Reviews of Environmental Contamination and Toxicology

VOLUME 197
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Arsenic Pollution and Remediation: An International Perspective

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Exposure to arsenic-contaminated drinking water is a major threat to human health. Millions of people across the world are exposed to arsenic-contaminated drinking water with concentrations far in excess of the 10 μg/L maximum permissible level established by the World Health Organization (WHO).

The major arsenic exposure pathway is believed to be via natural (geological) sources of contaminated groundwater. In addition, arsenic is introduced into the environment from anthropogenic sources, primarily metal mining and smelting activities, which pollute soils, sediments, and surface waters and groundwater worldwide. The implications for human health of arsenic exposure are serious, but neither are these implications fully understood nor are solutions for mitigation adequately evaluated or communicated.

The purpose of the six papers comprising this volume is to address this knowledge gap. These papers result from a project supported by the Chemistry and the Environment Division (VI) of the International Union of Pure and Applied Chemistry (IUPAC). They are consonant with and underpin the key IUPAC objectives of advancing the chemical sciences and the application of chemistry in service to mankind. IUPAC, in its role as an objective scientific, international, and nongovernmental body, in collaboration with international governmental bodies [e.g., United Nations Educational, Scientific and Cultural Organization (UNESCO) and the WHO] addresses many global issues involving the chemical sciences as well as issues that transcend pure science and have important sociopolitical implications. Arsenic contamination clearly has such implications.

The papers presented in this volume aim to review and analyze the status of arsenic pollution and consequential human exposure and to provide a practical guide to available arsenic remediation technologies. Moreover, we endeavor to advise on tools that support informed decision making when choosing avenues for arsenic mitigation. Such decision making cannot be solely concerned with arsenic treatment technologies, and the papers therefore seek to highlight and provide guidance on arsenic treatment technologies in the context of varying scenarios that can inform effective mitigation policies.

The authors of these papers have a diversity of knowledge, research experience, and interests, all of which contributed to assembly of this volume. The team’s expertise in epidemiology (Harry Caussey); risk assessment and toxicology (Nick
Priest); environmental chemistry (Hemda Garelick, Huw Jones, and Zoltán Galbács); environmental geochemistry (Eugena Valsami-Jones and Agnieszka Dybowska); analytical chemistry (Joerg Feldmann); bioremediation (Poransawan Visoottiviseth); environmental engineering (Feroze Ahmed, Rita Földényi, Nora Kováts, and Gábor Borbély); and environmental management (Bryan Ellis, Hemda Garelick, and Md. Khoda Bux) was critical in analyzing effects of and solutions to arsenic water pollution on exposed populations.

Key points addressed by each successive paper are these:

- Health risks of arsenic contamination, with reference to the technical challenges associated with optimizing arsenic remediation approaches that are acceptable to arsenic-polluted communities.
- Overview of the global status of arsenic pollution sources, both natural and anthropogenic, and behavior of arsenic in groundwater and surface waters. Information is provided on modes of formation and release of arsenic and the corresponding implications to environmental mobility and toxicity of different arsenic chemical species.
- Effects of high spatial and temporal variation of arsenic contamination and the consequential need for cheap, quick, onsite (field kits) analytical techniques that accurately portray the degree and nature of contamination so critical to remediation efforts are discussed.
- A variety of potential remediation technologies for arsenic removal are described. To be effective, particularly in developing countries with the greatest arsenic contamination, such methods must be reliable, cost-effective, and sustainable.
- The range of mitigation options available for arsenic reflects the complexity of its chemistry. Appraising suitable arsenic remediation technologies is itself a sizable challenge. In this paper, we address, through multi-criteria approaches, the factors relevant to evaluating mitigation options.
- The final paper of the series shares the challenges faced by three countries with arsenic-contaminated regions in addressing and remediating sources of arsenic contamination.

“Access to safe water is a fundamental human need and, therefore, a basic human right. Contaminated water jeopardizes both the physical and social health of all people. It is an affront to human dignity. Yet even today, clean water is a luxury that remains out of the reach of many.”

These words, spoken by Kofi Annan, then Secretary General of the United Nations, on World Water Day, March 22, 2001, sadly remain equally relevant in 2007. We, the authors, believe that this situation cannot be allowed to persist and hope that this series of papers will redress it, in some small way.

November 2007

Hemda Garelick and Huw Jones
Acknowledgements

The authors thank the IUPAC division of Chemistry and the Environment and its president, Dr Ken Racke, for its financial support and for providing a framework cogent to development and implementation of the project which led to this volume. Special thanks are extended to Dr Yehuda Shevah whose drive and commitment made this project possible.

We thank John Koushappas, Paula Newland, and Yvette Brown from the School of Health and Social Sciences Learning and Technical Unit at Middlesex University for their support in managing the web-based activities for our team and for their technical support in production of figures.

We, herewith, express our full appreciation to the independent referees of the papers—Prosun Bhattacharya, The Royal Institute of Technology, Stockholm, Sweden; Eton Codling, USDA, ARS, Beltsville, MD, USA; Karen Hudson-Edwards, Birkbeck College, London, UK; Jake Peters, USGS, Atlanta, GA, USA; and David Polya, The University of Manchester, UK—who provided comprehensive reviews and constructive feedback.

Very special thanks are owed to R.E.C.T. editor Dr. David Whitacre, who tirelessly and meticulously reviewed the manuscripts and who provided the team with outstanding editorial support.
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Introduction to Arsenic Contamination and Health Risk Assessment with Special Reference to Bangladesh

Deoraj Caussy(*) and Nicholas D. Priest

I Introduction

Arsenic is a metalloid element that occurs in nature in both organic and inorganic compounds. The valence of arsenic is three in arsenite (As (III)) and five in arsenate (As (V)) compounds. Arsenic compounds are poisonous and can be categorized into

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one of three broad categories, all with different toxicities: organic arsenic compounds, inorganic arsenic compounds and their solutions, and arsine gas. Of these, inorganic compounds are of relatively greater toxicological significance for human health than are organic compounds. Toxicological profiles for arsenic compounds have been published both in standard texts (Ishinishi et al. 1986) and in reports produced for international and governmental agencies (IARC 1982; ATSDR 2000; IPCS 2001). The reader is referred to these for a comprehensive description of the toxicokinetics and toxicity of arsenic. The purpose of this chapter is to review the salient health risks posed by arsenic contamination, with special reference to the technical challenges for optimizing arsenic remediation in ways that are acceptable to communities in Bangladesh.

II Exposure Pathways for Arsenic

Humans may be exposed to inorganic arsenic via air, water, food, and soil. Arsenic is widely distributed in nature as sulfides in minerals, as dissolved salts in groundwater, surface waters, and seawater, and in soils, sometimes consequent to its anthropogenic extraction and usage. Arsenic compounds are extracted from minerals and have been widely used as therapeutic agents, now mostly in the form of antiparasitic drugs, as pesticides, in the manufacture of glass, as an alloying agent, in the manufacture of some dyes, and as gallium arsenide for production of crystals in the semiconductor industry (for more information on arsenic sources, see “Arsenic Pollution Sources,” later in this volume).

Most food products usually contain less than 250 µg/kg arsenic. However, seafood such as demersal fish, crustaceans, and marine algae may contain up to ~100 mg/kg arsenic. The low levels in plants contrast with the much higher levels (~40 mg/kg) in soil and, under normal soil conditions, may reflect the insolubility of many arsenic-containing minerals such as pyrite. The average U.S. daily dietary intake for humans is estimated to be 10–20 µg arsenic. In Japan, however, where the seafood content of the diet is high, intakes are much larger (70–370 µg/d).

In addition to ingestion from food and drinking water, air is also a potential source for human exposure to arsenic. Significant intakes by inhalation occur in residents living near industrial sources where exposures to arsenic trioxide are possible; however, this is unlikely to be a significant source of exposure to natural forms of arsenic in

<table>
<thead>
<tr>
<th>Location</th>
<th>Recorded arsenic concentration (µg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nova Scotia, Canada</td>
<td>50</td>
</tr>
<tr>
<td>California, Romania, New Zealand</td>
<td>40–1,300</td>
</tr>
<tr>
<td>Japan</td>
<td>1,700</td>
</tr>
<tr>
<td>Cordoba, Argentina</td>
<td>3,400</td>
</tr>
<tr>
<td>Taiwan, China</td>
<td>1,800</td>
</tr>
<tr>
<td>Bangladesh</td>
<td>300</td>
</tr>
<tr>
<td>USA domestic water sources</td>
<td>&lt;10 (99%)</td>
</tr>
</tbody>
</table>

*Source: IPCS 2001.*
Bangladesh. Consumption of arsenic-contaminated water, however, poses a significant threat to human health in some parts of the world. The global nature of arsenic contamination in groundwater has been summarized in the report of the British Geological Survey (BGS 2001). Table 1 summarizes the arsenic levels found in groundwater in selected parts of the world. Arsenic contamination is particularly notable in deltaic plains of river basins in Asia, including the Brahmaputra Gangetic River and the Red River-Mekong deltas (Fig. 1). Groundwater contamination is further discussed later in this volume (see “Arsenic Pollution Sources”).

Bangladesh is among the countries most affected by arsenic contamination in the WHO (World Health Organization) South-East Asia Region. The origin of arsenic contamination in the Bengal River Basin can be traced to the deposition of arsenic-laden sediment in the Bengal deltas. This contaminated sediment has been carried by the Ganga and Brahmaputra Rivers from the Himalayan mountains to the delta over millions of years (McArthur 2002). These arsenic-containing sediments adhered to rocks and eventually formed the impervious layer of the aquifers in the delta. Increasing population growth led to the search for a source of drinking water free of microbial contamination, and, hence, the exploitation of aquifers that were subsequently found to be contaminated. Aquifer exploitation reached its
peak during the green revolution (McLellan 2002), when demand was compounded by the need for irrigation water. To meet growing needs, tube wells were sunk into the aquifers; in Bangladesh alone some 10 million tube wells were drilled into arsenic-contaminated aquifers. The consequence and tragedy was that some 40 million people were subsequently exposed to toxic levels of arsenic, sometimes exceeding the WHO Guideline value by a factor of 20 or more. Of the 64 districts within Bangladesh, 59 have reported unsafe levels of arsenic in groundwater (see the last chapter in this collection for more information on this topic). However, arsenic contamination is not homogeneous, and the correlation between arsenic concentrations in water, and disease prevalence, is less than perfect. Considerable geographic variability is apparent (Fig. 2). Most of the heavily contaminated areas are in the lower deltaic plain of the Brahmaputra River basin.

III Health Effects of Exposure to Inorganic Arsenic

The health outcomes resulting from arsenic exposure depend on the dose, modality, and duration of exposure, as well as the source and chemical type of arsenic (Caussy et al. 2003a). The major health effects of arsenic have been reviewed elsewhere (IPCS 2001). Arsenic is toxic following both acute and chronic intakes. However, drinking contaminated water is only likely to produce effects under conditions of chronic intake. Chronic effects produced by the ingestion of inorganic arsenic include skin lesions, disturbances of the peripheral nervous system, anemia and leukopenia, liver damage, circulatory disease, and cancer. Many of these effects have been observed in populations that consumed contaminated water, including populations in Taiwan, Argentina, and Bangladesh (IPCS 2001).
Under conditions of chronic intake, skin lesions are characterized by keratosis and melanosis of varying severity. These lesions are usually manifested on the palms of the hands and soles of the feet. Keratotic lesions may, at some stage, become malignant (by a process that remains unclear and may involve induction by factors other than arsenic) (IARC 1982) and result in squamous cell skin cancer (Caussy 2003a). Such tumors are commonly multifocal and develop throughout the body. Basal cell carcinoma has also been described in cases that display chronic arsenical dermatitis.

Chronic exposure to arsenic may also damage the peripheral nervous system. Such damage is characterized by a peripheral neuritis affecting mainly the upper and lower limbs. This neuritis results in a reduced sense of touch, in numbness and in paraesthesia, characterized by a “pins-and-needles” sensation. Nevertheless, such effects have only been seen in arsenic-exposed workers and may not occur in populations exposed only to lower arsenic concentrations in drinking water. Not surprisingly, such neuropathy has not been widely reported from Bangladesh. Similarly, mucous membrane lesions and liver damage have only been described in arsenic-exposed workers.

Disturbances in the hematopoietic system have been noted in subjects with arsenic-induced skin lesions, and in these subjects the skin lesions may be accompanied by anemia and leukopenia. Arsenic-induced anaemia is not associated with iron deficiency and is aplastic. In addition to blood damage, arsenic exposure also damages other components of the circulatory system. Toxic myocardial effects and peripheral blood vessel damage leading to atrophic acrodermatitis and gangrene of the extremities have been described. Gangrene of the extremities, known as black-foot disease, has been seen, but only in Taiwanese populations that have ingested arsenic. Therefore, the linkage between blackfoot disease and arsenic, as a sole causal agent, is uncertain.

IV  Bioavailability of Ingested Inorganic Arsenic

The bioavailability of arsenic is a key determinant in health outcomes of exposure, because it is related to the ability of arsenic to be liberated from ingested matrices (e.g., soil, water, and food) and thus enter into the bloodstream where it exerts its toxic effects (Caussy et al. 2003a). Most information on the bioavailability of arsenic is derived from human and animal observations. Studies from human volunteers and animal experiments show about 90% of ingested, dissolved, inorganic arsenic salts are absorbed from the gut and enter the bloodstream. This percentage is much higher than for most other nonessential elements and, in part, is a feature of the similar chemical properties of the arsenate (\(\text{AsO}_4^{2-}\)) and phosphate (\(\text{PO}_4^{2-}\)) ions. Phosphate is an essential body component and is avidly absorbed from the gut. Arsenate appears to follow many metabolic pathways that exist for phosphate, including deposition in the body at sites of phosphate incorporation. Arsenic also avidly binds to sulphydryl groups on proteins and other biomolecules. Nevertheless, the exact
fraction of arsenic uptake tends to depend upon gut contents and the presence, or otherwise, of phosphate absorption inhibitors such as aluminum salts, calcium carbonate, and lanthanoid salts, all of which inhibit arsenic uptake. In contrast, the fractional uptake of arsenic incorporated within organic compounds (organic arsenic) is reported to be lower than for dissolved inorganic species.

Arsenic, absorbed within the gastrointestinal tract, first passes through the liver and then enters the bloodstream and is distributed to the body (Caussy 2003a). Some arsenic accumulates in tissues and the remainder is excreted, mostly in urine. Although the affinity of arsenic for tissues, its kinetics of deposition, redistribution, and excretion, are arsenic species dependent, all species are rapidly cleared from blood and become evenly distributed among body tissues. Only a few tissues selectively concentrate or retain arsenic: liver, lungs, skin, nails, hair, and the skeleton (Table 2). Because skeletal muscle mass comprises a large proportion of body weight, a considerable amount of body arsenic is present in this tissue, despite its rather low concentrations.

The data presented in Table 2 reflect exposure of the general Japanese population to organic arsenic intake from seafood, rather than tissue distributions acquired from ingestion of inorganic arsenite (As (III)) and/or arsenate (As (V)) in contaminated drinking water. Following the intake of inorganic arsenic, more is likely to be deposited in liver and the skeleton, but less in kidneys. However, the deposition and retention of arsenic species is complicated by metabolic and redox processes within the body. These processes result in the oxidation of arsenite to arsenate, the metabolic reduction of arsenate to arsenite, and the methylation of arsenite in the

Table 2 Measured tissue concentrations of arsenic and implied organ content for human organs collected in Japan

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Arsenic concentration (µg/kg)</th>
<th>Tissue content (µg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone</td>
<td>96</td>
<td>450</td>
</tr>
<tr>
<td>Brain</td>
<td>34</td>
<td>41</td>
</tr>
<tr>
<td>Hair</td>
<td>174</td>
<td>3</td>
</tr>
<tr>
<td>Heart</td>
<td>41</td>
<td>11</td>
</tr>
<tr>
<td>Large intestine</td>
<td>25</td>
<td>8</td>
</tr>
<tr>
<td>Small intestine</td>
<td>22</td>
<td>12</td>
</tr>
<tr>
<td>Kidney</td>
<td>41</td>
<td>11</td>
</tr>
<tr>
<td>Liver</td>
<td>42</td>
<td>65</td>
</tr>
<tr>
<td>Lung</td>
<td>47</td>
<td>40</td>
</tr>
<tr>
<td>Muscle</td>
<td>29</td>
<td>700</td>
</tr>
<tr>
<td>Nail</td>
<td>892</td>
<td>3</td>
</tr>
<tr>
<td>Pancreas</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td>Skin</td>
<td>64</td>
<td>180</td>
</tr>
<tr>
<td>Spleen</td>
<td>21</td>
<td>3</td>
</tr>
<tr>
<td>Stomach</td>
<td>22</td>
<td>3</td>
</tr>
<tr>
<td>Teeth</td>
<td>78</td>
<td>3</td>
</tr>
<tr>
<td>Uterus</td>
<td>36</td>
<td>2</td>
</tr>
</tbody>
</table>

Source: Ishinishi et al. (1986).
liver to form monomethyl arsenic acid (MMA) and dimethyl arsenic acid (DMA). These transformations affect both tissue deposition and retention.

Results of human volunteer studies conducted with oxidized inorganic arsenic salts, such as those present in most contaminated drinking water, suggest that arsenic administered as As(V) is excreted according to the following pattern: 0.66 with a biological retention half-time ($T_{b/2}$) of 2.1 d; 0.3 with $T_{b/2}$ of 9.5 d; and 0.037 with $T_{b/2}$ of 38 d. In addition, arsenate substitutes for phosphate and is deposited in the skeleton, where it may be retained for a much longer period, perhaps as long as ~5,000 d. Of the excreted fraction, about half comprises inorganic ions and about half as DMA/MMA, with a preponderance of DMA; however, these proportions vary with age, gender, and intake composition. Unchanged arsenite is usually excreted faster than arsenate; the $T_{b/2}$ of organic arsenic in the body is about 20 hr. However, this apparent difference between arsenate and arsenite may be an artefact of the acidic conditions in the stomach, which adjusts the oxidation state of ingested ions. More exhaustive studies will probably reveal little or no difference between the bioavailability and biokinetic behavior of ingested soluble arsenate and arsenite ions.

V Application of the Health Risk Paradigm for Arsenic Contamination in the Bengal River Basin

The health risk paradigm can be viewed as comprising two components (Fig. 3): health risk assessment and risk management. The risk assessment paradigm classically involves a four-step process consisting of hazard identification, dose–response assessment, exposure assessment, and risk characterization (NRC 1983). The risk assessment process uses data from many sources, including animal experiments, in vitro studies and human epidemiological observations. The epidemiological gaps for applying the health risk assessment paradigm to arsenic contamination has been reviewed elsewhere (Caussy 2003b, c; Caussy and Than Sein 2006). Two uncertainties, linked with exposure and health effects, temper the risk assessment process, and these are discussed next.

A Hazard Identification

The purpose of hazard identification is to qualitatively characterize the relationship between arsenic exposure and probable adverse health outcomes. This analysis primarily relies on data derived from epidemiological and toxicological databases. Global epidemiological studies (of cross-sectional, case-control, and cohort study designs) have shown that exposure to unsafe levels of arsenic represents a definite health hazard, including arsenicosis and cancer (ATSDR 2000). In the absence of animal data, epidemiological data have been used to calculate the lowest observable adverse effect level (LOAEL) for arsenicosis. This LOAEL value is defined as the lowest dose needed to induce melanosis or keratosis, or both, and various sources estimate the value as
10–20 μg/kg/d (ATSDR 2000). Regional variations in the derived LOAEL value exist. The lowest LOAEL value (0.04 μg/kg/d) was observed in Mexico (Cebrian et al. 1983), whereas the highest value (18 μg/kg/d) was observed in exposed West Bengal populations (Chakraborty and Saha 1987). For an Asiatic 60-kg adult, this dose corresponds to a daily intake of \(0.900\) μg, an amount equivalent to a water concentration of about 600 μg/L, assuming a daily consumption of 1.5 L. Existing LOAEL data suggest that arsenic intakes are much higher than those derived using the WHO water quality standard; this may reflect continuing uncertainty in the LOAEL and a protective conservatism built into the WHO standard.

**B Dose–Response Assessment**

In dose–response assessment, the dosage of arsenic needed to induce predefined adverse health effects is quantified. Both experimental animal data and epidemiological data are routinely used to build the dose–response curve.

Statistical modelling has been used with cancer endpoints to assess dose response following arsenic exposure and concomitant intake. For example, Chen et al. (1985) and Chen and Wang (1990) used an ecological study to investigate the association between arsenic and cancer mortality. The relationship they studied was between arsenic concentration in water from more than 83,000 wells and reported cancer incidence from a cancer registry. Using regression analysis, the investigators
demonstrated a dose-dependent association between cancer mortality and arsenic exposure. These data, collected in Bangladesh and West Bengal, were inadequate to estimate dose–response functions for skin or internal cancers, largely because the duration of exposure to tube well water is less than the 20-yr induction period typically needed. However, this limitation has been addressed by using surrogate data on cancer incidence from Taiwan to exposure dose from Bangladesh (Yu and Ahsan 2004). Results show that the risk of skin and non-skin cancer steadily increased with increasing dose of arsenic exposure.

C Exposure Assessment

Because most arsenic is eventually excreted in urine, urinary excretion levels may, with reservation, be used to estimate total arsenic intake from inhaled, ingested, and skin absorption sources. Such intake measurements provide a meaningful estimate for toxicity assessment, unless the diet contains significant levels of much less toxic organic arsenic. Under conditions of normal arsenic exposure, levels of arsenic in urine range from about 5 to 50 \( \mu \text{g}/\text{L} \), implying total intakes of about 10–100 \( \mu \text{g}/\text{d} \), primarily from the consumption of either drinking water or seafood.

The speciation of arsenic, in natural sources of drinking water, is variable and depends upon dissolved oxygen levels, pH, and other factors. In general, arsenic is extracted from its aquifer under reducing conditions and reaches the surface as As (III), but on exposure to air is oxidized to As(V). Organic arsenic concentrations are low in most groundwaters but may be higher in biota-containing surface waters.

Data for arsenic exposure assessment in Bangladesh have been largely derived from environmental monitoring of water in tube wells. Monitoring by various agencies in Bangladesh indicates that 59 of the 64 districts have arsenic concentrations in groundwater in excess of the prevailing national standard of 50 \( \mu \text{g}/\text{L} \) (BGS 2001). The predicted intake is high, and in some places, water levels exceed the WHO guideline values by a factor of 30. Such high levels pose serious implications for remediation technologies.

Although tube wells have been in use for many years, neither the concentration nor duration of exposure to individuals can accurately be determined, partly because arsenic concentration data are often unavailable for the entire exposure period, as well as because the exposed population obtained drinking water from multiple sources. Furthermore, the concentration of arsenic fluctuates in the same tube well with time (NRC 1999). Moreover, when arsenic concentration was measured, testing methods used were either qualitative, semiquantitative, or constituted methods that did not conform to approved procedures. Because many of the methods utilized were not validated, most exposure data must be regarded as anecdotal. Biological monitoring of human beings, through measurement of arsenic levels in hair and nails, is limited, partly as a result of the cumbersome nature of the test and partly because of the inability of the markers to detect arsenic from exposure events more than 9 mon before analysis (NRC 1999; IPCS 2001).
Such samples are also subject to environmental contamination that conflates the assessment of body burden.

The existing exposure data are also deficient in being limited to a single matrix: water. A proper exposure assessment requires food chain monitoring capable of delivering insights on dose, species, and bioavailability of arsenic in commonly consumed food, particularly rice, which may contain significant levels of inorganic arsenic.

### D  Risk Characterization

The main risk of arsenic to human health is noncancerous and cancerous skin lesions (NRC 1999; IPCS 2001). The first and most common manifestation of arsenic poisoning is dermal lesions in the form of keratosis and melanosis. However, correctly diagnosing arsenic-induced dermal lesions from similar lesions mimicking arsenicosis requires special skills. Until recently, no universal method existed for diagnosing and classifying arsenicosis. Misdagnoses of arsenic-induced skin lesions should be minimized with the introduction of uniformity of case definition (Caussy 2005).

It has been concluded from epidemiological assessment of arsenic poisoning in the Bengal Basin that some 12 of 40 million exposed subjects are projected to develop some form of skin lesion within 10–20 yr of exposure (Caussy and Than Sein 2006). An alternative method, based on dose–response analysis, projects the count of arsenicosis cases in Bangladesh to amount to 1,864,000 (Yu et al. 2003). To date, the reported clinical cases fall short of these projected numbers, either because cases are undiagnosed, are unreported, or exposed persons have not yet manifested the disease. In part, the estimates vary because standard diagnostic criteria are lacking. The picture is further confused by data that show poor correlation between arsenic exposure levels in drinking water and manifestation of arsenicosis, i.e., low incidence of disease in areas with apparently high water levels of arsenic and vice versa. If such variability is confirmed, then additional factors are involved, either in the induction of disease or in the assessment of arsenic intake (e.g., inadequate consideration of arsenic levels in food). A strategy for effective remediation requires sufficient future work to establish reliable causation and dose response for arsenicosis in Bangladesh.

Arsenic-induced skin cancers require a latency of about 20 yr before manifestation. Hence, skin cancers have not yet been widely reported in Bangladesh populations exposed to arsenic. To reliably link arsenic as the causal agent for skin cancer, a cancer incidence rate statistically greater than the prevailing background level is required. It is commonly assumed (by regulators) that carcinogens induce their effects in a linear function with dose. Accordingly, exposure to arsenic may be assumed to present a finite risk of skin cancer irrespective of exposure. Experience with patients treated with Fowler’s solution (which contains arsenic) shows the absolute risk of skin cancer to be about 4%/g of arsenic intake. Although fraught
with uncertainty, one can extrapolate this risk to low-exposure environmental scenarios. The absolute risk of developing skin cancer after 30 yr of arsenic ingestion at the 10 μg/L WHO drinking water limit is $\sim 6 \times 10^{-4}$, which is a low but significant level of risk.

Epidemiologically based estimates of cancer incidence from arsenic exposure have been completed for Taiwan (Yu et al. 2003). Results produced by these authors indicate that the principal chronic debilitating conditions resulting from arsenic poisoning are cancers of the skin, lungs, and bladder (Chen et al. 1992). Estimates of the projected number of cancers from Bangladesh have been reported (Yu et al. 2003). To obtain these projections, the authors used the exposure rate from Bangladesh, the cancer incidence rate from Taiwan, and the dose–response data from West Bengal. It was estimated that 125,590 skin cancers and 3,250 internal organ cancers may occur in Bangladesh (Molla et al. 2004). Life-table analysis by this author, also using the exposure data from Bangladesh and the cancer incidence rate from Taiwan, has shown that there will be at least a doubling of lifetime mortality risk of internal cancer as a result of drinking arsenic-contaminated water.

By virtue of its chronic nature, the morbidity and mortality associated with arsenic disease have serious debilitating consequences on those affected. The consequences of morbidity and mortality to arsenicosis can be captured in a single measurement called the disability-adjusted life years lost (DALY). One estimate found a total of 1908 DALY to be associated with arsenicosis, thus highlighting the importance of the problem (Molla et al. 2004).

**VI  Risk Mitigation for Arsenic Contamination**

Results of the risk assessment process form the basis for risk management. With the exception of cancer, all arsenic-induced toxic effects are likely to have exposure thresholds that can be managed by the imposition of appropriate exposure limits (e.g., WHO guideline limit for drinking water). Risk management relies on legislative mandates, cost-benefit considerations, sociocultural acceptance of control measures, and availability of suitable control options (NRC 1983).

**A  Control Options**

Water, suitable for drinking and cooking, is a basic necessity in Bangladesh where contamination is widespread in rural areas and remote villages. Although control options are available and control technology is improving, each option has advantages and limitations, and no single control option is practical under all circumstances.

Among the most widely used options for securing safe water is exploitation of deep aquifers. Aquifers generally yield microbiologically safe water, but they are prone to contamination by arsenic and other heavy metals. Therefore, in addition to
the cost of sinking a tube well in an aquifer, one must consider the risk of finding impure water in the deep aquifer and the continuing cost to regularly monitor the aquifer for water purity during its natural life. The latter measure is important because arsenic-free aquifers can become contaminated at any time.

Although supplies of piped arsenic-free water are rapidly becoming available to all parts of West Bengal, the high cost limits its use in many areas. Rainwater harvesting is also viable in areas with abundant rainfall but is of limited use in arid areas.

Classical methods for purifying water of arsenical contamination rely on filtration, adsorption, and precipitation. Simple domestic filters, such as the “three-jug filter,” are commonly used in the region and are reasonably effective provided that the initial concentrations of arsenic in the water are not too high. Similarly, “tea-bag” adsorption and medium-level purification plants have been effectively employed. Although these methods can be applied widely, certain epidemiological and risk assessment issues must be resolved before their use.

First, the methods must undergo environmental technology verification (ETV) by competent internationally recognized bodies and national control authorities. These entities validate that the particular method or technology employed accomplishes its aim. Furthermore, performance of each technology or method must be validated with cogent coprecipitant molecules such as iron, phosphates, or nitrates that may compete with arsenic during water purification (Chen et al. 1999).

Second, the lower and upper concentration limits of arsenic that can be removed by each remediation method must be realistically defined. The current WHO drinking water guideline is 10 µg/L, but the prevalent national standard in Bangladesh is set at the higher and more achievable level of 50 µg/L. The success of risk mitigation depends on both technological feasibility and cost. Although, in countries of the Bengal River Basin, technologies may reach the standard of 50 µg/L for drinking water, it is important that, wherever possible, such technologies should be capable of yielding arsenic-safe water to a lower limit of at least 10 µg/L, in anticipation of future requirements. Furthermore, the cost of newer remediation technologies must be affordable to consumers. Nevertheless, a technology that removes 50% of arsenic from water should not be rejected only because it is incapable of reducing arsenic levels to the drinking water guideline value, because a reduction of 50% comprises a significantly lower risk of adverse effects including cancer.

B Socioeconomic Factors

Even if mitigation technology is feasible and affordable, ultimate success is dependent on acceptance by the target population. Epidemiological observations from Bangladesh demonstrate that consumption of arsenic-free water could be managed by simply directing villagers to switch from an arsenic-positive (red-painted) to an arsenic-negative (green-painted) tube well in the same village. However, cultural
beliefs prevent widespread acceptance of this approach (Hanchett et al. 2002; van Geen et al. 2002). A study of water supplies in Bangladesh shows that tube wells are regarded by local populations to be inherently safe because their historic advent coincided with the disappearance of waterborne bacterial pathogens (Caldwell et al. 2003). Studies sponsored by the World Bank have shown that arsenic-affected communities are willing to pay for arsenic-safe water. The challenge, then, is to establish the acceptability to communities of remediation technologies, taking into account their beliefs, attitudes, and practices.

C Legal and Ethical Considerations

Risk management aimed at reducing arsenic concentration to safe levels carries both legal and ethical implications. If risk mitigation measures are predicated on national governmental policy, then national authorities must have the infrastructure and manpower to monitor and enforce the imposed standards. Similarly, the affected community has a right to demand uniform application of such standards. In the absence of national policies or guidelines for managing arsenic risk, the international vendors that market arsenic removal technologies must be ethical to ensure that their products conform to international standards.

Summary

The problem of arsenic contamination in the Bengal River Basin illustrates a classic conundrum in environmental health, namely, that development projects can have double effects: on one hand development of tube wells eliminated bacterial pathogens and on the other it exposed the population to poisoning from arsenic. Thus, in future development projects the full health risk of a project must be considered during the planning, implementation, and decommissioning phases (Caussy 2003b; Caussy et al. 2003b). If such a holistic approach would have been followed, the mass contamination in the Bengal River Basin, in which millions of people were and are exposed to unsafe levels of arsenic, could have been averted. Although definite knowledge gaps in applying risk assessment steps for arsenic contamination exist, arsenic clearly poses a serious health problem and economic consequences to the affected population of the Bengal River Basin. It is binding on the international community to alleviate the problem through remediation measures to reduce arsenic exposure.

One Environmental Sustainability Millennium development goal is to increase the proportion of population with sustainable access to an improved water source (Bartram et al. 2005). Providing water with safe levels of arsenic to affected communities of the Bengal River Basin will directly contribute to improved community health.