Throwing the Baby out with the Drinking Water: Unintended Consequences of Arsenic Mitigation Efforts in Bangladesh

Erica Field, Rachel Glennerster and Reshmaan Hussam*

February 14, 2011

Abstract

The 1994 discovery of arsenic in ground water in Bangladesh prompted a massive public health effort to test all tubewells in the country and convince nearly one-quarter of the population to switch to arsenic-free drinking water sources. According to numerous sources, the campaign was effective in leading the majority of households at risk of arsenic poisoning to abandon backyard wells in favor of more remote tubewells or surface water sources, a switch widely believed to have saved numerous lives. We investigate the possibility of unintended health consequences of the wide-scale abandonment of shallow tubewells due to higher exposure to fecal-oral pathogens in water from arsenic-free sources. Significant small-scale variability of arsenic concentrations in ground water allows us to compare trends in infant and child mortality between otherwise similar households in the same village who did and did not have an incentive to abandon shallow tubewells. While child mortality rates were similar among households with arsenic-contaminated and arsenic-free wells prior to public knowledge of the arsenic problem, post-2000 households living on arsenic-contaminated land have 27% higher rates of infant and child mortality than those not encouraged to switch sources, implying that the campaign doubled mortality from diarrheal disease. These findings provide novel evidence of a strong association between drinking water contamination and child mortality, a question of current scientific debate in settings with high levels of exposure to microbial pathogens through other channels.

JEL: C81,C93, O12, O16

*The authors are from Harvard (Field) and MIT (Glennerster and Hussam). Please direct correspondence to efield@latte.harvard.edu. We thank Mahnaz Islam, Pronita Saxena and Rudmila Rahman for superb field work, Save the Children for enabling this study, and the Nike Foundation, KSG Center of International Development Empowerment Lab, and Harvard Sustainability Science Program for financial support.

1 Introduction

Water contamination is a central cause of illness in developing countries. The primary type of contamination in most settings is fecal-oral pathogens which lead to diarrheal disease, the second most common cause of infant and child mortality worldwide. However, in Bangladesh and a handful of other countries, carcinogenic heavy metals naturally leaching into ground water is a parallel concern. Based on tests conducted by the British Geological Survey (BGS) in 1998, an estimated 20 million Bangladeshis had been drinking shallow tubewell water that contained above the government's recommended maximum arsenic concentration of 50 μg per liter, and many more above the level recommended by the World Health Organization of 10 μg per liter. Although the health effects of chronic low-level exposure to arsenic are poorly understood, many believe the Bangladeshi population to be in danger of serious health effects from long-term arsenic poisoning.[1]

The subsequent international effort to move households away from water sources contaminated with arsenic constitutes one of the most successful public health campaigns in recent history in terms of scale, speed and success rate. In 1999, with help from international donors and NGOs, the Bangladeshi government initiated a massive campaign to test over five million tubewells throughout the country and conduct awareness-building activities encouraging households to abandon contaminated sources. According to household survey data from the Bangladesh Demographic and Health Survey (BDHS), by 2004 not only was there a high level of awareness of arsenic contamination among households in endemic regions, but the majority of households had stopped drinking from wells that were known to be contaminated.[2] In a strikingly short amount of time, awareness-building efforts alone led an estimated 23% of the population to transition from backyard pumps to less convenient

[1]See, for instances, media coverage in the New York Times ("Death by Arsenic: A special report; New Bangladesh Disaster: Wells That Pump Poison", November 10, 1998), the Economist ("A nation poisoned", December 20, 2001) and the British Medical Journal ("Half of Bangladesh population at risk of arsenic poisoning", March 25, 2000).

[2]For instance, in Barisal division where our study takes place, 46% of households had been drinking from contaminated tubewells in 1999 and only 1% were doing so in 2004. Furthermore, 78% of BDHS respondents had heard about the problem of arsenic in drinking water in 2004 (BDHS data, authors' tabulations).

drinking water sources including more remote tubewells or surface water sources.

However, as we investigate in this paper, in the process of switching, millions of households may have substantially increased their exposure to water-borne disease. In a setting such as Bangladesh where surface water is heavily contaminated with fecal bacteria, which causes diarrheal disease, cholera, dysentery and other potentially fatal water-borne diseases, backyard tubewells are widely considered "the most appropriate technology in terms of microbiologically clean water" (Lokuge et al., 2004). Not only are shallow tubewells protected from the surface, and therefore have very low rates of fecal contamination compared to ponds or dugwells, but by virtue of being located close to the residence, they minimize water storage time, which is highly correlated with pathogen levels since water becomes contaminated at a rapid rate in storage (Wright et al., 2004). Since distance to water source is also likely to reduce the overall amount of drinking water consumed, morbidity and mortality from diarrheal disease are also likely to increase when households switch to less convenient sources (Pruss et al., 2002).

For both reasons, recent successful public health efforts to move households away from shallow tubewells are likely to have unintentionally increased infant and child mortality among those that discovered arsenic in their groundwater. The extent to which this occurred depends on the marginal effect of clean drinking water on mortality from water-borne disease (the most important of which is diarrheal disease), a question of scientific ambiguity and intense policy debate in settings with high levels of exposure to microbial pathogens through other sources such as bathing, dish-washing and secondary drinking water sources. That is, in settings in which diarrheal disease is endemic, drinking water source improvements may be insufficient to interrupt transmission of waterborne pathogens, which also occurs via ingestion of contaminated food and other beverages, person-to-person contact, and by direct or indirect contact with infected feces.

To investigate this question, we quantify the impact on infant and child mortality of switching water sources in response to the arsenic testing and awareness campaign in one subdistrict of Bangladesh (Barisal). To do so, we make use of the high degree of natural

variation within villages in the rate of arsenic in shallow groundwater, which is uncorrelated with observable measures of land quality within small distances. This small-scale variability enables us to employ a difference-in-difference estimation strategy using data from a random sample of 3100 households spread across 155 villages that compares households living relatively close to one another who tested positive versus negative for arsenic contamination, and track the change in health outcomes of children born before versus after well-testing took place.

To identify households that were encouraged to switch to more distant water sources, we collected water samples from the drinking water kept in each household's kitchen and from the closest shallow tubewell. Consistent with census data collected by the government in 1999, our tests of the latter indicate that over 65% of households were drawing water from arsenic-contaminated shallow tubewells prior to 2000. However, only 1% of households in our sample tested positive for arsenic in their stored drinking water, implying that over two-thirds switched from shallow tubewells to alternative arsenic-free water sources between 2000 and 2009. Since there is no piped water in these rural villages, arsenic-free water sources include either deep tubewells, uncontaminated shallow tubewells in neighboring houses, or surface water sources.

We then estimate the trends in infant and child mortality with a village fixed effect specification that absorbs differences in mean characteristics between relatively exposed and relatively unexposed villages arising from potential correlations between the spatial clustering of arsenic contamination at a macro level and characteristics such as income that may influence health. In doing so, our identification strategy relies on the assumption that the spatial distribution of arsenic contamination is quasi-random within distances as small as villages, which these and other data support.[3]

Our estimates indicate that, while infant and child mortality rates were almost identical in contaminated versus uncontaminated households before 2000, these outcomes diverged

[3]For instance, in a large multi-village dataset from the Araihazar subdistrict, the spatial distribution of arsenic is orthogonal to observable household characteristics within but not across villages (Madajewicz et al., 2007).

sharply immediately after. Post-2000, households with arsenic-contaminated wells – those likely to have switched sources – exhibit a 27% increase in infant and child mortality relative to those in the same village with arsenic-free wells. This figure implies that the abandonment of shallow tubewells approximately doubled rates of diarrheal disease in the population of switchers.

We also undertake a similar exercise at the national level using data from the 2004 BDHS. Just as we did in 2009, the 2004 BDHS collected drinking water samples from each household and tested them for arsenic contamination. While the majority of households had already switched to arsenic-free drinking water by 2004, 8% of households spread across 29% of villages were still drinking from arsenic-contaminated tubewells despite public health efforts to change behavior. We make use of this variation to test whether households that we know did *not* switch away from backyard tubewells exhibit relatively lower rates of infant mortality after 2000 compared with households that can be presumed to have switched to more distant sources. While this approach raises concerns about the endogeneity of switchers, we view it as a consistency check on the more tightly identified estimates in Barisal that also allows us to look in more detail at potential mechanisms through which switching may adversely effect health.

Indeed, consistent with our estimates from Barisal, switchers have significantly higher rates of infant and child mortality after but not before the well-testing campaign relative to non-switchers. Furthermore, the negative effect of abandoning shallow tubewells appears to be equally large when deep tubewells are available as an alternative source. This provides evidence that clean but remote water sources are poor substitutes for backyard tubewells in terms of mortality risk, either due to high rates of recontamination in storage or more frequent use of water from secondary surface water sources.

Although it is difficult to argue that non-switchers identified in the BDHS data are a representative subsample of households, observable characteristics and pre-campaign mortality levels are similar across the two comparison groups, so the pattern does not reflect simple convergence in child mortality between low- and high-SES households that happens

to coincide with the well-testing campaign. In addition, when we use BDHS verbal autopsy data to classify causes of death, we find that abandoning shallow tubewells is associated with an increase in deaths due to diarrheal disease but find no such divergence in mortality from pneumonia or fever, further strengthening evidence of a causal relationship between shallow tubewells and child mortality as opposed to general trends in mortality between switchers and non-switchers that are unrelated to water source.

Together, these two sets of results provide novel evidence of a strong link between improved drinking water sources and mortality from diarrheal disease in settings with high risk of exposure to fecal contamination through other channels. Given the potentially small benefit offered by drinking microbiologically safe water in settings where there is constant exposure to fecal matter through bathing, food preparation, and dish washing, the marginal health benefit of protected water sources in countries such as Bangladesh is to date an unresolved question in the public health literature. The debate has become particularly heated in light of previous results showing little difference in rates of diarrhea by water source in such settings (Esrey and Habicht, 1986; Lindskog et al., 1987; Caldwell et al., 2003; Kremer et al., 2010). Hence, in addition to contributing to the current debate over arsenic mitigation efforts in Bangladesh, our results have important policy implications for more general efforts to reduce infant and child mortality in the most afflicted settings.

Our results also highlight the need to proceed cautiously when issuing public health recommendations when there is insufficient information concerning competing risks. In the case of rural Bangladesh, should the use of shallow tubewells contaminated with arsenic continue to be discouraged, given the current absence of equally clean and convenient alternative water sources? Our results suggest that continued efforts to do so could have dire consequences for the health of infants and children, which need to be weighed carefully against the less understood health consequences of chronic low-level arsenic exposure.

2 Background

2.1 Public health efforts surrounding shallow tubewells

Largely because of its geographic vulnerability to flooding combined with its high population density, Bangladesh has historically had one of the highest incidence of water-borne viral and parasitic infections and corresponding infant and child mortality in the world. To reduce chronic cholera and diarrheal disease outbreaks, an estimated 8.6 million shallow tubewells were constructed throughout the country from the 1970s to the 1990s.[4] These efforts succeeded in moving an estimated 95% of rural Bangladeshis from parasite-infected surface water to protected ground water (Caldwell et al., 2003).

Unfortunately, these improvements in sanitation were short-lived due to the discovery of arsenic in the major shallow aquifers.[5] Geologists first discovered traces of arsenic in Bangladesh groundwater in 1987, and physical manifestations of arsenicosis, the disease caused by substantial ingestion of arsenic, were first documented in 1994. Three years later, the World Health Organization (WHO) publicly declared groundwater arsenic contamination to be a "major public health issue," and issued a grant to address the emergency.

In 1998, BGS conducted a nationwide study measuring levels of contamination in a sample of shallow tubewells across Bangladesh. Results indicated that 21 million people (15% of the population) were in grave danger, drinking water with more than 50 ppb (μg) As, and 42 million in lesser danger, drinking water with more than 10 ppb As.[6] In the late 1990s and early 2000, the Bangladeshi government, along with UNICEF and a host of other aid organizations, conducted a blanket screening of all shallow tubewells in contaminated regions of the country. Wells that tested contaminated (1.4 million) were painted red and those that tested safe (3.3 million) were painted green (Johnston, 2006).

[4]Tubewell construction was funded by the Bangladeshi government, UNICEF, World Bank, and numerous other public and private organizations, and also financed privately by households.

[5]Arsenic-bearing sediments buried in the aquifers come from rocks that eroded from the Himalayas and were deposited in the low-lying areas which now make up West Bengal and Bangladesh. Arsenic sediment is released into ground water by a natural process called "oxyhydroxide reduction".

[6]This estimate has more recently been increased by the Government of Bangladesh to 30 million and 70 million, respectively (WHO, 2008).

Households were and continue to be strongly encouraged to stop drinking from red tubewells and switch to alternative sources (Jakariya, 2007). Potential alternatives include deep tubewells, piped water, dug wells, treatment of surface water, rainwater harvesting, sharing of safe shallow tubewells, and treatment of arsenic contaminated water. Among these, deep tubewells are one of the most commonly promoted alternatives. Although they are prohibitively expensive for most households to build, between 1998 and 2006, the Arsenic Mitigation Water Supply Project built over 9,000 deep tubewells across 1800 villages in Bangladesh where sufficiently deep aquifers could be found.[7]

Unfortunately, analyses of post-construction deep tubewell water found that arsenic can leach into the wells over time (Feroze Ahmed, 2002; WorldBank, 2007). A fear that further use of deep tubewells would lead to arsenic contamination of Bangladesh's deep aquifers led the 2004 National Policy for Arsenic Mitigation report to stress a "preference of surface water over groundwater as a source for water supply." According to a World Bank evaluation, this report had a notable effect on patterns of water usage "effectively foreclos[ing] use of the less costly option of tubewells as a safe source for small communities, leaving the less popular dug wells, rainwater harvesting, and pond sand filters as options for other areas. Many dug wells were abandoned, and some communities installed new shallow wells (with uncertain arsenic levels) or reverted to surface water from ponds (where water quality is suspect)" (WorldBank, 2007).

Though less emphasized among policymakers, the sharing of safe tubewells has been a relatively popular option in some parts of the country, including the heavily studied district of Araihazar. VanGeen et al. (2002) report that 43% of exposed individuals in Araihazar preferred switching to a nearby safe shallow tubewell over other alternatives such as deepening their well (31%) or using surface water (20%).[8] Within two years of well testing in the district, Schoenfeld (2005) reports that approximately 30% of individuals exposed to greater than 50 ppb As and 15% of individuals using unknown (unpainted) wells switched to nearby

[7]The cost of constructing deep tubewells in most locations is estimated to be well over $500, while the cost of constructing shallow tubewells is estimated to be $38 (Caldwell et al., 2003).

[8]The authors also note, however, that Araihazar District has more shallow tubewells than the rest of Bangladesh.

green-painted wells.

Finally, nationwide public education campaigns about the presence and dangers of arsenic have been widespread since 1999.[9] The impact of these educational campaigns are reportedly considerable: 80% of the population is aware that arsenic may be a danger in groundwater (relative to less than ten percent in the late 1990s), and 70% of households report changing their behavior to avoid arsenic (UNICEF, 2008).

2.2 Health benefits of switching away from shallow tubewells

Arsenic is a known carcinogen that has been shown in laboratory studies to cause or catalyze several forms of cancer, particularly of the lung and bladder (Kozul et al., 2009; Suzuki et al., 2008; Rossman et al., 2002).[10] Hence, it is generally accepted that exposure to high levels of arsenic (> 100 μg) will lead to a major increase in cancer-related deaths and morbidity in the older adult population. There is a notable lack of hard evidence on the health effects at the lower end of the exposure doses. However, in large part due to the long latency of most arsenic-related health problems, the National Research Council concludes that "arsenic-related disease due to chronic exposure through drinking water has a relatively low incidence" in settings with low average life expectancy such as Bangladesh (Research Council, 2001).

One exception to this perspective are recent results from an epidemiological study following over 10,000 adults in the Araihazar District in Bangladesh, which reported very high mortality associated with arsenic exposure (Argos et al., 2010). The authors estimated that approximately 20% of all deaths documented over nine years were attributable to arsenic, with mortality rates nearly 70% higher for those exposed to arsenic concentrations of

[9]Programs focus on raising awareness of the impact of arsenic ingestion, alternative safe water sources, remedial measures against poisoning, and the understanding that arsenicosis is not contagious (BMOH, 2004). During the testing campaign of 1999-2000, UNICEF had its tubewell testers spend their waiting time sharing basic information about arsenic, dispelling common myths, and then directly showing the villagers the result of the well test. In more recent years, UNICEF has established an educational curriculum integrating hygiene and sanitation with arsenic awareness and also involved the community in choosing alternative water sources best suited to their needs.

[10]Field studies have also found a strong dose-response relationship between skin cancer and arsenic exposure through drinking water (Chen et al., 2006; Mazumdar et al., 1998; Tucker et al., 2001).

over 150 ppb relative to those exposed to less than 10 ppb. However, an important caveat to this study not addressed by the authors is that arsenic concentrations in groundwater are not orthogonal to socio-economic status in this setting. As shown in Madajewicz et al. (2007), due to the spatial clustering of arsenic across the 54 villages in this study area, prior to testing households with uncontaminated wells happen to have significantly higher average income and assets (with 42% more assets and 16% more expenditures) compared to households living on contaminated land.[11] Although the differences disappear when accounting for village fixed effects, the Argos et al. (2010) study fails to do so and, as a result, mortality differentials found in their study are almost certainly biased upwards.[12]

On the other end of the spectrum, the calculations by Lokuge et al. (2004) of the disease burden from arsenic exposure that take into account only "strong causal evidence" from existing studies estimate that arsenic-related disease leads to the loss of 174,174 disability-adjusted life years (DALYs) per year among the population exposed to arsenic concentrations of more than 50 ppb, which amounts to 0.3% of the disease burden, compared with diarrheal disease which accounts for between 7.2% and 12.1% of the total disease burden.

Researchers almost universally agree that the relationship between arsenic exposure and morbidity and mortality in younger populations is minimal. One highly publicized study of children in Araihazar found that arsenic exposure inhibits the mental development of children (Wasserman et al., 2004), but the estimates face the same bias that the Argos et al. (2010) study faces so should be interpreted with caution. Similarly, a handful of studies

[11] VanGeen et al. (2003) and Ahsan et al. (2006) describe these spatial patterns in detail, though not as they relate to SES. VanGeen et al. (2003) notes that "Most of the wells with the lowest As concentrations are located in the northwestern portion of the study area", which appears to contain higher SES villages. According to Madajewicz et al. (2007), "Arsenic is released when the accumulation of plant matter during the formation of river delta deposits drives groundwater to anoxia. The process may generate 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. Wells are located within small, densely inhabited villages. The surrounding fields are fairly uniform geologically, while the dispersion of incomes and wealth within villages is large."

[12] Furthermore, age is significantly higher and the number of relatives in the study is significantly lower among high-concentration households (Madajewicz et al., 2007). Age is less of a concern in terms of bias since the Argos et al. (2010) estimates control for age. However, households with fewer social network connections are likely to have higher mortality due to a deficit of informal insurance and health care networks.

have reported reproductive health consequences of arsenic exposure, although the evidence is mixed (Vachter, 2008; Tofail et al., 2009; Milton et al., 2005; Liaw et al., 2008).

In general, since arsenic exposure also tends to be correlated at a macro level with socioeconomic conditions influencing child development measures, causality cannot be easily inferred from studies that show a correlation between arsenic exposure and various health outcomes (Tofail et al., 2009).

2.3 Health costs of switching away from shallow tubewells

Although abandoning shallow tubewells contaminated with arsenic is likely to have a measurable latent effect on reducing mortality in older populations, given the relatively high burden of diarrheal disease, it could come at a significant cost to the health of younger populations. Because shallow tubewells are supplied to individual households (generally built in the backyard close to the residence), they are an extremely convenient water source, which increases the frequency with which water is collected and therefore reduces water storage time and increases water consumption. Storage time is an important determinant of contamination with fecal matter, as water that is not stored properly is continuously exposed to dirty hands and cups or utensils, and previous studies find strong correlations between distance from water source and diarrheal disease (Esrey, 1996). Inconvenience also implies a potential decrease in the amount of water consumed (Hoque et al., 1989), which can have important health consequences for children facing dehydration from diarrheal disease. In fact, according to one previous study, the quantity of water used is a better predictor of child health than the quality of water used (Esrey, 1996).[13]

The only water sources equally convenient to shallow tubewells are surface water sources such as ponds that are also likely to be close to the residence. However, while they are free of arsenic, these sources are significantly more likely to be contaminated with fecal matter. While water filtering and cleaning methods can address point of use contamination,

[13]Using experimental methods, Kremer et al. (2010) estimate in rural Kenya that on average water quality deteriorates by one third between point-of-source and point-of-use.

survey data indicate that these have largely been abandoned in rural Bangladesh since the construction of shallow tubewells (Caldwell et al., 2003).

Taking into account all of these changes in risk exposure, Lokuge et al. (2004) estimate that abandonment of shallow tubewells would increase a household's risk of diarrheal disease by 20%. Until now, there has been no empirical estimation of this possibility and health messages promoted by governmental and non-governmental agencies continue to stress the importance of moving away from shallow tubewells that are contaminated with arsenic.[14]

3 Estimation Strategy

3.1 Data and Setting

To study trends in child mortality, we capitalize on extensive household survey data, including reproductive and child health outcomes for all children in the household, that were collected by the authors in 2007 as part of an impact evaluation of an adolescent empowerment program currently being implemented in one district in the southeast of the country. The data set covers 155 villages and 3093 households in Barisal District of Barisal Division, one of the areas most heavily contaminated with arsenic in the country. According to village-level well testing data collected by the government in 1999, over 70% of tubewells in the area were contaminated.[15]

Barisal was also a relatively "successful" region in terms of the public health campaign that followed. Data from the BDHS reveal a uniquely high rate of switching away from contaminated water sources in Barisal, attributed largely to the geology of the region, which made it possible to construct deep tubewells in almost all villages. According to esti-

[14]For instance, the Bangladeshi Ministry of Health recently had the following message posted on its web site: "The public health of the country is now facing a severe threat as a section of existing tube-wells are contaminated with arsenic. *Now time has come to return to our old habit. Because we can keep ourselves safe from arsenic pollution by drinking surface water."* (March, 2010)

[15]Correspondingly, in a 2000 nationally representative household survey on arsenic contamination, 28% of respondents reporting arsenic symptoms were in Barisal, which contains 9% of the country's population (Caldwell et al., 2006).

mates from the Bangaldeshi Government's National Arsenic Mitigation Information Center (NAMIC), there is currently one deep tubewell per approximately 100 households in rural Barisal.

The full household survey, of which we use a part, collected data from 9,048 households in three districts and five subdistricts of Barisal. Households included in the study were randomly drawn from within the five participating sub-districts in a two-stage sampling process in which villages were first sampled from the universe of villages containing more than 50 and fewer than 500 households, and then 20 households per village were selected at random from village-level census data.[16] Only one of the three districts, Barisal District, is contaminated with arsenic. Hence, our present analysis is restricted to the 3158 households in Barisal district. For the purpose of this analysis, these households were revisited in 2009 for water testing and a brief survey of water use and arsenic awareness, and 3093 households were successfully surveyed at follow-up. In order to link data on child health histories with water source, we also tested each household's closest shallow tubewell for arsenic level and collected survey data on household water sources before and after the 1999-2000 well testing campaign, in addition to respondents' knowledge about arsenic contamination.

Our analysis sample includes all children born in the present home between 1980 and 2007 to heads of households with complete arsenic survey information.[17] The final sample encompasses 2817 households and 11,766 children, 3,685 of whom reside in low concentration households and 8,081 in high concentration households.[18]

[16]Households were eligible for random selection only if they included at least one adolescent girl. Villages of medium size were included in the sample frame because this was seen as an appropriate size for the adolescent girls program the survey was designed to evaluate.

[17]The 2697 children (18%) born after 1980 but before the household moved into the current residence are dropped from the analysis, although the results are robust to including them. As predicted, the point estimate falls but remains statistically significant. We also exclude from the sample 167 individuals whose mother's age at birth is less than twelve years, greater than 45 years, or missing, and one household is dropped because identifying data do not match well between the baseline and arsenic surveys.

[18]Numbers are specific to defining high concentration as those households with wells with greater than 60 ppb As according to our closest-well test results.

3.2 Identification Strategy

Our identification strategy makes use of the fact that there is significant small-scale variability of arsenic concentrations in ground water uncorrelated with observable land characteristics (Yu et al., 2003).[19] In particular, an estimated 88% of contaminated wells are located within 100 meters of an uncontaminated well (VanGeen et al., 2003), giving rise to substantial within-village variation in contamination: in 47% of villages in our sample between 20 and 80% of wells are contaminated, and in 65% between 10 and 90% of wells are contaminated. Within a village, local pockets of contamination are impossible to predict as they have not been found to be correlated with any observable features of the land.[20] Hence, while certain villages contain a much higher percentage of contaminated groundwater than others, within a village it is impossible for households to know whether a given property is situated on contaminated groundwater prior to digging the well and testing it. This variation in well contamination makes it possible to compare otherwise identical households residing close to one another who are and are not encouraged to abandon shallow tubewells in 1999 based on revealed arsenic exposure in a difference-in-difference (DID) estimation strategy.

We define a binary level of arsenic exposure using two methods. The first, denoted "measured contamination," categorizes wells (and implicitly households) as contaminated if the concentration of arsenic in the shallow tubewell closest to the surveyed household, as tested by a standard arsenic testing kit, is greater than 60 ppb when measured by our field team in 2009.[21] The second, denoted "reported contamination", categorizes households

[19]Small-scale variation in arsenic levels is due to heterogeneity of near-surface geology and the resulting biogeochemical environments, both of which are uncorrelated with agricultural land quality.

[20]Because of this difficulty, encouraging households to build new shallow tubewells on uncontaminated land is not a viable policy alternative, although it has been explored extensively. There are many times village-level observable features of land such as the permeability of nearby soils that predict probabilities of arsenic contamination for all households in the village, but such characteristics do not provide information about within-village location of pockets of arsenic in underground aquifers (Madajewicz et al., 2007).

[21]To test arsenic levels in water samples, we used the Wagtech Digital Arsenator testing kit in a laboratory setting. We chose 60 ppb as the cutoff to reflect the 50 ppb WHO cutoff, taking into account an estimated 2% per year increase in arsenic levels, so that contaminated wells in our sample are those believed to have tested above 50 ppb in 1999. Relatively constant groundwater As concentrations have been reported in a number of time series studies in the area (VanGeen et al., 2003).

as contaminated if any of the shallow tubewells ever used by the household are reported in survey data collected in 2009 to have tested positive for arsenic, been painted red, been deemed unsafe for drinking, been abandoned, or been built less than three years before the survey.[22] The 2% of households that lack information on shallow tubewell use because of non-response are categorized using the "measured contamination" method for both measures. The two measures of contamination correspond for 87% of households.

Since there is some concern that households underreport use of contaminated wells, we favor the "measured contamination" variable over the "reported contamination" variable.[23] In the case that underreporting of contaminated wells is unrelated to household characteristics associated with child health outcomes, this measurement error will bias our estimate downward. However, if there is a more complicated reporting bias – for instance, if households that are more aware of health risks are more prone to hide contaminated wells – there is risk that our estimates are biased upwards.

A key assumption in our "measured contamination" method is that distance is a valid proxy for utilization (i.e. that the closest shallow tubewell for a given household was in fact the main source of drinking water prior to the arsenic awareness campaign), which is probably a fairly accurate assumption since most households have only one shallow tubewell in the interior courtyard close to their dwelling and convenience has been shown to be an important predictor of amount of water consumed. Although households could also lie about which well is the closest, our "measured contamination" method is less prone to such biases since enumerators were instructed to visually inspect the area surrounding each dwelling to identify the closest well, and typically backyard tubewells are quite close to the dwelling and highly visible.

To test the validity of our key identifying assumption regarding the quasi-random

[22]The latter condition is included under the assumption that wells installed recently were built to replace contaminated wells.

[23]Survey data also show evidence of underreporting: Our survey data on history of shallow tubewell use, when compared to our measured contamination, indicates a tendency to underreport use of highly contaminated wells prior to their being tested. The "reported contamination" method also lacks complete data, as many individuals responded "Don't Know" to relevant survey questions.

nature of variation in arsenic exposure, Table 1, Panel 1 presents a host of time-invariant sample characteristics, with mean values shown separately for those in low concentration households and those in high concentration households based on both measured and reported contamination. All averages are regression-controlled means that account for village fixed effects, as do reported t-statistics of the differences in means across samples. Characteristic of the rural population in this area, households in our sample are relatively poor and uneducated: Mothers completed an average of three and a half years of schooling and fathers completed nearly four. The mean monthly income of a household was approximately $11.40, with 40% of households working primarily in agriculture and 15% of households working primarily in business. Households owned on average less than one acre of land and lived in a home with fewer than three rooms. However, the majority is not destitute: approximately 90% of respondents reported having sufficient food for the family in a given week, and more than half (54%) of households had some type of outstanding loan.

In terms of similarity of our comparison groups on observable characteristics, baseline differences across low and high contamination households are small and statistically insignificant, supporting our identification strategy. Only one variable out of 23 - whether Muslim - is significantly different across the two subsamples at the 10% level, and under the measured contamination measure but not the reported contamination measure, and the point estimate of the difference is extremely small. Other measures of socio-economic status indicate that the samples are balanced on income and wealth, and an F-test of joint significance indicates that the samples are balanced on observables within villages ($p = 0.54$). Nonetheless, we present all estimates with and without controlling for a number of family background variables to reduce the scope for imbalance to bias our estimates. Interestingly, as shown in Appendix Table 1, the same exercise conducted without accounting for cross-village variation shows a high degree of imbalance, as is also observed in other study areas such as Araihazar. In our setting, however, spatial clustering across villages produces a pattern in which arsenic contamination is disproportionately concentrated in relatively well-off villages.

In terms of endogenous variables, differences in infant and child mortality across sub-

samples are evident from sample means alone: High concentration households have higher rates of infant and child mortality over the entire period, although we see no difference in fertility, sex ratios, or the timing of births, all of which could potentially be influenced by differences in child mortality and complicate the interpretation of trend differences. Interestingly, individuals in low contamination households reported statistically significantly higher home values than those in high contamination households, which is presumably a causal effect of having a contaminated well. Most households list at least two sources of drinking and cooking water, and about 70% report that the closest shallow tubewell was tested and painted during the campaign of 1998-2000, consistent with estimates from national data.

3.3 Estimating equation

We test for changes in infant and child mortality that correspond to the timing of the testing campaign by estimating the following difference-in-difference equation for individual i in household j and village v, which includes village fixed effects (θ):

$$Y_{ijv} = \alpha_{ijv} + \gamma * HighConc_{jv} + \delta * EarlyLifeExp_{ijv} + \beta * (HighConc_{jv} * EarlyLifeExp_{ijv}) + \theta_v + \epsilon_{ijv} \quad (1)$$

$HighConc$ is a dummy variable taking the value of one if the individual is in a household exposed to arsenic contamination. $EarlyLifeExp$ denotes the fraction of a child's life below the age-of-death cutoff being considered in the outcome variable that he or she was exposed to microbiologically unsafe drinking water from surface sources or deep tubewells as a result of the testing campaign in 1999-2000. Hence, for under 1 mortality, $EarlyLifeExp$ is simply a dummy variable that takes a value of 1 if the child was born after 2000 and 0 if born before 2001, but for under 2 mortality, $EarlyLifeExp$ takes a value of 1 if the child was born after 2000, 0.5 if born in 2000, and 0 if born before 2000.[24] Since it is difficult to verify in exactly which of these two years the majority of households was tested, we also run

[24]The maximum number of years of exposure is the mortality interval (of one, two, or five years) over which infant and child deaths are being measured in each outcome variable.

analogous estimates using 1999 as a cutoff point in place of 2000. Although the estimates are robust to either cutoff, we choose 2000 as our preferred specification since we presume that behavioral change towards alternative drinking water sources had at least a slightly lagged response. Standard errors are clustered at the household level.

We are interested in the coefficient estimate of β, describing the change in mortality due to abandoning shallow tubewells. Proper identification relies on the assumption that other natural processes or human interventions occurring over the observed time period did not differentially affect infant and child mortality rates for households exposed to high concentrations versus low concentrations of arsenic. The high degree of variation in arsenic exposure across very small distances and the similarity across comparison groups in relevant baseline characteristics and mortality levels prior to revelation of arsenic contamination lend credibility to this assumption. To test this assumption, we also run a placebo check described in Section 4.1 in which we test whether an alternative cutoff well above 60 ppb produces similar patterns within a subsample of households that were *all* encouraged to abandon shallow tubewells (those with arsenic concentrations higher than 60 ppb). Since this specification compares switchers with switchers, we should observe a significant effect of the cutoff only if unobservable determinants of mortality are correlated with arsenic in groundwater.

Along with the parsimonious specification, we also estimate versions of Equation 1 with controls for the individual's sex, parity, birth year, and birth year squared, and a wider set of control variables that includes age of mother at birth, mother's education, father's education, years since birth of last child, solvency, land size, number of rooms in house, electricity, whether Muslim, and monthly income per capita.

4 Results

Figures 1-3 present the trends in one, two, and five year mortality between 1978 and 2007 based on the raw data using the measured contamination method to divide the sample

into switchers and non-switchers. For smoothness, mortality rates are averaged across two-year periods. For the most part mortality trends in high concentration households closely follow those in low concentrations households until 1998-1999, at which point they begin to diverge. Both child and infant mortality rates are substantially higher among individuals in high concentration households relative to those in low concentration households immediately after the arsenic testing campaign (2000-2001), and these differences are sustained to the time of the survey in 2007 (though there is some indication of convergence in the last two-year interval). This suggests that most switching (and the resulting mortality effects of exposure to microbiologically unsafe water) occurred immediately after the campaign.

Table 2 presents the corresponding regression results from equation (1) for infant, under two, and under five mortality using our measured contamination measure. As reflected in the mortality graphs, the coefficient estimates indicate a substantial and statistically significant increase in mortality after 2000 among individuals living in households with high levels of arsenic in their shallow tubewells. These results are robust to the inclusion of both the basic and full set of controls (detailed in Section 3.3).

Referring to the full control specification (columns 3, 6 and 9) of Table 2, an additional year of exposure to the post-campaign environment for an individual with a contaminated shallow tubewell is associated with a 2 percentage point (27%) increase in the likelihood of death within 12 months, a 3.2 percentage point (33%) increase in the likelihood of death within two years, and a 3.9 percentage point (28%) increase in likelihood of death within 5 years. This implies that mortality from diarrheal disease, which was estimated to account for approximately one-quarter of deaths under age five in 2000 (Morris et al., 2003), approximately doubled after the well-testing campaign for households that abandoned backyard tubewells.

These estimates are large in comparison to the increased burden of diarrheal disease that is predicted in response to the abandonment of shallow tubewells in the projections of Lokuge et al. (2004) (20%), although there are several possible sources of discrepancy. First, the Lokuge et al. (2004) estimate was taken directly from a study by Esrey (1996) that

was based on DHS data from eight countries, all of which have diarrhea prevalence below that of rural Bangladesh. Projections were based on the simple correlation between access to improved water supply and reported incidence of diarrhea in children under 5, which could produce downward biased estimates of the causal effect of changes in water supply on diarrheal disease if improved water services are, conditional on income, targeted to areas with highest rates of mortality from diarrheal disease. This is particularly problematic since the Esrey (1996) study was based on extremely small samples within each country.

A potentially more appropriate benchmark is the reduction in rates of diarrheal disease that are associated with the widescale construction of tubewells in rural Bangladesh, which has roughly fallen in half since the 1970s. Unfortunately, as noted by Caldwell et al. (2003), it is unclear how much of a role can be attributed to the use of tubewells given the concomitant adoption of public health measures such as immunization, antibiotics and oral rehydration therapy (ORT). Still, it is worth noting that, according to autopsy data from the demographic surveillance site of Matlab, diarrheal disease accounted for an estimated 47% of deaths to children ages 1-4 in 1966-1977, then fell to 34% of deaths in 1978-1987, and by 1999 accounted for only 20% of deaths (Baqui et al., 1994), suggesting that the adoption of shallow tubewells could have reduced mortality from diarrheal disease by as much as 57% (or, correspondingly, reverting to surface water sources would increase diarrheal disease by 135%). Hence, we take 20-135% (which encompasses our estimates of $\sim$100%) as an appropriate range of possible mortality increases due to the abandonment of shallow tubewells.[25]

The results reported in Table 3, using the reported rather than measured contamination of the household water source prior to the testing campaign, show very similar patterns. The estimates are consistently larger in magnitude under the reported contamination method, which could be driven by either higher precision or reporting bias, as described in Section 3.2.[26]

[25]Data from 1966-1987 reported by the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDR, B) Demographic Surveillance System - Matlab: Registration of Demographic Events.

[26]Furthermore, mortality patterns are similar across gender (unreported), although the sample sizes are too small to draw precise comparisons.

4.1 Robustness Checks

Our estimates are robust to a number of alternative specifications and placebo checks, the results of which are presented in Appendix Tables 2-7. Appendix Table 2 presents the same regressions as Table 2, replacing the individual with the household as the unit of observation, such that the outcome variable is fraction of deaths in the household under a certain age, and early life exposure is now measured as the fraction of children born after 2000. Coefficient estimates are noisier but virtually unchanged. The regressions in Appendix Table 3 include a binary in place of a continuous measure of early life exposure, in which exposure is equal to 1 if the child was born after 2000 and zero otherwise. Again, the results are noisier but very similar, as we would expect.

Since approximately one-third of households report both well testing and switching drinking water sources after 2000, Appendix Table 4 makes use of survey data on the year in which a household's well was reportedly tested to try to gain precision on the anticipated date of switching within a given household. Here, we replace the binary indicator of a child being born after 2000 with an indicator of a child being born after the household's closest well was tested, according to survey reports. In this specification, the DID estimate is comparable in magnitude and *gains* precision, as one would expect if we take the survey reports at face value. However, because there is no way to confirm reports of testing dates, there is a possibility of non-random measurement error biasing these results.

In the regressions reported in Appendix Table 5, we run a falsification test in which we exclude households with arsenic contamination levels below 60 ppb (non-switchers) and construct a false cutoff point of 100 ppb. We then estimate a DID regression analogous to Equation 1 in which we compare households above and below 100 ppb. Since all of those households were equally encouraged to switch sources after 1999, we should see no difference in trends if our previous estimates truly reflect the causal effect of switching water sources. In contrast, if level of arsenic contamination in groundwater is correlated with unobservable characteristics of the household that are giving rise to differential time trends in child and infant mortality, we should expect to see positive and significant point estimates on the

interaction terms in both regressions. As the estimates reveal, we see no significant effect on mortality of having arsenic levels above 100 ppb relative to having arsenic levels between 50 and 100 ppb, which reduces the likelihood that our estimates reflect differential time trends in mortality that are correlated with a household's level of arsenic exposure through mechanisms other than switching drinking water sources. Since we only observe a significant DID estimate when the true cutoff for well-switching is used, we can deduce that the estimate reflects the causal effect of changing water sources rather than time trends in unobservables correlated with arsenic exposure.

Appendix Table 6 shows Equation 1 estimated only for households whose nearest well was built more than eight years ago. Exclusion of recently installed wells ensures that all individuals in the sample had access to the existing shallow tubewell prior to the testing campaign, and subsequent decisions on water source and usage would have been made with consideration of the campaign. The DID estimate is significant and larger in magnitude than those of the original specification: Obtaining drinking water from surface sources or deep tubewells since birth is associated with a 2.6 percentage point increase in likelihood of death within one year, a 3.8 percentage point increase for two years, and a 4.1 percentage point increase for five years. While the estimates are in theory more accurate, since year of well construction is likely subject to recall bias and potential misreporting, it is possible that estimates that take account of these reports are biased, so our preferred estimates are those in Table 2. Finally, Appendix Table 7 uses 1999 instead of 2000 as the cutoff date of the campaign, with very similar results.

4.2 Nationwide trends in DHS data

We next look for nationwide evidence of changes in risk of diarrheal disease attributable to the arsenic mitigation campaign using an analogous estimation strategy with national data on infant and child mortality and water sources available in the 2004 BDHS. The 2004 BDHS tested household drinking water for arsenic contamination and found that 8% of households distributed across 29% of BDHS villages had not switched to arsenic-free drinking

water sources in spite of the massive campaign efforts. That is, the presence of arsenic in their drinking water confirms that these households were still using contaminated shallow tubewells in 2004 even though deep tubewells existed in at least 18% of these affected villages.

We make use of this within-village variation in household response to the arsenic mitigation campaign to test whether child mortality trends before and after 2000 look worse for households that switched to arsenic-free sources relative to those that continued to drink from shallow tubewells. If switching away from shallow tubewells is associated with greater exposure to microbiologically contaminated water, we would expect child mortality to increase with early life exposure for those households in arsenic-contaminated villages who switched to arsenic-free water post-campaign.

Because we are restricting ourselves to within-village comparisons, our analysis sample is implicitly restricted to the 29% of villages in which at least one household is still drinking from an arsenic-contaminated source.[27] Hence, although we cannot observe in the BDHS data whether well water in a specific village is contaminated with arsenic, our sample is necessarily restricted to a subset of villages in which arsenic contamination is present by virtue of the fact that we observe it at least once in the data. Based on the spatial concentration of arsenic deposits in our data from Barisal, for villages in which arsenic is present, the median rate of contamination is 77%, and in only 25% of villages are less than half of shallow tubewells contaminated. Hence, although in the BDHS we are unable to distinguish whether households with clean drinking water in 2004 have switched away from contaminated shallow tubewells or continue to drink from tubewells that were never contaminated, in our subsample of "exposed" villages, we can assume that the majority of households with arsenic-free drinking water in 2004 are households that were encouraged to abandon shallow tubewells.[28] This ambiguity also implies that we are underestimating mortality effects of switching since not all households with arsenic-free water switched sources around the time

[27] In no villages are *all* sampled households drinking contaminated water.

[28] Though the BDHS does ask about current drinking water sources, we cannot make use of these data since the category "tubewell" does not clearly distinguish between deep and shallow tubewells, nor does it distinguish between backyard tubewells and tubewells in neighboring houses. Finally, given public health efforts, there is some concern that people misreport drinking water sources they have been told to avoid.

of the campaign.

Our sample contains all births reported in the birth history module of the DHS that occurred between 1990 and 2004.[29] Our final dataset consists of 19,919 children born in 361 villages of Bangladesh, but our effect is estimated off of 6,003 births in 104 villages in which we observe at least one household drinking arsenic-laden water. Our outcome of interest being child and infant mortality, we observe all deaths occurring under 12 months, under 24 months and under 60 months.

We estimate the following difference-in-difference equation for individual i in household j and village v, which includes village fixed effects (θ):

$$Y_{ijv} = \alpha_{ijv} + \gamma * ArsenicFree_{jv} + \delta * EarlyLifeExp_{ijv} + \beta * (ArsenicFree_{jv} * EarlyLifeExp_{ijv}) + \theta_v + \epsilon_{ijv} \quad (2)$$

In this regression, *ArsenicFree* is an indicator that household drinking water is free of arsenic when tested in 2004, our proxy for whether a household switched water sources after the well-testing campaign (in this sense, it is the opposite of the *HighConc* variable of Equation 1). As in the previous set of regressions, we are interested in the coefficient estimate on the interaction between being born after the well-testing campaign and being a "switcher" household (arsenic-free). If our identifying assumption holds, this coefficient captures the change in mortality from switching to a less convenient water source.

As shown in Appendix Table 8, regression-controlled means (that account for village fixed effects) of a wide range of household and respondent characteristics are very similar across arsenic-exposed and arsenic-free households. However, to account for potential differences between switchers and non-switchers, our regressions control for the following household and child characteristics: sex, parity, birth year, birth year squared, age of mother at birth, education of mother, education of father, mean birth interval, household wealth (solvency), amount of land owned by household, number of rooms in house, whether household

[29] We limit our sample to individuals born in 1990 or later to minimize noise by restricting the comparison to mothers of the same age range and also to minimize measurement error in reported death age. However, the estimates are robust to expanding the period of observation by at least 5 years.

has electricity, and whether Muslim.[30] Standard errors are clustered at the household level.

Table 4 presents the coefficient estimates for Equation 2 with full controls. We present results for both exposure after 1999 and exposure after 2000, as it is ambiguous how quickly the campaign led to switching. As expected, the coefficient estimate on the interaction term is positive: Among households that are arsenic-free in 2004, one additional year of exposure to the post-campaign environment is associated with at least a 1.8 percentage point increase in likelihood of death within one year, a 2.5 percentage point increase in likelihood of death within two years, and 3.2 percentage point increase in likelihood of death within five years. The two-year mortality estimate is significant at the 10% level and the 5-year mortality estimate is significant at the 5% level, and the magnitudes of the estimates are similar to our estimates from Barisal (Table 2). The results are qualitatively similar using the 1999 versus the 2000 cutoff, indicating that switching behavior was spread across both years.

As described earlier, one shortcoming of our *ArsenicFree* measure is that we cannot distinguish switcher households from households that were never exposed to arsenic in groundwater, and so are underestimating the effect of abandoning tubewells potentially by a great deal. Hence, to gain more precision in identifying switchers, in the next set of regressions we make use of information provided by village leaders on the primary source of water for households in each village. In villages in which the primary water source is identified to be anything other than shallow tubewells, households with arsenic-free drinking water are more likely to be switchers than households in villages in which the primary water source is shallow tubewells. Furthermore, we can look separately at switcher households that most likely moved to surface water sources compared to those who most likely moved to deep tubewells in order to estimate the relative impact of switching to alternative sources.

Table 5 presents separate regression estimates for these three categories of villages: those in which the main source of drinking water is piped water into or outside of the house (column 1), those in which the main source of drinking water is deep tubewells (column 2), and those in which the main source of drinking water is some type of surface water source

[30]Regression estimates without controls produce very similar and in most cases statistically robust results.

(ponds, lakes, streams, etc.) (column 3). As expected, the difference-in-difference estimate is small in magnitude and insignificant in villages in which arsenic-free households are most likely using piped water, which is relatively safe in terms of exposure to fecal matter, and is large and significant in villages in which arsenic-free households are most likely to be drinking from deep tubewells or surface water sources. This suggests that the patterns we are observing in the DHS data are not driven by convergence in mortality rates over time between switcher and non-switcher households, which is a concern due to the endogeneity of switching behavior. Interestingly, there is little difference in the negative effect of switching away from shallow tubewells when the alternative source is surface versus deep tubewells, possibly indicating that the higher rate of fecal contamination in surface water relative to deep tubewell water at the source reduces to similar levels when measured at the point of use, consistent with previous studies in other settings.

Our final exercise with the BDHS makes use of detailed verbal autopsy data collected for the majority of child and infant deaths reported between 1998 and 2004 in order to verify that the patterns on child mortality we observed in the previous regression estimates are driven by deaths due to an increase in water-borne illnesses, as our interpretation implies.[31] Using these reports, we classify infant and child deaths into proximate causes of death due to water-borne pathogens, pneumonia, and fever, and run regressions analogous to Equation 2 in which the dependent variable is now a specific cause of mortality. These estimates are presented in Table 5 in columns 4-6. As expected, we observe a significant DID estimate of switching to arsenic-free drinking water on deaths attributable to water-born illnesses, but no concurrent pattern with respect to deaths attributed to fever or pneumonia. Not only does this provide an important consistency check on our interpretation of the child mortality patterns, but it minimizes the likelihood that our estimates reflect simple convergence in infant and child mortality between relatively high and relatively low SES households.

[31] Due to nonresponse, the BDHS verbal autopsy data are only available for 572 of the 606 infant and child deaths that we observe in the data between 1998 and 2004.

5 Conclusion

While the arsenic mitigation campaign in Bangladesh has been heralded by the international medical community as a life-saving effort, our estimates indicate substantial negative health consequences of public health efforts to move Bangladeshi households away from shallow tubewells as sources of drinking water. Using data from a district in Bangladesh in which shallow tubewells were readily abandoned for less convenient but arsenic-free deep tubewells, we find that households with an incentive to switch sources experienced a significant increase in the rate of infant and child mortality after arsenic levels were revealed. Hence, evaluation of future public health interventions need to reconsider efforts to convince households to abandon shallow tubewells when alternatives that are equally safe in terms of water-borne pathogens are not readily available.

Perhaps most importantly, our findings provide rigorous evidence of substantial benefits in terms of reductions in infant and child mortality to point-of-source improvements in water quality in a setting of endemic diarrheal disease.

References

Ahsan, H., Y. Chen, F. Parvez, M. Argos, I. Hussain, H. Momotaj, D. Levy, A. VanGeen, G. Howe, and J. Graziano (2006). Health effects of arsenic longitudinal study (heals): Description of a multidisciplinary epidemiologic investigation. *Journal of Exposure Science and Environmental Epidemiology 16*, 191.

Argos, M., T. Kaira, P. Rathouz, Y. Chen, B. Pierce, F. Parvez, T. Islam, A. Ahmed, M. Rakibuz-Zaman, R. Hasan, G. Sarwar, V. Slavkovich, A. van Geen, J. Graziano, and H. Ahsan (2010). Arsenic exposure from drinking water, and all-cause and chronic-disease mortalities in Bangladesh (HEALS): A prospective cohort study. *The Lancet 376*, 267.

Baqui, A., R. Black, A. Mitra, H. Chowdhury, K. Zaman, V. Fauveau, and R. Sack (1994). *Matlab: Women, Children and Health*. Dhaka: ICDDR,B.

BMOH (2004). National policy for arsenic mitigation 2004. *Bangaldesh Ministry of Health Compendium of Environment Statistics of Bangladesh 2004*, 388.

Caldwell, B., J. Caldwell, S. Mitra, and W. Smith (2003). Tubewells and arsenic in Bangladesh: Challenging a public health success story. *International Journal of Population Geography 9*, 23.

Caldwell, B., W. Smith, K. Lokuge, G. Ranmuthugala, K. Dear, A. Milton, M. Sim, J. Ng, and S. Mitra (2006). Access to drinking water and arsenicosis in Bangladesh. *Journal of Health Population and Nutrition 24*(3), 336.

Chen, Y., J. Graziano, F. Parvez, I. Hussain, H. Momotaj, A. van Geen, G. Howe, and H. Ahsan (2006). Modification of risk of arsenic-induced skin lesions by sunlight exposure, smoking, and occupational exposures in Bangladesh. *Epidemiology 17*(4), 459.

Esrey, A. (1996). Water, waste, and well-being: A multicountry study. *American Journal of Epidimiology 143*(6), 608.

Esrey, S. and J. Habicht (1986). Epidemiologic evidence for health benefits from improved water and sanitation in developing countries. *Epidemiologic Reviews 8*, 117.

Feroze Ahmed, M. (2002). Alternative water supply options for arsenic affected areas of Bangladesh. *Conference Paper, ITN-Bangladesh, Centre for Water Supply and Waste Management*.

Hoque, B., S. Huttly, K. Aziz, M. Patwary, and R. Feachem (1989). Tubewell water consumption and its determinants in a rural area of Bangladesh. *Journal of Tropical Medicine and Hygiene 92*(3), 197.

Jakariya, M. (2007). Arsenic in tubewell water of Bangladesh and approaches for sustainable mitigation. *Doctoral Thesis, KTH-International Groundwater Arsenic Research Group*.

Johnston, R. (2006). Arsenic in Bangladesh. *UNICEF WES Section Briefing*.

Kozul, C., H. Ely, R. Enelow, and J. Hamilton (2009). Low-dose arsenic comprises the immune response to influenza A infection in vivo. *Environmental Health Perspectives 117*(9), 441.

Kremer, M., J. Leino, E. Miguel, and A. Zwane (2010). Spring cleaning: Rural water impacts, valuation and property rights institutions. *Quarterly Journal of Economics*.

Liaw, J., G. Marshall, Y. Yuan, C. Ferreccio, C. Steinmaus, and A. Smith (2008). Increased childhood liver cancer mortality and arsenic in drinking water in northern Chile. *Cancer Epidemiology, Biomarkers and Prevention 17*(8), 1982.

Lindskog, U., P. Lindskog, and S. Wall (1987). Water supply, sanitation and health education programmes in developing countries: Problems of evaluation. *Scandinavian Journal of Social Medicine 15*, 123.

Lokuge, K., W. Smith, B. Caldwell, K. Dear, and A. Milton (2004). The effect of arsenic mitigation interventions on disease burden in Bangladesh. *Environmental Health Perspectives 112*(11), 1172.

Madajewicz, M., A. Pfaff, A. van Geen, J. Graziano, I. Hussein, H. Momotaj, R. Sylvi, and H. Ahsan (2007). Can information alone change behavior? Response to arsenic contamination of groundwater in Bangladesh. *Journal of Development Economics 84*(2), 731.

Mazumdar, D., J. Das Gupta, A. Sandra, A. Pal, A. Ghose, and S. Sarkar (1998). Chronic arsenic toxicity in West Bengal - the worst calamity in the world. *Journal of Indian Medical Association 96*(1), 4.

Milton, A., W. Smith, B. Rahman, Z. Hasan, U. Kulsum, K. Dear, M. Rakibuddin, and A. Ali (2005). Chronic arsenic exposure and adverse pregnancy outcomes in Bangladesh. *Epidemiology 16*(1), 82.

Morris, S., R. Black, and L. Tomaskovic (2003). Predicting the distribution of under-five deaths by cause in countries without adequate vital registration systems. *International Journal of Epidemiology 32*, 1041.

Pruss, A., D. Kay, L. Fewtrell, and J. Bartram (2002). Estimating the burden of disease from water, sanitation, and hygiene at a global level. *Environmental Health Perspectives*, 537.

Research Council, N. (2001). *Arsenic in drinking water: 2001 update*. Washington, DC: National Academic Press.

Rossman, T., A. Uddin, F. Burns, and M. Bosland (2002). Arsenite cocarcinogenesis: an animal model derived from genetic toxicology studies. *Environmental Health Perspectives 110*(Suppl 5), 749.

Schoenfeld, A. (2005). Area, village, and household response to arsenic testing and labeling of tubewells in Araihazar, Bangladesh. *Doctoral Thesis, Columbia University* (80).

Suzuki, S., L. Arnold, T. Ohnishi, and S. Cohen (2008). Effects of inorganic arsenic on the rat and mouse urinary bladder. *Toxicological Sciences 106*(2), 350.

Tofail, F., M. Vahter, J. Hamadani, B. Nermell, S. Huda, M. Yunus, M. Rahman, and S. Grantham-McGregor (2009). Effect of arsenic exposure during pregnancy on infant development at 7 months in rural Matlab, Bangladesh. *Environmental Health Perspectives 117*(2), 288.

Tucker, S., F. Li, R. Wilson, D. Byrd, S. Lai, Y. Tong, and L. Loo (2001). Relationship between consumption of arsenic-contaminated well water and skin disorders in Huhhot, Inner Mongolia. *Final Report, University of Texas, Department of Dermatology*.

UNICEF (2008). Arsenic mitigation in Bangladesh. *UNICEF Brief*.

Vachter, M. (2008). Health effects of early life exposure to arsenic. *Basic and clinical pharmacology and toxicology 102*, 204.

VanGeen, A., H. Ahsan, A. Horneman, R. Dhar, Y. Zheng, I. Hussain, K. Ahmed, A. Gelman, M. Stute, H. Simpson, S. Wallace, C. Small, F. Parvez, V. Slavkovich, N. Lolacono, M. Becker, Z. Cheng, H. Momotaj, M. Shahnewaz, A. Seddique, and J. Graziano (2002). Promotion of well-switching to mitigate the current arsenic crisis in Bangladesh. *Bulletin of the World Health Organization* (80), 732.

VanGeen, A., Y. Zheng, R. Versteeg, M. Stute, A. Horneman, R. Dhar, M. Steckler, A. Gelman, C. Small, H. Ahsan, J. Graziano, I. Hussain, and K. Ahmed (2003). Spatial variability of arsenic in 6000 tube wells in a 25 km sq area of Bangladesh. *Water Resources Research 39*(5), 1.

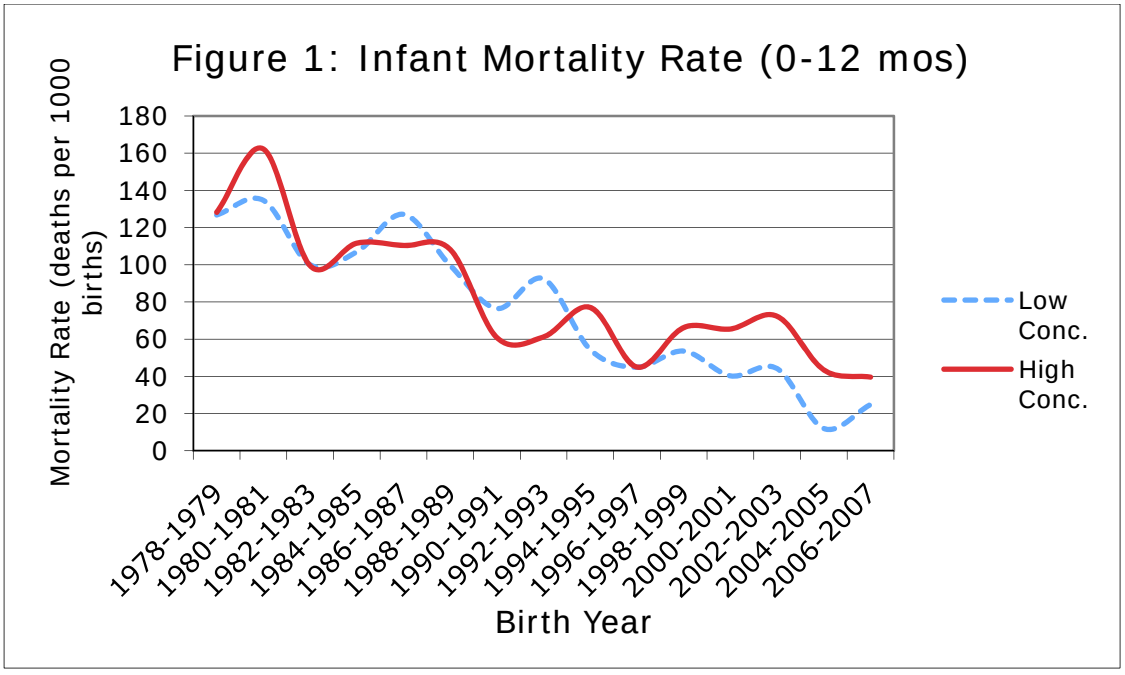
Wasserman, G., V. Liu, F. Parvez, H. Ahsan, P. Factor-Litvak, A. vanGeen, V. Slavkovich, N. Lolacono, Z. Cheng, I. Hussain, H. Momotaj, and J. Graziano (2004). Water arsenic exposure and children's intellectual function in Araihazar, Bangladesh. *Environmental Health Perspectives 112*(13), 1329.

WHO (2008). An interview with Mahmuder Rahman: Bangladesh's arsenic agony. *Bulletin of the World Health Organization 86*(1), 11.

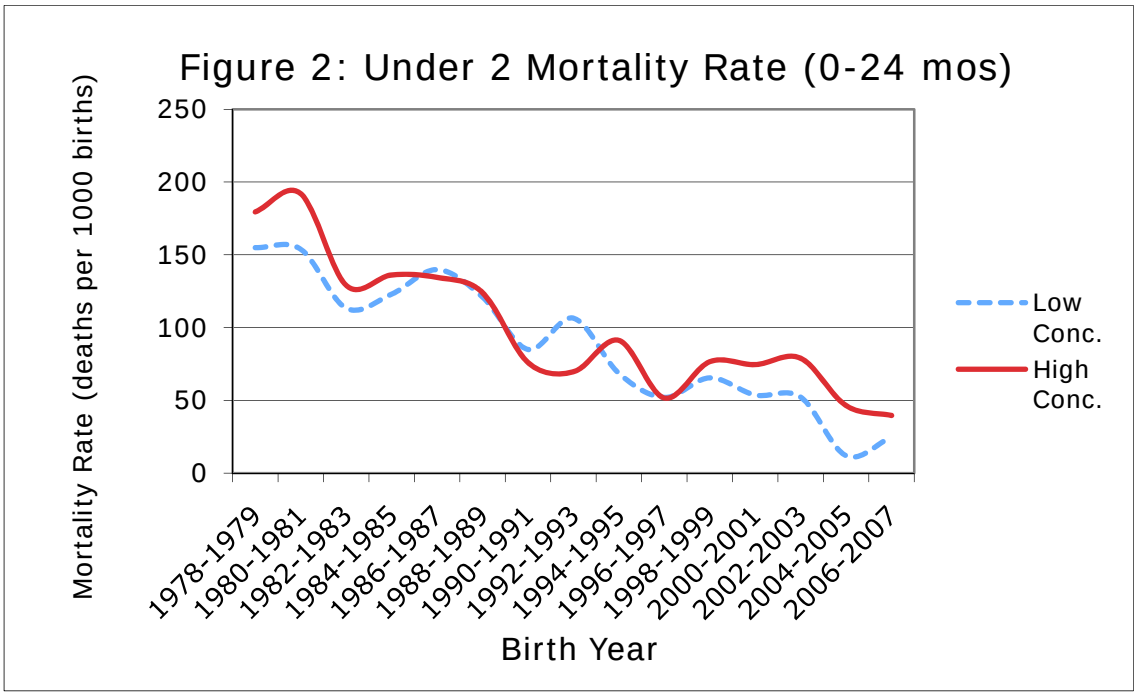
WorldBank (2007). Implementation completion and results report on a credit in the amount of SDR 24.2 million to Bangladesh for arsenic mitigation water supply. *Environment and Water Resources Unit, South Asia Region*.

Wright, J., S. Gundryl, and R. Conroy (2004). Household drinking water in developing countries: A systematic review of microbiological contamination between source and point-of-use. *Tropical Medicine and International Health 9*(1), 106.

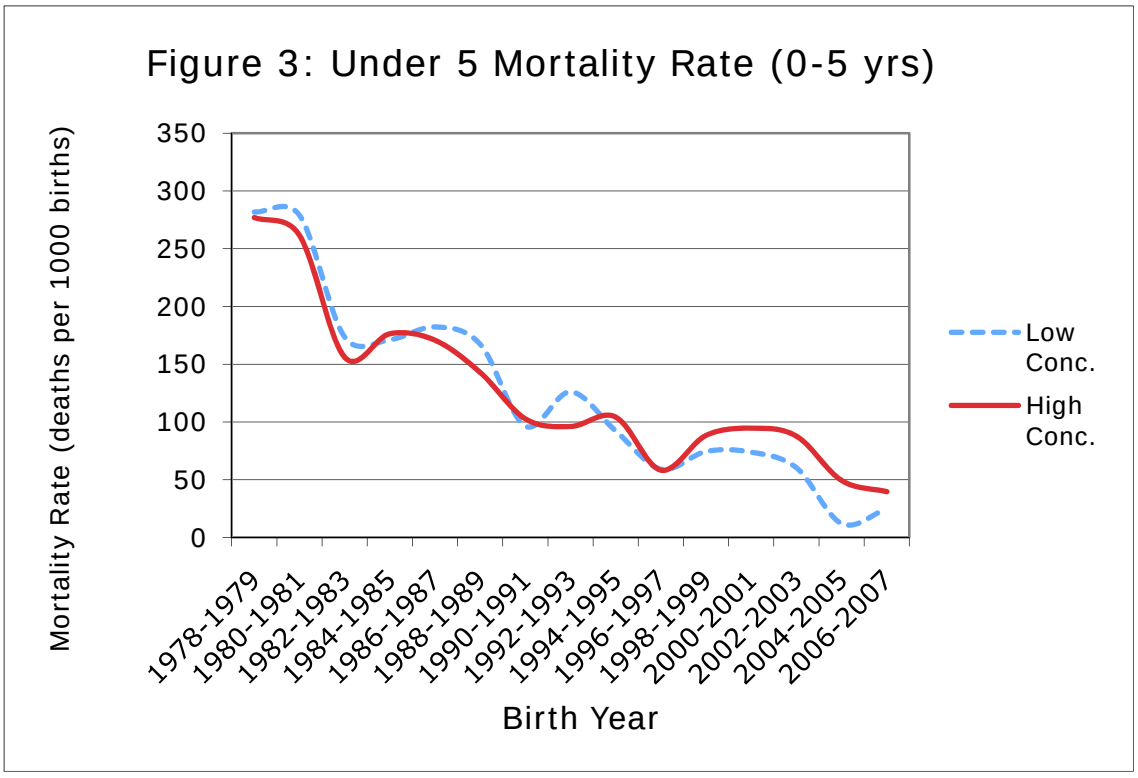
Yu, W., C. Harvey, and C. Harvey (2003). Arsenic in groundwater in Bangladesh: A geostatistical and epidemiological framework for evaluating health effects and potential remedies. *Water Resources Research 39*, 1146.



Notes: Data from the 2007 Kishoree Kontha Baseline Survey and 2009 Household Arsenic Survey in Barisal subdistrict, Bangladesh. Sample includes 11,766 births across 2,817 households in 162 villages. “Infant mortality rate” is deaths between 0 and 12 months of age per 1000 births observed in each two-year period. Average mortality rates are calculated controlling for village means. “Low conc” are households in which arsenic concentration of closest shallow tubewell < 60ppb, as measured in the 2009 survey, and “High conc” are households with >60ppb arsenic concentrations.



Notes: Data from the 2007 Kishoree Kontha Baseline Survey and 2009 Household Arsenic Survey in Barisal subdistrict, Bangladesh. Sample includes 11,766 births across 2,817 households in 162 villages. “Under 2 mortality rate” is deaths between 0 and 24 months of age per 1000 births observed in each two-year period. Average mortality rates are calculated controlling for village means. “Low conc” are households in which arsenic concentration of closest shallow tubewell < 60ppb, as measured in the 2009 survey, and “High conc” are households with >60ppb arsenic concentrations.



Notes: Data from the 2007 Kishoree Kontha Baseline Survey and 2009 Household Arsenic Survey in Barisal subdistrict, Bangladesh. Sample includes 11,766 births across 2,817 households in 162 villages. “Under 5 mortality rate” is deaths between 0 and 60 months of age per 1000 births observed in each two-year period. Average mortality rates are calculated controlling for village means. “Low conc” are households in which arsenic concentration of closest shallow tubewell < 60ppb, as measured in the 2009 survey, and “High conc” are households with >60ppb arsenic concentrations.

Table 1. Sample Means

	Measured Contamination				Reported Contamination			
	Panel I: Exogenous variables							
VARIABLES	(1) High Conc.	(2) N	(3) Low Conc.	(4) T-statistic	(5) High Conc.	(6) N	(7) Low Conc.	(8) T-statistic
Age of mother	38.58	1928/889	38.01	1.5	38.44	2332/485	38.17	0.65
Age of mother at earliest birth	21.03	1928/889	21.14	-0.4	21.03	2332/485	21.22	-0.61
Education of mother	3.43	1928/889	3.47	-0.31	3.46	2332/485	3.37	0.53
Education of father	3.76	1928/889	3.85	-0.44	3.78	2332/485	3.81	-0.15
Mean birth interval	2.28	1928/889	2.29	-0.17	2.27	2332/485	2.32	-0.87
Solvency	0.66	1928/889	0.68	-1.2	0.66	2332/485	0.69	-1.42
Land size (acres)	0.85	1928/889	0.85	0.04	0.85	2332/485	0.85	-0.02
Number of rooms in house	2.75	1928/889	2.75	0.02	2.76	2332/485	2.69	1.28
Electricity	0.39	1928/889	0.39	0.01	0.39	2332/485	0.40	-0.40
Muslim	0.97	1928/889	0.96	1.84	0.97	2332/485	0.96	0.58
Fraction of children living in household	0.80	1654/775	0.82	-1.6	0.81	2011/418	0.81	-0.18
Respondent's age	42.63	1928/889	42.31	0.64	42.54	2332/485	42.48	0.11
Male respondent	0.16	1928/889	0.16	0.08	0.16	2332/485	0.18	-1.14
Sufficiency of food per week	0.92	1928/889	0.93	-1.37	0.92	2332/485	0.93	-0.49
Outstanding loan	0.54	1928/889	0.54	0.13	0.54	2332/485	0.53	0.58
Years living in house	25.89	1928/889	24.89	1.55	25.73	2332/485	25.21	0.74
Years living in village	30.49	1547/729	30.26	0.3	30.28	1868/408	31.07	-0.94
Mean monthly income of household	11.37	1928/889	11.35	0.04	11.38	2332/485	11.28	0.21
Head of household works in agriculture	0.43	1928/889	0.41	0.55	0.41	2332/485	0.46	-1.57
Head of household works in business	0.15	1928/889	0.16	-0.62	0.16	2332/485	0.14	1.20
	Panel II: Endogenous variables							
Arsenic concentration (ppb)	94.71	1928/889	31.96	54.71	80.61	2332/485	47.50	19.29
Fraction of deaths under 12 mo.	0.07	1928/889	0.05	2.24	64.50	2332/485	44.77	2.84
Fraction of deaths under 24 mo.	0.08	1928/889	0.06	2.73	54.39	2332/485	54.36	2.77
Fraction of deaths under 60 mo.	0.09	1928/889	0.08	2.3	91.49	2332/485	74.09	2.13
M:F sex ratio	0.42	1928/889	0.47	-3.51	0.44	2332/485	0.45	-1.05
Number of offspring in family	4.21	1928/889	4.05	1.67	4.19	2332/485	4.03	1.57
Number of drinking sources used	2.05	1928/889	2.05	0.45	2.05	2332/485	2.04	1.21
Number of cooking sources used	2.24	1928/889	2.25	-0.3	2.25	2332/485	2.22	1.28
Whether closest well tested	0.69	1700/836	0.70	-0.44	0.72	2091/445	0.55	6.88
Whether closest well painted	0.68	1542/772	0.65	1.39	0.71	1922/392	0.49	8.24
Value of house ($)	2050.62	1835/864	2308.14	-2.82	2125.35	2230/469	2169.73	-0.44

Notes:
(1) All averages and t-statistics are calculated controlling for village means.
(2) 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.
(3) In columns 1-4 ("measured contamination"), high concentration versus low concentration defined according to field test of shallow tubewell closest to residence. High concentration households are those with tubewells that contain arsenic concentrations greater than 60ppb. In columns 5-8 ("reported concentration"), high concentration households are those who report that their well tested positive for arsenic concetration, or (if household has no recollection of well being tested or test result) if closest shallow tubewell currently contains arsenic concentration greater than 60ppb.

Table 2. Measured arsenic contamination and early life exposure to post-campaign environment

	Death under 12 mo.			Death under 24 mo.			Death under 60 mo.		
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
High concentration	0.00785	0.00606	0.00461	0.00998	0.00763	0.00563	0.00533	0.00132	-0.00105
	[0.00723]	[0.00722]	[0.00712]	[0.00815]	[0.00812]	[0.00797]	[0.00962]	[0.00958]	[0.00940]
Early life exposure	-0.0403***	-0.00116	0.000706	-0.0597***	-0.00495	-0.00763	-0.0941***	0.00453	-0.00675
	[0.00879]	[0.0127]	[0.0127]	[0.00930]	[0.0146]	[0.0146]	[0.0112]	[0.0189]	[0.0188]
High concentration * Early life exposure	0.0142	0.0163	0.0203*	0.0249**	0.0279**	0.0317***	0.0300**	0.0346**	0.0389***
	[0.0117]	[0.0117]	[0.0117]	[0.0121]	[0.0121]	[0.0120]	[0.0139]	[0.0138]	[0.0138]
Sex		0.0292***	0.0279***		0.0284***	0.0267***		0.0261***	0.0239***
		[0.00499]	[0.00499]		[0.00529]	[0.00528]		[0.00596]	[0.00594]
Parity		0.00308*	0.00329		0.00442***	0.00431*		0.00622***	0.00344
		[0.00157]	[0.00216]		[0.00167]	[0.00221]		[0.00176]	[0.00236]
Birth year		-0.00636***	-0.00693***		-0.00781***	-0.00889***		-0.0130***	-0.0150***
		[0.00204]	[0.00207]		[0.00208]	[0.00213]		[0.00207]	[0.00211]
(Birth year)^2		0.00006	0.00009		0.00007	0.000117		0.000135*	0.000239***
		[0.00007]	[0.00008]		[0.00008]	[0.00008]		[0.00008]	[0.00008]
Age of mother at birth			-0.000243			-0.000197			0.00008
			[0.000540]			[0.000576]			[0.000622]
Mother's education			0.00117			0.00178			0.00257
			[0.00148]			[0.00155]			[0.00165]
Father's education			-0.00146			-0.00163			-0.00208*
			[0.00107]			[0.00114]			[0.00126]
Years since birth of last child			-0.0107***			-0.0134***			-0.0172***
			[0.00178]			[0.00188]			[0.00204]
Solvency			0.000313			-0.00162			0.00178
			[0.00556]			[0.00599]			[0.00633]
Land size (acres)			0.00474***			0.00419**			0.00383**
			[0.00164]			[0.00170]			[0.00192]
No. of rooms in house			-0.0133***			-0.0143***			-0.0152***
			[0.00268]			[0.00298]			[0.00317]
Electricity			-0.00936			-0.0104			-0.0123*
			[0.00615]			[0.00667]			[0.00732]
Muslim			0.0115			0.0208			0.0168
			[0.0145]			[0.0148]			[0.0157]
Monthly income per capita			0.000214			0.000231			0.000324
			[0.000313]			[0.000328]			[0.000356]
Mean among offspring with zero exposure in households with low arsenic concentration	0.0764882	0.0764882	0.0764882	0.0961826	0.0961826	0.0961826	0.1410808	0.1410808	0.1410808
	[0.26582]	[0.26582]	[0.26582]	[0.29489]	[0.29489]	[0.29489]	[0.34819]	[0.34819]	[0.34819]
Observations	11766	11766	11766	11766	11766	11766	11766	11766	11766

Notes:
(1) Robust standard errors in brackets. *** p<0.01, ** p<0.05, * p<0.1. OLS regressions, linear probability models. Data from the 2007 Kishoree Kontha Baseline survey in Barisal subdistrict. An observation is a live birth. All specifications use village fixed effects and are clustered at the
(2) High concentration equal to 1 if closest shallow tubewell to residence revealed in field test to have arsenic concentration above 60ppb.
(3) Early life exposure measures the fraction of offspring's life before the specified age of death cutoff (12 mos, 24 mos, 60 mos) in which he/she was potentially exposed to water from the new source (fraction of years below cutoff lived post-2000).

Table 3. Reported arsenic contamination and early life exposure to post-campaign environment

	Death under 12 mo.			Death under 24 mo.			Death under 60 mo.		
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
High concentration	0.00754	0.00702	0.00749	0.00703	0.00625	0.00640	-0.00747	-0.00924	-0.00926
	[0.00820]	[0.00815]	[0.00800]	[0.00919]	[0.00918]	[0.00898]	[0.0113]	[0.0113]	[0.0110]
Early life exposure	-0.0520***	-0.0123	-0.00779	-0.0679***	-0.0121	-0.0126	-0.115***	-0.0146	-0.0237
	[0.0112]	[0.0147]	[0.0147]	[0.0122]	[0.0168]	[0.0168]	[0.0142]	[0.0207]	[0.0208]
High concentration * Early life exposure	0.0256*	0.0266**	0.0266**	0.0297**	0.0306**	0.0311**	0.0488***	0.0505***	0.0515***
	[0.0131]	[0.0131]	[0.0132]	[0.0139]	[0.0140]	[0.0141]	[0.0159]	[0.0161]	[0.0161]
Sex		0.0291***	0.0278***		0.0282***	0.0265***		0.0259***	0.0236***
		[0.00499]	[0.00498]		[0.00530]	[0.00528]		[0.00596]	[0.00594]
Parity		0.00312**	0.00334		0.00446***	0.00436**		0.00625***	0.00352
		[0.00157]	[0.00216]		[0.00167]	[0.00221]		[0.00176]	[0.00237]
Birth year		-0.00641***	-0.00696***		-0.00790***	-0.00895***		-0.0131***	-0.0150***
		[0.00204]	[0.00207]		[0.00208]	[0.00213]		[0.00207]	[0.00211]
(Birth year)^2		0.00006	0.00009		0.00007	0.000119		0.000138*	0.000240***
		[0.00007]	[0.00008]		[0.00008]	[0.00008]		[0.0008]	[0.00008]
Age of mother at birth			-0.000244			-0.000198			0.00006
			[0.000540]			[0.000576]			[0.000622]
Mother's education			0.00116			0.00178			0.00263
			[0.00149]			[0.00155]			[0.00165]
Father's education			-0.00147			-0.00165			-0.00215*
			[0.00106]			[0.00114]			[0.00126]
Years since birth of last child			-0.0106***			-0.0133***			-0.0170***
			[0.00177]			[0.00187]			[0.00203]
Solvency			0.000415			-0.00162			0.00152
			[0.00557]			[0.00600]			[0.00633]
Land size (acres)			0.00476***			0.00421**			0.00387**
			[0.00163]			[0.00169]			[0.00192]
No. of rooms in house			-0.0134***			-0.0144***			-0.0152***
			[0.00268]			[0.00298]			[0.00317]
Electricity			-0.00925			-0.0103			-0.0122*
			[0.00616]			[0.00668]			[0.00733]
Muslim			0.0118			0.0217			0.0178
			[0.0145]			[0.0149]			[0.0157]
Monthly income per capita			0.000216			0.000235			0.000330
			[0.000315]			[0.000330]			[0.000358]
Mean among offspring with zero exposure in households with low arsenic	0.0782609	0.0782609	0.0782609	0.0971548	0.0971548	0.0971548	0.1492933	0.1492933	0.1492933
	[0.26867]	[0.26867]	[0.26867]	[0.29627]	[0.29627]	[0.29627]	[0.35653]	[0.35653]	[0.35653]
Observations	11766	11766	11766	11766	11766	11766	11766	11766	11766

Notes:

(1) See notes 1 and 3 of Table 2.

(2) High concentration equal to 1 if household reported in survey that their drinking water source tested positive for arsenic contamination or, if respondent could not recall whether well was tested or test results, if shallow tubewell closest to the residence revealed in field test to have arsenic concentration above 60 ppb.

Table 4. Arsenic in drinking water in 2004 and exposure to post-campaign environment

	1999 campaign cutoff			2000 campaign cutoff		
	(1)	(2)	(3)	(4)	(5)	(6)
VARIABLES	Death under 12 mo.	Death under 24 mo.	Death under 60 mo.	Death under 12 mo.	Death under 24 mo.	Death under 60 mo.
Arsenic free	-0.00949	-0.0178	-0.0190	-0.00833	-0.0171	-0.0176
	[0.0130]	[0.0138]	[0.0146]	[0.0126]	[0.0134]	[0.0141]
Born after campaign	-0.00475	-0.00539	-0.0175	-0.00838	-0.0148	-0.0232
	[0.0153]	[0.0164]	[0.0168]	[0.0157]	[0.0169]	[0.0172]
Arsenic free * Born after campaign	0.0176	0.0245*	0.0317**	0.0177	0.0287*	0.0347**
	[0.0139]	[0.0149]	[0.0151]	[0.0141]	[0.0152]	[0.0154]
Sex	0.0166***	0.0149***	0.0115***	0.0166***	0.0149***	0.0116***
	[0.00384]	[0.00400]	[0.00424]	[0.00384]	[0.00400]	[0.00424]
Parity	0.00880***	0.00990***	0.00997***	0.00877***	0.00987***	0.00994***
	[0.00209]	[0.00222]	[0.00229]	[0.00209]	[0.00222]	[0.00229]
Birth year	-0.00003	0.00325	0.00207	-0.000704	0.00211	0.00166
	[0.00556]	[0.00577]	[0.00603]	[0.00633]	[0.00660]	[0.00695]
(Birth year)^2	-0.00009	-0.000215	-0.000210	-0.00006	-0.000164	-0.000187
	[0.000168]	[0.000174]	[0.000181]	[0.000191]	[0.000200]	[0.000209]
Age of mother at birth	-0.00189***	-0.00215***	-0.00200***	-0.00188***	-0.00213***	-0.00199***
	[0.000586]	[0.000617]	[0.000646]	[0.000587]	[0.000618]	[0.000646]
Mother's education	-0.00260***	-0.00295***	-0.00272***	-0.00261***	-0.00297***	-0.00273***
	[0.000802]	[0.000842]	[0.000874]	[0.000803]	[0.000843]	[0.000875]
Father's education	0.000004	0.00007	-0.000570	-0.000002	0.00006	-0.000578
	[0.000629]	[0.000655]	[0.000677]	[0.000629]	[0.000654]	[0.000677]
Years since birth of last child	-0.00147*	-0.00162*	-0.00221***	-0.00147*	-0.00162*	-0.00221***
	[0.000799]	[0.000826]	[0.000851]	[0.000799]	[0.000827]	[0.000851]
Solvency	0.000256	0.000338	0.00128	0.000284	0.000384	0.00131
	[0.00456]	[0.00476]	[0.00498]	[0.00457]	[0.00477]	[0.00499]
Land size (acres)	0.00121	0.000993	0.000639	0.00122	0.00100	0.000644
	[0.000869]	[0.000872]	[0.000911]	[0.000870]	[0.000873]	[0.000912]
No. of rooms in house	-0.00448***	-0.00530***	-0.00660***	-0.00449***	-0.00531***	-0.00661***
	[0.00137]	[0.00141]	[0.00147]	[0.00137]	[0.00141]	[0.00147]
Electricity	0.000926	0.000491	-0.00230	0.000866	0.000409	-0.00237
	[0.00556]	[0.00576]	[0.00604]	[0.00556]	[0.00577]	[0.00604]
Muslim	-0.00334	-0.000825	0.00286	-0.00326	-0.000673	0.00297
	[0.00943]	[0.00962]	[0.0101]	[0.00943]	[0.00961]	[0.0101]
Mean among offspring born before the campaign in households with high arsenic concentration	0.0801173	0.0897654	0.1040445	0.0789935	0.0884559	0.1018434
	[0.27149]	[0.28586]	[0.30533]	[0.26974]	[0.28397]	[0.30245]
Observations	19919	19919	19919	19919	19919	19919

Notes:
(1) OLS regressions, linear probability models. Data from the 2004 Bangladesh Demogrpahic and Health Survey. Observation is a live birth. All specifications use village fixed effects and are clustered at the household level.
(2) "Arsenic-free" defined from survey field test of household drinking water, and equal to 1 if arsenic concentration less than 50ppb.
(3) In columns 1-3, "Born after campaign" equal to 1 if child born after 1999; in columns 4-6, "Born after campaign" equal to 1 if child born after 2000.

Table 5. Heterogeneity according to village-level water source and cause of death

VARIABLES	(1) Death under 60 mo.	(2) Death under 60 mo.	(3) Death under 60 mo.	(4) Water related death	(5) Death by pneumonia	(6) Death by fever
	Subsample: Villages where primary water source is piped water	*Subsample: Villages where primary water source is deep tubewell*	*Subsample: Villages where primary water source is surface water*	*Subsample: All births within past 8 years in villages where primary water source is deep tubewell or surface water*		
Arsenic free	-0.0444	-0.0553	-0.0388	-0.0960	-0.0183	-0.0160
	[0.0423]	[0.0528]	[0.0475]	[0.0659]	[0.0397]	[0.0531]
Born after 2000	0.0117	-0.101*	-0.0370	-0.0879	-0.0258	-0.0383
	[0.0490]	[0.0561]	[0.0661]	[0.0673]	[0.0426]	[0.0630]
Arsenic free * Born after 2000	0.0327	0.100**	0.139**	0.110*	0.0508	0.0475
	[0.0465]	[0.0485]	[0.0547]	[0.0638]	[0.0403]	[0.0582]
Sex	0.0229	0.00350	-0.00949	-0.00184	-0.00683	-0.0155
	[0.0147]	[0.0126]	[0.0221]	[0.00966]	[0.00876]	[0.0105]
Parity	0.00999	0.00138	-0.0134	0.00215	-0.00260	0.00435
	[0.00828]	[0.00582]	[0.00984]	[0.00406]	[0.00505]	[0.00513]
Birth year	0.0232	0.0317*	0.0516	0.134**	0.0563	-0.0240
	[0.0230]	[0.0190]	[0.0356]	[0.0568]	[0.0486]	[0.0703]
(Birth year)^2	-0.000775	-0.000925	-0.00174	-0.00322**	-0.00139	0.000376
	[0.000700]	[0.000581]	[0.00109]	[0.00131]	[0.00114]	[0.00157]
Age of mother at birth	0.000183	-0.000462	0.00122	0.000689	0.000901	-0.000719
	[0.00211]	[0.00165]	[0.00315]	[0.00104]	[0.00124]	[0.00113]
Mother's education	-0.00566**	-0.00392	-0.00707	-0.00216	-0.00152	-0.00418*
	[0.00241]	[0.00280]	[0.00493]	[0.00200]	[0.00174]	[0.00237]
Father's education	0.00403*	-0.00353*	-0.00484	-0.00229	-0.000778	0.000982
	[0.00214]	[0.00183]	[0.00311]	[0.00142]	[0.000994]	[0.00156]
Years since birth of last child	-0.00391	-0.000123	0.00344	0.000861	0.00134	0.00176
	[0.00307]	[0.00232]	[0.00643]	[0.00171]	[0.00144]	[0.00156]
Solvency	0.000568	0.00529	0.00903	0.00721	-0.00963	-0.00881
	[0.0165]	[0.0152]	[0.0259]	[0.0118]	[0.00845]	[0.0114]
Land size (acres)	-0.000870	-0.00133	-0.00361**	-0.000997	-0.00125	-0.000303
	[0.00163]	[0.00168]	[0.00155]	[0.000831]	[0.00101]	[0.000857]
No. of rooms in house	-0.00901**	0.00148	-0.000155	0.00257	0.00415	0.00561
	[0.00440]	[0.00419]	[0.00627]	[0.00362]	[0.00306]	[0.00391]
Electricity	-0.0372	0.00667	-0.0512	0.0247	-0.00625	-0.0104
	[0.0276]	[0.0187]	[0.0337]	[0.0167]	[0.0153]	[0.0175]
Muslim	0.00414	-0.0317	0.0399	0.0519**	0.0487	0.0152
	[0.0285]	[0.0359]	[0.0465]	[0.0260]	[0.0331]	[0.0319]
Mean among offspring born before the campaign in households with high arsenic concentration	0.0833333	0.15	0.1506849	0.1111111	0.037037	0.0740741
	[0.28233]	[0.36008]	[0.36022]	[0.32036]	[0.19245]	[0.26688]
Observations	1444	1787	928	1122	1122	1122

Notes:

(1) See notes to Table 4.

(2) Subsample for (1) is all villages in which primary water source reported by community leader to be piped water. Subsample of (2) is all villages in which primary water source reported by community leader to be a deep tubewell. Subsample of (3) is all villages in which primary water source reported by community leader to be surface water (surface wells, ponds, lakes, streams, etc.)

(3) Subample for (4), (5), and (6) are individuals born after 1997 in villages in which primary water source reported by community leader to b a deep tubewell or surface water. Verbal autopsy data are only collected for deaths since 1997, and are available for only 572 out of 606 reported infant and child deaths int he sample due to nonresponse.

Appendix Table 1. Sample means without village fixed effects

VARIABLES	High Conc.	N	Low Conc.	T-statistic
Panel I: Exogenous Variables				
Age of mother	38.48	1928/889	38.23	0.79
Age of mother at earliest birth	20.87	1928/889	21.47	-2.59
Education of mother	3.59	1928/889	3.11	3.73
Education of father	3.97	1928/889	3.40	0.001
Mean birth interval	2.30	1928/889	2.24	1.31
Solvency	0.65	1928/889	0.69	-1.91
Land size	0.85	1928/889	0.85	0.08
Number of rooms in house	2.79	1928/889	2.67	2.7
Electricity	0.44	1928/889	0.30	7
Muslim	0.96	1928/889	0.98	-1.67
Fraction of children living in household	0.81	1654/775	0.82	-0.95
Respondent's age	42.63	1928/889	42.31	0.79
Male respondent	0.16	1928/889	0.16	0.32
Sufficiency of food per week	0.91	1928/889	0.95	-3.2
Outstanding loan	0.56	1928/889	0.51	2.26
Years living in house	26.21	1928/889	24.20	3.67
Years living in village	30.61	1547/729	30.02	0.88
Mean monthly income of household	11.55	1928/889	10.97	1.47
Head of household works in agriculture	0.40	1928/889	0.47	-3.71
Head of household works in business	0.16	1928/889	0.15	1.2
Panel II: Endogenous Variables				
Arsenic concentration (ppm)	96.26	1928/889	28.60	65.34
Whether closest well tested	0.68	1700/836	0.71	-1.78
Whether closest well painted	0.67	1542/772	0.66	0.54
Fraction of deaths under 12 mo.	0.06	1928/889	0.06	1.59
Fraction of deaths under 24 mo.	0.07	1928/889	0.06	1.9
Fraction of deaths under 60 mo.	0.09	1928/889	0.08	1.15
M:F sex ratio	0.43	1928/889	0.46	-3.32
Number of offspring in family	4.18	1928/889	4.14	0.5
Number of drinking sources used	2.05	1928/889	2.06	-0.93
Number of cooking sources used	2.25	1928/889	2.23	1.28
Value of house ($)	2190.39	1835/864	2011.29	2.24

Notes:

(1) See notes to Table 1.

(2) High concentration and low concentration are defined according to measured concetration of arsenic in nearest shallow tubewell, with cutoff point of 60ppb.

(3) Sample means and mean differences do not account of village fixed effects.

Appendix Table 2. Measured arsenic contamination and early life exposure to post-campaign environment, household-level estimation

	Fraction of deaths per HH under 12 mo.			Fraction of deaths per HH under 24 mo.			Fraction of deaths per HH under 60 mo.		
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
High concentration	0.00695	0.00640	0.00407	0.0117	0.0110	0.00782	0.00831	0.00702	0.00354
	[0.00775]	[0.00774]	[0.00771]	[0.00815]	[0.00798]	[0.00793]	[0.00928]	[0.00873]	[0.00861]
Fraction born after 2000	-0.0476***	-0.0190	-0.0130	-0.0596***	-0.0247	-0.0163	-0.0844***	-0.0269	-0.0188
	[0.0151]	[0.0176]	[0.0176]	[0.0154]	[0.0175]	[0.0175]	[0.0176]	[0.0193]	[0.0192]
High conc. * Fraction born after 2000	0.0297*	0.0276	0.0317*	0.0256	0.0233	0.0286	0.0330	0.0304	0.0359*
	[0.0177]	[0.0183]	[0.0188]	[0.0181]	[0.0187]	[0.0192]	[0.0200]	[0.0208]	[0.0211]
M:F sex ratio		0.0116	0.00713		0.0128	0.00685		0.00947	0.00191
		[0.0109]	[0.0104]		[0.0110]	[0.0104]		[0.0116]	[0.0109]
Number of offspring in family		0.0155***	0.0176***		0.0177***	0.0199***		0.0202***	0.0239***
		[0.00171]	[0.00205]		[0.00184]	[0.00226]		[0.00219]	[0.00258]
Earliest child birth year in family		0.000356	-0.00108		0.000284	-0.00155*		-0.000694	-0.00258**
		[0.000623]	[0.000771]		[0.000688]	[0.000874]		[0.000789]	[0.000997]
Age of mother at earliest birth			0.00185***			0.00197***			0.00293***
			[0.000700]			[0.000727]			[0.000775]
Education of mother			0.00288*			0.00365**			0.00462**
			[0.00171]			[0.00170]			[0.00179]
Education of father			-0.00184*			-0.00201*			-0.00236**
			[0.00108]			[0.00113]			[0.00114]
Mean birth interval			-0.0103***			-0.0132***			-0.0154***
			[0.00273]			[0.00287]			[0.00311]
Solvency			0.000184			0.000536			0.00336
			[0.00531]			[0.00571]			[0.00624]
Land size (acres)			0.00376**			0.00339*			0.00325
			[0.00176]			[0.00176]			[0.00201]
Number of rooms in house			-0.0114***			-0.0121***			-0.0138***
			[0.00280]			[0.00293]			[0.00309]
Electricity			-0.00494			-0.00625			-0.00407
			[0.00562]			[0.00587]			[0.00663]
Muslim			0.00667			0.0130			0.00849
			[0.0112]			[0.0110]			[0.0129]
Monthly income per capita			0.000228			0.000267			0.000441
			[0.000281]			[0.000278]			[0.000321]
Mean among offspring with zero exposure in households with low arsenic concentration	0.0765661	0.0765661	0.0765661	0.0835267	0.0835267	0.0835267	0.1229698	0.1229698	0.1229698
	[0.26621]	[0.26621]	[0.26621]	[0.27700]	[0.27700]	[0.27700]	[0.32878]	[0.32878]	[0.32878]
Observations	2817	2817	2817	2817	2817	2817	2817	2817	2817

Notes:

(1) See notes to Table 2.

(2) An observation is a household. Outcome is fraction of live births that died before given age cutoff.

(3) High concentration defined by arsenic test of closest well to household ("measured contamination").

Appendix Table 3. Measured arsenic contamination and early life exposure to post-campaign environment, binary exposure

	Death under 12 mo.			Death under 24 mo.			Death under 60 mo.		
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
High concentration	0.00785	0.00606	0.00461	0.0135*	0.0110	0.00905	0.0128	0.00865	0.00661
	[0.00723]	[0.00722]	[0.00712]	[0.00777]	[0.00771]	[0.00756]	[0.00850]	[0.00840]	[0.00821]
Born after 2000	-0.0403***	-0.00116	0.000706	-0.0530***	-0.00814	-0.00578	-0.0692***	-0.00460	-0.00272
	[0.00879]	[0.0127]	[0.0127]	[0.00931]	[0.0135]	[0.0135]	[0.0106]	[0.0152]	[0.0152]
High concentration * Born	0.0142	0.0163	0.0203*	0.0158	0.0184	0.0233*	0.0162	0.0200	0.0255*
after 2000	[0.0117]	[0.0117]	[0.0117]	[0.0124]	[0.0124]	[0.0124]	[0.0138]	[0.0136]	[0.0137]
Sex		0.0292***	0.0279***		0.0287***	0.0270***		0.0269***	0.0243***
		[0.00499]	[0.00499]		[0.00528]	[0.00527]		[0.00588]	[0.00587]
Parity		0.00308*	0.00329		0.00442***	0.00425*		0.00620***	0.00335
		[0.00157]	[0.00216]		[0.00167]	[0.00221]		[0.00176]	[0.00236]
Birth year		-0.00636***	-0.00693***		-0.00842***	-0.00904***		-0.0134***	-0.0145***
		[0.00204]	[0.00207]		[0.00218]	[0.00222]		[0.00243]	[0.00246]
(Birth year)^2		0.00006	0.00009		0.000103	0.000131		0.000188**	0.000241***
		[0.00007]	[0.00008]		[0.00008]	[0.00008]		[0.00009]	[0.00009]
Age of mother at birth			-0.000243			-0.000192			0.00009
			[0.000540]			[0.000576]			[0.000622]
Mother's education			0.00117			0.00175			0.00253
			[0.00148]			[0.00155]			[0.00164]
Father's education			-0.00146			-0.00164			-0.00212*
			[0.00107]			[0.00114]			[0.00126]
Years since birth of last child			-0.0107***			-0.0135***			-0.0173***
			[0.00178]			[0.00188]			[0.00204]
Solvency (acres)			0.000313			-0.00165			0.00170
			[0.00556]			[0.00599]			[0.00633]
Land size			0.00474***			0.00420**			0.00387**
			[0.00164]			[0.00170]			[0.00193]
No. of rooms in house			-0.0133***			-0.0143***			-0.0152***
			[0.00268]			[0.00298]			[0.00317]
Electricity			-0.00936			-0.0105			-0.0125*
			[0.00615]			[0.00668]			[0.00734]
Muslim			0.0115			0.0211			0.0172
			[0.0145]			[0.0148]			[0.0157]
Monthly income per capita			0.000214			0.000227			0.000313
			[0.000313]			[0.000328]			[0.000356]
Mean among offspring with	0.0764882	0.0764882	0.0764882	0.090123	0.090123	0.090123	0.1173928	0.1173928	0.1173928
zero exposure in households	[0.26582]	[0.26582]	[0.26582]	[0.28641]	[0.28641]	[0.28641]	[0.32194]	[0.32194]	[0.32194]
with low arsenic									
Observations	11766	11766	11766	11766	11766	11766	11766	11766	11766

Notes:
(1) See notes to Table 2.

Appendix Table 4. Measured arsenic contamination and early life exposure to post-campaign environment based on when well tested

	Death under 12 mo.			Death under 24 mo.			Death under 60 mo.		
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
High concentration	0.00733	0.00541	0.00422	0.0134*	0.0107	0.00905	0.0129	0.00844	0.00672
	[0.00707]	[0.00706]	[0.00697]	[0.00760]	[0.00754]	[0.00740]	[0.00826]	[0.00815]	[0.00797]
Born after well tested	-0.0499***	-0.0200*	-0.0191	-0.0607***	-0.0235*	-0.0222*	-0.0766***	-0.0235	-0.0224
	[0.00914]	[0.0120]	[0.0120]	[0.00986]	[0.0132]	[0.0132]	[0.0110]	[0.0146]	[0.0146]
High concentration * Born	0.0244**	0.0273**	0.0307**	0.0236*	0.0273**	0.0317**	0.0233	0.0289**	0.0339**
after well tested	[0.0124]	[0.0124]	[0.0125]	[0.0132]	[0.0132]	[0.0133]	[0.0143]	[0.0143]	[0.0145]
Sex		0.0292***	0.0279***		0.0287***	0.0270***		0.0269***	0.0243***
		[0.00499]	[0.00499]		[0.00529]	[0.00527]		[0.00589]	[0.00587]
Parity		0.00311**	0.00330		0.00444***	0.00426*		0.00623***	0.00336
		[0.00158]	[0.00216]		[0.00167]	[0.00221]		[0.00176]	[0.00236]
Birth year		-0.00728***	-0.00801***		-0.00912***	-0.00991***		-0.0144***	-0.0157***
		[0.00194]	[0.00197]		[0.00210]	[0.00214]		[0.00234]	[0.00236]
(Birth year)^2		0.000109	0.000141**		0.000137*	0.000174**		0.000238***	0.000299***
		[0.00007]	[0.00007]		[0.00007]	[0.00007]		[0.00008]	[0.00008]
Age of mother at birth			-0.000254			-0.000201			0.00008
			[0.000542]			[0.000577]			[0.000623]
Mother's education			0.00116			0.00174			0.00252
			[0.00148]			[0.00155]			[0.00165]
Father's education			-0.00142			-0.00161			-0.00208*
			[0.00107]			[0.00114]			[0.00126]
Years since birth of last child			-0.0107***			-0.0135***			-0.0173***
			[0.00178]			[0.00188]			[0.00204]
Solvency			0.000327			-0.00161			0.00173
			[0.00556]			[0.00599]			[0.00633]
Land size (acres)			0.00470***			0.00418**			0.00384**
			[0.00164]			[0.00170]			[0.00192]
No. of rooms in house			-0.0132***			-0.0142***			-0.0151***
			[0.00268]			[0.00298]			[0.00317]
Electricity			-0.00908			-0.0102			-0.0122*
			[0.00615]			[0.00667]			[0.00733]
Muslim			0.0111			0.0207			0.0167
			[0.0145]			[0.0148]			[0.0157]
Monthly income per capita			0.000212			0.000223			0.000309
			[0.000313]			[0.000327]			[0.000356]
Mean among offspring with	0.0755723	0.0755723	0.0755723	0.088429	0.088429	0.088429	0.1147695	0.1147695	0.1147695
zero exposure in households	[0.26435]	[0.26435]	[0.26435]	[0.28396]	[0.28396]	[0.28396]	[0.31879]	[0.31879]	[0.31879]
with low arsenic									
Observations	11766	11766	11766	11766	11766	11766	11766	11766	11766

Notes:

(1) See notes to Table 2.

(2) Whether individual born after well tested based on survey reports of year that drinking water tested for arsenice.

Appendix Table 5. Control test: 100 ppb arsenic contamination cutoff

	Death under 12 mo.			Death under 24 mo.			Death under 60 mo.		
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
High concentration	0.00824	0.00700	0.00693	-0.00172	-0.00306	-0.00298	0.00259	0.00105	0.000459
	[0.00905]	[0.00905]	[0.00870]	[0.0102]	[0.0102]	[0.00987]	[0.0122]	[0.0121]	[0.0118]
Early life exposure	-0.0273***	-0.00425	0.00209	-0.0420***	0.0111	0.0122	-0.0684***	0.0306	0.0225
	[0.00897]	[0.0152]	[0.0152]	[0.00861]	[0.0166]	[0.0167]	[0.00932]	[0.0212]	[0.0213]
High concentration*Early life	-0.000970	-0.00178	-0.00143	0.0247	0.0233	0.0259	0.0125	0.0105	0.0147
exposure	[0.0181]	[0.0183]	[0.0181]	[0.0195]	[0.0196]	[0.0194]	[0.0201]	[0.0201]	[0.0199]
Sex		0.0315***	0.0302***		0.0304***	0.0287***		0.0288***	0.0265***
		[0.00627]	[0.00628]		[0.00651]	[0.00653]		[0.00727]	[0.00727]
Parity		0.00224	0.00246		0.00327	0.00291		0.00535**	0.00265
		[0.00197]	[0.00272]		[0.00203]	[0.00275]		[0.00215]	[0.00294]
Birth year		-0.00863***	-0.00853***		-0.00941***	-0.00982***		-0.0132***	-0.0145***
		[0.00252]	[0.00257]		[0.00260]	[0.00267]		[0.00254]	[0.00261]
(Birth year)^2		0.000175*	0.000179*		0.000127	0.000156		0.000152	0.000236**
		[0.00009]	[0.0001]		[0.000100]	[0.000104]		[9.89e-05]	[0.000103]
Age of mother at birth			0.0001			0.000392			0.000522
			[0.000659]			[0.000709]			[0.000755]
Mother's education			0.000779			0.00150			0.00153
			[0.00171]			[0.00179]			[0.00192]
Father's education			-0.00242**			-0.00203			-0.00194
			[0.00120]			[0.00130]			[0.00144]
Years since birth of last child			-0.0109***			-0.0135***			-0.0167***
			[0.00206]			[0.00216]			[0.00236]
Solvency			-0.00623			-0.00791			-0.000402
			[0.00669]			[0.00715]			[0.00755]
Land size (acres)			0.00476***			0.00361**			0.00350*
			[0.00178]			[0.00178]			[0.00206]
No. of rooms in house			-0.00974***			-0.0106***			-0.0114***
			[0.00318]			[0.00343]			[0.00367]
Electricity			-0.00867			-0.0122			-0.0160*
			[0.00704]			[0.00764]			[0.00838]
Muslim			0.0120			0.0230			0.0130
			[0.0167]			[0.0176]			[0.0189]
Monthly income per capita			0.000368			0.000329			0.000405
			[0.000341]			[0.000359]			[0.000403]
Mean among offspring with	0.0814126	0.0814126	0.0814126	0.0993445	0.0993445	0.0993445	0.1361418	0.1361418	0.1361418
zero exposure in households	[0.27349]	[0.27349]	[0.27349]	[0.29915]	[0.29915]	[0.29915]	[0.34298]	[0.34298]	[0.34298]
with low arsenic									
Observations	8120	8120	8120	8120	8120	8120	8120	8120	8120

Notes:

(1) See notes to Table 2.

(2) High concentration defined by arsenic test of closest well to residence being above 100 ppb.

(3) Sample restricted to households with arsenic concentrations greater than 60 ppb.

Appendix Table 6. Measured arsenic contamination and early life exposure to post-campaign environment excluding recent wells

	Death under 12 mo.			Death under 24 mo.			Death under 60 mo.		
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
High concentration	0.00537	0.00397	0.00133	0.00578	0.00373	0.000384	-0.00142	-0.00491	-0.00915
	[0.00959]	[0.00963]	[0.00941]	[0.0108]	[0.0109]	[0.0105]	[0.0129]	[0.0129]	[0.0125]
Early life exposure	-0.0450***	-0.0122	-0.00896	-0.0639***	-0.0165	-0.0177	-0.0939***	0.00948	-0.00245
	[0.0125]	[0.0171]	[0.0170]	[0.0133]	[0.0197]	[0.0195]	[0.0158]	[0.0248]	[0.0246]
High concentration*Early life	0.0208	0.0228	0.0263*	0.0305*	0.0339**	0.0379**	0.0306*	0.0355*	0.0413**
exposure	[0.0155]	[0.0154]	[0.0154]	[0.0161]	[0.0162]	[0.0160]	[0.0185]	[0.0185]	[0.0183]
Sex		0.0270***	0.0261***		0.0258***	0.0243***		0.0225***	0.0205***
		[0.00617]	[0.00615]		[0.00653]	[0.00650]		[0.00736]	[0.00734]
Parity		0.00280	0.00281		0.00367*	0.00371		0.00537**	0.00382
		[0.00188]	[0.00258]		[0.00202]	[0.00269]		[0.00218]	[0.00287]
Birth year		-0.00780***	-0.00833***		-0.00887***	-0.00996***		-0.0139***	-0.0161***
		[0.00258]	[0.00261]		[0.00264]	[0.00269]		[0.00260]	[0.00264]
(Birth year)^2		0.000121	0.000133		0.000116	0.000155		0.000154	0.000253**
		[0.0001]	[0.0001]		[0.000102]	[0.000104]		[0.0001]	[0.000102]
Age of mother at birth			0.000113			0.000117			0.00005
			[0.000642]			[0.000689]			[0.000747]
Mother's education			0.00370**			0.00462**			0.00528***
			[0.00181]			[0.00188]			[0.00200]
Father's education			-0.00194			-0.00260*			-0.00307**
			[0.00133]			[0.00139]			[0.00153]
Years since birth of last child			-0.00966***			-0.0123***			-0.0160***
			[0.00219]			[0.00232]			[0.00254]
Solvency			-0.00211			0.000383			0.00653
			[0.00702]			[0.00749]			[0.00783]
Land size (acres)			0.00475***			0.00367**			0.00292
			[0.00174]			[0.00176]			[0.00203]
No. of rooms in house			-0.0183***			-0.0194***			-0.0193***
			[0.00332]			[0.00353]			[0.00369]
Electricity			-0.00798			-0.0123			-0.0171**
			[0.00729]			[0.00791]			[0.00869]
Muslim			-0.00472			0.00661			0.000819
			[0.0175]			[0.0181]			[0.0195]
Monthly income per capita			0.000355			0.000349			0.000449
			[0.000389]			[0.000397]			[0.000423]
Mean among offspring with zero	0.0837182	0.0837182	0.0837182	0.1031593	0.1031593	0.1031593	0.145201	0.145201	0.145201
exposure in households with low	[0.27704]	[0.27704]	[0.27704]	[0.30426]	[0.30426]	[0.30426]	[0.35245]	[0.35245]	[0.35245]
arsenic concentration									
Observations	7746	7746	7746	7746	7746	7746	7746	7746	7746

Notes:

(1) See notes to Table 2.

(2) Households with closest wells that were, according to survey data, constructed less than 8 years ago, are dropped from the sample.

Appendix Table 7. Measured arsenic contamination and early life exposure to post-campaign environment, 1999 campaign cutoff

	Death under 12 mo.			Death under 24 mo.			Death under 60 mo.		
VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
High concentration	0.00503	0.00328	0.00186	0.00940	0.00691	0.00487	0.00389	-0.00009	-0.00255
	[0.00745]	[0.00745]	[0.00735]	[0.00835]	[0.00831]	[0.00817]	[0.0101]	[0.0100]	[0.00986]
Early life exposure	-0.0460***	-0.00514	-0.00597	-0.0567***	0.00241	-0.00287	-0.0965***	-0.00272	-0.0150
	[0.00852]	[0.0123]	[0.0123]	[0.00898]	[0.0132]	[0.0133]	[0.0112]	[0.0173]	[0.0173]
High concentration*Early life	0.0241**	0.0264**	0.0296***	0.0229**	0.0260**	0.0294**	0.0289**	0.0337**	0.0376***
exposure	[0.0112]	[0.0112]	[0.0111]	[0.0115]	[0.0115]	[0.0115]	[0.0137]	[0.0137]	[0.0136]
Sex		0.0290***	0.0277***		0.0280***	0.0264***		0.0265***	0.0242***
		[0.00499]	[0.00498]		[0.00534]	[0.00533]		[0.00594]	[0.00592]
Parity		0.00308*	0.00331		0.00439***	0.00431*		0.00626***	0.00341
		[0.00158]	[0.00216]		[0.00167]	[0.00221]		[0.00176]	[0.00236]
Birth year		-0.00622***	-0.00706***		-0.00765***	-0.00888***		-0.0138***	-0.0156***
		[0.00194]	[0.00198]		[0.00193]	[0.00199]		[0.00200]	[0.00203]
(Birth year)^2		0.00005	0.00009		0.00005	0.000109		0.000174***	0.000276***
		[0.00007]	[0.00007]		[0.00007]	[0.00007]		[0.00007]	[0.00007]
Age of mother at birth			-0.000248			-0.000198			0.00008
			[0.000541]			[0.000576]			[0.000622]
Mother's education			0.00121			0.00177			0.00257
			[0.00148]			[0.00155]			[0.00164]
Father's education			-0.00146			-0.00163			-0.00207
			[0.00107]			[0.00114]			[0.00126]
Years since birth of last child			-0.0107***			-0.0134***			-0.0172***
			[0.00177]			[0.00188]			[0.00203]
Solvency			0.000331			-0.00157			0.00176
			[0.00556]			[0.00599]			[0.00633]
Land size (acres)			0.00472***			0.00418**			0.00382**
			[0.00164]			[0.00170]			[0.00193]
No. of rooms in house			-0.0133***			-0.0143***			-0.0153***
			[0.00268]			[0.00298]			[0.00317]
Electricity			-0.00926			-0.0103			-0.0123*
			[0.00615]			[0.00667]			[0.00732]
Muslim			0.0113			0.0206			0.0169
			[0.0145]			[0.0148]			[0.0157]
Monthly income per capita			0.000217			0.000238			0.000321
			[0.000314]			[0.000328]			[0.000356]
Mean among offspring with	0.0796491	0.0796491	0.0796491	0.0962609	0.0962609	0.0962609	0.1458774	0.1458774	0.1458774
zero exposure in households	[0.27080]	[0.27080]	[0.27080]	[0.29401]	[0.29401]	[0.29401]	[0.35308]	[0.35308]	[0.35308]
with low arsenic concentration									
Observations	11766	11766	11766	11766	11766	11766	11766	11766	11766

Notes:

(1) See notes to Table 2.

(2) Early life exposure defined according to campaign date of 1999 rather than 2000.

Appendix Table 8. DHS Sample Means

VARIABLES	Arsenic free	N	Contaminated	T-statistic
		Panel I: Exogenous variables		
Age of mother	29.72	7345/541	29.47	0.5
Age of mother at earliest birth	20.02	7345/541	19.58	1.27
Education of mother	3.38	7345/541	3.32	0.26
Education of father	4.47	7345/541	4.68	-0.83
Mean birth interval	3.04	7345/541	3.12	-0.37
Solvency	0.51	7345/541	0.51	0.12
Land size (acres)	0.67	7345/541	0.70	-0.14
Number of rooms in house	2.93	7345/541	2.98	-0.51
Electricity	0.43	7345/541	0.47	-1.74
Muslim	0.89	7345/541	0.89	0.59
Years living in house	26.90	3070/205	26.02	0.7
Head of household works in agriculture	0.33	3135/215	0.38	-1.24
Head of household works in business	0.43	3135/215	0.43	0.19
Sufficient earnings	2.17	2901/197	2.13	0.62
		Panel II: Endogenous variables		
Arsenic concentration (ppb)	1.54	7345/517	6.76	-94.86
Whether well painted	0.35	6250/520	0.40	-1.92
Fraction of deaths under 12 mo.	0.06	7345/541	0.06	-0.01
Fraction of deaths under 24 mo.	0.07	7345/541	0.07	-0.33
Fraction of deaths under 60 mo.	0.08	7345/541	0.08	-0.2
M:F sex ratio	0.52	7345/541	0.49	1.25
Number of offspring in family	0.25	7345/541	0.26	-0.51
Deep tubewell mentioned as a source for drinking	0.04	7341/540	0.02	2.45

Notes:
All means account for village fixed effects
Solvency defined sufficient food consumption in whole year
Sufficient earnings defined as whether man believes his earnings sufficient for family, asked only of households in which husband interviewed.