 2,540 | 38.09 | 1,332 | 0.86 | 0.716 |
| F-test combined sample means | | | | | | 34.004 |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level and village fixed effects. *Sufficiency of food* defined as family members taking at least two meals a day last week; *solvency* defined as last week's expenses being within the budget. *Last well tested or painted* as reported by survey respondent. Responses to years lived in village were only collected starting mid-survey. High contamination households defined as those with tubewells that contain arsenic contamination greater than 60 ppb according to field tests of the shallow tubewells closest to the residence. *Fraction of deaths under 12, 24, and 60 mos* exclude children under the age cutoff in 2007, for whom mortality is censored.

Table B.3: Sample means: deep tubewell data collection

| VARIABLE | Mean | N | S.D. | Min | Max |
|---|--------|-------|--------|--------|----------|
| Tubewell age (years) | 19.00 | 1,092 | 6.45 | 10.00 | 48.00 |
| Tubewell depth (meters) | 899.06 | 1,085 | 106.52 | 405.00 | 1,500.00 |
| Depth calculated from pipes' length and numbers | 901.30 | 1,057 | 110.62 | 420.00 | 1,500.00 |
| Village share of public tubewells | 0.32 | 158 | 0.30 | 0.00 | 1.00 |
| Share of quasi-public tubewells | 0.97 | 158 | 0.07 | 0.50 | 1.00 |
| Number of tubewells per village | 6.74 | 162 | 4.83 | 0.00 | 23.00 |
| Number of tubewells per 1000 capita | 5.40 | 162 | 3.63 | 0.00 | 23.72 |
| Number of tubewells per village in 2000 | 3.83 | 162 | 3.20 | 0.00 | 16.00 |
| Number of tubewells per 1000 capita in 2000 | 3.57 | 162 | 3.16 | 0.00 | 21.08 |

Notes: Data from the 2016 tubewell data collection. Unless stated, numbers from 2007.

Table B.4: Sample means: deep tubewell data, by village contamination

| VARIABLE | High Contamination | | Low Contamination | | Diff. | P-value |
|-------------------------------------|--------------------|-----|-------------------|----|-------|--------------|
| | Mean | N | Mean | N | | |
| Tubewell age (years) | 19.24 | 117 | 17.77 | 41 | 1.47 | 0.136 |
| Tubewell depth (meters) | 904.49 | 117 | 891.83 | 41 | 12.65 | 0.359 |
| Village share of public tubewells | 0.28 | 117 | 0.41 | 41 | -0.12 | 0.094 |
| Share of quasi-public tubewells | 0.97 | 117 | 0.98 | 41 | -0.01 | 0.323 |
| Number of tubewells per village | 7.48 | 118 | 5.28 | 44 | 2.20 | 0.005 |
| Number of tubewells per 1000 capita | 5.76 | 118 | 4.90 | 44 | 0.86 | 0.102 |
| F-test combined sample means | | | | | | 2.524 |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the union level and union fixed effects. High concentration is a community average of above $60\mu\text{g}$ (results are very similar using $50\mu\text{g}$ as a cutoff or at least 50% or 60% of households with a level of above $60\mu\text{g}$). *Quasi-public tubewells* include public wells as well as private wells used by other households in the village.

Table B.5: Child mortality: effects by exposure to deep tubewells close to the residence; child died < 60 months from a water-related cause, including household fixed effects

| Tubewells within (meters): | 300 | 400 | 500 | 600 | 700 | 800 | 900 | 1,000 |
|------------------------------------|--------------------|---------------------|---------------------|----------------------|----------------------|---------------------|---------------------|---------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| High con. * Exposure | 0.028** (0.014) | 0.031** (0.014) | 0.030** (0.013) | 0.034** (0.014) | 0.028* (0.014) | 0.026* (0.015) | 0.022 (0.015) | 0.021 (0.015) |
| # Tubewells | 0.006 (0.007) | 0.001 (0.005) | -0.002 (0.005) | -0.001 (0.004) | -0.005 (0.004) | -0.004 (0.003) | -0.003 (0.003) | -0.003 (0.002) |
| High con. * # Tubewells | -0.010 (0.008) | -0.000 (0.006) | 0.003 (0.005) | 0.002 (0.004) | 0.005 (0.004) | 0.003 (0.003) | 0.003 (0.003) | 0.004 (0.002) |
| Exposure * # Tubewells | 0.014 (0.010) | 0.012* (0.007) | 0.008* (0.005) | 0.007** (0.003) | 0.007*** (0.003) | 0.005** (0.002) | 0.003* (0.002) | 0.003* (0.002) |
| High con. * Exposure * # Tubewells | -0.016 (0.011) | -0.016** (0.008) | -0.012** (0.005) | -0.010*** (0.004) | -0.009*** (0.003) | -0.007** (0.003) | -0.005** (0.002) | -0.005** (0.002) |
| Mean(Low con. & No exposure) | 0.039 | 0.039 | 0.039 | 0.039 | 0.039 | 0.039 | 0.039 | 0.039 |
| Observations | 10298 | 10298 | 10298 | 10298 | 10298 | 10298 | 10298 | 10298 |
| Household FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Controls | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |

Notes: See notes to table 1. # Tubewells is the number of deep tubewells within X meters of the residence in the year of the child's birth. Controls include the child's sex and birth order and the mother's age at birth. $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.6: Child mortality: effects by exposure to deep tubewells close to the residence; child died < 60 months from non-arsenic/non-water related cause.

| Tubewells within (meters): | 300 | 400 | 500 | 600 | 700 | 800 | 900 | 1,000 | 500 |
|------------------------------------|-------------------|-------------------|-------------------|------------------|-------------------|-------------------|-------------------|-------------------|-------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High con. | 0.000 (0.005) | -0.001 (0.005) | -0.000 (0.005) | 0.000 (0.005) | -0.001 (0.005) | 0.000 (0.005) | -0.000 (0.005) | -0.000 (0.005) | |
| High con. * Exposure | 0.003 (0.007) | 0.000 (0.008) | 0.003 (0.008) | 0.002 (0.008) | 0.003 (0.008) | -0.001 (0.008) | -0.001 (0.009) | 0.001 (0.009) | 0.010 (0.010) |
| # Tubewells | -0.003 (0.003) | -0.001 (0.003) | 0.000 (0.002) | 0.001 (0.002) | 0.000 (0.001) | 0.002 (0.001) | 0.001 (0.001) | 0.001 (0.001) | -0.003 (0.004) |
| High con. * # Tubewells | 0.001 (0.004) | 0.003 (0.003) | 0.001 (0.003) | 0.001 (0.002) | 0.001 (0.002) | 0.000 (0.002) | 0.000 (0.001) | 0.000 (0.001) | 0.003 (0.005) |
| Exposure * # Tubewells | 0.005 (0.004) | 0.003 (0.003) | 0.001 (0.003) | 0.001 (0.002) | 0.000 (0.002) | -0.001 (0.002) | -0.001 (0.002) | -0.000 (0.001) | 0.002 (0.003) |
| High con. * Exposure * # Tubewells | 0.001 (0.006) | 0.001 (0.005) | 0.000 (0.004) | 0.000 (0.003) | -0.000 (0.002) | 0.001 (0.002) | 0.001 (0.002) | 0.000 (0.002) | -0.002 (0.004) |
| Mean(Low con. & No exposure) | 0.021 | 0.021 | 0.021 | 0.021 | 0.021 | 0.021 | 0.021 | 0.021 | 0.021 |
| Observations | 10534 | 10534 | 10534 | 10534 | 10534 | 10534 | 10534 | 10534 | 10299 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Controls | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Household FE | | | | | | | | | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. We exclude all children that had not yet reached age 5. High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains an arsenic contamination greater than 60 μ g, according to field tests conducted in 2009. Exposure denotes the fraction of life that the child was potentially exposed to unsafe water (time lived after 2000). # Tubewells is the number of deep tubewells within X meters of the residence in the year of the child's birth. Controls include the child's sex and birth order, the mother's age at birth, the mother's and father's education. We also control for income and distance to the village center to ensure that distance to tubewells does not only proxy income or location. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.7: Child mortality: effects by exposure to deep tubewells close to the residence; child died < 60 months from an arsenic-related cause.

| Tubewells within (meters): | 300 | 400 | 500 | 600 | 700 | 800 | 900 | 1,000 | 500 |
|------------------------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High con. | 0.001 (0.006) | -0.002 (0.006) | -0.001 (0.006) | -0.001 (0.007) | 0.000 (0.007) | -0.001 (0.007) | -0.001 (0.007) | 0.000 (0.007) | |
| High con. * Exposure | -0.011 (0.012) | -0.007 (0.013) | -0.004 (0.013) | -0.002 (0.014) | -0.004 (0.013) | -0.004 (0.014) | -0.000 (0.014) | -0.000 (0.014) | 0.003 (0.016) |
| # Tubewells | 0.004 (0.007) | 0.000 (0.004) | 0.003 (0.004) | 0.003 (0.003) | 0.004 (0.003) | 0.001 (0.003) | 0.001 (0.002) | 0.001 (0.002) | 0.004 (0.007) |
| High con. * # Tubewells | -0.005 (0.007) | 0.002 (0.005) | 0.001 (0.004) | 0.000 (0.004) | -0.001 (0.003) | 0.000 (0.003) | -0.000 (0.002) | -0.000 (0.002) | -0.004 (0.007) |
| Exposure * # Tubewells | -0.001 (0.010) | 0.005 (0.006) | 0.002 (0.005) | 0.001 (0.004) | -0.000 (0.004) | 0.001 (0.004) | 0.002 (0.003) | 0.002 (0.003) | 0.002 (0.005) |
| High con. * Exposure * # Tubewells | 0.010 (0.011) | -0.001 (0.007) | -0.001 (0.005) | -0.002 (0.005) | -0.000 (0.004) | -0.001 (0.004) | -0.001 (0.003) | -0.001 (0.003) | -0.002 (0.006) |
| Mean(Low con. & No exposure) | 0.040 | 0.040 | 0.040 | 0.040 | 0.040 | 0.040 | 0.040 | 0.040 | 0.040 |
| Observations | 10534 | 10534 | 10534 | 10534 | 10534 | 10534 | 10534 | 10534 | 10299 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Controls | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Household FE | | | | | | | | | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. We exclude all children that had not yet reached age 5. High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains an arsenic contamination greater than 60 μ g, according to field tests conducted in 2009. Exposure denotes the fraction of life that the child was potentially exposed to unsafe water (time lived after 2000). # Tubewells is the number of deep tubewells within X meters of the residence in the year of the child's birth. Controls include the child's sex and birth order, the mother's age at birth, the mother's and father's education. We also control for income and distance to the village center to ensure that distance to tubewells does not only proxy income or location. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.8: Child mortality: full set of controls

| | Death < 12 months | | | Death < 24 months | | | Death < 60 months | | |
|------------------------------|--------------------|--------------------|--------------------|--------------------|--------------------|--------------------|--------------------|--------------------|---------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High con. | -0.001 (0.007) | -0.001 (0.007) | -0.002 (0.007) | 0.006 (0.008) | 0.006 (0.008) | 0.004 (0.008) | -0.002 (0.009) | -0.002 (0.009) | -0.007 (0.009) |
| Exposure | -0.001 (0.009) | | | 0.009 (0.011) | | | 0.036** (0.016) | | |
| High con. * Exposure | 0.024** (0.011) | 0.024** (0.011) | 0.026** (0.011) | 0.024** (0.011) | 0.024** (0.011) | 0.027** (0.011) | 0.033** (0.015) | 0.034** (0.015) | 0.041*** (0.015) |
| Mean(Low con. & No exposure) | 0.069 | 0.069 | 0.069 | 0.088 | 0.088 | 0.088 | 0.138 | 0.138 | 0.138 |
| Observations | 11979 | 11979 | 11979 | 11755 | 11755 | 11755 | 10810 | 10810 | 10810 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ | | | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. We exclude all children that had not yet reached age 1 in columns (1)-(3), age 2 in columns (4)-(6), and age 5 in columns (7)-(9). High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than 60 μ according to field tests conducted in 2009. Exposure denotes the fraction of life that the child was potentially exposed to unsafe water (time lived after 2000). Controls include the child's sex and birth order, the mother's age at first birth and birth and years since last birth, the mother's and father's education, the monthly income of the household head, the land size, number of rooms in the house, whether the house has electricity and distance to the village center. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.9: Child mortality: yearly exposure

| | Death < 12 months | | | Death < 24 months | | | Death < 60 months | | |
|------------------------------|--------------------|--------------------|--------------------|--------------------|--------------------|--------------------|--------------------|--------------------|--------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High con. | -0.001 (0.007) | -0.001 (0.007) | -0.002 (0.007) | 0.006 (0.008) | 0.006 (0.008) | 0.004 (0.008) | -0.002 (0.009) | -0.002 (0.009) | -0.006 (0.009) |
| Exposure | -0.001 (0.009) | | | 0.004 (0.006) | | | 0.007** (0.003) | | |
| High con. * Exposure | 0.024** (0.011) | 0.024** (0.011) | 0.025** (0.011) | 0.012** (0.006) | 0.012** (0.006) | 0.013** (0.006) | 0.007** (0.003) | 0.007** (0.003) | 0.008** (0.003) |
| Mean(Low con. & No exposure) | 0.069 | 0.069 | 0.069 | 0.088 | 0.088 | 0.088 | 0.138 | 0.138 | 0.138 |
| Observations | 11979 | 11979 | 11979 | 11755 | 11755 | 11755 | 10810 | 10810 | 10810 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ | | | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. We exclude all children that had not yet reached age 1 in columns (1)-(3), age 2 in columns (4)-(6), and age 5 in columns (7)-(9). High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than 60 μ g, according to field tests conducted in 2009. Exposure denotes the number of life years that the child was potentially exposed to unsafe water (years lived after 2000). Controls include the child's sex and birth order, the mother's age at birth, and the mother's and father's education. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.10: Child mortality: binary exposure

| | Death < 12 months | | | Death < 24 months | | | Death < 60 months | | |
|------------------------------|--------------------|--------------------|--------------------|-------------------|-------------------|-------------------|-------------------|------------------|-------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High con. | -0.001 (0.007) | -0.001 (0.007) | -0.002 (0.007) | 0.007 (0.008) | 0.007 (0.008) | 0.006 (0.008) | 0.003 (0.008) | 0.002 (0.008) | -0.000 (0.008) |
| Exposure | -0.001 (0.009) | | | 0.009 (0.010) | | | 0.029* (0.015) | | |
| High con. * Exposure | 0.024** (0.011) | 0.024** (0.011) | 0.025** (0.011) | 0.021* (0.011) | 0.021* (0.011) | 0.022* (0.011) | 0.027 (0.017) | 0.026 (0.017) | 0.028 (0.017) |
| Mean(Low con. & No exposure) | 0.069 | 0.069 | 0.069 | 0.085 | 0.085 | 0.085 | 0.120 | 0.120 | 0.120 |
| Observations | 11979 | 11979 | 11979 | 11755 | 11755 | 11755 | 10810 | 10810 | 10810 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ | | | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. We exclude all children that had not yet reached age 1 in columns (1)-(3), age 2 in columns (4)-(6), and age 5 in columns (7)-(9). High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than 60 μ g, according to field tests conducted in 2009. Exposure is 1 if the child is born in or after 2000. Controls include the child's sex and birth order, the mother's age at birth, and the mother's and father's education. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.11: Child mortality: imputation approach by Borusyak et al. (2022)

| | Death < 12 months | | | Death < 24 months | | | Death < 60 months | | |
|------------------------------|---------------------|---------------------|---------------------|---------------------|---------------------|---------------------|---------------------|-------------------|-------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High con. * Exposure | 0.024*** (0.008) | 0.024*** (0.009) | 0.024*** (0.009) | 0.031*** (0.009) | 0.026*** (0.010) | 0.027*** (0.010) | 0.053*** (0.011) | 0.029* (0.016) | 0.028* (0.016) |
| Mean(Low con. & No exposure) | 0.069 | 0.069 | 0.069 | 0.088 | 0.088 | 0.088 | 0.138 | 0.138 | 0.138 |
| Observations | 11979 | 11979 | 11979 | 11755 | 11755 | 11755 | 10810 | 10810 | 10810 |
| Household FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ | | | ✓ |

Notes: The table shows results from the difference-in-differences imputation approach with staggered adoption of treatment (Borusyak et al., 2022). Huber-White robust SEs clustered at the village level. We exclude all children that had not yet reached age 1 in columns (1)-(3), age 2 in columns (4)-(6), and age 5 in columns (7)-(9). Controls include the child's sex and birth order, the mother's age at birth, and the mother's and father's education. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.12: Child mortality: reported concentration

| | Death < 12 months | | | Death < 24 months | | | Death < 60 months | | |
|------------------------------|-------------------|-------------------|-------------------|-------------------|------------------|------------------|--------------------|--------------------|--------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High contamination | 0.002 (0.007) | 0.002 (0.007) | 0.003 (0.007) | 0.004 (0.008) | 0.005 (0.008) | 0.005 (0.008) | -0.008 (0.010) | -0.008 (0.010) | -0.008 (0.011) |
| Exposure | 0.002 (0.010) | | | 0.015 (0.013) | | | 0.033* (0.019) | | |
| High con. * Exposure | 0.019* (0.010) | 0.019* (0.010) | 0.018* (0.010) | 0.013 (0.012) | 0.013 (0.012) | 0.013 (0.012) | 0.035** (0.017) | 0.036** (0.017) | 0.036** (0.017) |
| Mean(Low con. & No exposure) | 0.070 | 0.070 | 0.070 | 0.091 | 0.091 | 0.091 | 0.144 | 0.144 | 0.144 |
| Observations | 11979 | 11979 | 11979 | 11755 | 11755 | 11755 | 10810 | 10810 | 10810 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ | | | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. We exclude all children that had not yet reached age 1 in columns (1)-(3), age 2 in columns (4)-(6), and age 5 in columns (7)-(9). High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than 60μ according to field tests conducted in 2009. Exposure denotes the fraction of life that the child was potentially exposed to unsafe water (time lived after 2000). Controls include the child's sex and birth order, the mother's age at birth, and the mother's and father's education. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.13: Child mortality: 50 μ g contamination cutoff

| | Death < 12 months | | | Death < 24 months | | | Death < 60 months | | |
|------------------------------|-------------------|-------------------|-------------------|--------------------|--------------------|--------------------|--------------------|--------------------|---------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High con. | 0.001 (0.007) | 0.001 (0.007) | -0.000 (0.007) | 0.009 (0.008) | 0.009 (0.008) | 0.007 (0.008) | -0.000 (0.010) | -0.001 (0.010) | -0.004 (0.010) |
| Exposure | 0.002 (0.010) | | | 0.008 (0.012) | | | 0.030* (0.017) | | |
| High con. * Exposure | 0.019* (0.010) | 0.019* (0.010) | 0.020* (0.011) | 0.024** (0.011) | 0.025** (0.011) | 0.027** (0.012) | 0.039** (0.017) | 0.040** (0.017) | 0.045*** (0.016) |
| Mean(Low con. & No exposure) | 0.069 | 0.069 | 0.069 | 0.087 | 0.087 | 0.087 | 0.138 | 0.138 | 0.138 |
| Observations | 11979 | 11979 | 11979 | 11755 | 11755 | 11755 | 10810 | 10810 | 10810 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ | | | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. We exclude all children that had not yet reached age 1 in columns (1)-(3), age 2 in columns (4)-(6), and age 5 in columns (7)-(9). High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than 50 μ g, according to field tests conducted in 2009. Exposure denotes the fraction of life that the child was potentially exposed to unsafe water (time lived after 1999). Controls include the child's sex and birth order, the mother's age at birth, and the mother's and father's education. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.14: Child mortality: 1999 campaign cutoff

| | Death < 12 months | | | Death < 24 months | | | Death < 60 months | | |
|------------------------------|---------------------|---------------------|---------------------|--------------------|--------------------|--------------------|--------------------|--------------------|--------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High con. | -0.003 (0.007) | -0.003 (0.007) | -0.004 (0.007) | 0.005 (0.008) | 0.005 (0.008) | 0.003 (0.008) | -0.003 (0.010) | -0.003 (0.010) | -0.007 (0.009) |
| Exposure | -0.006 (0.009) | | | 0.013 (0.011) | | | 0.034** (0.017) | | |
| High con. * Exposure | 0.028*** (0.009) | 0.028*** (0.009) | 0.030*** (0.009) | 0.023** (0.010) | 0.023** (0.010) | 0.026** (0.011) | 0.030** (0.014) | 0.031** (0.014) | 0.036** (0.014) |
| Mean(Low con. & No exposure) | 0.072 | 0.072 | 0.072 | 0.089 | 0.089 | 0.089 | 0.142 | 0.142 | 0.142 |
| Observations | 11979 | 11979 | 11979 | 11755 | 11755 | 11755 | 10810 | 10810 | 10810 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ | | | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. We exclude all children that had not yet reached age 1 in columns (1)-(3), age 2 in columns (4)-(6), and age 5 in columns (7)-(9). High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than 60 μ g, according to field tests conducted in 2009. Exposure denotes the fraction of life that the child was potentially exposed to unsafe water (time lived after 1998). Controls include the child's sex and birth order, the mother's age at birth, and the mother's and father's education. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.15: Child mortality: 2002 campaign cutoff

| | Death < 12 months | | | Death < 24 months | | | Death < 60 months | | |
|------------------------------|-------------------|-------------------|-------------------|-------------------|------------------|-------------------|--------------------|--------------------|--------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High con. | 0.001 (0.007) | 0.001 (0.007) | 0.000 (0.006) | 0.008 (0.007) | 0.008 (0.007) | 0.007 (0.007) | -0.000 (0.009) | -0.000 (0.009) | -0.003 (0.009) |
| Exposure | 0.000 (0.009) | | | 0.008 (0.011) | | | 0.044** (0.018) | | |
| High con. * Exposure | 0.022* (0.012) | 0.022* (0.012) | 0.023* (0.012) | 0.022 (0.013) | 0.021 (0.013) | 0.023* (0.014) | 0.045** (0.020) | 0.045** (0.020) | 0.049** (0.020) |
| Mean(Low con. & No exposure) | 0.066 | 0.066 | 0.066 | 0.083 | 0.083 | 0.083 | 0.127 | 0.127 | 0.127 |
| Observations | 11979 | 11979 | 11979 | 11755 | 11755 | 11755 | 10810 | 10810 | 10810 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ | | | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. We exclude all children that had not yet reached age 1 in columns (1)-(3), age 2 in columns (4)-(6), and age 5 in columns (7)-(9). High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains an arsenic contamination greater than $60\mu\text{g}$, according to field tests conducted in 2009. Exposure denotes the fraction of life that the child was potentially exposed to unsafe water (time lived after 2001). Controls include the child's sex and birth order, the mother's age at birth, and the mother's and father's education. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.16: Child mortality: hazard of dying among children

| | (1) | (2) | (3) |
|----------------------------------|-------------------|---------------------|---------------------|
| High con. | 1.006 (0.072) | 1.000 (0.072) | 0.975 (0.071) |
| Post-2000 | 0.862 (0.133) | 0.586*** (0.107) | 0.571*** (0.105) |
| High con. * Post-2000 | 1.336* (0.215) | 1.349* (0.218) | 1.381** (0.225) |
| Mean(Low con. & Before campaign) | 0.003 | 0.003 | 0.003 |
| Observations | 152909 | 152909 | 152909 |
| Village FE | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ |
| Controls | | | ✓ |

Notes: The table shows results from Cox proportional hazards models with Huber-White robust SEs clustered at the village level. Each observation is one person-year (each year of life from birth to death, or the year of data collection if still alive, in which case the data is censored for that person). The failure event is death. The sample includes all children born between 1980 and 2006. High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than $60\mu\text{g}$, according to field tests conducted in 2009. Post-2000 is an indicator that is 1 in the years 2000 to 2006.

Controls include the child's sex and birth order, the mother's age at birth, and the mother's and father's education. All regressions are adjusted for time trends (birth year).

$p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.17: Child mortality: placebo test, 100 μ g contamination cutoff

| | Death < 12 months | | | Death < 24 months | | | Death < 60 months | | |
|------------------------------|--------------------|-------------------|-------------------|---------------------|-------------------|-------------------|---------------------|-------------------|-------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High con. | -0.007 (0.008) | -0.007 (0.008) | -0.008 (0.008) | -0.012 (0.009) | -0.012 (0.009) | -0.013 (0.009) | -0.012 (0.012) | -0.012 (0.012) | -0.012 (0.012) |
| Exposure | 0.030** (0.013) | | | 0.040*** (0.015) | | | 0.063*** (0.022) | | |
| High con. * Exposure | -0.005 (0.015) | -0.005 (0.015) | -0.006 (0.015) | -0.004 (0.017) | -0.004 (0.017) | -0.006 (0.017) | 0.018 (0.021) | 0.017 (0.022) | 0.018 (0.022) |
| Mean(Low con. & No exposure) | 0.074 | 0.074 | 0.074 | 0.098 | 0.098 | 0.098 | 0.140 | 0.140 | 0.140 |
| Observations | 7023 | 7023 | 7023 | 6903 | 6903 | 6903 | 6378 | 6378 | 6378 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ | | | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. The sample includes all households for which the shallow tubewell closest to the residence contains arsenic contamination greater than 60 μ g, according to field tests conducted in 2009. We exclude all children that had not yet reached age 1 in columns (1)-(3), age 2 in columns (4)-(6), and age 5 in columns (7)-(9). High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than 100 μ g, according to field tests conducted in 2009. Exposure denotes the fraction of life that the child was potentially exposed to unsafe water (time lived after 2000). Controls include the child's sex and birth order, the mother's age at birth, and the mother's and father's education. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.18: Child mortality: continuous arsenic contamination

| | Death < 12 months | | | Death < 24 months | | | Death < 60 months | | |
|------------------------------|--------------------|-------------------|-------------------|--------------------|-------------------|--------------------|--------------------|-------------------|-------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| Exposure | 0.032** (0.015) | | | 0.044** (0.017) | | | 0.059** (0.023) | | |
| Arsenic | -0.000 (0.000) | -0.000 (0.000) | -0.000 (0.000) | -0.000 (0.000) | -0.000 (0.000) | -0.000* (0.000) | -0.000 (0.000) | -0.000 (0.000) | -0.000 (0.000) |
| Ars. * Exposure | -0.000 (0.000) | -0.000 (0.000) | -0.000 (0.000) | -0.000 (0.000) | -0.000 (0.000) | -0.000 (0.000) | 0.000 (0.000) | 0.000 (0.000) | 0.000 (0.000) |
| Mean(Low con. & No exposure) | 0.069 | 0.069 | 0.069 | 0.089 | 0.089 | 0.089 | 0.130 | 0.130 | 0.130 |
| Observations | 7023 | 7023 | 7023 | 6903 | 6903 | 6903 | 6378 | 6378 | 6378 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ | | | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. We exclude all children that had not yet reached age 1 in columns (1)-(3), age 2 in columns (4)-(6), and age 5 in columns (7)-(9). The sample includes all high-contamination households (the shallow tubewell closest to the residence contains arsenic contamination greater than 60 μ g according to field tests conducted in 2009). Arsenic is the continuous Arsenic contamination in μ g. Exposure denotes the fraction of life that the child was potentially exposed to unsafe water (time lived after 2000). Controls include the child's sex and birth order, the mother's age at birth, and the mother's and father's education. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.19: Elderly mortality: hazard of dying among adults age 30+ and age 40+

| | Age 30+ | | | Age 40+ | | |
|---------------------------------|---------------------|---------------------|---------------------|---------------------|---------------------|---------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| High con. | 0.951 (0.139) | 0.962 (0.150) | 0.956 (0.151) | 0.890 (0.137) | 0.886 (0.144) | 0.883 (0.144) |
| Post-2000 | 2.793*** (0.411) | 2.415*** (0.358) | 2.400*** (0.357) | 3.043*** (0.469) | 2.644*** (0.416) | 2.620*** (0.410) |
| High con. * Post-2000 | 1.223 (0.181) | 1.212 (0.186) | 1.238 (0.193) | 1.322* (0.201) | 1.327* (0.210) | 1.361* (0.219) |
| Mean(Low con. & After campaign) | 0.024 | 0.024 | 0.024 | 0.032 | 0.032 | 0.032 |
| Observations | 115,450 | 115,450 | 115,450 | 75,807 | 75,807 | 75,807 |
| Village FF | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ |

Notes: The table shows results from Cox proportional hazards models with Huber-White robust SEs clustered at the village level. Each observation is one person-year (each year of life from birth to death, or the year of data collection if still alive, in which case the data is censored for that person). The left panel includes all person-years aged 30 or higher and the right panel includes all person-years aged 40 or higher. High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than 60 μ g, according to field tests conducted in 2009. Post-2000 is an indicator that is 1 in the years 2000 to 2017. Controls include the adult's gender and the education of the household head and his spouse. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.20: Elderly mortality: hazard of dying among adults age 50+

| | Village-specific baseline hazards | | | After moved into Household | | | 1980-2007 | | |
|---------------------------------|-----------------------------------|---------------------|---------------------|----------------------------|---------------------|---------------------|---------------------|---------------------|---------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| High con. | 0.786 (0.128) | 0.759 (0.135) | 0.747 (0.135) | 0.828 (0.181) | 0.866 (0.181) | 0.823 (0.171) | 0.874 (0.149) | 0.885 (0.166) | 0.861 (0.163) |
| Post-2000 | 3.223*** (0.563) | 2.650*** (0.462) | 2.628*** (0.464) | 3.250*** (0.606) | 3.229*** (0.612) | 3.029*** (0.576) | 3.019*** (0.458) | 2.700*** (0.450) | 2.576*** (0.434) |
| High con. * Post-2000 | 1.586*** (0.271) | 1.664*** (0.308) | 1.695*** (0.314) | 1.561** (0.324) | 1.503** (0.292) | 1.577** (0.312) | 1.405** (0.240) | 1.408* (0.256) | 1.422* (0.265) |
| Mean(Low con. & After campaign) | 0.048 | 0.048 | 0.048 | 0.048 | 0.048 | 0.048 | 0.049 | 0.049 | 0.049 |
| Observations | 45,555 | 45,555 | 45,555 | 30,238 | 30,238 | 30,238 | 33,520 | 33,520 | 33,520 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ | | | ✓ |

Notes: The table shows results from Cox proportional hazards models with Huber-White robust SEs clustered at the village level. Each observation is one person-year (each year of life from birth to death, or the year of data collection if still alive, in which case the data is censored for that person). The failure event is death. The sample includes all person-years age 50 or higher. High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than 60 μ g, according to field tests conducted in 2009. Post-2000 is an indicator that is 1 in the years 2000 to 2017 for the first and second panel, and is 1 for years 2000 to 2007 for the third panel. The first panel presents stratified estimates that allow the baseline hazard function to differ by village. The second panel excludes all person-years before a person moved into a given household. The third panel restricts the sample to years 1980-2007. Controls include the adult's gender and the education of the household head and his spouse. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.21: Elderly mortality: mortality under 80 among adults age 50+ from...

| Tubewells within (meters): | Any | | | Water | Arsenic | Any | | | | | | | |
|------------------------------------|---------------------|-------------------|-------------------|-------------------|-------------------|---------------------|---------------------|---------------------|--------------------|--------------------|--------------------|-------------------|-------------------|
| | (1) | (2) | (3) | (4) | (5) | 300 | 400 | 500 | 600 | 700 | 800 | 900 | 1,000 |
| High con. | 0.027 (0.051) | 0.063 (0.044) | 0.077* (0.046) | -0.015 (0.041) | 0.010 (0.029) | -0.042 (0.051) | -0.048 (0.054) | -0.053 (0.060) | -0.057 (0.054) | -0.044 (0.054) | -0.061 (0.055) | -0.052 (0.054) | -0.047 (0.053) |
| Exposure | 1.528*** (0.295) | | | | | | | | | | | | |
| High con. * Exposure | 0.323** (0.154) | 0.232* (0.120) | 0.192 (0.129) | 0.130 (0.146) | -0.095 (0.135) | 0.312* (0.175) | 0.347* (0.189) | 0.407* (0.219) | 0.463** (0.231) | 0.443** (0.208) | 0.454** (0.207) | 0.417* (0.214) | 0.391* (0.224) |
| # Tubewells | | | | | | -0.070** (0.031) | -0.047** (0.022) | -0.036** (0.018) | -0.023* (0.013) | -0.014 (0.010) | -0.015* (0.009) | -0.010 (0.007) | -0.008 (0.007) |
| High con. * # Tubewells | | | | | | 0.069** (0.033) | 0.044* (0.023) | 0.038** (0.019) | 0.029** (0.014) | 0.018* (0.009) | 0.019** (0.009) | 0.013* (0.007) | 0.010 (0.006) |
| Exposure * # Tubewells | | | | | | 0.069 (0.114) | 0.025 (0.078) | 0.082 (0.066) | 0.075 (0.049) | 0.050* (0.029) | 0.040* (0.022) | 0.027 (0.020) | 0.021 (0.021) |
| High con. * Exposure * # Tubewells | | | | | | -0.093 (0.128) | -0.062 (0.087) | -0.092 (0.076) | -0.089 (0.058) | -0.062 (0.038) | -0.052* (0.030) | -0.035 (0.026) | -0.026 (0.024) |
| Mean(Low con. & No exposure) | 0.108 | 0.108 | 0.108 | 0.000 | 0.000 | 0.108 | 0.108 | 0.108 | 0.108 | 0.108 | 0.108 | 0.108 | 0.108 |
| Observations | 864 | 860 | 860 | 860 | 860 | 666 | 666 | 666 | 666 | 666 | 666 | 666 | 666 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Controls | | | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. The sample includes all adults aged 50+ at the time of the survey or the time of death. We exclude all adults that have not yet reached age 80 and for whom mortality is censored. We also exclude all adults that moved into the residence not at least one year before turning 80, i.e., for whom we do not know the concentration status of the closest water source. High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than 60μ according to field tests conducted in 2009. Exposure denotes the fraction of vulnerable life (age 50-80) that the adult was potentially exposed to unsafe water (time lived after 2000). Controls include the gender of the adult, as well as the education of the household head and his spouse. We also control for income and distance to the village center in the tubewell regressions to ensure that distance to tubewells does not only proxy income or location. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

Table B.22: Elderly mortality: hazard of dying among adults age 50+

| | Placebo Test | | | Continuous Arsenic contamination | | |
|-----------------------|------------------|------------------|------------------|----------------------------------|---------------------|---------------------|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| High con. | 0.936 (0.170) | 0.819 (0.207) | 0.812 (0.202) | | | |
| Post-1990 | 1.266 (0.323) | 0.812 (0.218) | 0.812 (0.218) | | | |
| High con. * Post-1990 | 1.161 (0.241) | 1.254 (0.306) | 1.261 (0.312) | | | |
| Arsenic | | | | 1.000 (0.003) | 1.000 (0.003) | 1.000 (0.003) |
| Post-2000 | | | | 5.544*** (1.799) | 3.813*** (1.339) | 4.013*** (1.372) |
| Ars. * Post-2000 | | | | 0.999 (0.003) | 1.000 (0.004) | 1.000 (0.003) |
| Mean | 0.005 | 0.005 | 0.005 | 0.060 | 0.060 | 0.060 |
| Observations | 31,040 | 31,040 | 31,040 | 26,309 | 26,309 | 26,309 |
| Village FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Birth year FE | | ✓ | ✓ | | ✓ | ✓ |
| Controls | | | ✓ | | | ✓ |

Notes: The table shows results from Cox proportional hazards models with Huber-White robust SEs clustered at the village level. Each observation is one person-year (each year of life from birth to death, or the year of data collection if still alive, in which case the data is censored for that person). The failure event is death. All regressions restrict the sample to person-years age 50 or higher, and stratified estimations allow the baseline hazard function to differ by village. The left panel restricts the sample to all years before 2000 (the year of the campaign) and defines a hypothetical campaign year of 1990. High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains arsenic contamination greater than $60\mu\text{g}$, according to field tests conducted in 2009. Post-1990 is an indicator that is 1 in the years 1990 to 2000. The right panel instead restricts the sample to high-contamination households (the shallow tubewell closest to the residence contains arsenic contamination greater than $60\mu\text{g}$ according to field tests conducted in 2009). Arsenic is the continuous Arsenic contamination in μg . Post-2000 is an indicator that is 1 in the years 2000 to 2017. Controls include the adult's gender and the education of the household head and his spouse. All regressions are adjusted for time trends (birth year). $p < 0.10^*$, $p < 0.05^{**}$, $p < 0.01^{***}$.

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