Towards an Assessment of the Socioeconomic Impact of Arsenic Poisoning in Bangladesh

prepared in cooperation with the Department of Health in Sustainable Development
# TABLE OF CONTENTS

Acknowledgements ........................................................................ 1

1. Introduction ............................................................................. 3

2. The importance of safe water supply to the household ................. 3

3. Health effects of arsenic in drinking water .................................................. 5
   3.1 Non-cancer health effects .............................................................. 5
   3.2 Cancer health effects .................................................................. 6
   3.3 Treatment of arsenicosis sufferers ............................................... 8

4. Mitigation of arsenic in drinking water ................................................... 8
   4.1 Groundwater ............................................................................. 8
   4.2 Surface water ............................................................................ 10
      4.2.1 Surface water treatment ........................................................ 10
      4.2.2 Rainwater ........................................................................... 11
   4.3 Technology choice ...................................................................... 12

5. Arsenicosis and economic status: the poor suffer most ................... 12
   5.1 The relationship between household income and prevalence of arsenicosis 12
   5.2 Explanations for the role of socioeconomic status in arsenic poisoning ...... 13

6. The socioeconomic impact of arsenic poisoning: coping and steps towards modelling ........................................................................ 14
   6.1 Coping with the impact of arsenic poisoning: introductory elements ........... 15
   6.2 The policy question: the arsenic poisoning and its socioeconomic impact ........ 16

7. A sub-model of the epidemiology of arsenicosis at village level ............. 16
   7.1 Epidemiological model .................................................................. 17
   7.2 Assignment of prevalence and incidence ........................................... 18
   7.3 Natural history of arsenicosis ............................................................ 19
   7.4 Model results ............................................................................. 20

8. Sub-model of arsenicosis and its socioeconomic impact on village households 21
   8.1 Socioeconomic household survey at T0 ........................................... 21
   8.2 Coping ...................................................................................... 22
   8.3 Modeling the impact of mitigation methods ..................................... 24

9. Remaining challenges for modelling ......................................................... 25
   9.1 Including other health and social effects ........................................ 25
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1. Introduction

Access to a safe water supply is one of the most important determinants of health and socioeconomic development (Cvjetanovic 1986). This recognition of the importance of safe water supplies has led to an emphasis on the provision of appropriate facilities in developing countries. In the 1970s, it was realised that Bangladesh’s population density and lack of access to adequate sanitation had led to severe microbiological contamination of surface water, resulting in high levels of morbidity and mortality. Bangladesh’s government and population, supported by international agencies, have since then installed about 4 million tubewells¹ to tap better quality groundwater sources. This tube well initiative is said to have contributed significantly to the halving of infant mortality over a 36-year, from 151/000 in 1960 to 83/000 in 1996. The under-five mortality rate also dropped from 247/000 to 112/000 in that period (UNICEF 1998).

However, in 1993, drinking water samples from tubewells were found to contain high levels of arsenic. The area containing the worst arsenic concentrations stretches across the central section of Bangladesh from Chapai Nawabganj in the west to Brahmanbaria in the east, although there are isolated areas affected in other regions, particularly the Greater Sylhet area in the north-east. The exact number of persons at risk from arsenic poisoning is not known with accuracy. The British Geological survey (1998) estimates the total population affected as 18.5-22.7 million based on the assumption that the population affected is proportional to the number of wells contaminated.

The arsenic hazard will have an important impact on the health, social and economic status of the population. The primary purpose of this paper is to propose a methodology to analyse the health effects, how people cope with the socioeconomic consequences of the disease and to predict the beneficial effects of various alternative mitigation methods. In the next section the importance of a safe water supply will be discussed from the household perspective. Section 3 outlines the health impact of arsenic ingestion from drinking water, the suggested treatment protocol and the options for mitigation programmes. Section 4 investigates current technologies that mitigate arsenic levels to reduce in drinking water. We focus on the link between arsenicosis and economic status in section 5. In section 6, we address the issue of households’ coping strategies, and discuss steps towards modeling this coping behavior. Section 7 then sets out a framework for analysis of the socioeconomic impact of arsenicosis and presents results of an epidemiological model that attempts to predict the health impact of arsenic poisoning over a 30-year time frame. Further challenges for modeling of arsenicosis and its impact on households are discussed in section 8. We conclude in section 9.

2. The importance of safe water supply to the household

Water intended for human consumption should be both safe and wholesome. This has been defined as water that is free from pathogenic agents, free from harmful chemical substances, pleasant to taste, i.e. free from colour and odour, and usable for domestic purposes (Park 1997). Without ample safe drinking water, communities cannot be healthy.

¹ The figure of 11 million was stated by Dr Quazi Quamruzzaman, Dhaka Community Hospital, in his presentation at WHO in September 1999. The difference with the figure of 4 million above may be due to difference in the definition of tubewells, however.
The various mechanisms by which the provision of a safe water supply is transformed into health benefits are reviewed by Cvjetanovic (1986). His conceptual framework shows that an investment in water supply and sanitation gives an improvement in the quantity and/or quality of water available to the household. This yields direct health benefits resulting from improved nutrition, personal hygiene, and the interruption of water-related disease. Bradley classified the relationship between water and diseases into four categories; see Box 1 (Bartram 1996).

The health benefits from reducing water-related disease can in some circumstances be transmuted into a greater work capacity, which may contribute to increased production and hence to overall economic development.

According to Becker (1971, 1981) the household uses time, labour and purchased goods to create commodities for the household. The household attempts to produce safe water for consumption, dependent on time and resource constraints. Safe water for household use is dependent on the time and labour used in the collection of water; the time and resources used in boiling/sterilizing the water and in managing water within the household. Households may not have access to safe water supplies because the financial, labour or time and energy costs of collection and management are too high, either punctually or perpetually.

The provision of a local safe water supply source is likely to considerably reduce the burden of producing safe water for the household. The labour cost of collecting water is borne largely by women and girls, who are responsible for domestic chores in most developing countries. It has been found in Kenya that carrying water may account for up to 85% of total daily energy intake of females (Dufaut 1990). A number of physical ailments may result from carrying heavy loads, including head, neck and spinal problems (Dufaut 1990). Clearly there is considerable health benefit to be gained from decreasing women’s weight-bearing responsibilities. Also, Krishna (1990) points to the indirect health benefits that may be gained from mothers having greater time to spend on child-care. The extent of benefit relates to service level - proximity to point of use - and to reliability.

Access to safe water will also depend on non material factors, such as basic hygiene knowledge, social position and water quality. Basic hygiene knowledge and high water quality support/facilitate access to safe water. It is said that these factors alter the efficiency of the household as a safe water producer. Social factors affecting access to water supply sources will also determine the ability of the household to produce safe water. Lower caste households may not have access to high quality water supply sources due to cultural norms which embrace principles of social exclusion. Conversely, higher caste households, may be unwilling to share high quality water supply sources with lower caste households, and instead may choose alternative sources of lower quality water. In other social contexts, the effects on higher castes may be adverse, if they are socially excluded from water sources used by lower castes.

**Box 1: Water-related disease classification**

Water borne diseases occur when a pathogen or dangerous chemical is contained in water that is utilised for human consumption. Water-washed diseases are the result of an insufficient supply of water for the maintenance of hygienic health; almost all infectious water-borne disease is therefore also water-washed, but the group also includes non-water-borne diseases which are reduced by improved hygiene, such as scabies, trachoma and conjunctivitis. Water-based disease arises from transmission of a pathogen where water is the intermediate host (e.g. schistosomiasis). Water related insect vector diseases are those diseases that are transmitted by insects that breed in water or live and infect humans near water (e.g. malaria).
3. Health effects of arsenic in drinking water

World Health Organization (WHO) recommendations on the acceptability and safety of levels of arsenic in drinking water have dropped twenty-fold, from a concentration of 0.2 mg per litre in 1958 to 0.01 mg per litre in its 1993 Drinking Water Guidelines. But according to Bangladesh Standards for Testing Institution (BSTI, 1989) the maximum permissible limit for arsenic (As) is 0.05 mg per litre.

There is no widely accepted complete definition of what constitutes arsenicosis. Inorganic arsenic is a classified carcinogen (IARC 1980) that also has a multitude of non-cancer effects. The widespread effects of arsenic are perhaps responsible in part for the lack of a widely accepted care definition for arsenicosis. Furthermore, some symptoms of arsenicosis (such as shortness of breath) may be observationally indistinguishable from the health effects of other illnesses. It is not possible here to undertake a comprehensive review of the health effects of arsenic contamination of drinking water. The International Programme on Chemical Safety (IPCS) Environmental Health Criteria (EHC) is making a comprehensive health effect assessment, which will be finalised in 2000. Rather, the purpose of this section is to highlight some of the main findings of the literature on health effects, especially with respect to predictive use of the available information. In addition, arsenic poisoning may be acute or chronic. In the context of community drinking water supply, only chronic exposure is relevant. Acute poisoning is therefore not discussed further.

3.1 Non-cancer health effects

According to the National Research Council report (p89, 1999): “Arsenic exposure interferes with the action of enzymes, essential actions, and transcriptional events in cells in the body, and a multitude of multisystemic non-cancer effects might ensue.” The most widely noted non-cancer effects of chronic arsenic consumption are skin lesions. The first symptoms to appear after initiation of exposure are hyperpigmentation (dark spots on the skin) and hypopigmentation (white spots on the skin). Some physicians collectively refer to these symptoms as melanosis. Hyperpigmentation commonly appear in a raindrop pattern on the trunk or extremities, but also on mucous membranes such as the tongue (Yeh 1973). Over time arsenic exposure is associated with keratoses on the hands and feet. Keratosis is a condition where the skin hardens and develops into raised wart-like nodules. These nodules become more pronounced over time, sometimes reaching 1cm in size (National Research Council 1999). Tseng et al. (1977) noted that skin cancers often appear at the sites of existing keratoses. The time from exposure to manifestation is debated in the literature (see National Research Council 1999). It is likely that differing exposures to arsenic accounts for the heterogeneity in observations. The youngest age reported for patients with hyperpigmentation and keratosis is 2 years (Rosenberg 1974). For Bangladesh, Guha Mazumder et al. (1998) suggests a minimum time gap of five years between first exposure and initial cutaneous manifestations. The distinctive appearance of these skin lesions has meant they have been used as indicators of arsenic exposure, when it has not been possible to ascertain arsenic concentrations in well water.

Arsenic has been associated with a multitude of other non-cancer health effects. Arsenic is associated with peripheral vascular disease (blackfoot disease) in China (Province of Taiwan) (Tseng 1977). This condition results in gangrene in the extremities and usually occurs in conjunction with skin lesions. Other cardiovascular problems such as hypertension (Chen et al. 1995) and ischemic heart disease have been found to be associated with arsenic.
Research into organ damage has concentrated mainly on the liver. Guha Mazumder et al. (1988) found evidence of liver enlargement and non cirrhotic portal fibrosis among a small sample of severely affected arsenic patients in West Bengal. In a later study, Guha Mazumder et al. (1997) also suggested pulmonary health effects. They found restrictive lung disease among 53% of a small sample of severely affected arsenic patients in West Bengal.

In terms of haematological effects, anaemia is commonly cited (National Research Council 1999). Another widely suggested health effect is diabetes mellitus. Rahman et al. (1998) found a significant dose response relationship between arsenic exposure and diabetes mellitus among those suffering from keratoses in Dhaka, Bangladesh.

3.2 Cancer health effects

Hutchinson (1887) identified arsenic as a carcinogen because of the high number of skin cancers occurring on patients treated with arsenicals. The International Agency for Research on Cancer (IARC 1980) classified inorganic arsenic compounds as skin and lung (via inhalation) carcinogens. In the period following this classification, concerns have grown over the possibility of arsenic in drinking water causing a number of other cancers.

The strongest epidemiological evidence on skin cancer effects comes from studies of arsenic contamination of drinking water in China (Province of Taiwan). Villages in south-western China (Province of Taiwan) switched from surface water to arsenic contaminated well water for drinking in the 1920s. An early study by Tseng et al. (1968) found evidence of a dose response relationship between concentration of arsenic in drinking water and prevalence of skin cancer. IPCS (1981) estimated skin cancer risk from life-time exposure to arsenic in drinking water at 5% for 0.2 mg of arsenic per litre, based on the findings of Tseng et al. (1977). Based on the increased incidence of skin cancer observed in the population in China (Province of Taiwan), the US Environmental Protection Agency (1988) has used a multistage model that is both linear and quadratic in dose to estimate the lifetime skin cancer risk associated with the ingestion of arsenic in drinking water. With this model and data on males, the concentrations of arsenic in drinking-water associated with estimated excess lifetime skin cancer risks of $10^{-4}$, $10^{-5}$, and $10^{-6}$ are 0.0017, 0.00017, and 0.000017 mg/l respectively. Considering other data and the fact that the concentration of arsenic in drinking-water at an estimated skin cancer risk of $10^{-5}$ is below the practical quantification limit of 0.01 mg/l as well as a view to reducing the concentration of arsenic in drinking-water, provisional guideline value of 0.01 mg/l is recommended (WHO 1996). The guideline value is associated with an excess lifetime risk for skin cancer of $6 \times 10^{-4}$ (i.e. six persons in 10,000).

High levels of arsenic in drinking water are also associated with a number of internal cancers. However, it is difficult to quantitatively establish risk in many of the studies, due to problems in measuring exposure to arsenic. Chen et al. (1985) calculated standardised mortality ratios (SMRs) for a number of cancers in 84 villages in south-western China (Province of Taiwan). Mortality from 1968-1986 was compared with age and sex adjusted expected mortality. Significantly increased mortality was observed among both males and females for bladder, kidney, lung, liver and colon cancers. However, the authors were not able to directly estimate arsenic concentrations in well water. Chen and Wang (1990) were able to use data on arsenic concentrations in 83,656 wells in 314 precincts and townships, collected from 1974-1976 in
China (Province of Taiwan). The authors used a multiple regression approach to control for socioeconomic confounding factors, and compared age adjusted mortality rates with average arsenic concentrations in each township. They found a significant relationship with arsenic concentration and mortality from cancers of the liver, nasal cavity, lung, bladder and kidney for both sexes. One problem with this study for the purpose of quantitative risk assessment, is that the authors do not report the methodology used for calculating the average arsenic concentration for each township or precinct.

Hopenhayn-Rich et al. (1998) examined SMRs for bladder, kidney, lung, liver and stomach cancers for 1986-1991 for 26 counties in the Cordoba Province in Argentina. The authors stratified counties into low, intermediate and high exposure groups based on arsenic levels in their drinking water. The low and intermediate exposure counties were defined by the authors. Data for arsenic levels in the two high exposure counties were given. These levels ranged from 0.04 mg/l to 0.43 mg/l. SMRs were calculated using age and sex specific national rates for Argentina. Significant exposure-response relationships were found for the cancer in the bladder, lung and kidney. It is unlikely that smoking is a confounding factor, as deaths from chronic obstructive pulmonary disease (indicative of smoking) were not related to arsenic concentrations.

The above-mentioned studies all utilised an ecological design and are thus susceptible to bias from confounding factors. However, the bladder and lung cancer results of these studies are also confirmed by cohort studies which may be less susceptible to this form of bias. These studies are also useful in providing data on the latency period of internal cancers. Cuzick et al. (1992) studied a cohort of patients treated with Fowler’s solution (potassium arsenite) in England from 1945-1969. In the follow up until 1991, a significant excess of bladder cancer mortality occurred. In addition, a subset of patients had exhibited skin lesions when examined in 1970. It was found that all patients who subsequently died of bladder cancer had also suffered skin lesions. Even after stratifying this subset according to dose group, the finding that all cases had skin lesions was highly significant. The authors suggested this provided evidence that skin lesions are a useful biomarker for susceptibility to internal cancers. The period between first exposure and death from bladder cancer varied from 10 years to over 20 years.

Tsuda et al. (1995) followed up a cohort of 454 residents in Japan who had used industrially polluted water for five years. The authors separated the cohort into low, medium and high exposure groups, based on arsenic concentration in local wells. The low group was exposed to less than 0.05 mg/l, the medium exposure group 0.05-0.99 mg/l and the high exposure group greater than 1 mg/l. A significant excess of cancers occurred only in the group exposed to an arsenic concentration greater than 1 mg/l. This finding may be because the small sample size is unable to detect significant excess deaths in the medium exposure group. There may also be underestimation of the effect due to the relatively short period of exposure. Significant excess deaths from lung cancer (nine deaths) and urinary tract cancer (two from bladder cancer and one renal pelvis cancer) were observed in the high exposure group. In contrast to the findings of Cuzick et al. (1992), the authors found excess cancer mortality among those both with and without skin lesions present: “The results demonstrate that negative skin signs are no assurance of low risk for cancer development.” (Tsuda et al. p207, 1995). The authors note that the period from first exposure to death from lung cancer varied from 11 to 35 years, with a mean of 26.7 years.
To conclude, the results from studies of cancer indicate strong evidence that exposure to arsenic is related to skin, lung and bladder cancer, although more established assessment on health effect of arsenic is being prepared by IPCS/EHC. It is likely that arsenic causes a number of other cancers, but thus far epidemiological evidence has not been consistent for other sites in the body.

3.3 Treatment of arsenicosis sufferers

Guha Mazumder (1996) outlines a treatment regime for those suffering from arsenicosis. It is suggested that the first stage in treating those with arsenicosis should be the immediate cessation of consumption of arsenic contaminated water. Once this has been achieved the emphasis should be on the provision of a diet high in protein (preferably meat) and vitamins, to aid the methylation of inorganic arsenic in the body. The chelating agents DMPS (dimercaptopropane sulphonate) and DMSA (dimercaptosuccinic acid) are recommended as treatment drugs. DMPS is considered more efficacious than DMSA in aiding the elimination of arsenic from the body, but DMSA is preferred for wide spread application because of its lower risk of side effects (Angle 1995). However, Guha Mazumder (1996) notes that these drugs are very expensive. Chelation therapy has been demonstrated to be effective in the treatment of acute arsenic poisoning. Its value in treatment of chronic poisoning remains undemonstrated. Palliative care may be the only affordable treatment in rural areas of Bangladesh, where expensive drugs and protein-rich diets are unlikely to be available to the vast majority. In the case of keratosis, application of ointment containing salicylic acid can help to soften the skin and ease the pain of the patient.

4. Mitigation of arsenic in drinking water

This section will analyse the technologies that can be used to provide safe drinking water in rural Bangladesh. The available options for safe water can be classified by source: groundwater, surface water and rainwater. Recent years have seen increasing acceptance of strategies for incremental improvement in environment and health in general and of demand-driven approaches in water supply and sanitation in particular. It is inappropriate therefore to pursue a single “master” technological solution but rather to inform communities and individuals of alternatives and their characteristics, to “offer” a selection of alternatives. The mitigation measures outlined below should not be seen as a list from which the most preferable should be selected, but the basis for development of a portfolio of alternatives.

4.1 Groundwater

The simplest and most immediately achievable option is the sharing of tubewells that are currently low or free from arsenic. Arsenic-containing wells may still be used safely for laundry-washing for example, and a simple colour coding (e.g. “traffic lights”) may have a significant impact on community arsenic exposure if carefully and continuously backed up by awareness raising and educational activities. However, in the most highly contaminated areas no tubewells will contain safe levels of arsenic. Furthermore, colour-coding would have to be monitored carefully over time, as tubewells with previously safe test results may be later found to contain increased levels of arsenic. The principal costs of such an approach relate to the
ongoing testing and labelling of wells and of continuous awareness raising and education. These may be borne by the community or an outside agency. In practice the household burden of water collection is likely to increase (i.e. a greater average distance will be travelled in order to collect the same volume of water).

The other alternative for groundwater supply is the development of deep tubewells. The British Geological Survey (1998) found only two out of 280 tubewells below 200 m in Bangladesh to be contaminated with high levels of arsenic. The principal costs of such an approach relate to the costs of developing deep tubewells. There include costs of training and equipping drilling teams as well as the direct costs of drilling per se including a proportion of unsuccessful bores. These may be borne by the community or an outside agency. If contaminated wells remain in use for other purposes such as laundry then ongoing awareness-raising and education will be essential. If new wells are appropriately sited then the household burden of water collection may be constant or even decrease. Deep tubewells have been in use for years in coastal areas because of high salinity in shallow aquifers. However, it is not possible to exploit this technology in all areas because rock formations may make drilling unfeasible.

An alternative option for groundwater supply is treatment of arsenic contaminated tubewell water to make it safe for consumption. However, at the moment arsenic removal using the bucket treatment method being tested (see Boxes 2 and 3) is regarded as a alternative for the transitional period until a “permanent” solution is found.

**Box 2: Danida Research Project on Arsenic Removal in Bangladesh**

Danida has conducted a research in Noakhali in Bangladesh since November 1998 on removal of arsenic using a mix of 200 mg/l alum and 1.5 mg/l KMnO₄ introduced into a large bucket (18l), of which the supernatant is drained off after 1-1.5 hrs. into a bucket standing beneath it. Cost of chemicals for an average family is Tk. 10/US $0.2 a month. Lab tests show a removal from 1.1 mg/l As to 0.016 mg/l In the field level reality check, As ranging from 0.12-0.45 mg/l was reduced to 0.02-0.04 consistently. Though well within the Bangladesh standard, the removal efficiency was considerably less that in the laboratory. Stirring (time, mixing efficiency – paddle stick instead of cane stick) are supposed to make the difference and Danida is checking this out in a field situation at the moment. Danida has also designed a two-bucket column (total investment cost for the set is Tk. 300/US $6) which circumvents the resuspension of the settled solids. Danida reports that 50-80% of the two-bucket systems deliver water within Bangladesh Standards (Danida 2000).

Co-precipitation is a well known phenomenon and has been the subject of a small study by Water Aid in East Madaripur near Chittagong. Fe ranges from 0-10 mg/l. In the first phase of the study it seemed that removal rates were very good. However, upon further study it was found that some wells showed very low removal rates. It seems that salinity has a detrimental effect on removal. Hardness may possibly have an effect as well

The Danida and Water Aid studies also look into the sustainability of methods at the household level. Apart from initial acceptance of a suitable method, households will also have to apply the technique consistently and properly to continue to avail of the benefits of arsenic avoidance.
WHO PAHO - Pan American Center for Sanitary Engineering and Environmental Sciences (CEPIS) in Peru has developed a technology called ALUFLOC for arsenic removal at the household level and it has been tested in Argentine. ALUFLOC is a sachet containing chemicals that are added to a bucket of arsenic contaminated tubewell water. After about an hour the treatment process is complete and the water is safe for consumption. Preliminary field results suggest that ALUFLOC is effective in reducing arsenic content to safe levels. However, it is necessary to optimise the product for treating tubewell water with a concentration of arsenic greater than 1mg/l. The cost of the technology is estimated at US $0.15 per bucket treated, given the assumption of production at an industrial level. The cost of such an approach relate to the ongoing need for awareness-raising and education; the cost of treatment materials (including manufacture, distribution etc); and the costs of additional household expenditure upon equipment (e.g. additional buckets) and of time. It may however be deployed rapidly and costs may be borne by the community, outside agency or may be subsidised.

4.2 Surface water

Surface waters (rainwater, rivers, lakes etc) are typically low in arsenic and therefore potentially attractive drinking water sources in arsenic-rich areas. However surface waters are frequently contaminated with human and animal faecal matter, and other material and are unsafe for this reason. This risk initially led to the preference for groundwater sources in Bangladesh. The critical issues in arsenic-rich areas therefore concern whether treating surface water for faecal contamination can be reliably achieved at a lower overall cost than that of securing groundwater from low-arsenic sources or through treatment to remove arsenic.

4.2.1 Surface water treatment

Treatment of surface water can be achieved by several means. Slow sand filtration, for example, is a typical method for treatment for rural areas and small towns. The water passes slowly through a large tank filled with sand and gravel. Fine particles are filtered out and micro-organisms are inactivated by a thin layer formed on the surface of the bed (Schmutzdecke). The latter consists of retained organic and inorganic material and a huge variety of biologically active micro-organisms which break down organic matter. Reservation is given for the validity of this method in terms of the sustainability in Bangladesh, despite the relatively limited requirement in terms of maintenance. The reasons include the fact that this method of treatment may require too much maintenance for water presenting high turbidity (more than 30 mg/l) and carries a bacteriological risk when not operated properly. However,
Pond Sand Filters\(^2\) are still useful as an option in Bangladesh especially in the coastal belt where there are few alternatives.

The key elements in the decision-making process leading to the selection of this technology as opposed to ground water deals with the costs involved not only in terms of capital investment in infrastructure, but also the cost of maintenance, including supervisory support. If arsenic-containing wells are maintained in use then ongoing awareness raising and education will be required. The household burden of water collection is likely to increase (as the number of available sources is likely to decrease) unless opportunity of capital investment is taken to develop piped distribution.

### 4.2.2 Rainwater

Rainwater harvesting\(^3\) is a recognised water technology in use in many developing countries around the world (WHO/IRC 1997). UNICEF has promoted dispersion of the technology since 1994 in Bangladesh. The rainwater is collected using either a sheet material rooftop and guttering or a plastic sheet and is then diverted to a storage container. Water is not collected during the first few minutes of a rainstorm to avoid contamination by dust, insects, bird dropping, etc.

The costs of rainwater harvesting will be largely capital intensive, and will be dependent on the availability of suitable roofing, materials for guttering and storage tanks. Rainwater use has proven to be successful elsewhere, such as in China (Province of Taiwan), Sri Lanka and Thailand.

In some circumstances there is the possibility of chemical contamination of the collected water, particularly where air pollution is a major problem and where bacteriological contamination may be caused by bird drops. There is also the possibility of contamination particularly when the water is stored for long periods due to the intrusion of insects, etc. Health inspections are needed regularly to ensure that the water is of good quality. However, these reservations might be less problematic as rainwater quality in many circumstances is at least as good as the piped water distributed in many towns in Bangladesh.

The above are only examples of technologies that might be considered as an alternative for the current ground water abstraction. There are other low-cost technologies to be considered in a decision-making process such as springs, infiltration galleries, etc.

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\(^2\) The pond sand filter (PSF) is a manually operated filtration unit built by the side of a pond which treats pond water by using the principle of slow sand filtration. Recent bacteriological examination of the PSFs in Bangladesh show that these are not functioning too badly when maintained well (Chowdhury 2000).

\(^3\) In a country with a mean annual rainfall in excess of 1500mm this is an attractive mitigation technology. Rooftop catchments are most suited to family requirements as corrugated iron roofs are ever-present in Bangladesh. Rainwater is collected in storage containers of various sizes above and below ground. Even a jar (200-1000 litre) will allow a high degree of convenience and water security during 7-9 months of the year (Heijnen 1999).
4.3 Technology choice

The choice between these technologies should take into account their cost effectiveness in providing arsenic free and microbiologically safe drinking water. Different options may have very different balances of cost between for example capital and recurrent costs and may impact differently on the household costs of water management. However, the criteria of sustainability and acceptance by rural users must be incorporated in the calculation of cost effectiveness, in order to aid the decision making process over which mitigation method(s) to implement.

5. Arsenicosis and economic status: the poor suffer most

5.1 The relationship between household income and prevalence of arsenicosis

The Asia Arsenic Network conducted a demographic survey of Samta Village in the Jessore District of Bangladesh in 1998 (Tani,1999a;b). A graphical representation of household income data versus arsenicosis prevalence, using data collected in this survey, showed a negative relationship. In an attempt to further quantify this relationship a regression analysis was conducted using the same data. Prevalence of arsenicosis was based on evidence of skin lesions in the surveyed population. Household income was measured on an annual basis. It is based on a self reported estimate by the household head for a given period. If the period reported was less than one year, this estimate was multiplied by the appropriate factor to obtain the annual income for the household. As most villagers are subsistence farmers, these estimates should be considered as lower bounds for income, as it is likely that at least some villagers will not have included the monetary value of produce consumed within the household. Prevalence data were grouped by annual household income class using ranges of 10,000 Taka. Data for households with an income above 170,000 Taka were excluded due to small sample size: typically one household per income class. Therefore, the data consisted of 17 household income classes. There were no arsenicosis patients in any of the income classes above the 140,000-150,000 Taka household income range.

A logit regression form was found to be appropriate (Box 4). The coefficient for household income is significant beyond the 1% level (p=0.0078). The regression indicates the negative relationship between arsenicosis and household income. Increasing levels of household income are associated with a lower prevalence of arsenicosis. The estimated $\alpha$ and $\beta$ coefficients from this regression are used to calculate the prevalence estimates that are presented in Figure 5.1 along with the observed data. It can be seen from reference to Figure 5.1 that the burden of arsenicosis falls mainly on those living in low income households. This relationship between household income and health damage by arsenic was hinted at, although not quantitatively proven, by Tani (1999a).

\[ P_i = \frac{1}{1+e^{-(\alpha + \beta Y_i)}}. \]

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4 The errors terms were not found to be heteroskedastic using a Koenker test (Koenker 1981):
\[ E_i^2 = -3.652174 + 0.188817Y_i \]
\[ R^2 = 0.14 \quad N=17; \quad F= 2.4431; \quad p= 0.1389 \]
\[ (11.85142) \quad (0.120801) \]

Where $E_i^2$ is the error terms from the Logit regression. The null hypothesis of homoskedasticity could not be rejected at the 5% significance level. (p = 0.13).

5 Prevalence estimates are obtained using the formula: $P_i = \frac{1}{1+e^{-(\alpha + \beta Y_i)}}$. See Intriligator (1978).
### Box 4: Logit regression results

The logit regression as it would ensure that the prevalence estimates from the regression would remain within the range of 0-1. The logit regression uses the log odds ratio as the dependent variable,\(^1\) and household income \((Y_i)\) as the explanatory variable.

\[
\frac{\ln P_i}{(1-P_i)} = -0.0968 - 0.07168Y_i \\
(2.295) \quad (0.023)
\]

\[R^2 = 0.385\]  
\[N = 17\]  
\[F = 9.388541\]

\(^1\) \(\ln P_i/(1-P_i)\) = the log of the odds ratio of arsenicosis. The odds ratio is the ratio of the probability that the person will have arsenicosis to the probability that the person will not have arsenicosis.

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### 5.2 Explanations for the role of socioeconomic status in arsenic poisoning

This finding of a statistically significant relationship between arsenicosis and household income may be explained by a number of possible causal factors. According to the National Research Council: “Variability in arsenic metabolism appears to be important in understanding the human response. There is evidence that methylating capacity differs among individuals and population groups. Different capacities would result in variations in tissue concentrations of arsenic. Also, environmental factors, particularly diet, might be important in explaining susceptibility.” (National Research Council, p193, 1999).

In humans, the liver methylates inorganic arsenic that is consumed in drinking water. The resulting arsenic metabolites are excreted in the urine. Differences in methylating efficiency may be the reason for variations in arsenic retained in the body, and thus susceptibility to arsenic poisoning. In this context, it will also be important to establish gender differences in exposure and effect. Social roles are likely to affect amount and duration of exposure, and the issue of gender differences in susceptibility to environmental contaminants and carcinogens is increasingly being addressed (IOM 1998).

Hsueh et al. (1995) found that chronic carriers of hepatitis B with liver dysfunction had a significantly increased prevalence of skin cancer. This evidence indicates that those with liver disorders have a diminished ability to methylate inorganic arsenic, as suggested by Buchet et al. (1984). Therefore, one hypothesis would be that there is a negative relationship between the prevalence of liver disorders and household income. However, this could not be verified using the data from the Samta demographic survey.

Evidence suggests that the role of nutrition may also be important in determining methylation efficiency and toxicity to arsenic retained in the body. Yang and Blackwell (1961) studied nutritional factors in the blackfoot endemic region of China (Province of Taiwan). Their results indicated that residents of this region consume a diet low in protein, and in particular the amino acid methionine. Vahter and Marafante (1987) found that a low amount of methionine or protein in the diet decreased methylation of inorganic arsenic in the rabbit. In
addition, insufficient vitamin intake, in particular vitamin B12, might reduce the ability of the
body to methylete arsenic (Buchet and Lauwerys 1985). For these reasons, women, whose
nutritional levels are frequently deficient in South Asian countries for reasons linked with
cultural norms and reproductive function, may be at particular risk.

It is also suggested that zinc and selenium may provide protection against the toxic effects of
accumulated levels of arsenic in the body (National Research Council 1999). It is suggested
that the diet of blackfoot disease patients in China (Province of Taiwan) is deficient in zinc and
selenium (Pan et al. 1996). The National Research Council (1999) notes that there is still
uncertainty over the relative importance of the various nutritional factors, and calls for more
research into the issue.

The Asia Arsenic Network (AAN) researchers indicated however, that household income
might serve as a good proxy for nutritional intake in Samta Village (Tani, 1999a;b).
Consequently, they ascribed an important part of the negative relationship between prevalence
and household income to this explanation. For an accurate estimation of nutritional intake, it
is also important to take into account patterns of intra-household food distribution, which may
favour or disadvantage various family members by age or sex, and which cannot be seen
through the general category of household income.6

However, household income might also be related to water practices in the village. The
importance of water storage techniques has been highlighted by Alaerts (1999). He highlights
the example of the water storage practices of people in the Laxipur area of Bangladesh. In this
area water is stored in small vessels to allow the iron oxide to settle on the bottom of the vessel
and this enhances the concentration of adsorbed arsenic in the sludge. Higher income
households might have greater storage facilities for their tubewell water and might
consequently be able to store the water for longer. If higher income households are using
water that has been allowed to settle for a period before drinking, then this could help to
explain the observed arsenicosis-income relationship.7 Unfortunately, we are currently not
informed about longer-term water storage practices in Samta Village.

In Bangladesh it is likely that access to tubewell drinking water will be at least partially
determined by social status. Therefore, the observed relationship between arsenicosis
prevalence and household income could be due to social barriers to access to arsenic-free water
for poor households.

6. The socioeconomic impact of arsenic poisoning: coping and steps towards
modelling

Previous sections have highlighted the clear linkages between socioeconomic status (of a
household) and arsenic exposure. The latter is both a determinant and an outcome of the former, and
vice-versa. Thus low socioeconomic status may pre-dispose households and individuals to both

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6 This is important given the often poorer nutritional status of women and girls compared to men in some South-East
Asian countries. Appropriate gender analysis of data, as well as disaggregation of data by sex, are needed to ascertain
any differences in arsenicosis attributable to nutritional status.

7 This is especially valid where there is excess iron in the water. Storage of 200-500 litre is recommended as first partly
(subsidized) step towards arsenic avoidance, and it will also help in collecting better rainwater, and to get water for the
week during the end of the dry season (Heijnen 1999).
exposure and to the adverse effects of exposure. The effects of exposure will impact adversely on households through a variety of means and conspire to increase pre-disposition to further exposure and farther adverse effects.

6.1 **Coping with the impact of arsenic poisoning: introductory elements**

Household coping mechanisms as a result of illness have been discussed, among others, by Pryer (1989) and Sauerborn et al. (1996). The latter have been used in our further analysis.

Arsenic poisoning in communities has numerous effects, and families are likely to use a range of coping mechanisms. An important issue arises when families first become aware of the hazards of the use of arsenic-contaminated water. While an individual or household may not even be exposed to arsenic, or if exposed, onset of arsenicosis symptoms may not have started, how will families decide to protect themselves and ensure access to safe water? The absence of minimum information to enable/support household decision-making, for instance regarding the risk itself and the safety of the customary water source for drinking, laundry and other purposes will create inefficiencies and stress. Due to the use of nearby tubewells in the past, women and children saved countless hours of walking to more distant water sources. Are these savings threatened now, because of new demands on time in order reach safe water sources? Furthermore, are payments involved in acquiring safe water? And what are the opportunity costs (reduced time for work or going to the market, for cooking and other home activities) of this time allocation?

It is important to point out that the potential physical access to safe water is no guarantee that families can use this source. For example, access to safe water may be difficult for social reasons. Families may have to negotiate the use of water with others who own a hand pump, a yard connection (piped system), a storage tank, or a rainwater harvesting system with storage. However, class and caste relationships, such as rich-poor or landlord-tenant farmer, may impede sharing of water between families of different socioeconomic backgrounds. The use of financial incentives to enhance tubewell sharing may overcome some problems, however, social constraints may prove so strong that the efficacy of purely financial incentives is far from guaranteed.

Once family members become ill, various coping mechanisms come into play, depending upon the status of the afflicted person. A significant amount of attention is paid to the effects of illness of the breadwinner, usually the father. Coping with the burden of treatment costs constitutes a first important issue for the family. For Bangladesh, Pryer (1989) found earlier that “large” medical expenditure would be paid out of the sale of assets. It would need to be ascertained whether these assets are factors of production, such as land, that affect future income, or are smaller assets like beds, tables, chairs, fan or radio. It has also been found that intra-household labour substitution takes place to preserve income. For instance, family members such as the breadwinner’s wife and mother could put in extra hours. The children in the family could be involved in selling goods or foods at the market place. Another coping mechanism could be the reduction of food consumption or other consumption of other basic needs items (including expenditure for clothing, education, housing). It was also found by Pryer (1989) that some households would accumulate large consumption loans to finance lost income as a result of the breadwinner’s illness.\(^8\)

\(^8\) We refer to Carrin, Gray and Almeida (1999) for an analysis of coping with tuberculosis, based among others on Pryer’s findings.
Coping is also likely to be different between rural and urban areas. For instance, the economy of poor households in urban areas is likely to be linked much more closely with the urban manufacturing sector, offering in principle a wider variety of coping mechanisms. Evidence on coping with illness, but among urban slum residents in Dhaka-City is available from Desmet, Bashir and Sohel (1998). For daily wagers they found that “sacrificing holidays” is the first coping strategy following loss of income due to illness, followed by intra-household labour substitution, and foregoing consumption of commodities. Taking loans and using cash savings rank fourth and fifth in the list of coping strategies. It is interesting to note here that on the whole expenditure for basic needs items such as staple food, education, clothing and education does not seem to be affected.

6.2 The policy question: the arsenic poisoning and its socioeconomic impact

Given the millions of people in Bangladesh who are currently menaced by arsenic poisoning, health policy-makers need to devise policies capable of counteracting this threat. They need to show that an increasing number of people will suffer from arsenicosis if mitigation methods are not implemented rapidly. It is also important to demonstrate the social and economic effects on households with arsenicosis patients, and how mitigation methods can reduce the burden of those effects. One method of analysis would be a cohort study, selecting “control” (no intervention) and “intervention” villages (with implementation of mitigation methods) and tracking the effects of the disease on people’s health and livelihood, including coping mechanisms, over some period of time. Any study method on real populations would, however, only give results after long periods, which limits applicability to the immediate public health concern. In addition, it is questionable whether long term cohort follow-up would be achieved in a country where tracking of individuals is limited. Ethical considerations would preclude such studies as soon as it becomes apparent that mitigation methods do work and provide relief.

An alternative rapid method of analysis and of making predictions would be to apply simulation methodology which allows conducting experiments using a model of the real system (Budnick et al. 1997). A model could be established consisting of two sub-models addressing, respectively, the epidemiology of the disease and socioeconomic effects on households, for instance at village level. Any probability distributions for parameters and variables can be reconstructed via the generation of random numbers.\(^9\) We would first establish a base-line simulation run, mimicking what would happen in the village in the case of no-intervention. Subsequently, simulation runs can be done measuring the health and socioeconomic effects of interventions vis-à-vis the base-line. However, simulation modeling, even though one can “save” the lengthier process of a cohort study, still requires an appropriate data base. In practice, the latter is generally a combination of some retrospective material, results from recent surveys, and new rapid surveys.

7. A sub-model of the epidemiology of arsenicosis at village level

Let us suppose that we want to make predictions over a thirty year period (from year 0 or T0 to year 30 or T30) of the evolution of the disease and its health impact in a village of, say,

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\(^9\)This is often referred to as Monte-Carlo simulation.
100 households. This is an important prerequisite before any socioeconomic modelling can be undertaken. We introduce basic prevalence data on arsenicosis in T0, including the stage of arsenicosis that patients are in. Subsequently, we introduce the arsenicosis incidence rates, year by year. We also define the probability of moving between the various stages of the disease, and introduce the mortality rates associated with those stages.

As some minimum data are already available for Bangladesh, namely from the baseline epidemiological data for Samta Village in Jessore district (Tani, 1999a;b), we proceeded to establish an epidemiological sub-model, as follows.

A base case simulation was run to estimate the impact of arsenicosis on the village in the absence of any mitigation methods over a 30 year period. The length of the simulation period was chosen because of epidemiological evidence that internal cancer health effects tended to occur from around 20 years after exposure (Smith et al. 1998).

7.1 Epidemiological model

A model of a rural village containing 100 households was constructed. It is based partly on data collected from Samta Village, in the heavily arsenic contaminated Jessore district of Bangladesh together with published epidemiological literature. Arsenic concentrations in well water ranged from 0-1.37 mg/l, with the mode concentration (49% of wells) occurring in the range of 0.10-0.29 mg/l (Yokota et al. 1997). This is greatly in excess of the WHO recommended value of 0.01mg/l (Box 5). It is this mode value that is used in the estimation of cancer risks. For simplicity in modelling, each household is assumed to members: a father, mother, son and daughter. It is assumed that each household has been using arsenic polluted water since tubewell installation began in the 1970s. The natural history of arsenicosis is modelled over a 30 year period. A base case simulation of the model was run using the best available estimates for parameter values. For sensitivity analysis, high and low impact scenarios were simulated using extreme values for the uncertain incidence parameter. The main outcome measures analysed are the morbidity and mortality statistics at five-year time intervals. Several assumptions are used, either for necessary simplification of the model or because of data unavailability. Each household was assigned an income based on the Samta demographic study income data (Tani, 1999a;b). This assignment was achieved using the random sampling function of Microsoft Excel. These income figures were then used to calculate prevalence as discussed in the following section.

The modelling of arsenicosis can be considered in three sections. Firstly, assignment of prevalence at the beginning of the simulation, secondly, the assignment of incidence of new arsenicosis sufferers each year throughout the simulation and thirdly, the disease profile of arsenicosis sufferers for the 30 year period.

Arsenicosis is modelled as having a number of clinical manifestations. It is not possible to include all the adverse health effects from arsenic reviewed in section 3. It has been possible to include only skin lesions and cancers as the resulting health effects of arsenic exposure, as these are the health effects for which data was available for Bangladesh. The definition of health states used in the present analysis is based partly on the classification of patients used by the AAN researchers. They classified skin lesion suffers as 1, 2 or 3 based on the severity
The WHO Guidelines for Drinking-water Quality are intended as a basis for the development of national standards in the context of national environmental, social, economic and cultural conditions. The last edition of WHO guidelines for drinking water quality (1993) established 0.01 mg/l as a provisional guideline value for arsenic in drinking water. The fact that inorganic arsenic compounds are classified by IARC in Group 1 (carcinogenic to humans) on the basis of sufficient evidence for carcinogenicity in humans and limited evidence for carcinogenicity in animals was taken into consideration.

WHO has had a public position on arsenic in drinking water since 1958 when the first version of International Standards for Drinking-Water was published. In this instance the term "standards" was used to be applied to the suggested criteria of water quality. It established 0.20 mg/l as an allowable concentration in the category of toxic substances which, if present in drinking-water supplies at concentrations above certain levels, may give rise to actual danger to health. The updated standards in 1963 kept arsenic in the same category but established a stricter concentration of 0.05 mg/l. This value had kept being recommended for thirty years until it was superseded by 0.01 mg/l in 1993.

of their symptoms (Tani, 1999a;b). Those villagers classified as 1 were suffering from malanosis (hypopigmentation and hyperpigmentation). Villagers in category 2 were suffering from melanosis and keratosis on the hands and/or feet. Villagers in category 3 were the most severely affected and were suffering from melanosis and developed (late stage) keratosis on the hands and/or feet. This classification is similar to that proposed by A.W Khan (WHO 1997). He suggested that arsenicosis could be categorised into 3 sequential stages:

1) melanosis;
2) keratosis with or without anaemia, conjunctivitis, bronchitis, gastroenteritis and blackfoot disease;
3) developed keratosis and skin cancer.

This sequential stage theory of arsenicosis will be used to aid the modelling of arsenicosis over time, as discussed in the following sections.

7.2 Assignment of prevalence and incidence

Before the start of the simulation at time period T0, a random assignment of arsenicosis sufferers is generated. This assignment is based on the prevalence (see Box 6) data calculated from the Logit regression in section 4. The prevalence rates are a function of the assigned household income but there is random assignment within the household income class. Following this assignment sufferers are distributed among stage 1: melanosis (probability α), stage 2: keratosis (probability β) and stage 3: late stage keratosis (probability ?). These
probabilities are obtained from the proportions of arsenicosis sufferers in each category in Samta Village (Tani, 1999a,b).\textsuperscript{10}

In the years from T1 onwards, the incidence of disease (see Box 6) is used to simulate the new arsenicosis cases occurring each year. The incidence is defined as the number of new cases/population without arsenicosis. An incidence rate of 0.0038 is assumed for the base case scenario,\textsuperscript{11} with high and low impact scenarios also simulated using incidence rates of 0.0019 and 0.0076 respectively. Assignment of incidence is randomly generated. If a person is assigned as having developed arsenicosis, they are then assigned to the initial melanosis stage 1.

\begin{center}
\textbf{Box 6: Prevalence and incidence}
\end{center}

\begin{quote}
\textit{Prevalence} refers to the cases of the disease that exist at a specified instant of time or during some period of time.

\textit{Incidence} refers to the number of new cases of the disease that develop during some specified time interval.

Thus, a prevalence rate (or, more precisely, ratio) is the number of cases divided by the population at that instant of time, or, over a time period, it is the number of existing cases divided by the mid-period population during that period.

On the other hand, an incidence rate has as its numerator the number of new cases of the diseases that arise during the specified time interval and, as its denominator, the population in risk but free of the disease at the beginning of the time interval".

(Colton 1974)
\end{quote}

7.3 Natural history of arsenicosis

A state transition model is used to describe the disease profile of arsenicosis after the initial assignment of illness. State transition models are useful for analysing diseases that occur over an extended time period (Sonnenberg and Beck 1993). A state transition model requires each individual in the model to be placed in one of a number of clearly defined health states, which can include healthy and dead. The natural history of arsenicosis is modelled by transitions through different health states. As arsenic is a cumulative toxin, it is assumed that the severity of a patient’s illness increases over time if exposure continues (as in base case scenario). Patients are modelled as moving sequentially from melanosis through to late stage keratosis over time. Changes in patient health status are modelled as a transition from one health state.

\begin{itemize}
\item Melanosis (stage 1) probability = 0.586;
\item Keratosis (stage 2) probability = 0.262;
\item Late stage keratosis (stage 3) probability = 0.152.
\end{itemize}

\textsuperscript{10} This was derived from a prevalence formula, assuming that in certain villages prevalence was already 4.7\% in 1983.

\textsuperscript{11} Melanosis (stage 1) probability = 0.586; Keratosis (stage 2) probability = 0.262; Late stage keratosis (stage 3) probability = 0.152.
to another. Each possible transition has an associated transition probability. The structure of the state transition model is presented in Figure 7.1. Table 7.1 presents the transition probability estimates.

Each arrow in Figure 7.1 refers to a possible transition at the end of each year. The bold arrows indicate that the person has remained in the same health state. The health state 7 (dead) has been included in the model to allow for future modelling of mortality in the skin lesion health states. In the following simulations the risk of mortality in these health states is assumed to be zero. Therefore, in the base case simulations discussed in this paper, no individuals are in health state 7.

In addition to skin lesion health effects, a number of cancers are modelled. Skin cancer is assumed to be a hazard only in those with the most advanced stage of skin lesions. Assignment is modelled as a transition from late stage keratosis (3) to skin cancer (4). Skin cancer risk is assumed to begin at T10, ten years into the simulation. Whilst mechanistically the assignment of zero risk up to 10 years of exposure may be imprecise, it does reflect the information reported to date from Bangladesh and has simplified the modelling undertaken.

There is currently disagreement over whether internal cancers are a risk that is independent of skin lesions. Findings by Cuzick et al. (1992) indicate that internal cancers only occur for those with skin lesions, whereas Tsuda et al. (1995) found cancer occurrence among those without any skin lesions. Due to this uncertainty, a combined bladder and lung cancer risk is incorporated for both those with and without existing skin lesions. These internal cancers are modelled as appearing after T20, 20 years into the simulation. This risk is modelled for those without skin lesions as a transition from healthy (0) to the internal cancers health state (5). The modelling of internal cancers for those with skin lesions is achieved by assuming a transition from late stage keratosis (3) to internal cancers (5). The data used in the calculation of cancer transition probabilities is contained in Appendix I. Despite the existing uncertainty in the mechanisms of disease causation it would probably be most appropriate to model non-skin cancers as random events with probability increase with total exposure, and independent of skin manifestations; whilst modelling skin cancer as a progression from stage 3 keratosis. The approach taken to date is a simplification that will subsequently be refined.

Mortality from cancers is modelled as a transition probability from the cancer health state to the health state; dead (6). It is assumed that those in the cancer health state die in the following year. This assumption of 100% cancer mortality was necessary because the epidemiological data used was for cancer mortality from arsenic exposure. The latter reasonably reflects the reality in poor rural areas of Bangladesh.

7.4 Model results

Simulation results are presented at the end of the paper in Tables 7.2 to 7.4 and Figures 7.2 to 7.4 using the base case, high and low impact estimates of arsenicosis incidence. The figures highlight the health status of those exposed to arsenic. In the base case (Figure 7.2), 22.5% of the village is in one of the arsenicosis health states at the end of the 30 year simulation. 5.5% have died from skin or internal cancers and 0.5% of villagers have developed skin and internal cancers in the final year of the simulation. The prevalence of late stage keratosis has increased from 1% in T0 to 7.5% in T30 at the end of the simulation. This highlights the progressive
decline in health status of arsenicosis sufferers over time. The overall prevalence of skin lesions has increased from 8% in T0 to 16.5% in T30.

Using the low impact estimate for arsenicosis incidence of 0.0019 shows the expected reduction in arsenicosis health impact (Figure 7.3). 15.75% of the village is in one of the arsenicosis health states, as compared to 22.5% for the base case incidence simulation. Skin and internal cancer mortality is reduced by half a percentage point from the base case simulation to 5%. 0.5% have developed skin and internal cancers in the final year of the simulation. The overall prevalence of skin lesions in T30 is 10.25% as compared to 16.5% in the base case, reflecting the lower growth rate in number of arsenicosis sufferers.

Using the high impact estimate for incidence of 0.0076 means that 29.25% of the village ends the simulation in one of the arsenicosis health states (Figure 7.4). Cancer mortality is increased to 6.5% with 0.75% having developed cancers in the final year of the simulation. The overall prevalence of skin lesions in T30 is 22% as compared to 16.5% in the base case simulation.

8. Sub-model of arsenicosis and its socioeconomic impact on village households

8.1 Socioeconomic household survey at T0

Just as a minimum set of epidemiological data is needed for T0, an initial socioeconomic survey of households in the community is necessary to assess any changes in the socioeconomic position of households due to the disease. In the case of the Samta Village survey, this was undertaken at the level of the village as a whole. Alternatively, a survey among a sample of villagers can be envisaged.

A socioeconomic household survey will have to include at the minimum a number of basic indicators as indicated in Box 7.
Box 7: A number of basic indicators

a. demographic variables
   a.1 household size
   a.2 age and sex of household members
   a.3 marital status of adult members

b. household income and assets
   b.1 occupation and wages of all household members in the case of formal employment
   b.2 type of activity and cash income of all household members in the case of informal employment
   b.3 cash equivalent of consumption of self-produced food products
   b.4 assets, including land holdings, cash savings, animals/livestock
   b.5 amount of microcredits

c. household expenditure pattern
   c.1 food and clothing
   c.2 education
   c.3 health care
   c.4 water supply
   c.5 fuel
   c.6 maintenance costs (housing, water supply infrastructure)
   c.7 other

d. education
   d.1 children’s enrolment in primary, secondary or tertiary education (according to gender)
   d.2 school attendance (as a % of theoretical school attendance)

e. water supply
   e.1 source of drinking water (tap, tube well, other) and ownership
   e.2 water source for cooking
   e.3 water source for bathing
   e.4 time spent by each household member on collecting water and distance from source of each type of water

f. housing
   f.1 roof with non-permanent material
   f.2 thatched roof
   f.3 metal roof
   f.4 other

8.2 Coping

The following step is to study how diseased patients, predicted by the model, will cope with the illness, and what effects this will have on their socioeconomic status.
An immediate issue is whether affected persons have access to health care treatment and, in this case, how the costs will be paid. The following minimum set of variables could be measured on a per patient basis (Box 8).

### Box 8: Access to treatment and financing

1. **Stage of the disease (melanosis, keratosis stages 1 and 2, cancers)**
2. **No treatment or self-care, or treatment by provider**
3. **In the case of provider treatment**
   - **Patient cost of ambulatory care** (episodes of ambulatory care, treatment cost per episode)
   - **Patient cost of hospitalisation** (number of admissions, treatment cost per admission)
4. **Ways of financing patient cost**
   - **Regular household income**
   - **Additional income earned by other household members**
   - **Cash savings**
   - **Loan**
   - **Payment in kind**

Arsenicosis may lead to changes in work responsibilities inside and outside the home for all family members, and in the case of children to changes in school attendance. We propose to measure the following variables (Box 9) at the patient level.

### Box 9: Coping mechanisms at the household level

1. **Percentage of initial income reduction** (in the case of adult patient) or percentage of missed school-days (in the case of a child patient)
2. **Degree of reliance on family members in order to sustain household economy**
   - **Time input of other family members**
   - **Amount of income compensated by family members**
   - **Time input of schoolchildren and number of school-days missed**
3. **Other ways of coping**
   - **Reduction of consumption by different family members of staple food, non-basic food items, other consumption items**
   - **Use of cash savings**
   - **Sales of assets**
   - **Loan**
Arsenicosis may also lead patients and their families to react to demand more information about arsenicosis in their communities, or to request that the testing of water quality from tubewells be speeded up. This can be interpreted as a coping mechanism at the community level, which could be measured as follows (Box 10).

<table>
<thead>
<tr>
<th>Box 10: Coping at the community level</th>
</tr>
</thead>
<tbody>
<tr>
<td>c.1 number of meetings that took place at the community level to address arsenic poisoning (categories of households and household members that attend)</td>
</tr>
<tr>
<td>c.2 inclusion of arsenic messages into an existing health and education programme</td>
</tr>
<tr>
<td>c.3 number of tube wells tested and painted</td>
</tr>
</tbody>
</table>

As a result of these reactions at the community level, patients and their families may adjust their behaviour, measured as follows (Box 11).

<table>
<thead>
<tr>
<th>Box 11: Adjustment of behavior with regard to water use</th>
</tr>
</thead>
<tbody>
<tr>
<td>d.1 use of water by the family at safe tube wells</td>
</tr>
<tr>
<td>d.1.1 family member(s) are in charge of collecting the water</td>
</tr>
<tr>
<td>d.1.2 servants are in charge of collecting the water</td>
</tr>
<tr>
<td>d.2 reasons for continuation of use of unsafe water</td>
</tr>
<tr>
<td>d.2.1 excessive time demand on family members or servants, due to distance</td>
</tr>
<tr>
<td>d.2.2 no access due to social constraints</td>
</tr>
</tbody>
</table>

Conceptually, what is proposed above can be achieved. Data are not currently available, but could be collected so that the modelling can proceed. Using the suggested minimum set of indicators above, ways of coping with arsenicosis could be tracked for the community as a whole. Morbidity and mortality, as well as total income and asset loss for the community can be studied. With these data, it would also be possible to investigate which households, and of which socioeconomic background, are likely to have greater difficulty than others in coping with the illness. In other words, it can be analysed to which extent the situation of poor households would worsen as a result of the illness, and which specific members of these households would be most affected.

8.3 Modeling the impact of mitigation methods

A final step is the analysis of the impact of the three alternative mitigation methods mentioned above.
• **First**, there is the expected effect on the epidemiology of arsenicosis, or the health effects. Significant attention will need to be paid to modelling the progress (including reversal) of the disease in some patients, as a result of use of safe (or safer) water. It is still unclear at what stage the disease becomes irreversible, but it is accepted that it is reversible in the very early stage of the disease.

• **Secondly**, effects on changes in socioeconomic variables measuring the livelihood of households have to be studied. For instance, it should be possible to quantify expected change in important variables like treatment costs, household income and time allocation of household members. Any social or other impediments to access, especially for the poor and most vulnerable, despite the introduction of mitigation measures, will also have to be analyzed.

• **Thirdly**, the proposed financial contribution of households to the implementation of the alternative mitigation methods has to be clarified and tested for feasibility and acceptability. Co-financing options with central and local Government, and donors, also need to be explored, particularly where subsidies to the poor are shown to be the predominant way of financing mitigation measures.

• **Finally**, this analysis will also enable us to undertake a cost-effectiveness analysis of mitigation methods. Effectiveness could be measured, for instance, in terms of healthy days gained or in terms of Disability Adjusted Life Years (DALY) (Murray and Lopez 1994).

### 9. Remaining challenges for modelling

Above we have outlined the major components of a simulation model, and presented a first version of the epidemiological sub-model. This section will describe additional methodological and data problems, and will suggest where further work is needed to improve the realism of the modelling effort.

#### 9.1 Including other health and social effects

In the epidemiological sub-model presented above, we have only considered the cutaneous manifestations of arsenicosis and certain cancers. In reality, the likely health effects of exposure to arsenic in drinking water are more wide-ranging as noted in section 3. Epidemiological studies have pointed to the existence of risk from numerous cancers due to inorganic arsenic ingestion in water. However, the decision to only consider lung, bladder and skin cancer risks was due to the strength of epidemiological findings for these cancers across different populations and different ranges of arsenic dose exposure. A number of other sites have been suggested for cancer (kidney, liver and colon according to Chen et al., 1985). Other non-cancer health effects are noted as well, such as spontaneous abortions and still-births. These inferences may or may not be proven, but the main barrier to inclusion of the other health effects is a lack of adequate epidemiological data relating to their prevalence. The current figures for the health impact of arsenic will therefore underestimate the true disease burden on the village. Ideally, further extensions should be made to the sub-model, in terms of inclusion of other health effects. However, for this to be possible, dose response estimates of prevalence and incidence for village populations would have to become available.
When modelling the effect of mitigation methods, attention should be paid to the impact of clean water on other water-related diseases like diarrhoeal disease and cholera. It should be investigated to what extent different mitigation methods have different impacts upon water-related disease incidence and subsequent morbidity and mortality. Only if these impacts are shown to be similar, can one limit oneself to the differential impacts on arsenicosis.

Arsenicosis has a strong social dimension, affecting issues such as relationships within the family, and the mental health of the sick. Many arsenicosis sufferers have been ostracised at either the household or village level. The Harvard Public Health Review (1999) highlights the case of a patient suffering from skin lesions whose children were unwilling to eat the food she served, and whose husband eventually divorced her. Social and gender relations thus pose additional threats to security and wellbeing. It is essential to pay attention to these factors in assessing the effects of arsenicosis. The real impact on family and gender relations, and on livelihoods, of the stigma attached to arsenicosis sufferers, would constitute a further useful study.

9.2 Transition between stages of arsenicosis

The nature of arsenic as a cumulative toxin poses problems for modelling. In the epidemiological sub-model a simple disease profile has been used to illustrate patients becoming progressively more sick over time. This has been modelled as a probability of transition to the next stage (e.g. progression from the melanosis to the keratosis stage of arsenicosis.) An attempt to incorporate the cumulative toxic effect of arsenic has been included by the simplistic method of increasing the transition probabilities every time period of 10 years. However, this is only a proxy mechanism for analysing the effect of cumulative arsenic exposure over time.

In order to make arsenicosis fully time dependent in the model, one would need to have varying transition probabilities related to the extent (level, time) of arsenic exposure. In other words, transitional probabilities should be made a function of exposure. However, this would require a more complex model structure than was possible for the present research. Future extension to the model could make arsenicosis fully dependent on length of arsenic exposure. Conversely, in modelling the effects of mitigation methods the reverse transition probabilities should also be a function of exposure. This is because the recovery process should also be dependent on the length of arsenic exposure.

9.3 Unit of time in the simulation model

In the epidemiological sub-model, the time unit was a year. The latter may have to be reconsidered in that shorter time units (monthly or quarterly) may be more appropriate. Impacts on households and coping strategies may be different according to the time of onset of an episode of disease during the year. For instance, the burden of episodes of disease and

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12 One possible functional form could be a linear one, with a constant increase in the transition probability related to an increase in years of exposure: \( T_{ij} = \alpha t \) where \( T_{ij} = \) transition probability from health state \( i \) to health state \( j \); \( i = (1,2,3,4,5) \) and \( j = (1,2,3,4,5,6) \); \( \alpha = \) probability term; \( t \) = number of years of arsenic exposure. This indicates that the transition probability is a function of the number of years of arsenic exposure. In other words, there is a constant decrease in health over time due to the effects of arsenic exposure. However, if this is considered too simplistic, a more complex form could be utilised. An exponential functional form could be used if it is believed that the effects of arsenic become increasingly damaging as the length of exposure increases.
treatment may be different according to the agricultural season: a potential loss of working
time may constitute a greater burden at harvest time, but a lesser burden after harvest time
when cash income could be expected to be at its highest. Or, if payment of treatment costs
coincides with the beginning of the school year, families’ capacity to pay school fees or other
school expenses may be affected.

10. Conclusion

Attention has been drawn to the fact that ingestion of arsenic contaminated water leads to
serious skin disease and cancer in a significant number of exposed people. It is important to prevent
the increase of arsenicosis for both health and economic reasons. However, drug treatment to
eliminate arsenic from the body is expensive, leading to the conclusion that possibly palliative care,
including application of ointment in the case of keratosis, is the only affordable treatment in rural
areas of Bangladesh. Bangladeshi villagers affected by arsenicosis are likely to lose a significant
amount of productive time. In addition, the disease may become a burden on villagers’ overall
financial and time resources. We have shown, using econometric analysis, that the poorest suffer
the most from arsenicosis.

It is clear that mitigation methods need to be implemented urgently, not least for reasons of
poverty alleviation. It is important to assess the future health and socioeconomic impacts of
alternative mitigation methods, so that policy-makers in Bangladesh can take informed decisions.
To avoid the time implications of a prospective cohort study, we propose to apply simulation
methodology. An appropriate simulation model would consist of basically three components: first
an epidemiological sub-model forecasting the natural history of the disease in the absence of
mitigation measures; second a socioeconomic sub-model showing how households cope with the
disease in the absence of intervention; third, a component that addresses how alternative mitigation
methods halt the progression of the disease, and prevent or reduce economic losses.

The paper gives a detailed account of the construction and results of the epidemiological sub-
model. It also proposes to obtain socioeconomic information on households and their coping
mechanisms through the organization of simple household surveys; it therefore lists a minimum set
of core health and socioeconomic indicators to be considered. We hope this paper can be an input
into further discussions and decision-making about mitigating existing damage and preventing
further arsenic poisoning in Bangladesh. In particular, important discussion and analysis will be
needed on alternative financing strategies for hot-spot areas, thereby specifying the roles of donors,
local and central Government, and households.
BIBLIOGRAPHY


Figures and Tables
Figure 5.1 Estimates of relationship between prevalence and household income based on Logit regression results.
Figure 7.1 Structure of the State Transition Model

- Healthy
- Melanosis
- Keratosis
- Late stage keratosis
- Internal cancer
- Skin cancer
- Mortality from skin lesions
- Mortality from cancer

Transition probabilities:
- From T20: Healthy to Melanosis, Healthy to Keratosis, Healthy to Late stage keratosis, Healthy to Internal cancer, Healthy to Skin cancer.
- From T20: Melanosis to Keratosis.
- From T10: Keratosis to Late stage keratosis.
- From T20: Late stage keratosis to Internal cancer.
- From T20: Skin cancer to Mortality from cancer.
Table 7.1 Parameters used in the model

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevalence</td>
<td>Varies with income</td>
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</tr>
<tr>
<td>Incidence</td>
<td></td>
<td></td>
</tr>
<tr>
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<td>See footnote 7 for calculation of incidence rates</td>
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<tr>
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<td></td>
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Table 7.2: End period health status: percentage of village population in each health state

<table>
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<tr>
<th></th>
<th>T0</th>
<th>T5</th>
<th>T10</th>
<th>T15</th>
<th>T20</th>
<th>T25</th>
<th>T30</th>
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<td>88</td>
<td>85.5</td>
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</tr>
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<td>4</td>
<td>3.75</td>
<td>3.5</td>
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<td>4.25</td>
</tr>
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<td>7.5</td>
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<td>0.25</td>
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Figure 7.2 : Health status of arsenicosis affected population.
Base case incidence
Table 7.3: End period health status: percentage of village population in each health state

<table>
<thead>
<tr>
<th></th>
<th>T0</th>
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<th>T15</th>
<th>T20</th>
<th>T25</th>
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</tr>
<tr>
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<td>1.75</td>
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Figure 7.3: Health status of arsenicosis affected population.
High impact incidence

![Health status chart](image-url)
Table 7.4: End period health status: percentage of village population in each health state

<table>
<thead>
<tr>
<th>Health State</th>
<th>T0</th>
<th>T5</th>
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<th>T15</th>
<th>T20</th>
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<tr>
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<td>2.75</td>
</tr>
<tr>
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<td>3.25</td>
<td>3.25</td>
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Figure 7.4: Health status of arsenicosis affected population.
Low impact incidence

![Health status graph](image_url)
Appendix I

Calculation of cancer transition probabilities

Skin cancers

WHO calculated a lifetime risk of skin cancer of 5% for exposure to 0.2mg/l of arsenic in drinking water. This dose response model was calculated using data from Taiwan in Tseng (1977). This risk was used to calibrate the skin cancer transition probabilities in the model. A simulation was run to estimate the percentage of villagers in the late stage keratosis health state at the end of T30. This percentage was then used to calibrate the necessary transition probability to achieve a 5% skin cancer outcome in T30. It was found that a skin cancer transition probability of 0.0278 achieved close to 5% skin cancer mortality for the three incidence rates.

Internal cancers

Dose response models have not been calculated by WHO for internal cancers due to insufficient data on exposures, as discussed in section 3.2. Therefore, it has been necessary to estimate internal cancer transition probabilities using alternative methods. The transition probabilities calculated for internal cancers use data on Standardised Mortality Ratios calculated by Smith et al (1998), combined with data on average annual cancer incidence for India. This method of calculation assumes that mortality rates from arsenic cancers in Chile are directly proportional to Indian cancer incidence.

Smith et al calculated SMRs for those aged over 30 for a region of Chile exposed to high levels of arsenic in drinking water. They calculated population weighted averages of arsenic concentrations for drinking water for each of the 5 year periods from 1950-1994. The mean of these 5 year averages = 0.28 mg/l. This figure lies within the mode concentration of well water in Samta Village of 0.10-0.29 mg/l (Yokota et al. 1997). Therefore, it was assumed that the arsenic exposures are comparable. We then took the mean of the SMRs calculated by Smith et al for bladder and lung cancers:

<table>
<thead>
<tr>
<th>Cancer Type</th>
<th>SMR</th>
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<tbody>
<tr>
<td>SMR lung cancer, males</td>
<td>3.8</td>
</tr>
<tr>
<td>SMR lung cancer, females</td>
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</tr>
<tr>
<td><strong>Mean lung cancer SMR</strong></td>
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</tr>
<tr>
<td>SMR bladder cancer, males</td>
<td>6.0</td>
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<tr>
<td>SMR bladder cancer, females</td>
<td>8.2</td>
</tr>
<tr>
<td><strong>Mean bladder cancer SMR</strong></td>
<td><strong>7.1</strong></td>
</tr>
<tr>
<td><strong>Mean of lung and bladder SMRs</strong></td>
<td><strong>5.28</strong></td>
</tr>
</tbody>
</table>
The mean annual cancer incidence for bladder and lung cancers in India was then calculated for those aged over 30 (IARC 1987). India was used as the closest proxy in the absence of data for Bangladesh:

<table>
<thead>
<tr>
<th>Region</th>
<th>Mean annual lung cancer incidence per 100 000 for those aged 30 plus</th>
<th>Mean annual bladder cancer incidence per 100 000 for those aged 30 plus</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bangalore</td>
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<tr>
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<tr>
<td>Nagpur</td>
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<tr>
<td>Poona</td>
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<td>10.12</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
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<td>Nagpur</td>
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<td>1.77</td>
</tr>
<tr>
<td>Poona</td>
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<tr>
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<td><strong>5.048</strong></td>
</tr>
<tr>
<td><strong>Mean for both sexes</strong></td>
<td><strong>20.626</strong></td>
<td><strong>9.038</strong></td>
</tr>
<tr>
<td><strong>Mean bladder and lung cancer for both sexes</strong></td>
<td><strong>14.83</strong></td>
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</table>