

Arseniasis in the World: From Endemic to Pandemic

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Arsenic is a ubiquitous element in the crust of the earth. It is transported in the environment mainly by water. Humans are exposed to ingested and inhaled arsenic through environmental, occupational, dietary and medicinal exposures. Although arsenic is one of the oldest poisons which have been identified and used since ancient time, chronic arsenic poisoning was documented as an endemic environmental disease in Taiwan, Chile, and Japan in the early 1950s. Due to the rapid growth of the human population, severe surface water pollution, global warming and climate change, more and more world populations are using groundwater as the main drinking water source. The arsenic level in underground water varies significantly in different geographical areas. The global disease burden of chronic arsenic poisoning from drinking groundwater has significantly expanded from several confined endemic areas to a pandemic environmental calamity all over the world in the late 20th century. The number of victims exposed to arsenic from drinking water is rapidly increasing. Hundreds of millions of people are living in areas where the arsenic content in drinking water exceeds the maximal contamination level

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recommended by the World Health Organization. This chapter describes the endemic health hazards related to long-term exposure to arsenic in Taiwan and the current status of pandemic environmental disaster of arsenic in drinking water in the world.

PROLOGUE: A MYSTERIOUS DISEASE

Blackfoot disease as shown in Fig. 1 is an end-stage peripheral vascular disorder, which was endemic in a limited area along the southwest coast in



(1)



(2) World Health Organization Regional Office for South-East Asia 2005.



(3) (Ahamed *et al.*, 2006).⁵⁸

Fig. 1. Blackfoot disease/gangrene in southwestern Taiwan (1), West Bengal, India (2) and Bangladesh (3).

Taiwan. The disease was once called “black dry snake” by the local inhabitants to denote the mysterious discoloration of the affected extremities which then become black and well demarcated from the adjacent unaffected tissues, with the lesions gradually from the toes through the foot to the leg like the crawling of a snake. Clinically, the disease starts with numbness, coldness or both of one or more extremities and usually progresses to ulcer, black discoloration, and gangrene of the affected parts.¹ The disease was first reported as progressing “spontaneous gangrene” by surgeons in Kaohsiung Medical College in Taiwan.² At the end stage of the disease, spontaneous amputation of the distal parts of the affected extremities is common. The disease affects mostly the foot, occasionally the leg, and rarely the hand. Blackfoot disease is accompanied by lancinating, gnawing or burning pain, which made the patients unable to sleep or rest at ease. Another dreadful characteristic of the blackfoot disease is that it continues to worsen even after the affected limb has been surgically removed. It may spread up to the lower and even upper leg. Repeated removal of affected parts caused severe physical and psychological damages to the patients.

The pathological features of blackfoot disease have been extensively examined.³ The disease was compatible with two distinct histological types including thromboangiitis obliterans (30%) and arteriosclerosis obliterans (70%), although in terms of gross anatomy they all showed the same appearance of dry gangrene of the affected extremities with or without sharp demarcation, depending on the extent of secondary infection. The fundamental vascular changes that are common in both groups of blackfoot disease is an unduly developed severe arteriosclerosis, (leading to pure arteriosclerotic gangrene of the extremities) and thromboangiitis obliterans, another disease superimposed on the pre-existing or co-existent arteriosclerosis.

Sporadic cases of blackfoot disease were found in southwestern Taiwan in the early 20th century. However, no attention was paid to the disease until more and more people fell victim to it in the early 1950s. Most blackfoot disease patients reported before 1961 were living in a confined endemic area, including four townships — Pei-Men, Hsueh-Chia, Pu-Tai and I-Chu — in southwestern Taiwan as shown in Fig. 2. After an intensive survey, there were more blackfoot disease patients identified after 1961. A total of 2252 blackfoot disease patients were reported with the

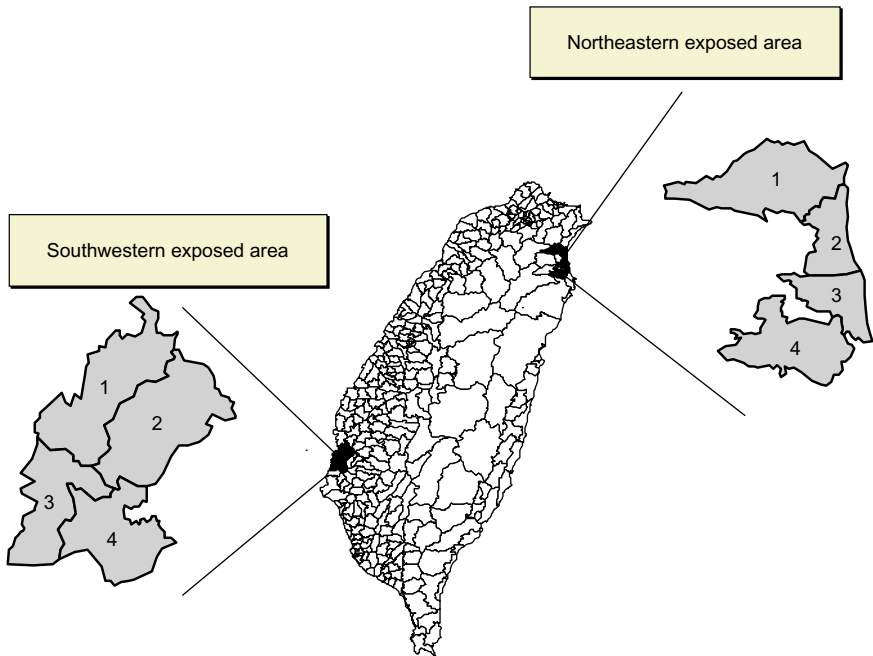


Fig. 2. Map of southwestern and northeastern arsenic-exposed areas in Taiwan. There are four townships in the southwestern exposed area: Pu-Tai (1), I-chu (2), Pei-Men (3) and Hsueh-Chia (4), and four townships in the northeastern exposed area: Chiao-Hsi (1), Chuang-Wei (2), Wu-Chieh (3) and Tung-Shan (4).

disease onset from 1912 to 1990.⁴ The prevalence of blackfoot disease was 5.57, 3.87, 2.02, and 0.64 per 1000 population in Pei-Men, Hsueh-Chia, Pu-Tai and I-Chu Township, respectively.

The etiology of blackfoot disease has been intensively investigated by two major investigation teams from the National Taiwan University and the US Naval Medical Research Unit 2, separately. The lifestyle and living condition of the residents in the endemic area were very similar to those who lived in southern Taiwan. However, the soil of the endemic area had a high salt content, which was not suitable for crops, especially rice. The majority of the residents took a mixture of rice and dried sweet potato chips as the staple food. The residents in most villages of the endemic area used well water as the main drinking water resources. Both shallow wells

and artesian wells were used in this area. In a study on the physicochemical characteristics of the drinking water in the endemic area,⁵ high arsenic content in the artesian well water was considered most likely to be the main causal factor of blackfoot disease. The arsenic concentrations of water samples from the artesian wells in the endemic area were all equal to or greater than 350 $\mu\text{g/L}$, and the arsenic concentrations of the water samples from the shallow wells in the endemic and non-endemic areas were all less than 300 $\mu\text{g/L}$.

In an epidemiological study carried out in five townships of the endemic area,^{6,7} 109 villages may be classified into four groups according to their drinking water sources: 39 used artesian wells only (Group I); 30 used both shallow and artesian wells (Group II); 38 used shallow wells only (Group III); and two used surface water (Group IV). Among them, 39 (89.5%) Group I villages and 19 (63.3%) Group II villages had blackfoot disease patients; but no blackfoot disease patient was found in Groups III and IV villages. The prevalence of blackfoot disease was 4.88 and 2.03 per 1000 population in Groups I and II villages. Blackfoot disease patients were found among users of 89 (45.6%) artesian wells in Group I villages and 29 (21.3%) artesian wells in Group II; however, no blackfoot disease patient was found among users of shallow wells in Groups II and III. The consumption of artesian well water was concluded as the main cause of blackfoot disease.

In a community-based case-control study of 353 pairs of blackfoot disease patients and matched unaffected controls,⁸ the length of residence in the endemic villages was significantly associated with the risk of developing the blackfoot disease in a dose-response relationship. While all (100%) blackfoot disease patients had used artesian well water as the principal source of drinking water during the 15 years before onset, only 233 (66%) matched controls had consumed artesian well water. In another community-based case-control study of 241 blackfoot disease patients and 759 matched healthy controls,⁹ there was a significant dose-response relationship between the risk of blackfoot disease and the duration of consumption of artesian well water. Based on the abovementioned studies, the consumption of high arsenic artesian well water was considered the most important risk predictor of blackfoot disease. As artesian wells were widely used in early 20th century in the endemic area, the peak occurrence

of blackfoot disease was observed in late 1950s. The induction period of developing the blackfoot disease after the consumption of artesian well water was estimated to be as long as 30 or more years with individual variations.

In a community-based cross-sectional survey of 40,421 residents in 37 villages in the blackfoot disease endemic area of southwestern Taiwan,¹⁰ the prevalence of blackfoot disease was significantly associated with the arsenic concentration in well water, showing a dose-response relationship in both males and females. The higher the level of arsenic in the drinking water, the higher the prevalence was of the blackfoot disease. Also, the older the age group, the higher, the prevalence of the disease. The age effect may reflect the increased cumulative exposure to arsenic in drinking water as well as the aging process.

In an analysis of cardiovascular mortality in 1973–1986 among the residents in 42 villages of the blackfoot disease-endemic area,¹¹ a significant dose-response relationship between arsenic in drinking water and age-adjusted mortality from peripheral vascular disease, mainly blackfoot disease, was observed. The higher the arsenic level in drinking water was, the higher the age-adjusted mortality from severe peripheral vascular disease. In another comprehensive health survey in three hyperendemic villages of blackfoot disease, the subclinical peripheral vascular disease of 263 male and 319 female adult residents was examined using Doppler ultrasonography.¹² The prevalence of the disease was found to be associated with cumulative arsenic exposure from drinking water in a dose-response relationship after adjustment for age, gender, body mass index, cigarette smoking, and serum levels of total cholesterol and triglycerides. This study used a more objective and sensitive tool to define peripheral vascular disease rather than clinical diagnosis based on severe manifestation of blackfoot disease.

These studies have well documented that the main cause of mild, severe and lethal peripheral vascular disease in the endemic area is the arsenic in drinking water. The increased risk of peripheral vascular disease has been observed in other countries where high-arsenic drinking water was consumed.^{13,14} The overt blackfoot disease, a severe type of peripheral vascular disease, has also been observed, albeit rarely, in other studies.^{13,15} The long-term exposure to extremely high arsenic level in well water of the

blackfoot disease-endemic area in Taiwan, as compared with the short-term exposure to intermediately high arsenic levels in drinking water of other countries, might explain at least partly the different severity of peripheral vascular disease in different arsenic-exposed areas.

In a 15-year follow-up study on cause-specific mortality of 789 blackfoot disease patients, a significantly increased mortality from peripheral vascular disease, cardiovascular disease, and cancers of the lung, liver, bladder and skin was observed in the blackfoot disease patients as compared with the general population in the endemic area.⁹ The study suggests arsenic may also cause several lethal diseases including cancers.

HEALTH HAZARDS OF CHRONIC ARSENIC POISONING IN TAIWAN

There are two endemic areas of long-term exposure to arsenic from drinking water in Taiwan as shown in Fig. 2. Residents in the southwestern endemic area started using high-arsenic well water in the early 1910s. They shared few wells with their neighbors living in the same village. A public water supply system using surface water was implemented in this area since the early 1960s, but its coverage remained low until the early 1970s. Artesian well water was not used for drinking and cooking after the mid-1970s. The northeastern endemic area includes the townships of Chiao-Hsi, Chuang-Wei, Wu-Chieh and Tung-Shan.¹⁶ Residents in this area started using high-arsenic well water in the late 1940s. Each household had its own tube-well in the backyards. A public water supply system using surface water was implemented in this area in the early 1990s, and its coverage was as high as 95% in 2000.

The systemic health hazards of long-term exposure to arsenic in drinking water have been intensively investigated since the 1960s, especially after 1985 in Taiwan. During the last decades, several diseases have been well documented to be associated with chronic arsenic poisoning from drinking water showing a dose-response relationship as shown in Table 1. Early studies in Taiwan have reported clinical manifestations of chronic arsenic intoxication in humans, which are insidious in onset and dependent on the magnitude of the dose and duration of exposure. The most distinct characteristic of chronic arsenic toxicity is the classical skin lesions, including hyperpigmentation with depigmentation, hyperkeratosis

Table 1. Chronic Health Hazards Induced by Long-term Exposure to Arsenic in Drinking Water in Southwestern (SW) and Northeastern (NE) Endemic Area of Arseniasis in Taiwan

Arsenic-Induced Health Hazard	Endemic Area	Biological Gradient	Reference (Author, Year)
Blackfoot disease	SW	Yes	Tseng 1977, Chen <i>et al.</i> 1988a
Skin hyperpigmentation	SW	—	Tseng <i>et al.</i> 1968
Palmsolar hyperkeratosis	SW	—	Tseng <i>et al.</i> 1968
Skin cancer	SW	Yes	Tseng <i>et al.</i> 1968, Tseng 1977, Chen <i>et al.</i> 1988b, Wu <i>et al.</i> 1989
Lung cancer	SW/NE	Yes	Chen <i>et al.</i> 1988b, Wu <i>et al.</i> 1989, Chen <i>et al.</i> 2004, Chen <i>et al.</i> 2010a
Bladder cancer	SW/NE	Yes	Chen <i>et al.</i> 1988b, Wu <i>et al.</i> 1989, Chiou <i>et al.</i> 2001, Chen <i>et al.</i> 2010b
Kidney cancer	SW/NE	Yes	Chen <i>et al.</i> 1988b, Wu <i>et al.</i> 1989, Chen <i>et al.</i> 2010b
Liver cancer	SW	Yes	Chen <i>et al.</i> 1988b, Wu <i>et al.</i> 1989
Prostate cancer	SW	Yes	Chen <i>et al.</i> 1988b, Wu <i>et al.</i> 1989
Ischemic heart disease	SW	Yes	Chen <i>et al.</i> 1996, Hsueh <i>et al.</i> 1998, Tseng <i>et al.</i> 2003
Cerebral infarction	NE	Yes	Chiou <i>et al.</i> 1997
Peripheral vascular disease	SW	Yes	Tseng <i>et al.</i> 1996
Macrovascular diseases	SW	—	Wang <i>et al.</i> 2003
Microvascular diseases	SW	—	Wang <i>et al.</i> 2003
Abnormal peripheral microcirculation	SW	—	Tseng <i>et al.</i> 1995
Carotid atherosclerosis	SW/NE	Yes	Wang <i>et al.</i> 2002, Wang <i>et al.</i> 2007
QT prolongation (EKG)	SW	Yes	Wang <i>et al.</i> 2009
Hypertension	SW	Yes	Chen <i>et al.</i> 1995
Goiter	SW	—	Chang <i>et al.</i> 1991
Diabetes mellitus	SW	Yes	Lai <i>et al.</i> 1994, Tseng <i>et al.</i> 2000
Posterior subcapsular lens opacity	SW	Yes	See <i>et al.</i> 2007
Pterygium	SW	Yes	Lin <i>et al.</i> 2008
Slow neural conduction	NE	—	Tseng <i>et al.</i> 2006
Retarded neurobehavioral development	NE	Yes	Tsai <i>et al.</i> 2003
Erectile dysfunction	NE	—	Hsieh <i>et al.</i> 2008

in the palms and soles, as well as Bowen's disease and skin cancer.¹⁸ There was an increasing prevalence of skin cancer with the biological gradient of arsenic level in drinking water. The co-existence of blackfoot disease and characteristic arsenic-induced skin lesions has been observed in southwestern Taiwan.¹⁰

In addition to blackfoot disease, several chronic cardiovascular effects of long-term exposure to ingested arsenic have been reported in both the southwestern and northeastern endemic areas in Taiwan. They include ischemic heart disease,^{18–20} cerebral infarction,¹⁶ peripheral vascular disease,¹² macrovascular and microvascular diseases,²¹ abnormal peripheral microcirculation,²² carotid atherosclerosis,^{23,24} QT prolongation in electrocardiography,²⁵ and hypertension.²⁶ Dose-response relationship has been observed between arsenic level in drinking water and prevalence of cardiovascular diseases. Long-term exposure to ingested arsenic has also been found to induce goiter,²⁷ diabetes mellitus,^{28,29} posterior subcapsular lens opacity, a specific type of cataract,³⁰ and pterygium, a unique eye disease,³¹ showing a biological gradient in the southwestern endemic area in Taiwan. Long-term exposure to arsenic in well water has also been found to be associated with an increased risk of slow neural conduction,³² retarded neurobehavioral development,³³ and erectile dysfunction³⁴ in the northeastern endemic area.

An increased mortality from various cancers was observed in the endemic area of blackfoot disease as compared with the general population in Taiwan,³⁵ and the elevated cancer mortality was significantly associated with the use of artesian well water showing a dose-response relationship.^{16,35} The increasing arsenic level in well water has been well documented to be associated with a biological gradient of mortality from cancers of the skin, lung, kidney, bladder, liver and prostate in the southwestern endemic area.^{11,36} Further studies in the northeastern endemic areas also showed a dose-response relationship between arsenic in well water and incidence of cancers of the lung, bladder and kidney.^{37–40}

The risk of cancers of the liver, lung, and urinary bladder associated with arsenic in drinking water in southwestern Taiwan has been intensively analyzed.⁴¹ The data have been used to set up the maximal contamination level of arsenic in drinking water by the World Health

Organization (WHO) and Environment Protection Agency of the USA (USEPA). Norms for drinking-water quality were initially recommended by WHO in 1958. The *International Standards for Drinking-Water* established 0.20 mg/L as an allowable concentration for arsenic in that year. In 1963, the standard was re-evaluated and reduced to 0.05 mg/L. This was maintained as WHO's "Guideline Value" in 1984; and many countries have kept this as the national standard or as an interim target. According to the last edition of the *WHO Guidelines for Drinking-Water Quality* (1993), inorganic arsenic is a documented human carcinogen and 0.01 mg/L was established as a provisional guideline value for arsenic. Based on health criteria, the guideline value for arsenic in drinking-water would be less than 0.01 mg/L. Because the guideline value is restricted by measurement limitations (0.01 mg/L), this is termed a provisional guideline value. The guidelines are intended for use as a basis for the development of national standards in the context of local or national environmental, social, economic, and cultural conditions. The implementation of the standards for arsenic in drinking water in the USA had been debated for 20 years. A new maximal contamination level of 0.01 mg/L was effective from February 2002 and complied with in 2006.

The health hazards of chronic arsenic poisoning appear to differ between individuals, populations and geographic areas. There is no universal definition of the disease caused by arsenic. This complicates the assessment of the burden on health of arsenic. Similarly, there is no method to identify those cases of internal cancer that are caused by arsenic as compared with cancers induced by other factors. The multiple health hazards in humans associated with the ingestion of arsenic in drinking water have been reported from many countries and comprehensively reviewed by the World Health Organization,^{42,43} Chen *et al.*,^{14,44,45} National Research Council,⁴⁶ International Agency for Research on Cancer,⁴⁷ Agency for Toxic Substances and Disease Registry,⁴⁸ and Wang *et al.*⁴⁹ The explanations for the geographical variation in health hazards induced may include: (1) difference in arsenic concentration in drinking water; (2) difference in the starting age and duration of arsenic exposure; (3) difference in the quantity of water consumption; (4) difference in anthropometric characteristics including height and weight; (5) difference in lifestyle such as

alcohol consumption and cigarette smoking, nutritional status, and disease risk factors other than ingested arsenic; (6) difference in genetic composition of exposed populations; and (7) completeness and accuracy of health hazard identification, arsenic exposure assessment, competing diseases and causes of death, and study designs.

Molecular and genomic epidemiological studies have identified several biomarkers of susceptibility to arsenic-induced health effects. Arsenic methylation capability, folate and carotenoids intake, and genetic polymorphisms of enzymes involved in xenobiotic metabolism, DNA repair, and oxidative stress are associated with the development of arsenic-induced health hazards in Taiwan.⁵⁰ Further exploration of gene-environment and gene-gene interactions on the development of arsenic-induced health hazards is in urgent need.

ARSENIC IN GLOBAL ENVIRONMENTS

Arsenic and its compounds are ubiquitous in nature and have both metallic and nonmetallic properties. Both trivalent and pentavalent forms are the most common oxidation states. Arsenic compounds are classified into three groups: inorganic arsenic compounds, organic arsenic compounds, and arsine gas. The most common trivalent inorganic arsenic compounds are arsenic trioxide, sodium arsenite and arsenic trichloride; and pentavalent inorganic compounds include arsenic pentoxide, arsenic acid and metal arsenates. The common organic arsenic compounds include arsanilic acid, methylarsonic acid (MMA), dimethylarsinic acid (cacodylic acid, DMA), arsenobetaine, arsenocholine and arsenosugars. Arsenic appears in nature primarily in the form of sulfides in association with the sulfides of ores of silver, lead, copper, nickel, antimony, cobalt and iron. Trace amounts of arsenic are found in soils and other environmental media.^{43,46,47}

Arsenic is transported in the environment mainly by water. In oxygenated surface water, arsenic usually occurs as arsenate; but under reducing conditions such as in deep well waters, the predominant forms are arsenites. Arsenic is introduced into water through the dissolution of minerals and ores, and the concentrations of arsenic in groundwater in some areas are elevated as a result of erosion from local rocks. Industrial

effluents also contribute arsenic to water in some areas. The level of arsenic in uncontaminated stream water ranges from 0.1 to 1.7 $\mu\text{g/L}$, and in groundwater it ranges from 0.1 to 7500 $\mu\text{g/L}$ in the world.⁴⁷ The inorganic arsenic in water may be methylated to MMA and DMA through biological activity. Marine organisms may transform inorganic arsenic into organic arsenic, such as arsenobetaine, arsenocholine, arsonium phospholipids and arsenosugars. Similarly, inorganic arsenic is in the pentavalent form in oxygenated soil and in the trivalent form under reducing conditions. Leaching of arsenate is slow due to its binding to hydrous oxides of iron and aluminium. Inorganic arsenic in the soil may be bi-methylated as methylarsines and released into the air. Nevertheless, airborne arsenic is mainly inorganic. Arsenic concentrations in uncontaminated soil range generally 0.2–40 mg/kg. The levels may be as high as 100–2500 mg/kg in the vicinity of copper smelters. In the soil of orchards that used arsenical pesticides widely in the past, the arsenic levels may range 200–2500 mg/kg.^{43,47}

Arsenic released to the atmosphere comes from both natural and anthropogenic sources. Volcanic activity is the main natural source of airborne arsenic, and only a minor proportion is exudated from vegetation and windblown dusts. Anthropogenic emission of arsenic to that comes principally from metal smelting, fossil fuel combustion, and pesticide use. Global natural emissions were estimated as 7900 tons per year, while anthropogenic emissions amount to 23,600 tons per year.⁴⁷ Arsenic oxide (III) collected as a by-product in the metal smelting is used for the manufacture of virtually all arsenicals. Arsenic was mainly used for the manufacture of pesticides, specifically insecticides, before they were replaced by other preparations. Arsenic is used commercially as antifungal wood preservatives, and in the manufacture of pharmaceuticals, glass, sheep-dips, leather preservatives, poisonous baits, pigments and alloys. Gallium arsenide and indium arsenide are used in the production of certain semiconductor devices. Arsanilic acid and its derivatives are added to cattle and poultry feed as growth-stimulating agents. As a consequence of the many different uses of arsenic and arsenicals, humans are exposed to ingested and inhaled arsenic through environmental, occupational, dietary and medicinal exposures. However, drinking water is the greatest threat to public health from arsenic.⁴⁷

ENVIRONMENTAL CALAMITY OF ARSENIC IN DRINKING WATER: AN EMERGING PANDEMIC

High arsenic concentration in well water has been found in many countries all over the world. Chronic arsenic poisoning from drinking water is now considered the largest environmental catastrophe, much more prominent than the nuclear power plant event at Chernobyl, Russia or the chemical plant explosion at Bhopal, India. Arseniasis is becoming an emerging epidemic in the world.^{51,52} Taken together with the historical discovery of chronic arsenic poisoning from groundwater in Taiwan and other countries, groundwater sources throughout the world that are used for drinking-water should be tested for arsenic.

The delayed health effects of exposure to arsenic, the lack of common definitions and local awareness, as well as the poor reporting in affected areas are major problems in determining the extent of the problem of arsenic in drinking water. Reliable data on exposure and health effects are rarely available, but it is clear that there are many countries in the world where arsenic in drinking water has been detected at concentration greater than 0.01 mg/L. These include Argentina, Australia, Bangladesh, Burkina Faso, Cambodia, Chile, China, Hungary, India, Mexico, Peru, Romania, Thailand, the United States of America, and Vietnam. Countries where adverse health effects have been documented include Bangladesh, China, India (West Bengal), and the United States of America.

Some large populations exposed to high arsenic drinking water have been identified. Seven of 16 districts of West Bengal were reported to have groundwater arsenic concentrations above 0.05 mg/L; the total population in these seven districts is over 34 million.⁵³ It has been estimated that the population actually using arsenic-rich water of above 0.05 mg/L is more than one million and of above 0.01 mg/L is 1.3 million.⁵⁴ According to a British Geological Survey study in 1998 on shallow tube-wells in 61 of the 64 districts in Bangladesh, 46% of the samples had arsenic level above 0.010 mg/L and 27%, above 0.050 mg/L. When combined with the estimated 1999 population, it was estimated that the number of people exposed to arsenic concentrations above 0.05 mg/L is 28–35 million and the number of those exposed to more than 0.01 mg/L is 46–57 million.⁵⁵ USEPA has estimated that some 13 million of the population of USA,

mostly in the western states, are exposed to arsenic in drinking water at 0.01 mg/L, although the concentrations appear to be typically much lower than those encountered in areas such as Bangladesh and West Bengal.⁵⁶

Historically, surface water sources in Bangladesh have been contaminated with micro-organisms, causing a significant burden of disease and mortality. Consequently, during the 1970s the United Nations Children's Fund (UNICEF) worked with the Department of Public Health Engineering of Bangladesh to install tube-wells to provide what was presumably a safe source of drinking water for the population. By 1997, UNICEF indicated in its country report for Bangladesh that it had surpassed its goal of providing 80% of the population by 2000 with access to "safe" drinking water in the form of tube-wells, ring-wells and taps. In 1997, a project authorized by the government of Bangladesh found about 62% of the 32,651 tube-wells sampled had concentrations greater than 100 $\mu\text{g/L}$. It was estimated that of the 125 million inhabitants of Bangladesh, between 35 million and 77 million are at risk of drinking contaminated water. According to some estimates, arsenic in drinking water will cause 200,000–270,000 deaths from cancer in Bangladesh alone.⁵⁷ A United Nations Foundation grant for 2.5 million approved in July 2000, will enable UNICEF and WHO to support a project to provide clean drinking-water alternatives to 1.1 million people in three of the worst affected subdistricts in Bangladesh. The project utilizes an integrated approach involving communication, capacity building for arsenic mitigation of all stakeholders at subdistrict level and below, tube-well testing, patient management, and provision of alternative water supply options.

The most important remedial action is prevention of further exposure by providing safe drinking water. The cost and difficulty of reducing arsenic in drinking water increases as the targeted concentration lowers. It varies with the arsenic concentration in the source water, the chemical matrix of the water including interfering solutes, availability of alternative sources of low arsenic water, mitigation technologies, and the amount of water to be treated. Control of arsenic contamination is more complex where drinking water is obtained from many individual sources (such as hand-pumps and wells) and in rural areas. Low arsenic water is only needed for drinking and cooking. Arsenic-rich water can be used safely for laundry and bathing.

Discrimination between high-arsenic and low-arsenic sources by painting the hand-pumps (e.g. red and green) was found to be an effective and low cost means to rapidly reducing exposure to arsenic and this should be accompanied by effective health education.

Alternative low-arsenic sources such as rain water and treated surface water may be available and appropriate in some circumstances. Where low arsenic water is not available, it is necessary to remove arsenic from drinking water. The technology for arsenic removal for piped water supply is moderately costly and requires technical expertise. It is inapplicable in some urban areas of developing countries and in most rural areas world-wide. There are no proven technologies for the removal of arsenic at water collection points such as wells, hand-pumps and springs. Simple technologies for household removal of arsenic from water are few and have to be adapted to, and proven sustainable in each different setting.

CONCLUSION

The chronic arsenic poisoning from drinking water was well documented in southwestern Taiwan in the early 1960s. In addition to blackfoot disease and skin cancer, long-term exposure to arsenic in drinking water has been found to induce systemic health hazards including internal cancers, cardiovascular diseases, metabolic diseases, eye diseases, and retarded neurobehavioral development. As more and more populations change major water sources from surface water to groundwater, the environmental disaster of arsenic poisoning has expanded from endemic to pandemic public health issues. Intensive global collaboration in the comprehensive survey of arsenic-contaminated groundwater sources and mitigation of chronic arsenic poisoning is in urgent need.

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