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Article

Diarrhoeal Health Risks Attributable to Water-Borne-Pathogens in Arsenic-Mitigated Drinking Water in West Bengal are Largely Independent of the Microbiological Quality of the Supplied Water

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Abstract: There is a growing discussion about the possibility of arsenic mitigation measures in Bengal and similar areas leading to undesirable substitution of water-borne-pathogen attributable risks pathogens for risks attributable to arsenic, in part because of uncertainties in relative pathogen concentrations in supplied and end-use water. We try to resolve this discussion, by assessing the relative contributions of water supply and end-user practices to

water-borne-pathogen-attributable risks for arsenic mitigation options in a groundwater arsenic impacted area of West Bengal. Paired supplied arsenic-mitigated water and end-use drinking water samples from 102 households were collected and analyzed for arsenic and thermally tolerant coliforms [TTC], used as a proxy for microbiological water quality, We then estimated the DALYs related to key sequelae, diarrheal diseases and cancers, arising from water-borne pathogens and arsenic respectively. We found [TTC] in end-use drinking water to depend only weakly on [TTC] in source-water. End-user practices far outweighed the microbiological quality of supplied water in determining diarrheal disease burden. [TTC] in source water was calculated to contribute <1% of total diarrheal disease burden. No substantial demonstrable pathogen-for-arsenic risk substitution attributable to specific arsenic mitigation of supplied waters was observed, illustrating the benefits of arsenic mitigation measures in the area studied.

Keywords: arsenic in groundwater; mitigation; water-borne pathogens; health risk substitution; disease burden; West Bengal

1. Introduction

Millions of tube-wells have been drilled in the Bengal Delta both in West Bengal, India and in Bangladesh during the past few decades with the aim of providing pathogen-free drinking water. However, arsenic in these groundwaters constitutes a major health risk to millions of people using these waters for drinking, cooking or irrigation [1–4]. Hyperpigmentation and keratosis are amongst the most visible and widespread of the water-borne-arsenic-attributable diseases, whilst cancers of the skin, lung, bladder and liver are currently amongst the most serious [5]. To combat the arsenic crisis in West Bengal, the provision of alternative water sources is the main arsenic mitigation strategy. A key issue for public-health protection is that, in so improving water-supply services, consideration must also be given to the degree of public-health risk substitution, notably from water-borne pathogens.

There are, however, contrasting published views on the importance or otherwise of such risk substitution. Most published research on the health impacts of water and sanitation has focused on the incidence of diarrheal diseases [6] because these diseases quantitatively represent the most serious health risks arising from water-borne pathogens [7]. Esrey [8] and Clasen and Cairncross [9] both conclude that the prevalence of diarrheal diseases in developing countries, similar to (peri-)rural Bengal, is not related to improvements in water supply but rather to the status of improvements in sanitation and hygiene. In contrast, Howard *et al.* [10], using quantitative microbial risk assessment (QMRA) based upon measurements of pathogens in water supplies from arsenic mitigation schemes in Bangladesh, calculated that disease burden arising from water-borne pathogens, especially for waters from pond sand filters and rain water harvesting, were typically orders of magnitude more important than that arising from groundwater arsenic.

Additionally, Lokuge *et al.* [11] concluded that the substitution of unimproved water supplies for arsenic-bearing groundwaters could potentially lead to an overall increase in water-supply attributable disease burden. Lokuge's [11] conclusion is based, in part, on an estimate from Pruss *et al.* [12],

derived in turn from Esrey [8], that such substitution of water supplies would result in a 20% increase in diarrheal disease incidence. Although this 20% figure is indeed derivable from Esrey's [8] data, it is also apparent from a cursory examination that it is associated with a large uncertainty which makes it statistically indistinguishable from 0% Esrey [8] themselves did not consider the observed difference to be significant, and concluded that the lack of significant difference arose because of confounding factors, principally related to sanitation and hygiene in end-user households. Hence Lokuge *et al.* [11] usage of this 20% datum is subject to question but, equally, the lack of power of Esrey's [8] study, which covered many different countries and continents, indicates that derivation of an accurate figure might require a more geographically-focused study, explicitly considering the differences between the microbial quality of supplied water and water at the point of use.

The aim of our work, therefore, was to obtain data to help resolve these differences and, in particular, to test, in a well-constrained geographically focused groundwater-arsenic-impacted area:

(i) Can pathogen-attributable health risks arising from water at point-of-use (*i.e.*, end-use) be predicted from the microbiological quality of supplied (as opposed to end-use, household) water? or, as Esrey [8] hypothesizes, is post-supply contamination of water (either outside and/or inside the household) more important to the microbiological quality of end-use water than the microbiological quality of the supplied water?

(ii) Have specific arsenic-mitigation measures led to a net overall increase in water-borne attributable health risks, as has been suggested by Lokuge *et al.* [11] for water supply improvements in nearby Bangaldesh?

The area selected for this study, Chakdha Block of Nadia District, West Bengal fulfils the requirements of being (i) geographically focused; (ii) an arsenic impacted area [1,13,14]; (iii) the site of several different arsenic mitigation strategies [15]; (iv) well studied with background information available [14–16] and (v) straightforward logistically. We calculated disability adjusted life years (DALYs) as a mean of comparing disease burden arising from the two water-borne hazards: arsenic and pathogens. These calculations require the measurement of distributions of arsenic and pathogens in drinking water. Whilst arsenic concentration can be used directly in these calculations, in the absence of a comprehensive method to estimate the specific pathogens for waterborne-pathogen-attributable health risks, we used thermally tolerant colliforms [TTC] as a proxy, being the indicator organisms for the reference pathogens used for modeling the pathogen-attributable health risks [17].

2. Materials and Methods

2.1. Sample Collection and Analysis

We collected paired water samples (supplied water; household drinking water) in pre-washed (5% Aristar nitric acid; then 18 MΩ deionized water), sterilized (121 °C/15 psi/30 min) HDPE bottles from 102 households in Chakdha block, West Bengal supplied by one of the three most common arsenic mitigation options in the area, viz.—tap water supply (TW) (n = 40), deep tubewells (DTW) (n = 29) and arsenic removal treatment plants (TP) (n = 33). In our study area, the tap water supply, also known locally as piped water supplies (PWS) are predominately sourced from groundwater, and pumped untreated, then stored and supplied during specific times of the day to communal taps. Arsenic removal

treatment plants typically use absorption or adsorption based chemical treatment methods on hand-pumped tubewells. No documentation of chlorination treatment for any of the water supplies was found. For the purposes of this study, "source water" is defined as the water supply from whence the drinking water was collected and "household drinking water", abbreviated throughout to "end-use" water, is defined as the water, typically stored in a container inside the household, which was ultimately used for drinking. We noted the type of container used to store drinking water in each household and completed an observer-administered questionnaire as a part of a related study, from which, information on the type of household (mixed/mud), the level of education of the respondent and the frequency of cleaning of household water containers (weekly/daily) were collected. After collection, we stored the water samples in an ice box and transported it to the Indian Institute of Chemical Biology (IICB), Kolkata where they were either refrigerated for subsequent arsenic analysis or analyzed within 4–6 h of collection for TTC.

We estimated the abundance of TTC using an Oxfam Del Agua® portable water testing kit by a membrane filtration method. In this method, water-borne bacteria from 100 mL samples were collected on pre-sterilized membrane filters (25.4 mm diameter; 0.45 μm pore-size), the filters were then incubated in a lauryl sulfate broth media at 44 °C for 18 h and the yellow colonies formed counted and recorded as individual thermo-tolerant coliforms. To establish the precision of the method we have done replicate analysis, including by different operators, while we validated the method through a comparison with analyses of replicate samples by the All India Institute of Hygiene and Public Health (AIIHPH) by a maximum probable number (MPN) technique following that of the Environment Agency [18]. In each incubation cycle we included a negative control consisting of sterile water, processed in the same way as the samples. We analyzed water samples for total arsenic by inductively coupled plasma mass spectrometry (ICP-MS) (Agilent 7500 Series ICP-MS) at the University of Manchester following the method outlined by Mondal *et al.* [14].

2.2. Statistical Analysis

We calculated the overall summary statistics for $[TTC]_{end\text{-}use}$ and then stratified by each of the risk factors: (i) whether or not $[TTC]_{source}$ was zero; (ii) the type of mitigation option from which the water was collected; (iii) the type of house; (iv) the education level of the householder; (v) the type of container used to store the drinking water and (vi) the frequency of cleaning of the container. We have used cross tabulation to estimate the mean and standard deviation of $[TTC]_{end\text{-}use}$ stratified by these factors. Due to significant over-dispersion in the data, we used Quasi Poisson regression to model the dependence of $[TTC]_{end\text{-}use}$ counts on $[TTC]_{source}$. We screened the predictors for inter-relationships using Pearson's Chi-squared test of association and variables found to have strong associations (a strong association between the education of the respondent with the type of house ($p < 0.005$) was observed as well as the type of storage container with its cleaning frequency ($p < 0.05$)) were dropped as a check against multi-co-linearity.

The following model Equation (1) was fitted to the data:

$$E([TTC]_{end\text{-}use}) = \lambda;\ Var([TTC]_{end\text{-}use}) = \phi\lambda \quad (1)$$

where

$$\log\lambda = \beta_0 + \beta_1([TTC]_{source} = 0) + \beta_2([TTC]_{source} - 1)([TTC]_{source} > 0) + \gamma_2(\text{Mitigation} = \text{TP}) + \gamma_3(\text{Mitigation} = \text{DTW}) + \delta_3(\text{Type of house} = \text{Mud}) + \theta_2(\text{Container cleaning frequency} = \text{Weekly}) + (\gamma\beta)_2([TTC]_{source} > 0)(\text{Mitigation} = \text{TP}) + (\gamma\beta)_3([TTC]_{source} > 0)(\text{Mitigation} = \text{DTW}) + (\gamma\delta)([TTC]_{source} > 0)(\text{Type of house} = \text{Mud}) + (\gamma\theta)_2([[TTC]_{source} > 0)(\text{Container cleaning frequency} = \text{Weekly}) \quad (2)$$

where E and Var denote the estimated value and variance respectively, and where β_0, β_1, γ_2, γ_3, δ_3, θ_2, $(\gamma\beta)_2$, $(\gamma\beta)_3$, $(\gamma\delta)$ and $(\gamma\theta)_2$ are empirically determined model parameters, the latter 4 representing interaction parameters.

Note that β_1 measures the shift in the average values of $[TTC]_{end\text{-}use}$ depending upon whether or not TTC was present at source and β_2 represents a linear effect of $[TTC]_{source}$ on $[TTC]_{end\text{-}use}$ given that $[TTC]_{source}$ is non-zero. Other potential confounders included in the model are the type of mitigation option employed by the family, the type of house and the frequency with which the storage container for water was cleaned. The model allows for an interaction between whether or not TTC is present at source and each of the other risk factors. We used Cook's distance to determine influential values (typically outliers) and to test the robustness of the list of significant model parameters to the deletion of these values. We also used analysis of deviance to find the relative predictive power of each of the factors towards $[TTC]_{end\text{-}use}$.

To explore the presence of a possible non linear effect of $[TTC]_{source}$ on $[TTC]_{end\text{-}use}$, we refitted the above model after categorising $[TTC]_{source}$ as 0, 0–2, 2–8 and 8–80 CFU/100 mL. We also fitted a Tobit model to the $[TTC]_{end\text{-}use}$ values with $[TTC]_{end\text{-}use} > 0$ to find the significant predictors (Tobit analysis is a technique borrowed from econometrics where it has been used to find significant predictors of expenditure on a commodity given that not all consumers may have a propensity to buy it. The parallel here is that not all households have positive $[TTC]_{end\text{-}use}$). We employed Simulation Extrapolation (SIMEX) to explore the consequences of measurement error in $[TTC]_{source}$ resulting in misclassification error. The effect of up to 8% misclassification error in $[TTC]_{source} = 0$ being classified as positive was explored. Jackknife estimates of variance were also used. We used the statistical software package R for all statistical calculations.

2.3. Risk Models and Input Parameters

We estimated the pathogen-attributable health risks in terms of DALYs using the QMRA model of Howard [10] (Figure 1) and compared it to arsenic-attributable DALYs calculated using the risk model of Mondal [16] (Figure 2). In the absence of comprehensive dose-response data for sequela arising from exposure to water-borne pathogens and to arsenic, we have restricted our calculations to sequelae considered to be the dominant detrimental health outcomes arising from each agent, viz. diarrheal diseases for water-borne pathogens and combined skin, lung, liver and bladder cancers for arsenic.

The input parameters for the estimation of pathogen concentrations based on the *E. coli* concentration obtained from the measured TTC for the QMRA model are summarized in Table 1. Following Howard [10], no distinction is made between pathogen concentrations arising from post-supply contamination outside the household and that arising from within the household. The dose-response relationships for the model reference pathogens based on the reported human-feeding-trial data are summarized in Table 2 along with the μDALYs per reference pathogen microbial infection applied to

the QMRA model based on the data by Havelaar and Melse [19] and Howard [10]. The outcome, severity, duration and probability of infection data are based on the values given by Havelaar and Melse [19] while the susceptible fraction data were obtained from Howard [10] for the population of Bangladesh.

Figure 1. Schematic of the QMRA model [10] for pathogen-attributable health risks.

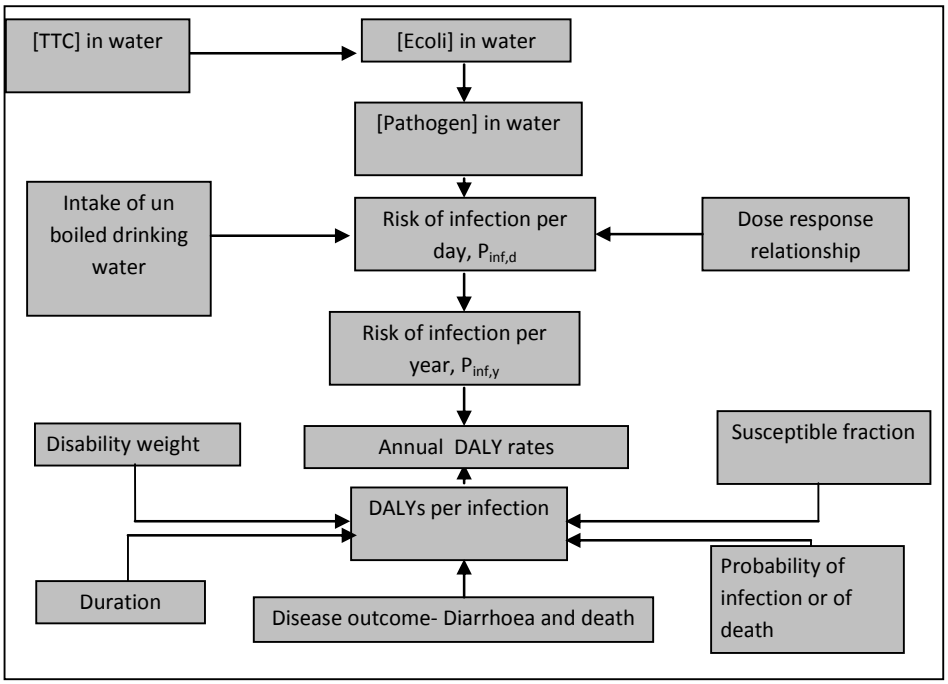


Figure 2. Schematic of the model for arsenic attributable health risks [16].

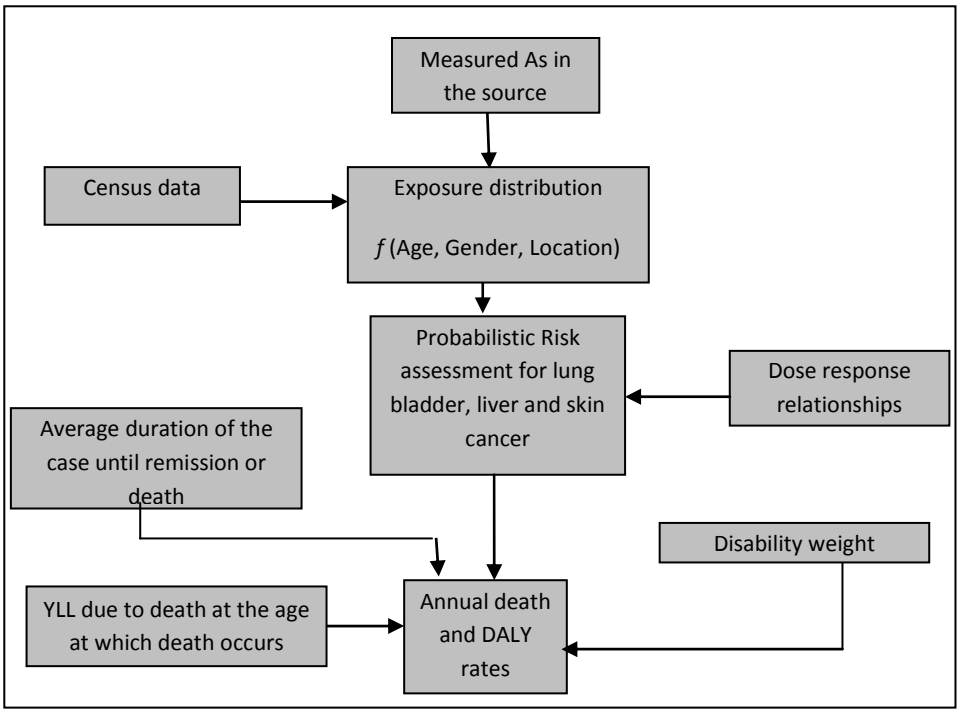


Table 1. Input parameters for exposure assessment for QMRA model.

Input variable	Fitteddistribution	Parametervalue	Reference
$[TTC]_{environmental\ origin}$	lognormal	15% (7.5%, 30%) [b]	Howard [10]
[*E.Coli*]:[Viruses]	lognormal	10^5 (10^4, 10^6) [b]	Howard [10]
[*E.Coli*]:[Bacteria]	lognormal	10^5 (10^4, 10^6) [b]	Howard [10]
[*E.Coli*]:[Protozoa]	lognormal	10^6 (10^5, 10^7) [b]	Howard [10]

Notes: [a] The remainder of the thermally tolerant coliforms are considered to be of faecal origin and assumed to be *E.Coli* for the purposes of calculating a pathogen concentration; [b] Mean (5th percentile, 95th percentile).

Table 2. Microbial dose-response data based upon literature cited by Howard [10] and µDALYs per reference pathogen microbial infection after Havelaar and Melse [19].

Model reference pathogen	Concentration estimate basis	Ref. [a]	P_{infl} [b]	Outcome	S [c]	D [d]	P_{out} [e]	X_{sus} [f]	µDALY per case
Virus	Rota-viruses	[g]	27%	Mild diarrhoea	0.10	0.02	0.856	1.6%	27.4
				Severe diarrhoea	0.23	0.02	0.144		10.6
				Death	1.00	30 [h]	0.0023		11.0
Bacterium	*E. coli* O157 H7	[i]	1%	Watery diarrhoea	0.06	0.009	0.53	9%	28.8
				Bloody diarrhoea	0.39	0.015	0.47		247
				Death	1.00	13.2 [j]	0.0023		2730
Protozoan	Crypto-sporidium	[k]	2.8%	Watery diarrhoea	0.06	0.02	0.9877	7.1%	95.1
				Death	1.00	13.2 [j]	0.01		93.7

Notes: [a] Basis for infectivity estimation; [b] Probability of infection for a dose of 1; [c] Severity; [d] Duration; [e] Probability of outcome, viz. infection or death, *cf.* P_{inf} of Howard [10]; [f] Susceptible fraction; [g] Human feeding trial of rotavirus model of Gerba [20] (cited by [10]); [h] Average death at age of 1 was assumed for rotavirus infected diarrhea [2] leading to loss of 30 years of healthy life based on the data of the GBD study [21]; [i] Human feeding trial of *Shigella dysenteri* model of Holcomb [22] (cited by [10]); [j] The mean loss of life associated with 1 fatal case of cryptosporidiosis and *E.coli* O157 infection was estimated as 13.2 years [2]; [k] Human feeding trial for *C.parvum* model of Messner [23] (cited by [10]).

The input parameters for the arsenic risk assessment model were same as described by Mondal *et al.* [16] with incidence rates being calculated based on the NRC [24] dose-response function, which in turn is based on the Weibull multistage model, predicting the incidence rate of arsenic induced lung, bladder, skin and liver cancer as a function of age, gender and arsenic concentration in drinking water.

3. Results and Discussion

3.1. Quality Control

Arsenic concentration in a standard water reference material (SRM 1640) was measured as 27.8 ± 0.4 µg/L (n = 3) in agreement with the certified value of 26.6 ± 0.4 µg/L. Analyses of arsenic for ARS29-32 groundwater samples, undertaken as part of an Inter Laboratory Quality Evaluation [25], were found to be within 12% of the indicative values in the range 66–330 µg/L.

The mean deviations between duplicate samples analyzed by the same operator for TTC (n = 14) and between the replicate analyses by different operators (n = 8) was better than 6%. The mean

deviation between the analysis done by the method outlined here and by an MPN method at AIIHPH (n = 37) was 37%. No non-zero TTC values were found for any negative controls (n = 18).

3.2. Water Analysis

Table 3 presents the arithmetic mean ± SD, median (95% CI) and percentiles (95% CI) for $[TTC]_{source}$, $[TTC]_{end\text{-}use}$ and arsenic (As) in drinking water for the whole study area. We found $[TTC]_{end\text{-}use}$ to be typically greater than $[TTC]_{source}$ for most of the paired samples with almost no correlation between them (Figure 3). The mean and median $[TTC]_{source}$ for DTWs in this study (0 CFU/100mL) are very comparable to that observed by Howard *et al.* [10] in similar areas in Bangladesh. The observed median arsenic concentration, 28 µg/L is comparable to that previously observed by Mondal *et al.* [16] of 27.9 µg/L (n = 2171). We found no significant correlation between As and $[TTC]_{source}$ (Figure 4) and observed TTC and As in source water to be the highest for the treatment plants among the three different mitigation options. A Kruskal-Wallis test to determine the differences in $[TTC]_{source}$ between the three different mitigation options, showed DTWs to have significantly lower $[TTC]_{source}$ ($p < 0.05$) compared to TP and TW.

Table 3. Summary of water analyses.

Parameter	**$[TTC]_{source}$**	**$[TTC]_{end\text{-}use}$**	**[As]**
Unit	**CFU/100 mL**	**CFU/100 mL**	**µg/L**
Number of samples	102	102	102
Mean ± S.D.	5.6 ± 16	56 ± 162	40 ± 40
Median (95% CI)	0 (0–1)	7 (2–18)	28 (19–37)
5th percentile	0 (0–0)	0 (0–0)	0.76 (0–2)
25th percentile	0 (0–0)	0 (0–2)	7 (4–14)
75th percentile	2 (1–5)	36 (24–69)	57 (46–89)
95th percentile	41 (12–80)	256 (126–843)	112 (100–143)

Table 4 presents $[TTC]_{end\text{-}use}$ stratified by the different risk factors for this study. We found $[TTC]_{end\text{-}use}$ was higher for households with positive $[TTC]_{source}$ than for those with $[TTC]_{source} = 0$. $[TTC]_{end\text{-}use}$ was found to be highest for the water sourced from TW for the studied area. It was also noted that people using TW mostly use the water for drinking, cooking and washing (75%) whereas people using the DTW and TP used the water either only for drinking (23%) or for drinking and cooking (75%). We also observed that $[TTC]_{end\text{-}use}$ was higher for respondents living in mud houses having primary education and storing the water in big mouth containers such as buckets and *hari.* But it was found to be lower for households cleaning the storage container weekly rather than daily for the overall data, perhaps reflecting differences in the sampled populations using daily and weekly cleaning regimes.

Figure 3. Cross-plot of thermally tolerant coliforms [TTC] in supplied and end-use drinking water from households in Chakda Block, West Bengal, India. The correlation between $[TTC]_{end\text{-}use}$ and $[TTC]_{source}$ is extremely poor—thus $[TTC]_{source}$ is an extremely poor predictor of $[TTC]_{end\text{-}use}$ and hence of water-borne pathogen attributable health risks to householders. For the majority of households, $[TTC]_{end\text{-}use} >> [TTC]_{source}$ suggesting that post-supply contamination, either during transport to the household or within the household is an important process.

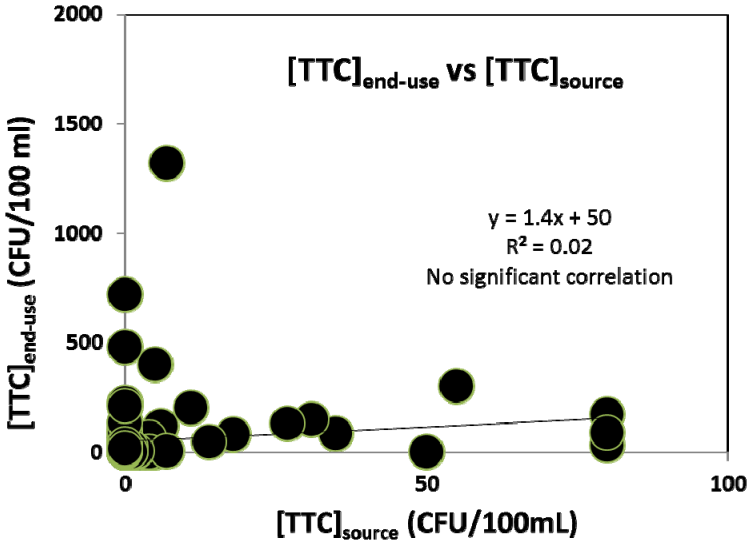


Figure 4. Cross-plot of arsenic and thermally tolerant coliforms in water supplied to households in the Chakdha, West Bengal, India study area through three different arsenic mitigation options. There is no significant correlation between [As] and $[TTC]_{source}$ for any of Tap Water Supply (●; $R^2 = 0.034$), Arsenic Treatment Plant (■, $R^2 = -0.002$), Deep Tube Wells (▲, $R^2 = 0.064$) of for the dataset as a whole ($R^2 = 0.003$). R^2 refers to the correlation for the simple least squares linear regression between [As] and $[TTC]_{source}$ for the relevant dataset.

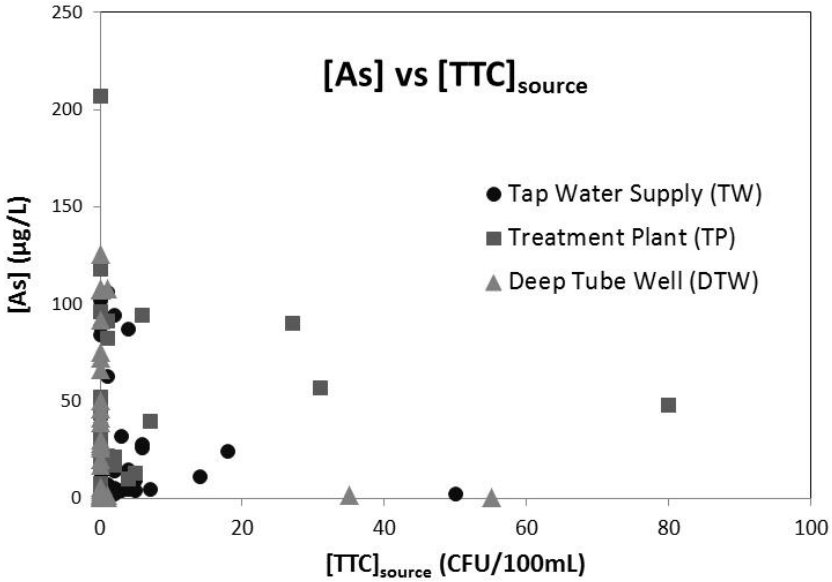


Table 4. $[TTC]_{end\text{-}use}$ classified by independent or predictor variables.

Variable	Classification criteria	Mean $[TTC]_{end\text{-}use}$	SD $[TTC]_{end\text{-}use}$
[TTC]source	=0	45	114
	>0	77	207
Mitigation type	TW (tap water)	101	239
	TP (treatment plant)	38	51
	DTW (deep tube well)	21	55
Highest level of education	Higher	39	123
	Middle	4.0	0.0
	Primary	89	250
	Nil	28	37
Type of storage container	Bottle	38	73
	Jug	2.7	4.6
	Pitcher	46	129
	Jar	17	30
	Bucket	129	301
	Hari	260	198
Cleaning frequency	Daily	72	192
	Weekly	34	80

Table 5 presents the results of fitting the regression model. The model, when refitted after dropping outliers and influential values, produced similar results. The results indicate that the linear term for $[TTC]_{source}$ is significantly positive (p-value = 0.002), that is, among those with positive $[TTC]_{source}$, $[TTC]_{end\text{-}use}$ has a significant linear relationship with $[TTC]_{source}$ after adjusting for other confounders. The estimate for the linear term is 0.05301 (95% confidence interval = (0.025, 0.0933)) indicating that if all other factors remaining constant, a unit increase in $[TTC]_{source}$ will lead to a 1.054440 (=exp(0.05301)) fold increase in $[TTC]_{end\text{-}use}$. In general, we can say that an x unit increase in $[TTC]_{source}$ will lead to an exp(0.05301x) fold increase in $[TTC]_{end\text{-}use}$ if the source water is contaminated. It was further noted that DTW users have significantly lower $[TTC]_{end\text{-}use}$. Cross tabulations of the predictors revealed that DTW users are more likely to live in mud houses, and have higher education levels. None of the other interactions were statistically significant at the 5% level. However the direction of the estimated coefficients suggests that positive $[TTC]_{source}$ leads to lowest $[TTC]_{end\text{-}use}$ for those using water from treatment plants (TP) followed by tap water (TW) and then deep tube wells (DTW). Positive $[TTC]_{source}$ leads to lower $[TTC]_{end\text{-}use}$ for those living in mixed houses than for those living in mud houses. The main effect and interactions are marginally significant for the cleaning frequency of the storage container. If $[TTC]_{source} = 0$, then weekly cleaning leads to a 0.45 (=exp(−0.79202)) fold reduction in $[TTC]_{end\text{-}use}$ whereas if $[TTC]_{source} > 0$, weekly cleaning leads to a 1.8 fold increase in $[TTC]_{end\text{-}use}$.

An analysis of variance showed that the maximum variation in $[TTC]_{end\text{-}use}$ was explained by the linear term for $[TTC]_{source}$ (15%) followed by the type of mitigation from where the water is sourced (10%) and the interaction between the type of house and whether or not $[TTC]_{source} > 0$ (4%). While the model allowing for a nonlinear effect of $[TTC]_{source}$ was found to have better predictive power over the linear effect model (p value from Analysis of Deviance = 0.02), we observed no evidence of

significant contrasts after correcting for multiple comparisons. Based on the results of the Tobit analysis we found a highly significant effect of the linear term for $[TTC]_{source}$ (p-value = 0.0004). The analysis did not find evidence for a significant effect of any of the other risk factors. Finally the output from application of the SIMEX algorithm showed that the interactions were no longer marginally significant but the other estimates and p-values continued to be similar.

Table 5. Regression model [a] of $[TTC]_{end\text{-}use}$.

Variable	Estimate	Standard Error	t	Pr (>|t|) [b]
(Intercept)	1.48749	1.32	1.125	0.26
(if $[TTC]_{source} = 0$)	2.93330	1.37	2.145	0.035 *
($[TTC]_{source} - 1$) * (if $[TTC]_{source} > 0$)	0.05301	0.02	3.167	0.0021 **
(Type of mitigation = TP)	−0.61338	0.43	−1.415	0.16
(Type of mitigation = DTW)	−1.75188	0.57	−3.084	0.0027 **
(Type of house = Mud)	−0.17066	0.53	−0.322	0.75
(Cleaning frequency = Weekly)	−0.79202	0.45	−1.751	0.084
(if $[TTC]_{source} > 0$) * (Type of mitigation = TP)	−0.77046	1.10	−0.703	0.48
(if $[TTC]_{source} > 0$) * (Type of mitigation = DTW)	0.87905	1.08	0.813	0.42
(if $[TTC]_{source} > 0$) * (Type of house = Mud)	2.23692	1.48	1.507	0.14
(if $[TTC]_{source} > 0$) * (Cleaning frequency = weekly)	1.41970	0.80	1.764	0.081

Notes: [a] Dispersion parameter for quasi poisson family taken to be 51.374; [b] Significance codes: 0 < *** < 0.001 < ** < 0.01 < * < 0.05 < 0.1 << 1.

3.3. Calculated Health Risks

Table 6 summarizes the input parameters estimated in this study for the calculation of disease burden. Table 7 gives a comparison of disease burdens, expressed as DALYs, arising from the measured arsenic and TTC, and based on the input parameters estimated in this study and rest of the parameters as detailed in previous sections along with the DALYs reported by previous authors and publications.

Table 6. Input parameters for DALY estimation.

Input variable	Unit	Type of fitted distribution	Parameters	Value	Source
$[TTC]_{source}$	CFU/100 mL	Extreme value	Location parameter	1.24	This study
			Scale parameter	4.76	This study
$[TTC]_{end\text{-}use}$	CFU/100 mL	Extreme value	Location parameter	18.34	This study
			Scale parameter	48.46	This study
[As]	µg/L	Beta General	Shape parameters	0.64, 3.50	This study
			Minimum	0.2	This study
			Maximum	254.78	This study
Water intake	L/person/day	lognormal	Mean	2.50	Mondal [14]
			Standard deviation	1.03	Mondal [14]

Table 7. Comparison of disease burdens (expressed in μDALYs/person/year) from exposure to waterborne arsenic or pathogens or global country-wide estimates.

Study Type	Study area	Secula	μDALYs	Reference
$[TTC]_{source}$	Chakdha Block, West Bengal	Diarrhoea	174 (11,611) [a]	This study
$[TTC]_{end-use}$	Chakdha Block, West Bengal	Diarrhoea	1574 (260,2578) [a]	This study
$[TTC]_{end-use}$ (modeled)	Chakdha Block, West Bengal	Diarrhoea	1252 (555,2282) [a]	This study
Global Burden of Disease (GBD) study	India	Diarrhoea	14530	WHO [21]
[TTC] from shallow tube wells in dry season	Bangladesh	Diarrhoea	10 (0, 5600) [b]	Howard [10]
[TTC] from pond sand filters	Bangladesh	Diarrhoea	~ 3000 (100, 10000) [b]	Howard [10]
Global Burden of Disease (GBD) study	Bangladesh	Diarrhoea	15980	WHO [21]
[As] in mitigated waters	Chakdha Block, West Bengal	Lung, bladder, liver and skin cancer	238 (127, 400) [a]	This study
[As] in pre-mitigation tube wells	Chakdha Block, West Bengal	Lung, bladder, liver and skin cancer	580 (146, 1168) [a]	Calculated from Mondal [16]
Global Burden of Disease (GBD) study	India	Lung, bladder, liver and skin cancer	2450	WHO [21]
[As] in shallow tube wells	Bangladesh	Lung, bladder and skin cancer	~ 1000 (300, 3000) [b]	Howard [10]
[As] in tube well water	Bangladesh	Lung, bladder and skin cancer	385 [c]	Lokuge [11]
Global Burden of Disease (GBD) study	Bangladesh	Lung, bladder, liver and skin cancer	780	WHO [21]

Notes: [a] Median μDALYs with 25th and 75th percentiles; [b] Based on the figures of Howard [10] with 90% confidence interval; [c] Considering only Bangladesh population exposed to drinking water with [As] > 50 μg/L.

3.4. Water Quality

Against the WHO [26] guideline of absence of any fecal pollution as the standard for microbial water quality, the presence of TTC in 25% of the collected source water samples and 50% of the end-use household water samples confirms the potential risk of water-borne diseases from drinking water contamination for the studied area. We also found the arsenic concentration in the collected arsenic mitigated drinking water to be higher than the WHO provisional guideline value of 10 μg/L for 68% of the samples and higher than the Indian permissible limit of 50 μg/L for 29% of the samples, indicating that the water from the arsenic mitigation options neither completely arsenic free nor even compliant with these guidelines/regulations. The significant inverse correlation between As and pathogens (*E.coli*) for shallow tube wells of Bangladesh observed by van Geen *et al.* [27] could be a potential concern for health risk substitution for arsenic mitigation options, but although we observed a very weak inverse relationship of As with $[TTC]_{source}$ (Figure 4) we found the relationship not to be significant.

Based on the regression analysis we observed that the contamination of end-use water largely depends on whether the source water is contaminated or not rather than any other predictors. Hence the fate of $[TTC]_{end\text{-}use}$ is predicted in a different way for $[TTC]_{source} = 0$ and $[TTC]_{source} > 0$. Once the source water is contaminated, $[TTC]_{end\text{-}use}$ increases exponentially irrespective of other factors. In a study in Sierra Leone, Clasen and Bastable [28] observed a 200 fold increase in $[TTC]_{end\text{-}use}$ with respect to $[TTC]_{source}$ when the source was contaminated compared to when the source was not contaminated.

Significantly high $[TTC]_{end\text{-}use}$ compared to $[TTC]_{source}$ (Figure 3) supports evidences from previous studies that have shown the importance of fecal contamination of drinking water after collection associated with household storage linked to water handling and hygiene practices. Eshcol *et al.* [29] illustrated 36% households have their water being contaminated by fecal coliforms on storage within 24–36 h of collection for a study at Andhra Pradesh, India (n = 50). We observed, the overall $[TTC]_{end\text{-}use}$ to be higher for people living in mud houses (Table 4). The type of house- mixed or mud as observed in this study, acts as a proxy for wealth, reflects the sense of personal hygiene, with mixed being better than mud suggests that contamination within the household is a likely factor in controlling the end-use water contamination.

The significant difference in $[TTC]_{source}$ between the three different mitigation options and TP and TW being significantly higher than DTW suggests that much of the pathogen loading in TP and TW is attributable to contamination arising from storage of waters during the treatment for arsenic removal and/or in the distribution network of tap water supply. The overall $[TTC]_{end\text{-}use}$ is found to be lower for DTW users, in spite of the fact that DTW users more likely dwell in mud houses.

Our study shows that wide-mouth open containers such as buckets or Hari had higher potential risk of contamination than narrow-mouth vessels such as bottles, jugs or pitchers (Table 4)—we speculate that the contamination could arise while fetching water from wide-mouth containers by dipping. Clasen and Bastable [28] indicated that using a tap or spigot to access water is protective of stored water quality compared with water accessed by dipping.

3.5. Disease Burden—Pathogens in Source vs. End-Use Water

DALYs arising from TTC in source water accounts for only 11% (174 μDALYs/person/year/ 1574 μDALYs/person/year) of the DALYs resulting from TTC in end-use household drinking water (Table 7) indicating that the microbial contamination of source water is only a small factor for diarrheal disease incidence arising from drinking water from arsenic mitigated sources in the study area. Irrespective of whether water-borne pathogens originate in supplied water or from household contamination, calculated risks from water-borne pathogens (1574 μDALYs/person/year; or using modeled $[TTC]_{end\text{-}use}$, 1252 μDALYs/person/year) represent only around 10% of the total diarrheal disease burden (14,530 μDALYs/person/year) reported by GBD for India [30] thus drinking water would seem to be a minor contributor to diarrheal diseases in Chakdha relative to other causes. This is consistent with Esrey's [8] large cross sectional study on 8 developing countries, which concluded that (i) calculated predictions of reduction in diarrheal disease incidence may be invalid where they are based only on the improvement of microbiological quality of (improved) supplied water; (ii) health benefits from improved water supply were less important than improvements in sanitation; and (iii) improved sanitation was the governing factor reducing diarrheal disease incidence. Pruss [12]

further noted that the transmission of water-borne-pathogens may be linked to a lack of water linked in turn to inadequate personal hygiene and use of contaminated water for cleaning [12]. In this study, we observed that the [TTC]/100 mL in pond water (median 3490, interquartile range 1680–7125, n = 6) and dugwell water (median 1440, interquartile range 1120–2442, n = 5), both extensively used for washing and bathing in Chakdha block, were extremely high and may represent an important source of exposure to water-borne pathogens unrelated to the quality of either supplied or end-use drinking water.

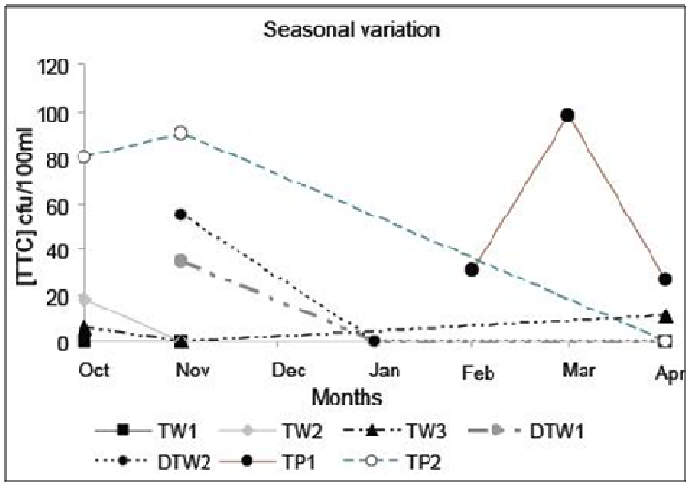
The links between water and health are complex and errors may arise from: (i) for developing countries recall-data on diarrhoea prevalence from cross-sectional studies may be too insensitive with chances of misclassification [8]; (ii) the GBD estimate is for the whole of India and there could be possible local variation in diarrheal disease incidences; and (iii) potential flaws of the DALY approach, given the present availability of data [19]. Important but almost inevitable shortcomings are the uncertainties associated with the model input parameters which can be reduced with further research in the fields of country specific Pathogen: *E coli* ratios and country specific probabilities of infection for reference pathogens as well a more quantitative understanding of differences in post-supply pathogen contamination arising from outside and inside households. Notwithstanding this, verification of the microbial quality of drinking-water by testing for TTC as an indicator of faecal pollution provides conclusive evidence of recent faecal pollution that should not be present in drinking-water. While TTC is a useful indicator, it has limitations. Enteric viruses and protozoa are more resistant to disinfection; consequently, the absence of TTC will not necessarily indicate freedom from these organisms [26]. Under certain circumstances, such as high levels of viral and parasitic diseases in the community it may be desirable to include the estimation of more resistant microorganisms, such as bacteriophages and/or bacterial spores [26] as indicator organisms. In addition, it is important to note that, although diarrheal diseases are probably the largest contributor to the disease burden from water, sanitation and hygiene, it cannot entirely be attributed to these factors because it is also transmitted through food and through air [12].

Lastly, we note that water quality can vary rapidly, and all systems are subject to occasional failures [26] which can have a disproportionate impact on disease. Rainfall can greatly increase the levels of microbial contamination in source waters and waterborne outbreaks often occur following rainfall [26] and this is apparent from the study on seasonal variation for the observed seven sources (Figure 5). Results of the daily, continuous monitoring of Cryptosporidium in drinking water in the UK showed that the Cryptosporidium concentration generally followed a continuous trend, however, in some cases a trend break was detected which dominated the mean health risk hence frequent monitoring rather than the point data as obtained for this study would be required to detect such events [30]. Thus, whilst we report here relative magnitudes of pathogen and arsenic attributable health risks calculated from our small study, the uncertainties on the calculated risks are considerable and further work, including stochastic treatment of the QMRA [30] is required to obtain a more reliable result.

Notwithstanding these limitations to the model DALY calculations, because we do observe a statistically significant, though weak, relationship between microbiological quality of source and end-use water where $[TTC]_{source}$ is > 0, implies that whilst sanitation and hygiene are important in combating diarrheal diseases, degradation of source water quality because of arsenic mitigation can result in increased diarrheal disease incidence. In the study, this is estimated to be 5% (25th and 75th percentile: 2%, 20%)—this is substantially lower than the 20% figure used by Lokuge *et al.* [11] to

estimate increase in diarrheal incidences when there is a shift from existing water sources that are arsenic contaminated to arsenic mitigated options having potential for microbiological contamination. We also note that our datum is significantly higher than 0, reflecting that arsenic mitigated water supplies can be prone to microbiological contamination. This indicates that efforts to minimise microbiological contamination at source, for example chlorination of the source water for deactivating water-borne pathogens, done on a frequent and regular basis and accompanied by appropriate monitoring in order for its effectiveness, are indicated [2]. Additional treatment at household level is indicated for the more effectively reduction of diarrheal disease incidences. Among the various approaches to household-based water treatment are heat and UV radiation, sedimentation, filtration and chemical treatment (coagulation, flocculation, precipitation, adsorption and disinfection) [28].

Figure 5. Seasonal variation in $[TTC]_{source}$ for water supplied from arsenic removal treatment plants (TW), deep tube wells (DTW) and tap waters (TP) in Chakdha Block, West Bengal, India. The lines are a visual aid to link samples from the same well but are not intended to indicate a determined trend. Nevertheless, the data suggest that other than the occasional spike (TP1, March), $[TTC]_{source}$ is most commonly highest in October/November near the end of the monsoon season (June–October).



3.6. Overall Change in Disease Burden Arising from Arsenic Mitigation

We observe (Table 7) that DALYs calculated as arising from As in mitigated waters in this study (238 µDALYs/person/year) are less than half of those calculated for As in pre-mitigation wells (580 µDALYs/person/year) in the same area using data reported from previous studies [16]. Both these values are substantially less those arising (1000 µDALYs/person/year) from unmitigated shallow wells in similar areas in Bangladesh. The arsenic mitigations considered here therefore have clearly resulted in a substantial reduction in drinking water arsenic attributable health risks in the study area.

Although DALYs arising from pathogens in end-use water (1574 µDALYs/person/year; or using modeled $[TTC]_{end\text{-}use}$, 1252 µDALYs/person/year) are higher than any of the average arsenic-attributable values calculated, the regression model derived here demonstrates that only a small fraction of those DALYs, viz. 174 µDALYs/person/year, can be attributed to pathogens in the supplied water, as opposed to pathogens acquired post-supply, either outside or inside households.

Thus, the combined DALYs arising from both $[TTC]_{source}$ and As in mitigated wells in this study (424 μDALYs/person/year) represents, on average, a reduction in disease burden as a result of the arsenic mitigation methods—we thus find no evidence in this small study for significant risk substitution resulting in a net increase in overall water-borne disease burden for the sequelae considered here.

4. Conclusions

This small study on fecal contamination of arsenic mitigated source and end-use water for an arsenic impacted area of West Bengal, India demonstrates that the microbiological quality of end-use water in such areas can be largely independent of that of the source water. In part because of this, the specific arsenic mitigations studied here have not been found to substantially result in risk substitution, although the DALY estimates made have significant uncertainties. The results indicate that far greater public health benefits are to be made through improving sanitation and hygiene facilities and practice and through the removal or avoidance of arsenic than from only improving the microbiological quality of supplied water.

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Author Contributions

The study was design and conceived by Debapriya Mondal, David Polya and Ashok Giri Fieldwork and microbiological analysis was carried out in West Bengal by Debapriya Mondal, Babli Halder, Mayukh Banerjee and Nilanjana Banerjee under the supervision of Ashok Giri. Chemical analysis was carried out by Debapriya Mondal under the supervision of David Polya. Statistical analysis was carried out by Debapriya Mondal, Bhaswati Ganguli and Maitreya Semanta under the supervision of Sugata Sen Roy. The manuscript was largely written by Debapriya Mondal and David Polya but all authors contributed to the writing and review of the manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

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