The Lifesaving Benefits of Convenient Infrastructure: Quantifying the Mortality Impact of Abandoning Shallow Tubewells Contaminated by Arsenic in Bangladesh

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Abstract

We document the consequences of a public health campaign which led to the sudden abandonment of local water infrastructure by one-fifth of Bangladesh's population. Households who experienced quasi-randomly distributed arsenic contamination, and thus were likely to abandon their shallow tubewells, saw 28% greater child and 47% greater elderly mortality post-campaign than those who not motivated to shift. Sudden mortality increases are driven by diarrheal disease, with no change in arsenic-related deaths. Mortality changes depend on the distance to alternative clean water infrastructure: those with an (arsenic and pathogen-free) deep tubewell within 300m of their home experience no increase in mortality, but mortality rises as households are forced to walk further for safe water access. Our results quantify the mortality benefits of water infrastructure and underscore the importance of physical proximity to, rather than just access to, pathogen-free water sources.

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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. Unsafe water sources are recognized as a primary risk factor for diarrheal disease (Troeger et al., 2018). However, the magnitude of the direct impact of improved water infrastructure on child and elderly mortality remains disputed in both the economic and the public health literature given 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 recent history of Bangladesh offers a unique opportunity to causally identify the impact of water infrastructure on mortality, as the discovery of naturally-leaching arsenic in the groundwater led a large number of (plausibly exogenous) households to rapidly abandon arsenic contaminated backyard wells for more distant primary water sources. Based on tests conducted by the British Geological Survey (BGS) in 1998, an estimated 20 million Bangladeshis were exposed to well water that contained more than the government's recommended maximum arsenic level of 50ppb (μg) per liter (D G Kinniburgh, 2001). This prompted a government-initiated campaign in 1999 to test millions of tubewells nationwide and encourage households to abandon wells 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 contaminated tubewells dropped from 69% in 1999 to 1% in 2006 (Bangladesh Bureau of Statistics and Unicef (2006); D G Kinniburgh (2001)).

In this paper, we show that the sudden abandonment of convenient but arsenic-contaminated shallow tubewells in southwest Bangladesh was associated with a substantial *rise* in mortality among both children and the elderly, and that these deaths were caused by diarrheal rather than arsenicosis-related disease. While vulnerable to arsenic contamination from groundwater, shallow tubewells 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 fecal bacteria at source, but it also faces little risk of becoming contaminated at point of use: shallow tubewells are typically built within household compounds, reducing storage time.[1] Accordingly, shallow tubewells were constructed across Bangladesh in the

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

1970s and 1980s as a prophylactic to water-borne diseases such as cholera and dysentery, common to surface water or the more distant alternative water sources used prior to widespread tubewell construction (Prüss et al., 2002).

In our study context of Barisal, the vast majority of households had access to alternative pathogen-free water sources in the form of deep tubewells by the time of the campaign. What drives the increase in mortality is a households proximity (or not) to this alternative water source. Deep tubewells draw from deeper aquifers that are less vulnerable to arsenic contamination, making them also free of high levels of arsenic. However, because deep tubewells are expensive to build (at 8.5 times the cost of shallow tubewells), they are rarely constructed by individual households within a family compound. The use of arsenic and pathogen-free deep tubewells therefore requires that water be collected and stored in containers in the home, introducing the risk that drinking water becomes contaminated at the point of use. As such, our setting offers a unique opportunity to evaluate the mortality impacts of proximity, rather than merely access, to safe water infrastructure.

Distance to tubewells, both shallow and deep, is not random. However, the particular geography of Bangladesh creates unusual exogenous variation which we are able to exploit in our identification. 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 areas prior to the testing campaign (Smedley and Kinniburgh, 2001). We use both very local variation in the presence of arsenic, and the time discontinuity in the knowledge of arsenic contamination, to examine the impact of sudden changes in access to water infrastructure.

We collect novel data on the location and arsenic content of the closest shallow tubewells of approximately 3,000 households randomly sampled from 162 communities in the Barisal district of Bangladesh. We also collect data on the location and date of construction of deep tubewells in these communities. Our data allow us to compare households whose shallow tubewells tested positive for arsenic contamination (and who were therefore actively encouraged to abandon these convenient water sources) to their neighbors in the same village whose wells tested negative and were deemed safe for drinking. We combine this spatial variation with annual data on child and elderly mortality from before and after the public health campaign in a difference-in-difference specification. To understand the mechanisms at play we compare households who were a similar distance to a deep tubewell and who, post campaign, are and are not forced to rely on it because of quasi random variation in arsenic contamination.

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 water sources experienced a 28% increase in child mortality and 47% increase in elderly mortality relative to those with arsenic-free wells. This divergence is driven by water-related deaths only and coincides with the moment that contaminated households switched to more remote water sources.

While children are vulnerable to diarrheal disease but less susceptible to arsenic-induced cancers (Vahter, 2008; Liaw et al., 2008; Tofail et al., 2009), the elderly potentially face competing mortality risks from the two, making the impact of switching away from arsenic-contaminated but pathogen-free shallow tubewells *ex ante* ambiguous in this population. The long lead time between arsenic contamination and arsenic-related mortality indeed complicates this exercise among adults, but we find no evidence that elderly mortality is higher in households using arsenic-contaminated relative to uncontaminated water sources prior to the public health campaign. Nor do we find that abandoning arsenic wells is associated with improvements in adult life expectancy, the very motivation of the campaign. Instead, we document that the hazard of dying from diarrhea-related deaths for adults over 50 years old increases sharply after the campaign among households encouraged to abandon their wells.

Might households who shift away from their backyard water sources adopt behaviors to counter the increased exposure to pathogen-contaminated water? Keskin et al. (2017), for example, show that mothers in households with arsenic-contaminated wells increase the duration for which they breastfeed their infants.[2] However, the authors only find evidence for this behavioral shift among households who have no access to arsenic and pathogen-free water sources, as proxied by presence within a one mile radius. In contrast, nearly 100% of households in Barisal have alternative clean water infrastructure within one mile. This compensating behavior does not appear to occur among such households (perhaps because the dangers of contamination at point-of-use are less salient than those of contamination at point-of-source), consistent with the increase in infant mortality we document among those encouraged to switch away from their arsenic-contaminated wells. Indeed, our analysis finds that mortality effects increase linearly with distance even within the radius of one kilometer: while those who can access a clean well within 300m of their home experience no adverse mortality impacts from abandoning their backyard shallow tubewells, each additional 100m to a clean alternative source raises both child and elderly

[2]This 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.

mortality. This evidence underscores the importance of proximity, or convenience, beyond access alone to clean water infrastructure.[3]

This study makes three contributions. First, we provide evidence of 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. This speaks to a substantial economics literature that seeks 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), with widely varying results for the degree to which water infrastructure alone reduces mortality.[4] This mixed evidence is echoed in the policy-influential Disease Control Priorities 3 (Jamison, 2018), which 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 nor the WHO (Stenberg et al., 2021).[5] Our study provides new evidence that accessibility to pathogen-free water should meaningfully reduce not only infant and child 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),[6] the majority of studies

[3]An 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 that 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 pipe water, no other options for us. My children are often sick and weak. We are too poor to invest in a well." (Zhongming et al., 2021).

[4]Cutler and Miller (2005) attribute half the mortality declines in US cities in late 19 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 chlorination and filtration had no measurable impact on elderly mortality while filtration reduced infant mortality by 11%. There is evidence water infrastructure reduces deaths from cholera and typhoid (Anderson et al., 2021)but these diseases are a relatively small proportion of overall mortality. Data from 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 hypothesise part of this is due to increased water access.

[5]Kremer 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.

[6]There is ample reason to expect proximity to a clean water source to impact health. Re-

compare unimproved off-premises water sources to improved on-premises sources, and they examine diarrhea rates alone. Two studies (to our knowledge) vary distance to an improved source. Most closely related is Kremer et al. (2011), which randomizes the protection of springs in Kenya and finds that the extent of 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 non-tap water in Morocco and finds no impacts on health; the more developed context and the nature of the intervention, however, suggest distinct underlying mechanisms and policy implications. Our study context permits us to hold the pathogen-cleanliness of well sources constant—as shallow and deep tubewells are equally safe 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 tradeoff 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 contaminated shallow tubewells in contexts such as Bangladesh. 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, at least 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. This 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 easily accessible (Larsen, 2016).

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) 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.

2 Background and previous literature

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 ground water (Caldwell et al., 2003).

Efforts were halted and then reversed with the discovery of arsenic naturally leaching into Bangladesh's groundwater, from which shallow tubewells drew, in the mid-1990s. 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 BGS 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 50μg (D G Kinniburgh, 2001) and were determined to be in "grave danger." Following government screening of all shallow tubewells in endemic regions, 1.4 million were found to be contaminated (above 50μg) and painted red and 3.5 million were painted green to indicate they were safe (Ahmed et al., 2006). Households were strongly 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 may 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 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).[7] 200,000 functional deep tubewells were built by 2007, 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 that were as convenient as shallow tubewells were scarce. Because shallow tubewells are often built close within a family compound, they require less water storage time than less accessible water sources. A study in the Araihazar 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 intent of the 1999 public health campaign was to reduce arsenic exposure and thereby improve health. Those who switched away from their backyard wells were therefore not only exposed to less 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 50μg arsenic cutoff, permits a clean way to rule out that the mortality effects we estimate are due to delayed effects of increased 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 previous 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

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

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 cancers (Chen et al., 2006). These studies have resulted in broad scientific consensus that exposure to high levels of arsenic (>100μg) increases cancer-related deaths and morbidity in older adult populations. Recent studies also suggest that arsenic exposure is associated with higher cardio-vascular, cerebro-vascular and respiratory mortality risk in young adults (Moon et al., 2017; Rahman et al., 2019; Abdul et al., 2015).

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 Araihazar 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 150μg relative to those exposed to less than 10μg (Argos et al., 2010). An important shortcoming not addressed in the study is that 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 Araihazar were in fact concentrated in villages with significantly higher average income and assets.[8] These differences in arsenic disappear when accounting for mean levels of village income, suggesting that the Argos et al. (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),

[8] Van 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 potentially "a 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."

it documents significant impacts on cognition and income.

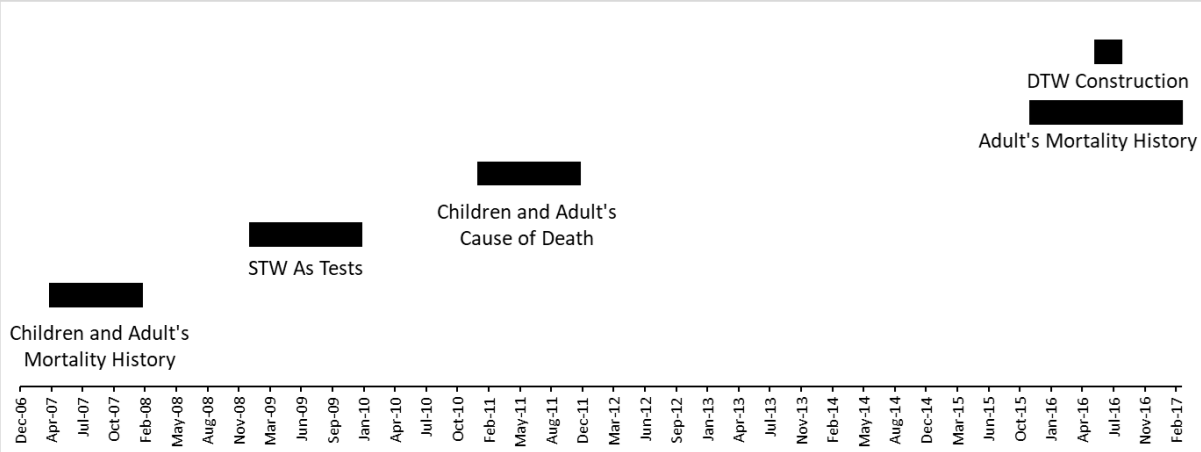
3 Estimation strategy

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

Figure 1 presents the timeline of the data collected for this study.

Figure 1: Timeline of Data Collections



We capitalize on extensive household survey data collected by the authors in 2007, 2011, and 2016. The 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 conntamination at source), households in Barisal were more 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 as well as the age of death of the household head and partner.[9] In 2009, we successfully revisited 3,138 (99%) of the households in our sample. At this time, each household's closest shallow tubewell was tested for arsenic using a standard field testing kit.[10] 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.[11] 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 death of the parents of the household head and partner.[12]

As we did not collect the timing of 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.[13] Our final sample encompasses 12,195 children and 2,422 adults (244,584 life years).

[9]The 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 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 (appendix table B.2), including no differences in child mortality.

[10]A research agency based in Dhaka, Data International, collected up to two samples per household using the Wagtech Digital Arsenator Arsenic kit, which has been found to be reliable in testing both As(III) and As(V) concentrations between 5-100μg (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.

[11]We 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.

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

[13]We drop 198 households because of missing or inconclusive arsenic measurements. The 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.

3.2 Identification strategy

Our identification strategy makes use of small-scale variability in arsenic concentrations in ground water (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 large scale contamination) are extremely hard to predict and do not appear to be correlated with observable features of the land. Combining our data on small-scale variability in arsenic concentration with the sharp time discontinuity in knowledge about arsenic contamination thus allows us to compare households residing close to one another who are and are not encouraged to abandon water sources in a difference-in-difference (DID) estimation strategy. Reflecting the binary nature of information provided to households, we define a binary level of arsenic exposure pre-2000, which categorizes households as contaminated (or "high concentration") if the concentration of arsenic in the shallow tubewell closest to the household is greater than 60μg when measured by our field team in 2009.[14]

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.3). All rows contain regression-controlled means that account for village fixed effects, as do reported 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.[15] The same exercise conducted without accounting for mean differences across villages shows a high degree of imbalance (appendix table B.4), as was observed in the Araihazar study area, highlighting the importance of our identification strategy of using

[14]We chose 60μg as the cutoff to reflect the 50μg 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 50μg in 1999. Relatively constant groundwater Arsenic concentrations have been reported in a number of time series studies (Van Geen et al., 2002). Since our survey data on the history of shallow tubewell use indicate a tendency to underreport use of highly contaminated wells, we deem "measured contamination" to be more conservative than reported contamination. Our results are similar, though noisier, when using reported contamination (appendix table B.3 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).

[15]Similarly, none of the 19 variables is significantly correlated with the continuous arsenic measure after controlling for village fixed effects.

within-village, rather than between village, spatial variation in arsenic concentrations.

3.3 Estimating equations

While we employ a difference-in-difference methodology to estimate the impact of abandoning shallow tubewells for both child and elderly mortality, we highlight important differences in the estimating equations. For children, we exploit between-child variation in the share of life (out of one, two, or five years) lived after the campaign 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 within which to calculate a share of post-campaign exposure; furthermore, the cumulative effect of arsenic exposure acts with an undetermined lag. For adults, we thus exploit variation both between adults and within an adult's lifetime by testing whether the *probability* of death among adults age 50 or older rises more for those encouraged to abandon wells following the campaign: in other words, we employ a Cox-hazard model in which the coefficient of interest is the hazard ratio of dying.

Specifically, 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 in a household exposed to arsenic above 60μg 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 as a result of 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 if born before 2000.[16] 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)).

[16]The 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.

Our coefficient of interest is β, the coefficient on the interaction between high concentration and exposure. Our identifying assumption is that no other events during the period differentially affect infant and child mortality rates for households that were (or were not) encouraged to stop using shallow tubewells. 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.[17]

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

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

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 60μg 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 both 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 both overall mortality and disaggregated into water- and arsenic-related mortality. We then test for heterogeneity by distance to alternative clean water sources, in the form of deep tubewells.

[17] We also show 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.

4.1 Time trends

Figure 2 presents the raw, unadjusted averages for under one year mortality between 1980 and 2007 by contamination status. Infant mortality trends in households with arsenic largely track those of households with clean wells until 1999, when they diverge sharply as infant mortality rises among individuals in households encouraged to abandon contaminated wells. 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 increased construction and accessibility of deep tubewells in Barisal. We find similar results for two-year and five-year mortality (appendix figures A.2 and A.3).[18]

Figure 2: 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 in each four-year period, which are plotted as cubic splines for smoothness. High contamination households defined as those with tubewells that contain arsenic contamination greater than 60μg 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.

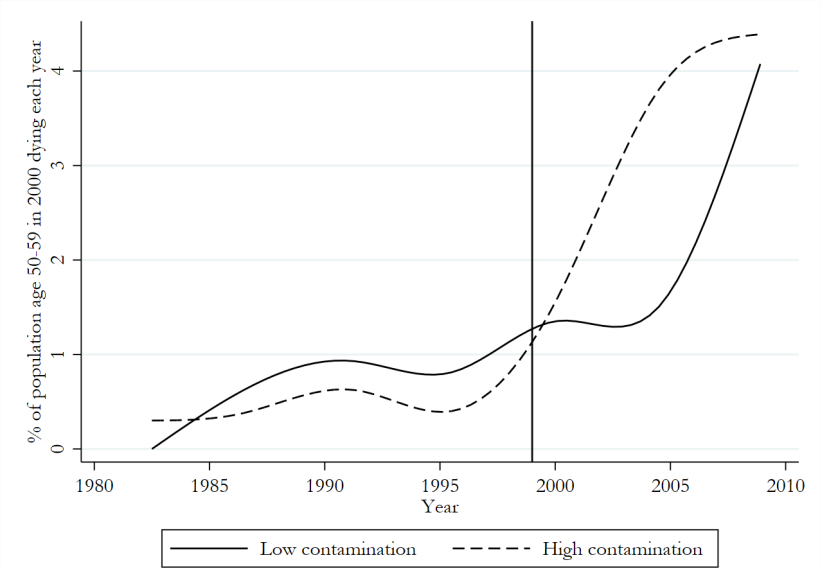
A parallel pattern is observed for mortality in older adults. Figure 3 plots the mortality rates of all adults age 50-59 in 2000 (born between 1941 and 1950) from 1980 onward[19]

[18] We observe a slight reversal in child mortality both among high- and low-contamination households for two-year and five-year mortality. These differences could be due both to mismeasurement of contamination status and low-contamination households switching water sources because of fear of arsenic.

[19] We do not observe any deaths after 2011 and exclude 2011 from the graph as the mortality outcomes are censored for households surveyed in 2011 but not 2016.

(i.e., the mortality every year among all individuals still alive at the beginning of that year). 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. As with childhood mortality, there is evidence that mortality begins to converge in later years in tandem with increased deep tubewell construction across the region.

Figure 3: 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 that are age predicted to be 50-59 years old in 2000 (the vulnerable age range) and alive at the beginning of a year that die in that year, plotted as cubic splines for smoothness. High contamination households defined as those with tubewells that contain arsenic contamination greater than 60μg according to field tests of the shallow tubewells closest to the residence. The figure shows the mean death rates across all high- and low-contamination households. $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

4.2 The effect of abandoning shallow tubewells on mortality

Table 1 presents the regression results from equation 1 for infant, under two, and under five mortality. Consistent with figure 2, the coefficient estimates indicate a substantial and statistically significant increase in mortality after 2000 among individuals with arsenic-contaminated tubewells. 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).[20]

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	-0.001	-0.002	0.006	0.006	0.004	-0.002	-0.002	-0.006
	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.009)	(0.009)	(0.009)
Exposure	-0.001			0.009			0.036**		
	(0.009)			(0.011)			(0.016)		
High con. * Exposure	0.024**	0.024**	0.025**	0.024**	0.024**	0.027**	0.033**	0.034**	0.039**
	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.015)	(0.015)	(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 an 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 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) water related (diarrhea and vomiting), 2) arsenic-related (spasms and pneumonia), and 3) not related to either water or arsenic (tetanus and accidents). The results on cause of death presented in table 2 suggest that the mortality effects we document are driven by water-related causes.

Table 3 presents the regression results from equation 2 for elderly mortality. Consistent with figure 3, we find no difference in the hazard of dying between contaminated and uncontaminated households prior to 2000. Post-2000, the hazard ratio of dying in households encouraged to switch water sources relative to those not encouraged to switch is 1.47 (column (3)): in other words, adults over 50 years of age in switching households are 47% more likely to die than their non-switching counterparts.[21]

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%) 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)).[22]

[20] The coefficient estimates are similar but noisier when including household fixed effects.

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

[22] We have very few adult cases of tetanus-related deaths or accidents.

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

	Water-related		Arsenic-related		Not water-/arsenic-related	
	Diarrhea	Vomiting	Spasms	Pneumonia	Tetanus	Accident
	(1)	(2)	(3)	(4)	(5)	(6)
High con.	-0.003	-0.006	-0.001	0.001	0.001	-0.000
	(0.005)	(0.004)	(0.004)	(0.005)	(0.003)	(0.003)
High con. * Exposure	0.011*	0.017**	0.004	-0.005	0.001	0.005
	(0.007)	(0.007)	(0.006)	(0.009)	(0.004)	(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.

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

	Any			Water	Arsenic
	(1)	(2)	(3)	(4)	(5)
High con.	0.853	0.841	0.838	0.498	1.251
	(0.143)	(0.154)	(0.156)	(0.301)	(0.758)
Post-2000	3.681***	2.947***	2.928***	7.193***	7.480***
	(0.612)	(0.484)	(0.476)	(3.407)	(3.842)
High con. * Post-2000	1.425**	1.444**	1.468**	2.600*	0.739
	(0.239)	(0.259)	(0.270)	(1.387)	(0.415)
Mean(Low con. & Before campaign)	0.048	0.048	0.048	0.009	0.011
Observations	45555	45555	45555	45555	45555
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 an arsenic contamination greater than 60μg according to field tests conducted in 2009. Post-2000 is an indicator that is 1 in 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 adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

In total, 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).

4.3 Heterogeneity by distance

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 greater construction and the health benefits of greater proximity. Our results thus far suggest a large mortality differential between having water infrastructure that is very local (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 distance to water source 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 have the same access to alternative water sources. Our identifying assumption is that households with the same number of deep tubewells within (ex.) 500m only experience a differential change in mortality as a result of having to shift away from their backyard tubewell due to differential arsenic concentration.

To perform this exercise, we use data collected in 2016 on the GPS location and age of all deep tubewells in our study area. We present summary statistics in appendix tables B.5 and B.6, which suggest that the construction of deep tubewells was much faster in villages with high arsenic levels. 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 a triple-difference version of equation 1 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 mortality of having an additional deep tubewell within a given radius for those encouraged to abandon their shallow tubewells.

We estimate the effect of having an additional deep tubewell on water-related deaths for a range of distance values. As a placebo test, we also estimate the impact on arsenic and non-water related deaths.

Table 4: Child mortality: effects by exposure to deep tubewells close to the residence; child died < 60 months from ...

	Water-related									Arsenic-related	Non-water-arsenic
Tubewells within (meters):	300	400	500	600	700	800	900	1000	500	500	500
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
High con.	-0.007	-0.009	-0.009	-0.010	-0.009	-0.009	-0.010	-0.010		-0.001	-0.000
	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.007)		(0.006)	(0.005)
High con. * Exposure	0.028***	0.029***	0.028**	0.030***	0.028**	0.027**	0.025**	0.025**	0.030**	-0.004	0.003
	(0.010)	(0.011)	(0.011)	(0.011)	(0.012)	(0.012)	(0.012)	(0.012)	(0.013)	(0.013)	(0.008)
# Tubewells	-0.008	-0.005	-0.003	-0.003	-0.002	-0.002	-0.002	-0.002	-0.002	0.003	0.000
	(0.005)	(0.004)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.002)	(0.005)	(0.004)	(0.002)
High con. * # Tubewells	0.011*	0.012**	0.008**	0.007**	0.005**	0.004*	0.003*	0.003*	0.003	0.001	0.001
	(0.006)	(0.005)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.001)	(0.005)	(0.004)	(0.003)
Exposure * # Tubewells	0.021**	0.014**	0.008**	0.008***	0.005**	0.004*	0.003*	0.003*	0.008*	0.002	0.001
	(0.010)	(0.006)	(0.004)	(0.003)	(0.002)	(0.002)	(0.002)	(0.002)	(0.005)	(0.005)	(0.003)
High con. * Exposure * # Tubewells	-0.025**	-0.020***	-0.013***	-0.011***	-0.008**	-0.006**	-0.005**	-0.004**	-0.012**	-0.001	0.000
	(0.011)	(0.007)	(0.005)	(0.004)	(0.003)	(0.003)	(0.002)	(0.002)	(0.005)	(0.005)	(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. # 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 as well as distance to the village center to ensure that distance to tubewells does not only proxy income or location. All regressions adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table 4 indicates that having access to an additional deep tubewell within 300 meters of the residence reduces water related child mortality by 2.5 percentage points (column (1)), fully offsetting the impact of being encouraged to abandon the closest shallow tubewell.[23] As to be expected, the water-related mortality benefits of deep tubewells decrease linearly with distance (columns(2)-(9)). Accordingly, the combined effect of $(HighCon \times Exposure) + (HighCon \times Exposure \times \#Tubewells)$ is significantly different from zero for a tubewell distance of 1000m but not 300m (although it should be noted that the coefficients on 300m v.s. 1000m are not significantly different). By contrast, we find no significant coefficient on the triple interaction in either of our placebo estimations (columns (11)-(12)): neither 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 is 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 re-estimate the effect of deep tubewell construction on water-related deaths with the inclusion of household fixed effects (column (11)). Estimates remain essentially unchanged, with strong and significant reductions on mortality *within* households from one additional deep tubewell. While table 4 only shows this result for the 500m category, appendix table B.7 imposes household fixed effects for each distance category, and again finds that the value of an additional deep tubewell declines as distance to the deep tubewell increases. However, it should be noted again that the coefficients on 300 meters v.s. 1000 meters are not significantly different. Meanwhile, we find no within-household effects for non-water (appendix table B.8) or arsenic-related (appendix table B.9) 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 distance grows, an additional deep tubewell yields

[23] The 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 $HighCon \times Exposure \times \#Tubewells$.

smaller and smaller reductions in the risk of dying among those encouraged to switch.[24]

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

	Any							
Tubewells within (meters):	300	400	500	600	700	800	900	1000
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
High con.	0.788	0.854	0.843	0.850	0.869	0.902	0.883	0.877
	(0.167)	(0.182)	(0.182)	(0.185)	(0.190)	(0.204)	(0.200)	(0.201)
Post-2000	2.415***	2.375***	2.351***	2.230***	2.248***	2.216***	2.267***	2.408***
	(0.420)	(0.435)	(0.425)	(0.406)	(0.406)	(0.423)	(0.440)	(0.489)
High con. * Post-2000	1.754***	1.722**	1.830***	1.885***	1.783***	1.634**	1.654**	1.596**
	(0.373)	(0.378)	(0.402)	(0.422)	(0.393)	(0.371)	(0.374)	(0.375)
# Tubewells	0.671*	0.869	0.935	0.964	0.976	0.980	0.980	0.986
	(0.146)	(0.123)	(0.087)	(0.064)	(0.053)	(0.047)	(0.041)	(0.040)
High con. * # Tubewells	1.713**	1.174	1.131	1.090	1.055	1.025	1.027	1.029
	(0.414)	(0.195)	(0.125)	(0.087)	(0.073)	(0.063)	(0.052)	(0.047)
Post-2000 * # Tubewells	1.641**	1.238	1.153	1.123*	1.090	1.070	1.057	1.040
	(0.354)	(0.172)	(0.107)	(0.075)	(0.060)	(0.051)	(0.043)	(0.040)
High con. * Post-2000 * # Tubewells	0.549**	0.798	0.830*	0.861*	0.904	0.946	0.950	0.962
	(0.132)	(0.132)	(0.092)	(0.068)	(0.062)	(0.057)	(0.047)	(0.044)
Mean(Low con. & Before campaign)	0.048	0.048	0.048	0.048	0.048	0.048	0.048	0.048
Observations	42123	42123	42123	42123	42123	42123	42123	42123
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 an arsenic contamination greater than 60μg 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 in 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 as well as distance to the village center in the tubewell regressions to ensure that distance to tubewells does not only proxy income or location. All regressions adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

4.4 Robustness checks

Our estimates are robust to a number of alternative specifications as detailed in the appendix. For infant and child mortality, using the extended set of controls (appendix table B.10), and replacing early life exposure with the number of years exposed (appendix table B.11) or a binary measure equal to 1 if the child was born in or after 2000 (appendix table B.12) produce very similar results. Using reported as opposed to measured contamination (appendix table B.13) or a 50μg cutoff (appendix table B.14) 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.15 and B.16) produce very similar results. Finally, we estimate the hazard of dying among

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

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.17). Consistent with the difference-in-difference estimations, we find no difference between contaminated and uncontaminated households in the hazard of a child dying prior to 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) and statistically significant. 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 60μg and estimate a placebo check in which we test whether an imaginary cutoff point of 100μg produces similar patterns within the subsample of households that we know were *all* encouraged to abandon shallow tubewells (appendix table B.18). If the level of arsenic contamination in groundwater is correlated with unobservable characteristics of the household 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 100μg relative to those between 60 and 100μg.

Finally, we explore several alternative explanations for the increase in 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.19). Second, we consider whether alerting families to switch water sources may have led to a larger migration of health-conscious households out of properties identified as being contaminated with arsenic. However, we do not find any differences in migration, as measured by number of years lived in the house, by contamination status (appendix table B.3).

For elderly mortality, we find smaller but significant effects when expanding the sample to adults age 40+ (appendix table B.20) and smaller and insignificant effects when expanding the sample to adults age 30+ (appendix table B.21), suggesting that the mor-

tality 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 in the data-intensive estimation with village-specific baseline hazards (appendix table B.22) or when excluding all person-years before the individual moved into the current residence (appendix table B.23). 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.24). Finally, we estimate a linear probability model for adults as similar in spirit 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.25).

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.26), 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.27).

5 Conclusion

We exploit the sudden abandonment of a large number of shallow tubewells in Bangladesh to identify the protective effects of convenient pathogen-free water relative to more distant alternatives. Infant, child and elderly mortality rise significantly among households who were encouraged to abandon nearby shallow tubewells and were thus forced to walk further to access alternative sources. We document these mortality effects despite the fact that the vast majority of households in our sample (81%) had an alternative pathogen-free source in the form of a deep tubewell within 1km of their home. Underscoring the importance of proximity, we find that having a deep tubewell within 300 meters (a 3-4 minute walk) fully 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.

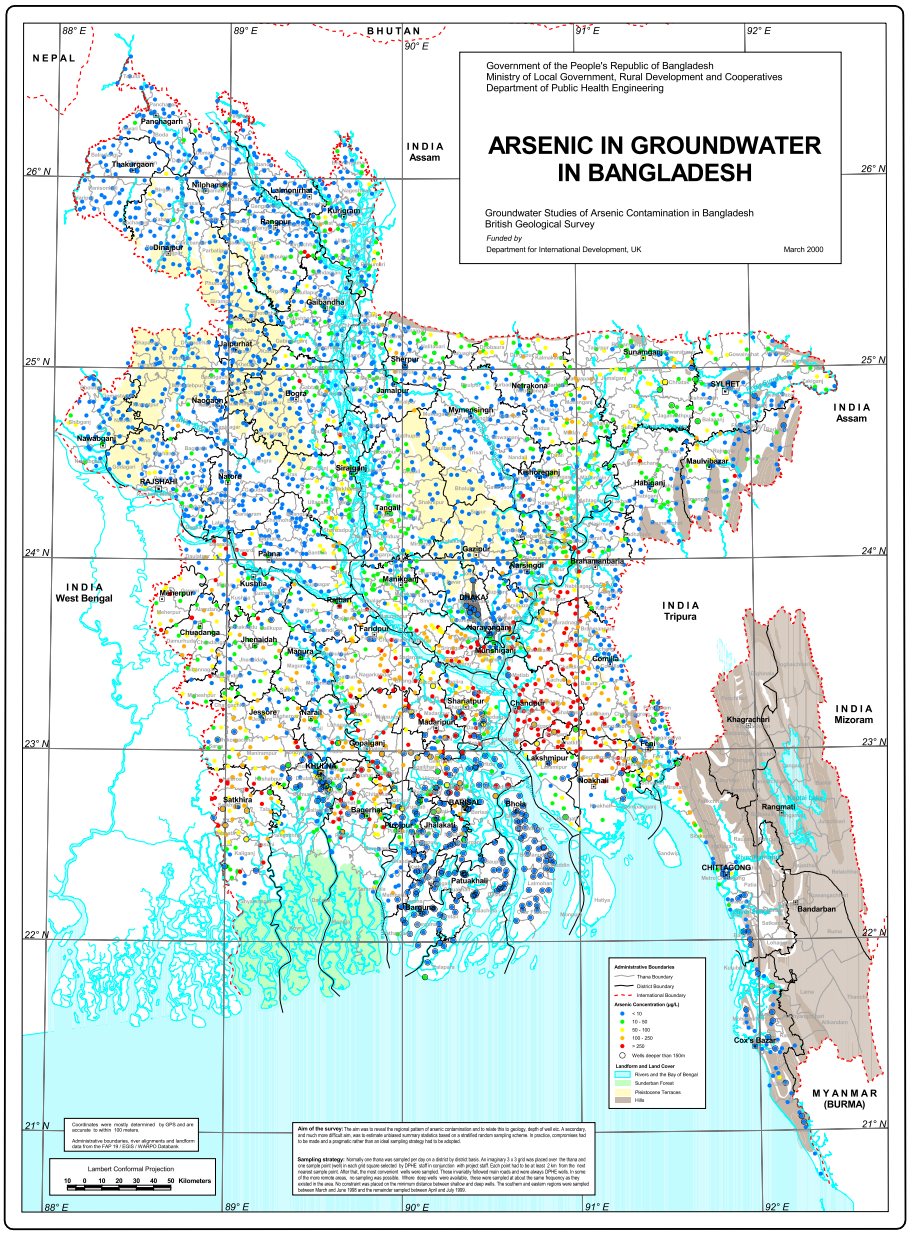
Our results strengthen the limited evidence that clean water infrastructure may generate significant mortality reductions among infants and children in vulnerable contexts, and provides new evidence of concomitant protective effects on the lifespan of the elderly. Beyond access alone, they demonstrate 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 further away water sources. We find relatively little evidence of large negative effects of arsenic consumption on [short-term] mortality, though we encourage caution in interpreting the magnitude of this finding, as recent studies have suggested that arsenic exposure has impacts on cognition and later-life income.

From a policy perspective, the results of this study 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 that are 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 in order 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 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.

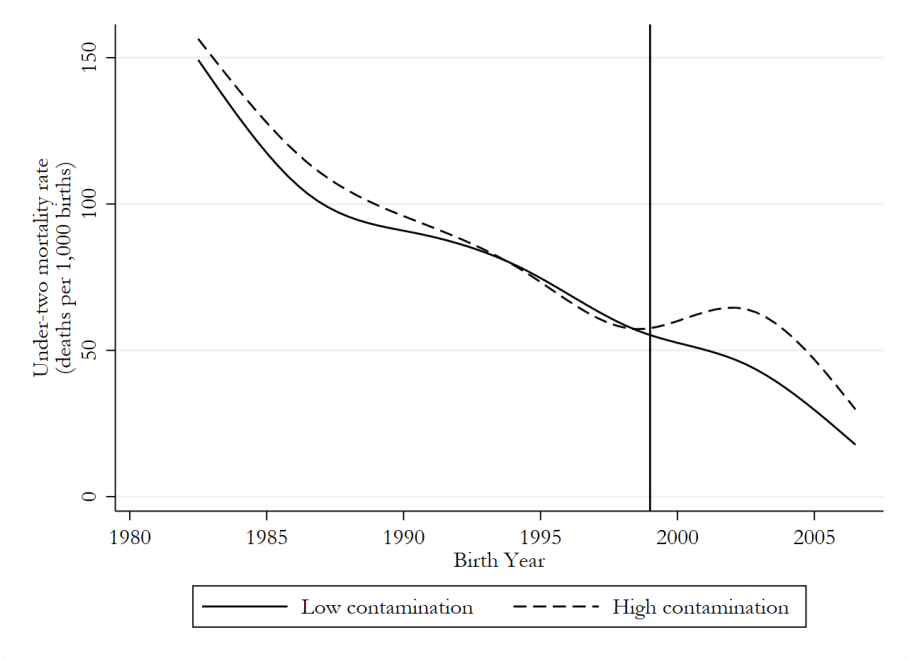
A Appendix figures

Figure A.1: Union-level arsenic contamination in Bangladesh



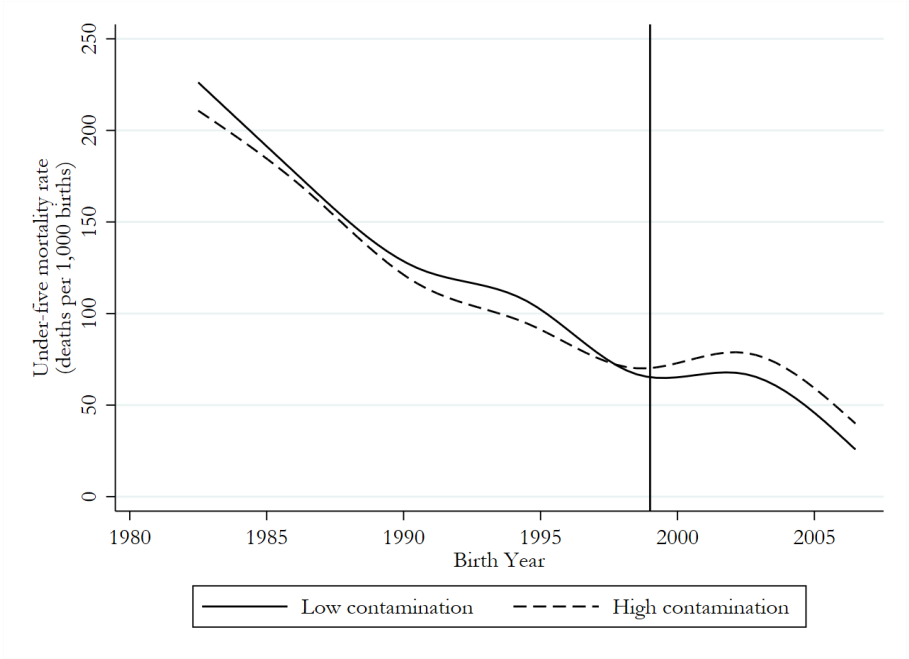
Notes: Source: Smedley (2000).

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



Notes: Data from our 2007 data collection and 2009 tubewell tests. "Under-two mortality rate" is deaths between 0 and 24 months of age per 1,000 births observed in each four-year period, which are plotted as cubic splines for smoothness. High contamination households defined as those with tubewells that contain arsenic contamination greater than 60μg 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.

Figure A.3: Under 5 mortality rate (0−5 yrs), four-year unadjusted averages



Notes: Data from our 2007 data collection and 2009 tubewell tests. "Under 5 mortality rate" is deaths between 0 and 60 months of age per 1,000 births observed in each four-year period, which are plotted as cubic splines for smoothness. High contamination households defined as those with tubewells that contain arsenic contamination greater than 60μg 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

	Survey Data		DHS Data			
VARIABLE	Mean	N	Mean	N	Diff.	P-value
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

	Survey Data		DHS data			
VARIABLE	Mean	N	Mean	N	Diff.	P-value
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 in the 2007 DHS

Panel I: Exogenous Variables

	Barisal		Rest of Bangladesh			
VARIABLE	Mean	N	Mean	N	Diff.	P-value
Age of mother	35.76	327	36.62	2,193	-0.86	0.028
Age of mother at earliest birth	16.85	327	17.19	2,193	-0.34	0.039
Education of mother	3.48	326	2.75	2,190	0.74	0.021
Education of father	4.67	326	4.00	2,192	0.67	0.075
Land size (acres)	0.82	327	0.76	2,179	0.06	0.852
Number of rooms in house	2.72	327	2.29	2,193	0.43	0.000
Electricity	0.28	310	0.51	2,113	-0.23	0.000
Muslim	0.87	327	0.91	2,193	-0.04	0.300
Years lived in house	23.00	310	23.84	2,116	-0.84	0.163
Head of household works in agriculture	0.28	327	0.31	2,193	-0.03	0.425
Head of household works in business	0.20	327	0.24	2,193	-0.03	0.274
F-test combined sample means						4.565

Panel II: Endogenous Variables

	Barisal		Rest of Bangladesh			
VARIABLE	Mean	N	Mean	N	Diff.	P-value
Number of births	5.11	1,130	5.22	7,892	-0.11	0.611
Mean birth interval	3.10	1,130	3.20	7,892	-0.10	0.171
Fraction of deaths under 12 mos	0.09	1,130	0.08	7,892	0.00	0.697
Fraction of deaths under 24 mos	0.11	1,129	0.09	7,892	0.01	0.217
Fraction of deaths under 60 mos	0.13	1,129	0.12	7,889	0.01	0.442
F-test combined sample means						2.384

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 sample 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.3: Sample means by measured contamination

Panel I: Exogenous Variables

	High Contamination		Low Contamination			
VARIABLE	Mean	N	Mean	N	Diff.	P-value
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

	High Contamination		Low Contamination			
VARIABLE	Mean	N	Mean	N	Diff.	P-value
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.4: Sample means by measured contamination, no village fixed effects

Panel I: Exogenous Variables

	High Contamination		Low Contamination			
VARIABLE	Mean	N	Mean	N	Diff.	P-value
Age of mother	42.10	2,512	41.85	1,316	0.25	0.532
Age of mother at earliest birth	18.18	1,560	18.48	852	-0.30	0.116
Education of mother	3.53	2,508	3.08	1,316	0.45	0.003
Education of father	4.82	2,462	4.14	1,293	0.68	0.001
Solvency	0.69	2,543	0.69	1,333	-0.00	0.921
Land size (arces)	0.82	2,521	0.81	1,322	0.01	0.914
Number of rooms in house	2.81	2,537	2.73	1,331	0.08	0.129
Electricity	0.45	2,541	0.32	1,332	0.13	0.000
Muslim	0.96	2,542	0.97	1,333	-0.01	0.402
Fraction of children living in household	0.73	2,515	0.73	1,318	-0.00	0.719
Respondent's age	41.63	2,542	41.10	1,333	0.54	0.248
Male respondent	0.18	2,543	0.16	1,333	0.01	0.480
Sufficiency of food per week	0.93	2,543	0.93	1,333	-0.01	0.614
Outstanding loan	0.56	2,534	0.52	1,332	0.04	0.089
Years lived in house	27.75	2,543	25.67	1,333	2.08	0.002
Years lived in village	31.66	2,044	30.31	1,074	1.35	0.073
Mean monthly income of household ($)	58.04	2,543	54.68	1,333	3.36	0.132
Head of household works in agriculture	0.41	2,470	0.47	1,296	-0.06	0.013
Head of household works in business	0.16	2,470	0.14	1,296	0.02	0.303
F-test combined sample means						2.250

Panel II: Endogenous Variables

	High Contamination		Low Contamination			
VARIABLE	Mean	N	Mean	N	Diff.	P-value
Arsenic concentration (ppb)	95.77	2,267	28.89	1,278	66.89	0.000
High reported concentration	0.89	2,543	0.41	1,333	0.47	0.000
Number of births	6.14	2,535	6.20	1,331	-0.06	0.651
Mean birth interval	5.22	2,525	5.04	1,319	0.18	0.397
Fraction of deaths under 12 mos	0.06	2,525	0.05	1,325	0.01	0.043
Fraction of deaths under 24 mos	0.07	2,516	0.06	1,318	0.01	0.048
Fraction of deaths under 60 mos	0.10	2,479	0.09	1,280	0.01	0.434
Deep tubewell $\leq$ 500 meters	0.56	2,418	0.48	1,247	0.08	0.000
Number of drinking sources used	1.55	2,543	1.72	1,333	-0.17	0.004
Closest well tested	0.79	2,104	0.78	1,171	0.01	0.667
Closest well painted	0.68	2,301	0.66	1,267	0.02	0.501
Value of house ($)	40.78	2,540	34.59	1,332	6.19	0.004
F-test combined sample means						97.368

Notes: The table shows results from OLS regressions with Huber-White robust SEs clustered at the village level. *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.5: 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.6: Sample means: deep tubewell data, by village contamination

	High Contamination		Low Contamination			
VARIABLE	Mean	N	Mean	N	Diff.	P-value
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 60μg (results are very similar using 50μg as a cutoff or at least 50% or 60% of households with a level of above 60μg). *Quasi-public tubewells* include public wells as well as private wells used by other households in the village.

Table B.7: Child mortality: effects by exposure to deep tubewells close to the residence; child died < 60 months from water-related cause, including household fixed effects

Tubewells within (meters):	300	400	500	600	700	800	900	1000
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
High con. * Exposure	0.028**	0.031**	0.030**	0.034**	0.028*	0.026*	0.022	0.021
	(0.014)	(0.014)	(0.013)	(0.014)	(0.014)	(0.015)	(0.015)	(0.015)
# Tubewells	0.006	0.001	-0.002	-0.001	-0.005	-0.004	-0.003	-0.003
	(0.007)	(0.005)	(0.005)	(0.004)	(0.004)	(0.003)	(0.003)	(0.002)
High con. * # Tubewells	-0.010	-0.000	0.003	0.002	0.005	0.003	0.003	0.004
	(0.008)	(0.006)	(0.005)	(0.004)	(0.004)	(0.003)	(0.003)	(0.002)
Exposure * # Tubewells	0.014	0.012*	0.008*	0.007**	0.007***	0.005**	0.003*	0.003*
	(0.010)	(0.007)	(0.005)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)
High con. * Exposure * # Tubewells	-0.016	-0.016**	-0.012**	-0.010***	-0.009***	-0.007**	-0.005**	-0.005**
	(0.011)	(0.008)	(0.005)	(0.004)	(0.003)	(0.003)	(0.002)	(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.8: 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	1000	500
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
High con.	0.000	-0.001	-0.000	0.000	-0.001	0.000	-0.000	-0.000	
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	
High con. * Exposure	0.003	0.000	0.003	0.002	0.003	-0.001	-0.001	0.001	0.010
	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.009)	(0.009)	(0.010)
# Tubewells	-0.003	-0.001	0.000	0.001	0.000	0.002	0.001	0.001	-0.003
	(0.003)	(0.003)	(0.002)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.004)
High con. * # Tubewells	0.001	0.003	0.001	0.001	0.001	0.000	0.000	0.000	0.003
	(0.004)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.001)	(0.001)	(0.005)
Exposure * # Tubewells	0.005	0.003	0.001	0.001	0.000	-0.001	-0.001	-0.000	0.002
	(0.004)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.002)	(0.001)	(0.003)
High con. * Exposure * # Tubewells	0.001	0.001	0.000	0.000	-0.000	0.001	0.001	0.000	-0.002
	(0.006)	(0.005)	(0.004)	(0.003)	(0.002)	(0.002)	(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 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). # 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 as well as distance to the village center to ensure that distance to tubewells does not only proxy income or location. All regressions adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.9: Child mortality: effects by exposure to deep tubewells close to the residence; child died < 60 months from arsenic related cause.

Tubewells within (meters):	300	400	500	600	700	800	900	1000	500
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
High con.	0.001	-0.002	-0.001	-0.001	0.000	-0.001	-0.001	0.000	
	(0.006)	(0.006)	(0.006)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	
High con. * Exposure	-0.011	-0.007	-0.004	-0.002	-0.004	-0.004	-0.000	-0.000	0.003
	(0.012)	(0.013)	(0.013)	(0.014)	(0.013)	(0.014)	(0.014)	(0.014)	(0.016)
# Tubewells	0.004	0.000	0.003	0.003	0.004	0.001	0.001	0.001	0.004
	(0.007)	(0.004)	(0.004)	(0.003)	(0.003)	(0.003)	(0.002)	(0.002)	(0.007)
High con. * # Tubewells	-0.005	0.002	0.001	0.000	-0.001	0.000	-0.000	-0.000	-0.004
	(0.007)	(0.005)	(0.004)	(0.004)	(0.003)	(0.003)	(0.002)	(0.002)	(0.007)
Exposure * # Tubewells	-0.001	0.005	0.002	0.001	-0.000	0.001	0.002	0.002	0.002
	(0.010)	(0.006)	(0.005)	(0.004)	(0.004)	(0.004)	(0.003)	(0.003)	(0.005)
High con. * Exposure * # Tubewells	0.010	-0.001	-0.001	-0.002	-0.000	-0.001	-0.001	-0.001	-0.002
	(0.011)	(0.007)	(0.005)	(0.005)	(0.004)	(0.004)	(0.003)	(0.003)	(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 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). # 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 as well as distance to the village center to ensure that distance to tubewells does not only proxy income or location. All regressions adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.10: 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	-0.001	-0.002	0.006	0.006	0.004	-0.002	-0.002	-0.007
	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.009)	(0.009)	(0.009)
Exposure	-0.001			0.009			0.036**		
	(0.009)			(0.011)			(0.016)		
High con. * Exposure	0.024**	0.024**	0.026**	0.024**	0.024**	0.027**	0.033**	0.034**	0.041***
	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.015)	(0.015)	(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 an 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 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 adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.11: 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	-0.001	-0.002	0.006	0.006	0.004	-0.002	-0.002	-0.006
	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.009)	(0.009)	(0.009)
Exposure	-0.001			0.004			0.007**		
	(0.009)			(0.006)			(0.003)		
High con. * Exposure	0.024**	0.024**	0.025**	0.012**	0.012**	0.013**	0.007**	0.007**	0.008**
	(0.011)	(0.011)	(0.011)	(0.006)	(0.006)	(0.006)	(0.003)	(0.003)	(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 an arsenic contamination greater than $60\mu g$ 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 adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.12: 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	-0.001	-0.002	0.007	0.007	0.006	0.003	0.002	-0.000
	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
Exposure	-0.001			0.009			0.029*		
	(0.009)			(0.010)			(0.015)		
High con. * Exposure	0.024**	0.024**	0.025**	0.021*	0.021*	0.022*	0.027	0.026	0.028
	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.017)	(0.017)	(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 an arsenic contamination greater than 60μg according to field tests conducted in 2009. Exposure is 1 if 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 adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.13: 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	0.002	0.003	0.004	0.005	0.005	-0.008	-0.008	-0.008
	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.010)	(0.010)	(0.011)
Exposure	0.002			0.015			0.033*		
	(0.010)			(0.013)			(0.019)		
High con. * Exposure	0.019*	0.019*	0.018*	0.013	0.013	0.013	0.035**	0.036**	0.036**
	(0.010)	(0.010)	(0.010)	(0.012)	(0.012)	(0.012)	(0.017)	(0.017)	(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 an 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 adjusted for time trends (birth year).
$p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.14: Child mortality: 50μg contamination cutoff

	Death < 12 months			Death < 24 months			Death < 60 months		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
High con.	0.001	0.001	-0.000	0.009	0.009	0.007	-0.000	-0.001	-0.004
	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.010)	(0.010)	(0.010)
Exposure	0.002			0.008			0.030*		
	(0.010)			(0.012)			(0.017)		
High con. * Exposure	0.019*	0.019*	0.020*	0.024**	0.025**	0.027**	0.039**	0.040**	0.045***
	(0.010)	(0.010)	(0.011)	(0.011)	(0.011)	(0.012)	(0.017)	(0.017)	(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 an arsenic contamination greater than 50μg 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 adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.15: 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	-0.003	-0.004	0.005	0.005	0.003	-0.003	-0.003	-0.007
	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.010)	(0.010)	(0.009)
Exposure	-0.006			0.013			0.034**		
	(0.009)			(0.011)			(0.017)		
High con. * Exposure	0.028***	0.028***	0.030***	0.023**	0.023**	0.026**	0.030**	0.031**	0.036**
	(0.009)	(0.009)	(0.009)	(0.010)	(0.010)	(0.011)	(0.014)	(0.014)	(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 an 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 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 adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.16: 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	0.001	0.000	0.008	0.008	0.007	-0.000	-0.000	-0.003
	(0.007)	(0.007)	(0.006)	(0.007)	(0.007)	(0.007)	(0.009)	(0.009)	(0.009)
Exposure	0.000			0.008			0.044**		
	(0.009)			(0.011)			(0.018)		
High con. * Exposure	0.022*	0.022*	0.023*	0.022	0.021	0.023*	0.045**	0.045**	0.049**
	(0.012)	(0.012)	(0.012)	(0.013)	(0.013)	(0.014)	(0.020)	(0.020)	(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 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 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 adjusted for time trends (birth year).
$p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.17: Child mortality: hazard of dying among children

	(1)	(2)	(3)
High con.	1.006	1.000	0.975
	(0.072)	(0.072)	(0.071)
Post-2000	0.862	0.586***	0.571***
	(0.133)	(0.107)	(0.105)
High con. * Post-2000	1.336*	1.349*	1.381**
	(0.215)	(0.218)	(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 an arsenic contamination greater than 60μg according to field tests conducted in 2009. Post-2000 is an indicator that is 1 in 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 adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.18: Child mortality: placebo test, 100μg contamination cutoff

	Death < 12 months			Death < 24 months			Death < 60 months		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
High con.	-0.007	-0.007	-0.008	-0.012	-0.012	-0.013	-0.012	-0.012	-0.012
	(0.008)	(0.008)	(0.008)	(0.009)	(0.009)	(0.009)	(0.012)	(0.012)	(0.012)
Exposure	0.030**			0.040***			0.063***		
	(0.013)			(0.015)			(0.022)		
High con. * Exposure	-0.005	-0.005	-0.006	-0.004	-0.004	-0.006	0.018	0.017	0.018
	(0.015)	(0.015)	(0.015)	(0.017)	(0.017)	(0.017)	(0.021)	(0.022)	(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 an arsenic contamination greater than 60μg 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 an arsenic contamination greater than 100μg 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 adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.19: 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**			0.044**			0.059**		
	(0.015)			(0.017)			(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 an arsenic contamination greater than 60μg according to field tests conducted in 2009). Arsenic is the continuous Arsenic contamination inμg. 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 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 40+

	(1)	(2)	(3)
High con.	0.890	0.886	0.883
	(0.137)	(0.144)	(0.144)
Post-2000	3.043***	2.644***	2.620***
	(0.469)	(0.416)	(0.410)
High con. * Post-2000	1.322*	1.327*	1.361*
	(0.201)	(0.210)	(0.219)
Mean(Low con. & Before campaign)	0.032	0.032	0.032
Observations	75807	75807	75807
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 sample includes all person-years age 40 or higher. High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains an arsenic contamination greater than 60μg according to field tests conducted in 2009. Post-2000 is an indicator that is 1 in years 2000 to 2017. Controls include the adult's gender and the education of the household head and his spouse. All regressions adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.21: Elderly mortality: hazard of dying among adults age 30+

	(1)	(2)	(3)
High con.	0.951	0.962	0.956
	(0.139)	(0.150)	(0.151)
Post-2000	2.793***	2.415***	2.400***
	(0.411)	(0.358)	(0.357)
High con. * Post-2000	1.223	1.212	1.238
	(0.181)	(0.186)	(0.193)
Mean(Low con. & Before campaign)	0.024	0.024	0.024
Observations	115450	115450	115450
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 sample includes all person-years age 30 or higher. High contamination is an indicator that is 1 if the shallow tubewell closest to the residence contains an arsenic contamination greater than 60μg according to field tests conducted in 2009. Post-2000 is an indicator that is 1 in years 2000 to 2017. Controls include the adult's gender and the education of the household head and his spouse. All regressions 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+, village-specific baseline hazards

	(1)	(2)	(3)
High con.	0.786	0.759	0.747
	(0.128)	(0.135)	(0.135)
Post-2000	3.223***	2.650***	2.628***
	(0.563)	(0.462)	(0.464)
High con. * Post-2000	1.586***	1.664***	1.695***
	(0.271)	(0.308)	(0.314)
Mean(Low con. & Before campaign)	0.048	0.048	0.048
Observations	45555	45555	45555
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 an arsenic contamination greater than 60μg according to field tests conducted in 2009. Stratified estimations allow the baseline hazard function to differ by village. Controls include the adult's gender and the education of the household head and his spouse. All regressions adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.23: Elderly mortality: hazard of dying among adults age 50+, excluding years before the household moved into the current location

	(1)	(2)	(3)
High con.	0.828	0.866	0.823
	(0.181)	(0.181)	(0.171)
Post-2000	3.250***	3.229***	3.029***
	(0.606)	(0.612)	(0.576)
High con. * Post-2000	1.561**	1.503**	1.577**
	(0.324)	(0.292)	(0.312)
Mean(Low con. & Before campaign)	0.048	0.048	0.048
Observations	30238	30238	30238
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 an arsenic contamination greater than 60μg according to field tests conducted in 2009. We exclude all person-years before a person moved into a given household. Controls include the adult's gender and the education of the household head and his spouse. All regressions adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.24: Elderly mortality: hazard of dying among adults age 50+, years 1980-2007

	(1)	(2)	(3)
High con.	0.874	0.885	0.861
	(0.149)	(0.166)	(0.163)
Post-2000	3.019***	2.700***	2.576***
	(0.458)	(0.450)	(0.434)
High con. * Post-2000	1.405**	1.408*	1.422*
	(0.240)	(0.256)	(0.265)
Mean(Low con. & Before campaign)	0.049	0.049	0.049
Observations	33520	33520	33520
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 an arsenic contamination greater than 60μg according to field tests conducted in 2009. Post-2000 is an indicator that is 1 in years 2000 to 2007. Controls include the adult's gender and the education of the household head and his spouse. All regressions adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.25: Elderly mortality: mortality under 80 among adults age 50+ from...

	Any			Water	Arsenic	Any							
Tubewells within (meters):						300	400	500	600	700	800	900	1000
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
High con.	0.034	0.065	0.074*	-0.007	0.013	-0.023	-0.026	-0.027	-0.031	-0.022	-0.039	-0.032	-0.024
	(0.046)	(0.041)	(0.042)	(0.041)	(0.031)	(0.050)	(0.053)	(0.058)	(0.054)	(0.053)	(0.055)	(0.053)	(0.053)
Exposure	1.523***												
	(0.262)												
High con. * Exposure	0.338**	0.262**	0.214*	0.160	-0.096	0.275	0.296	0.335	0.381	0.372*	0.381*	0.341	0.305
	(0.147)	(0.114)	(0.121)	(0.155)	(0.150)	(0.180)	(0.195)	(0.223)	(0.230)	(0.208)	(0.207)	(0.216)	(0.229)
# Tubewells						-0.068**	-0.047**	-0.035*	-0.020	-0.011	-0.013	-0.010	-0.006
						(0.032)	(0.023)	(0.018)	(0.014)	(0.010)	(0.009)	(0.007)	(0.007)
High con. * # Tubewells						0.068**	0.042*	0.034*	0.026*	0.017*	0.018**	0.013*	0.010
						(0.034)	(0.024)	(0.019)	(0.014)	(0.010)	(0.009)	(0.007)	(0.006)
Exposure * # Tubewells						0.067	0.031	0.078	0.066	0.044	0.036	0.024	0.016
						(0.114)	(0.080)	(0.067)	(0.049)	(0.030)	(0.023)	(0.021)	(0.022)
High con. * Exposure * # Tubewells						-0.083	-0.053	-0.072	-0.069	-0.049	-0.042	-0.026	-0.017
						(0.126)	(0.087)	(0.076)	(0.057)	(0.038)	(0.030)	(0.026)	(0.026)
Mean(Low	0.119	0.119	0.119	0.000	0.000	0.119	0.119	0.119	0.119	0.119	0.119	0.119	0.119
con.	746	741	741	741	741	597	597	597	597	597	597	597	597
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 age 50+ at the time of the survey or the time of death. We exclude all adults that have not yet have reached age 80 and for whom mortality is censored. We also exclude all adults that moved in 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 an 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 as well as distance to the village center in the tubewell regressions to ensure that distance to tubewells does not only proxy income or location. All regressions adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.26: Elderly mortality: hazard of dying among adults age 50+, placebo test

	(1)	(2)	(3)
High con.	0.825	0.863	0.891
	(0.179)	(0.205)	(0.218)
Post-2000	4.699***	3.634***	3.636***
	(0.997)	(0.779)	(0.760)
High con. * Post-2000	1.147	1.115	1.103
	(0.268)	(0.283)	(0.268)
Mean(Low con. & Before campaign)	0.028	0.028	0.028
Observations	26309	26309	26309
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. We restrict the sample to all years before 2000 (the year of the campaign) and define a hypothetical campaign year in 1990. 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 an arsenic contamination greater than 60μg according to field tests conducted in 2009. Post-1990 is an indicator that is 1 in years 1990 to 2000. Stratified estimations allow the baseline hazard function to differ by village. Controls include the adult's gender and the education of the household head and his spouse. All regressions adjusted for time trends (birth year). $p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

Table B.27: Elderly mortality: hazard of dying among adults age 50+, continuous arsenic contamination

	(1)	(2)	(3)
Arsenic	1.000	1.000	1.000
	(0.003)	(0.003)	(0.003)
Post-2000	5.544***	3.813***	4.013***
	(1.799)	(1.339)	(1.372)
Ars. * Post-2000	0.999	1.000	1.000
	(0.003)	(0.004)	(0.003)
Mean(Before campaign)	0.060	0.060	0.060
Observations	26309	26309	26309
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 high contamination households (the shallow tubewell closest to the residence contains an arsenic contamination greater than 60μg according to field tests conducted in 2009). Arsenic is the continuous Arsenic contamination inμg. The sample includes all person-years age 50 or higher. Post-2000 is an indicator that is 1 in years 2000 to 2017. Stratified estimations allow the baseline hazard function to differ by village. Controls include the adult's gender and the education of the household head and his spouse. All regressions adjusted for time trends (birth year).
$p < 0.10^{*}, p < 0.05^{**}, p < 0.01^{***}$.

References

Abdul, Khaja Shameem Mohammed, Sudheera Sammanthi Jayasinghe, Ediriweera PS Chandana, Channa Jayasumana, and P Mangala CS De Silva, "Arsenic and human health effects: A review," *Environmental toxicology and pharmacology*, 2015, *40* (3), 828–846.

Ahmed, Kazi Matin, Khan Hasan, Tanzeer Hasan, and Mahfuzur Khan, *Situation Analysis of Arsenic Mitigation 2009* 06 2010.

Ahmed, M Feroze, Satinder Ahuja, Mohammad Alauddin, Stephan J Hug, Jon R Lloyd, Alex Pfaff, Thomas Pichler, Chad Saltikov, Martin Stute, and Alexander van Geen, "Ensuring safe drinking water in Bangladesh," 2006.

Anderson, D Mark, Kerwin Kofi Charles, Daniel I Rees, and Tianyi Wang, "Water purification efforts and the black-white infant mortality gap, 1906–1938," *Journal of Urban Economics*, 2021, *122*, 103329.

Argos, Maria, Tara Kalra, Paul J Rathouz, Yu Chen, Brandon Pierce, Faruque Parvez, Tariqul Islam, Alauddin Ahmed, Muhammad Rakibuz-Zaman, Rabiul Hasan et al., "Arsenic exposure from drinking water, and all-cause and chronic-disease mortalities in Bangladesh (HEALS): a prospective cohort study," *The Lancet*, 2010, *376* (9737), 252–258.

Bangladesh Bureau of Statistics and Unicef, "Multiple Indicator Cluster Suvery 2006," 2006, p. 57.

BMOH, "National Policy for Arsenic Mitigation 2004," *Compendium of Environment Statistics of Bangladesh 2004*, 2004, (), 388–394.

Caldwell, Bruce, John Caldwell, S.N. Mitra, and Wayne Smith, "Tubewells and arsenic in Bangladesh: Challenging a public health success story," *International Journal of Population Geography*, 01 2003, *9*, 23 – 38.

_ , Wayne Smith, Kamalini Lokuge, Geetha Ranmuthugala, Keith Dear, Abul Milton, Malcolm Sim, Jack Ng, and S.N. Mitra, "Access to Drinking-water and Arsenicosis in Bangladesh," *Journal of health, population, and nutrition*, 10 2006, *24*, 336–45.

Chen, Yu, Joseph H Graziano, Faruque Parvez, Iftikhar Hussain, Hassina Momotaj, Alexander van Geen, Geoffrey R Howe, and Habibul Ahsan,

"Modification of risk of arsenic-induced skin lesions by sunlight exposure, smoking, and occupational exposures in Bangladesh," *Epidemiology*, 2006, *17* (4), 459–467.

Cutler, David and Grant Miller, "The role of public health improvements in health advances: the twentieth-century United States," *Demography*, 2005, *42* (1), 1–22.

Das, Sumonkanti, Hukum Chandra, and Unnati Rani Saha, "District level estimates and mapping of prevalence of diarrhoea among under-five children in Bangladesh by combining survey and census data," *PloS one*, 2019, *14* (2), e0211062.

Devoto, Florencia, Esther Duflo, Pascaline Dupas, William Parienté, and Vincent Pons, "Happiness on tap: Piped water adoption in urban Morocco," *American Economic Journal: Economic Policy*, 2012, *4* (4), 68–99.

Edmunds, WM, KM Ahmed, and PG Whitehead, "A review of arsenic and its impacts in groundwater of the Ganges–Brahmaputra–Meghna delta, Bangladesh," *Environmental Science: Processes & Impacts*, 2015, *17* (6), 1032–1046.

Esrey, Steven A, James B Potash, Leslie Roberts, and Clive Shiff, "Effects of improved water supply and sanitation on ascariasis, diarrhoea, dracunculiasis, hookworm infection, schistosomiasis, and trachoma.," *Bulletin of the World Health organization*, 1991, *69* (5), 609.

fei Lee, Lung, Mark R Rosenzweig, and Mark M Pitt, "The effects of improved nutrition, sanitation, and water quality on child health in high-mortality populations," *Journal of econometrics*, 1997, *77* (1), 209–235.

Galiani, Sebastian, Paul Gertler, and Ernesto Schargrodsky, "Water for life: The impact of the privatization of water services on child mortality," *Journal of political economy*, 2005, *113* (1), 83–120.

Geen, Alexander Van, Habibul Ahsan, Allan H Horneman, Ratan K Dhar, Yan Zheng, Iftikhhar Hussain, Kazi Matin Ahmed, Andrew Gelman, Martin Stute, H James Simpson et al., "Promotion of well-switching to mitigate the current arsenic crisis in Bangladesh," *Bulletin of the World Health Organization*, 2002, *80* (9), 732–737.

_ , Y Zheng, R Versteeg, M Stute, A Horneman, R Dhar, M Steckler, A Gelman, C Small, H Ahsan et al., "Spatial variability of arsenic in 6000 tube wells in a 25 km2 area of Bangladesh," *Water Resources Research*, 2003, *39* (5).

Goel, V, MS Islam, M Yunus, MT Ali, AF Khan, N Alam, ASG Faruque, G Bell, M Sobsey, and M Emch, "Deep tubewell microbial water quality and access in arsenic mitigation programs in rural Bangladesh," *Science of the total environment*, 2019, *659*, 1577–1584.

Hoque, BA, SR Huttly, KM Aziz, MY Patwary, and Richard G Feachem, "Tubewell water consumption and its determinants in a rural area of Bangladesh.," *The Journal of tropical medicine and hygiene*, 1989, *92* (3), 197–202.

Howard, Guy, M Feroze Ahmed, Abu Jafar Shamsuddin, Shamsul Gafur Mahmud, and Daniel Deere, "Risk assessment of arsenic mitigation options in Bangladesh," *Journal of Health, Population, and Nutrition*, 2006, *24* (3), 346.

Hug, Stephan J, Dominique Gaertner, Linda C Roberts, Mario Schirmer, Thomas Ruettimann, Thomas M Rosenberg, ABM Badruzzaman, and M Ashraf Ali, "Avoiding high concentrations of arsenic, manganese and salinity in deep tubewells in Munshiganj District, Bangladesh," *Applied Geochemistry*, 2011, *26* (7), 1077–1085.

Iqbal, Afrida Binth, Mohammad Moshiur Rahman, Dhiman Ranjan Mondal, Nadim Reza Khandaker, Haniyum Maria Khan, Gias Uddin Ahsan, Md Jakariya, and Md Mainul Hossain, "Assessment of Bangladesh groundwater for drinking and irrigation using weighted overlay analysis," *Groundwater for sustainable development*, 2020, *10*, 100312.

Jakariya, Md, "Arsenic in tubewell water of Bangladesh and approaches for sustainable mitigation." PhD dissertation, KTH 2007.

Jamison, Dean T, "Disease control priorities: improving health and reducing poverty," *The Lancet*, 2018, *391* (10125), e11–e14.

Johnston, Richard, Stephan J Hug, Jennifer Inauen, Nasreen I Khan, Hans-Joachim Mosler, and Hong Yang, "Enhancing arsenic mitigation in Bangladesh: Findings from institutional, psychological, and technical investigations," *Science of the Total Environment*, 2014, *488*, 477–483.

Keskin, Pinar, Gauri Kartini Shastry, and Helen Willis, "Water Quality Awareness and Breastfeeding: Evidence of Health Behavior Change in Bangladesh," *The Review of Economics and Statistics*, May 2017, *99* (2), 265–280.

Kinniburgh, P L Smedley D G, "Executive summary of the final report of Phase II, groundwater studies of arsenic contamination in Bangladesh," Technical Report 2001.

Kozul, Courtney D, Kenneth H Ely, Richard I Enelow, and Joshua W Hamilton, "Low-dose arsenic compromises the immune response to influenza A infection in vivo," *Environmental health perspectives*, 2009, *117* (9), 1441.

Kremer, Michael, Jessica Leino, Edward Miguel, and Alix Peterson Zwane, "Spring cleaning: Rural water impacts, valuation, and property rights institutions," *The Quarterly Journal of Economics*, 2011, *126* (1), 145–205.

_ , Stephen Luby, Ricardo Maertens, Brandon Tan, and Witold Wiecek, "Water Treatment and Child Mortality: A Meta-analysis and Cost-effectiveness Analysis," *University of Chicago, Becker Friedman Institute for Economics Working Paper*, 2022, (2022-26).

Kwong, Laura H, Ayse Ercumen, Amy J Pickering, Joanne E Arsenault, Mahfuza Islam, Sarker M Parvez, Leanne Unicomb, Mahbubur Rahman, Jennifer Davis, and Stephen P Luby, "Ingestion of fecal Bacteria along multiple pathways by young children in rural Bangladesh participating in a Cluster-Randomized trial of water, sanitation, and hygiene interventions (WASH benefits)," *Environmental science & technology*, 2020, *54* (21), 13828–13838.

Larsen, Bjorn, *Benefits and Costs of Drinking Water, Sanitation and Hygiene Interventions*, Copenhagen Consensus Center., 2016.

Liaw, Jane, Guillermo Marshall, Yan Yuan, Catterina Ferreccio, Craig Steinmaus, and Allan H Smith, "Increased childhood liver cancer mortality and arsenic in drinking water in northern Chile," *Cancer Epidemiology and Prevention Biomarkers*, 2008, *17* (8), 1982–1987.

Lokuge, Kamalini M, Wayne Smith, Bruce Caldwell, Keith Dear, and Abul H Milton, "The effect of arsenic mitigation interventions on disease burden in Bangladesh," *Environmental Health Perspectives*, 2004, *112* (11), 1172–1177.

Madajewicz, Malgosia, Alexander Pfaff, Alexander Van Geen, Joseph Graziano, Iftikhar Hussein, Hasina Momotaj, Roksana Sylvi, and Habibul Ahsan, "Can information alone change behavior? Response to arsenic contamination of groundwater in Bangladesh," *Journal of development Economics*, 2007, *84* (2), 731–754.

Mobarak, Ahmed Mushfiq and Alexander Van Geen, "The politics of poison," 2019.

Moon, Katherine A, Shilpi Oberoi, Aaron Barchowsky, Yu Chen, Eliseo Guallar, Keeve E Nachman, Mahfuzar Rahman, Nazmul Sohel, Daniela D'Ippoliti, Timothy J Wade et al., "A dose-response meta-analysis of chronic arsenic exposure and incident cardiovascular disease," *International journal of epidemiology*, 2017, *46* (6), 1924–1939.

NIPORT, MaA and ORC Macro, "Bangladesh demographic and health survey 2004," *Dhaka: National Institute of Population Research and Training, Mitra and Associates, and ORC Macro*, 2005.

Pitt, Mark M, Mark R Rosenzweig, and M Nazmul Hassan, "Identifying the costs of a public health success: Arsenic well water contamination and productivity in Bangladesh," *The Review of Economic Studies*, 2021, *88* (5), 2479–2526.

Prüss, Annette, David Kay, Lorna Fewtrell, and Jamie Bartram, "Estimating the burden of disease from water, sanitation, and hygiene at a global level.," *Environmental health perspectives*, 2002, *110* (5), 537.

Rahman, Mahfuzar, Marie Vahter, Nazmul Sohel, Muhammad Yunus, Mohammad Abdul Wahed, Peter Kim Streatfield, Eva-Charlotte Ekström, and Lars Åke Persson, "Arsenic exposure and age-and sex-specific risk for skin lesions: a population-based case–referent study in Bangladesh," *Environmental health perspectives*, 2006, *114* (12), 1847–1852.

_ , Nazmul Sohel, Fakir Md Yunus, Nurul Alam, Qamrun Nahar, Peter Kim Streatfield, and Mohammad Yunus, "Arsenic exposure and young adult's mortality risk: A 13-year follow-up study in Matlab, Bangladesh," *Environment international*, 2019, *123*, 358–367.

Ravenscroft, Peter, Ahammadul Kabir, Syed Adnan Ibn Hakim, A. K. M. Ibrahim, Sudhir Kumar Ghosh, Md Saifur Rahman, Firoza Akhter, and Md Abdus Sattar, "Effectiveness of public rural waterpoints in Bangladesh with special reference to arsenic mitigation," *Journal of Water, Sanitation and Hygiene for Development*, 07 2014, *4* (4), 545–562.

Sankararamakrishnana, Nalini, Divya Chauhana, R.T. Nickson, R.M Tripathid, and L. Iyengarb, "Evaluation of two commercial field test kits used for

screening of groundwater for arsenic in Northern India.," *Science of The Total Environment*, 2008, *401* (1-3), 162–167. PMID: 18514262.

Smedley, Pauline, "Arsenic in Groundwater in Bangladesh," Mar 2000.

Smedley, P.L. and David Kinniburgh, "Chapter 1. Source and behaviour of arsenic in natural waters," *United Nations Synthesis Report on Arsenic in Drinking Water*, 01 2001.

Smith, Allan H, Elena O Lingas, and Mahfuzar Rahman, "Contamination of drinking-water by arsenic in Bangladesh: a public health emergency," *Bulletin of the World Health Organization*, 2000, *78*, 1093–1103.

Stenberg, Karin, Rory Watts, Melanie Y Bertram, Kaia Engesveen, Blerta Maliqi, Lale Say, and Raymond Hutubessy, "Cost-effectiveness of interventions to improve maternal, newborn and child health outcomes: a WHO-CHOICE analysis for Eastern sub-Saharan Africa and South-East Asia," *International journal of health policy and management*, 2021, *10* (Special Issue on WHO-CHOICE Update), 706–723.

Tofail, Fahmida, Marie Vahter, Jena D Hamadani, Barbro Nermell, Syed N Huda, Mohammad Yunus, Mahfuzar Rahman, and Sally M Grantham-McGregor, "Effect of arsenic exposure during pregnancy on infant development at 7 months in rural Matlab, Bangladesh," *Environmental Health Perspectives*, 2009, *117* (2), 288.

Troeger, Christopher, Brigette F Blacker, Ibrahim A Khalil, Puja C Rao, Shujin Cao, Stephanie RM Zimsen, Samuel B Albertson, Jeffery D Stanaway, Aniruddha Deshpande, Zegeye Abebe et al., "Estimates of the global, regional, and national morbidity, mortality, and aetiologies of diarrhoea in 195 countries: a systematic analysis for the Global Burden of Disease Study 2016," *The Lancet Infectious Diseases*, 2018, *18* (11), 1211–1228.

UNICEF, "Arsenic Mitigation in Bangladesh," *UNICEF Brief*, 2008, ().

Vahter, Marie, "Health effects of early life exposure to arsenic," *Basic & clinical pharmacology & toxicology*, 2008, *102* (2), 204–211.

van Geen, Alexander, Jérôme Rose, S Thoral, Jean-Marie Garnier, Y Zheng, and Jean-Yves Bottero, "Decoupling of As and Fe release to Bangladesh groundwater under reducing conditions. Part II: Evidence from sediment incubations," *Geochimica et Cosmochimica Acta*, 2004, *68* (17), 3475–3486.

Vos, Theo, Stephen S Lim, Cristiana Abbafati, Kaja M Abbas, Mohammad Abbasi, Mitra Abbasifard, Mohsen Abbasi-Kangevari, Hedayat Abbastabar, Foad Abd-Allah, Ahmed Abdelalim et al., "Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019," *The Lancet*, 2020, *396* (10258), 1204–1222.

Wolf, Jennyfer, Sydney Hubbard, Michael Brauer, Argaw Ambelu, Benjamin F Arnold, Robert Bain, Valerie Bauza, Joe Brown, Bethany A Caruso, Thomas Clasen et al., "Effectiveness of interventions to improve drinking water, sanitation, and handwashing with soap on risk of diarrhoeal disease in children in low-income and middle-income settings: a systematic review and meta-analysis," *The Lancet*, 2022, *400* (10345), 48–59.

Yoshikawa, Thomas T., "Epidemiology and Unique Aspects of Aging and Infectious Diseases," *Clinical Infectious Diseases*, 2000, *30* (6), 931–933. _eprint: https://academic.oup.com/cid/article-pdf/30/6/931/7799871/30-6-931.pdf.

Yu, Winston H, Charles M Harvey, and Charles F Harvey, "Arsenic in groundwater in Bangladesh: A geostatistical and epidemiological framework for evaluating health effects and potential remedies," *Water Resources Research*, 2003, *39* (6).

Zhongming, Zhu, Lu Linong, Yao Xiaona, Zhang Wangqiang, Liu Wei et al., "Enhancing women's access to water, sanitation, and hygiene in Bangladesh," 2021.