Preface

The Society of Environmental Geochemistry and Health (SEGH) Fifth International Conference on Arsenic Exposure and Health Effects was held July 14 to 18, 2002 in San Diego, California. Both public and private groups sponsored the conference. In addition to SEGH and the University of Colorado at Denver, other sponsors included the US Environmental Protection Agency (US EPA), the Centers for Disease Control and Prevention (CDC), AWWA Research Foundation (AwwaRF), the Electric Power Research Institute (EPRI), and the National Institute of Environmental Health Sciences (NIEHS).

Over 220 people, including the speakers and poster presenters, attended the 5th conference. Of these, approximately one-third were non-US citizens; the largest groups were from Asia and Europe. The attendees included scientists from academia, industry, local, state, US federal and several foreign governments. The disciplines of geochemistry, chemistry, molecular biology, biochemistry, epidemiology and medicine were well-represented at the meeting. Several outstanding papers and posters presented at the conference generated lively discussion and debate, not only about scientific issues, but also social, public policy and regulatory issues. The interactions between the attendees both during and outside the sessions resulted in many new contacts between scientists.

There were 14 platform sessions and 4 poster sessions with 67 speakers and 130 posters. Panel discussions were held after each session to encourage discussion and debate. Two sessions were devoted to an overview of some US EPA and NIEHS arsenic programs. As in past conferences, the first report of elevated arsenic exposures in a new country was given. This time it was Nepal; Dr. Shrestha reported elevated arsenic concentrations in groundwaters in the Terai region.

The seeds of the conference(s) were sown at a meeting of the SEGH Executive Board in December 1991. They agreed to form an Arsenic Task Force similar to the SEGH Lead in Soil Task Force that had been formed in the 1980s. It was clear that there was a growing controversy regarding the proposed changes in the US EPA Maximum Contaminant Level (MCL) for arsenic in drinking water. This is the enforceable standard for drinking water. In addition to impacting on water utilities, the development of the standard would also have the possibility for significant economic impacts on the cleanup of superfund sites and on the electric power industry (because of arsenic in fly ash).

The Task Force was formed in 1992 and co-chaired by Willard Chappell and Charles Abernathy. An international conference seemed to be an excellent way to begin to compile the data and to determine what needed to be done in this area. The First SEGH International Conference on Arsenic Exposure and Health Effects was held in New Orleans in July, 1993. This conference was successful in attracting the top arsenic researchers in the world. It was followed by the Workshop on Epidemiology and Physiologically-Based Pharmacokinetics that was held in Annapolis, MD in June, 1994. Perhaps the most significant outcome of the 1994 Workshop was the realization that there are many arsenic hot spots in the world. Although not widely recognized by the scientific community at that time, significant public health problems existed in countries such as India, Thailand and China. The Second SEGH International Conference on Arsenic Exposure and Health Effects was held in June, 1995 in San Diego with a primary purpose being to highlight the global aspects of the problem

and most of the impacted countries were represented. At that time, the biggest recognized problem area was in West Bengal, India where an estimated 30 million people are at risk from arsenic exposure in the ground water.

The Third SEGH International Conference on Arsenic Exposure and Health Effects (July, 1998) was also held in San Diego and continued the theme of global impact of arsenic. In addition, two new countries with significant arsenic problems, Inner Mongolia and Bangladesh were represented. The attendees were to learn that the Bangladesh problem could be larger than the one in West Bengal with a possible 80 million people (two-thirds of the population) at risk. This situation caught the attention of the media later in 1998 with the publication of a front-page article in the Nov. 16, 1998 New York Times. The article was syndicated and published in newspapers around the world.

The SEGH Fourth International Conference on Arsenic Exposure and Health Effects (June, 2000) continued the focus on the global impact of arsenic, and also featured sessions on mechanisms of cancer carcinogenesis, metabolism, and water treatment technology. New impacted countries represented included Viet Nam.

The Fifth International Conference on Arsenic Exposure and Health Effects (July, 2002) introduced the participants to the problems in Nepal. Numerous speakers discussed advances in understanding the mechanisms of arsenic toxicity and carcinogenicity. The considerable advances in several areas were readily apparent and this monograph represents the state of the art. The organizers are deeply grateful to the authors for their fine work and to the sponsors for the support that made it possible.

We are also deeply appreciative of the fine efforts of Rosemary Wormington of the Environmental Sciences Program of the University of Colorado at Denver who put in long hours as conference coordinator. She kept the entire Conference going and, more than anyone else, is responsible for the success of this and the past conferences.

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Arsenic Exposure and Health Effects V
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Chapter 1

Groundwater arsenic exposure in India

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Abstract

The first report on arsenic in hand tubewells, dugwells and spring water was published in 1976 from India. It was reported that people were drinking arsenic-contaminated water in Chandigarh and different villages of Punjab, Haryana, Himachal Pradesh in northern India. High arsenic was found in the liver of those suffering from non-cirrhotic portal fibrosis (NCPF) and drinking arsenic-contaminated water. Arsenic groundwater contamination in the state of West Bengal first came to notice during July 1983. The problem first came to international attention after the international conference held in Calcutta during February 1995. Before Bangladesh's arsenic episode was discovered, West Bengal's arsenic problem was known as the world's biggest arsenic calamity. During July 1983, 16 patients with arsenical skin lesions were identified from one village in the district of 24-Parganas where people were drinking arsenic-contaminated water from their hand tubewells in West Bengal. The present arsenic situation from 38,865 km^2 of affected area with a population of 50 million in West Bengal up to August, 2002 is as follows: 3150 villages from 9 districts, 78 blocks/police stations have been identified where groundwater contains arsenic concentrations above 50 μg/l. On the basis of 125,000 water analyses by a laboratory method from the arsenic-affected areas it was estimated that more than 6 million people are drinking arsenic-contaminated water above 50 μg/l. So far, from our preliminary survey 8500 patients with arsenical skin lesions have been registered from 250 villages, and extrapolation of available data indicates that may be 300,000 people are suffering from arsenical skin lesions from 9 arsenic-affected districts of West Bengal. The source of arsenic is geologic. The mechanism of arsenic contamination from the source to the aquifer has not yet been established.

Groundwater arsenic contamination from industrial effluent discharge by a company producing paris-green (copper-aceto-arsenite) and the suffering of people in Behala – Calcutta came to notice during 1989. The highest arsenic concentration in soil near the effluent discharged point was found to be 10,000 μg/gm and the highest arsenic concentration in hand tubewell water was 38,000 μg/l. The total number of people using arsenic-contaminated water was 7000, and around 200 people were identified with arsenical skin lesions.

In the Rajnandgaon district of the state of Chattisgarh in India, a few villages were found where both dugwells and hand tubewells are arsenic contaminated. The source of arsenic is also geologic. The highest concentrations of arsenic found in the dugwells and hand tubewells were 520 and 880 μg/l, respectively. About 130 people were affected with arsenic poisoning. The number of people estimated to be at risk was 10,000.

About 1000 people are suspected to be suffering from arsenical skin lesions from the Semria Ojha Patty village of Sahapur police station in Bhojpur district of Bihar in the middle Ganga Plain. The magnitude of the problem in Bhojpur district hence in Bihar is unknown.

Keywords: arsenic in Ganga plain, industrial arsenic contamination, arsenic contamination in Bihar, source of arsenic

1. Introduction

Before the onset of the 21st century, groundwater arsenic contamination had already been reported in 20 countries, out of which four major incidents were from Asia. In order of severity of occurrence, these are Bangladesh, West Bengal (WB, India), Inner Mongolia (PR China) and Taiwan. Each year, new arsenic groundwater contamination incidents are being reported from Asian countries. For example, several places in China have been recently reported to be new problem areas (ESCAP–UNICEF–WHO Expert Group Meeting, 2001). Severe arsenic contamination has also been recently reported from Vietnam, where several million people are said to be at considerable risk of chronic arsenic poisoning (Berg et al., 2001). In a recent United Nations Economic and Social Commission for Asia and the Pacific–United Nations International Children's Emergency Fund–World Health Organization (UNESCAP–UNICEF–WHO) expert group meeting (ESCAP–UNICEF–WHO Expert Group Meeting, 2001), arsenic groundwater contamination was also reported from other countries including the Lao People's Democratic Republic, Cambodia, Myanmar and Pakistan. Groundwater arsenic contamination has also been reported from Nepal (Shrestha and Maskey, 2002).

In India, groundwater contamination incidents are increasing with time and the major incidents are in Gangetic Plain.

2. Groundwater arsenic contamination in northern India

A preliminary study on arsenic in dugwells, hand pumps and spring water (Datta and Kaul, 1976) was reported in 1976 from Chandigarh and different villages of Punjab, Haryana and Himachal Pradesh in northern India. Concentrations as high as 545 μg/l of arsenic were obtained in the water sample from a hand pump. Datta (1976) further reported high arsenic contents in the liver of five out of nine patients with non-cirrhotic portal hypertension (NCPH) who had been drinking arsenic-contaminated water. It was further stated (Datta, 1976) that "Cirrhosis (adult and childhood), non-cirrhotic portal fibrosis (NCPF) and extrahepatic portalvein obstruction in adults are very common in India and suggested that consumption of arsenic-contaminated water may have some role in the pathogenesis of these clinical states". To date, no further information on arsenic poisoning from northern India is available.

3. Groundwater arsenic contamination in West Bengal – India

After the incident of groundwater arsenic contamination in northern India in 1976, the next available information on arsenic contamination was from the state of West Bengal in 1983. An arsenic patient with skin lesions was identified by dermatologist Dr K.C. Saha (Docket No. 3/158/33/83) of the School of Tropical Medicine, Calcutta on 6th July 1983. The first arsenic contamination report on West Bengal (Garai et al., 1984) stated that 16 patients in 3 families were identified with arsenical skin lesions on 16th July 1983 and 28th July 1983 from one village in the district of 24-Parganas, who were drinking water from their hand tubewell. The highest arsenic concentration recorded in a tubewell was 1250 μg/l.

The same report stated that the condition of 2 patients was so severe that they needed hospitalisation. They had the symptoms of hyperpigmentation, hyperkeratosis, oedema, ascites, wasting, weakness, pain and a burning sensation in toes and fingers.

In 1984, the government of West Bengal constituted an investigation committee with some research workers of various organizations. In the same year after surveying the villagers, the government of West Bengal was informed by the committee about the extent, severity, possible causes of the disease and measures to prevent this disease. A copy of this report could not be traced; only a summary of the report was available from a published book (Saha, 2002).

During October 1984 Saha (Saha, 1984) reported 127 patients with arsenical skin lesions from 25 families (total members 139) from 5 villages in 3 districts (24 Parganas, Nadia, Bardhaman) of West Bengal. Out of the 127 affected members all had diffuse melanosis, 39% had spotted melanosis, and 37% and 12.6% had palmoplanter keratosis and dorsum keratosis respectively. This was the first report on skin lesions from arsenic patients of West Bengal. Next available report on magnitude of the arsenic problem from West Bengal was in the published paper of Saha & Poddar (Saha and Poddar, 1986) in 1986. The report stated 36 villages from 18 police stations/blocks of six districts are affected. These districts were 24 Parganas, Murshidabad, Nadia, Bardhaman, Malda & Midnapur. Although one patient from Ramnagar police station of Midnapur was reported in 1986 but later on it was found that Midnapur district is not affected and the patient was originally from affected district Nadia but settled in Midnapur. From 36 villages, water samples from 207 hand tubewell were analyzed and 105 (50.7%) showed arsenic concentration above 50 μg/l and highest concentration recorded was 586 μg/l. The report further stated that so far 1000 cases of chronic arsenical dermatosis were recorded from the affected villages and cutaneous malignancy was found in 3. Analysis of arsenic in hair, nail, skin-scale from the people in the affected villages confirmed arsenic toxicity and identified subclinical arsenic toxicity in some of the apparently unaffected members of the affected families.

During 1987 an epidemiological survey in 6 villages from 3 districts (24 Parganas, Bardhaman, and Nadia) revealed 197 patients with arsenical dermatosis in 48 families (Chakraborty and Saha, 1987). In some families all the members had symptom of dermatosis. Liver was enlarged in 34.5% and ascites present in 5.6% of the patients. The youngest patient was 14 months old. One patient developed skin cancer. Three deaths were reported due to chronic arsenic poisoning. Out of 71 water samples collected from tubewells of the affected villages, in 55 (77.5%) concentration of arsenic was higher than the Indian permissible limit (50 μg/l) of arsenic in drinking water. Mean arsenic concentration in 31 water samples collected from tubewells of the affected families were 640 μg/l and that of the 40 water samples collected from tubewells of the unaffected families were 210 μg/l. The study reported that lowest concentration of arsenic in water resulting dermatosis was found to be 200 μg/l.

During 1988 an epidemiological investigation (Guha Mazumder et al., 1988) from an arsenic affected of Ramnagar village, Baruipur Block, 24-Parganas showed evidence of chronic arsenical dermatosis and hepatomegaly in 62 (92.5%) out of 67 members of families who drank contaminated tubewell water (arsenic level 200–2000 μg/l). In contrast, only 6 (6.25%) out of 96 persons from the same area who drank safe water (arsenic level <50 μg/l) had non-specific hepatomegaly, while none had skin lesions.

Hepatomegaly occurred in all 13 patients who were studied in detail, although 5 had splenomegaly. Biopsies of liver tissue from these patients revealed various degrees of fibrosis and the expansion of the portal zone that resembled NCPF. Datta reported (Datta, 1976) similar incidents in 1976 from Chandigarh and its surroundings from northern India. In 1991, a report from the steering committee, government of West Bengal (Steering Committee Arsenic Investigation Project, 1991) described the regional geology, geomorphology, geohydrology, some borehole sample analyses and the clinical investigation of about 60 arsenicosis patients in arsenic-affected areas of West Bengal. It was also reported that water in the intermediate aquifer is polluted with arsenic. Neither the shallow (first) nor the deep (third) aquifers have been reported to have arsenic contamination. The sand grains in the arsenic-contaminated aquifer are generally coated with iron and arsenic-rich material.

During October 1994, a committee constituted by the government of West Bengal (Committee Constituted by Government of West Bengal, 1994) reported arsenic contamination in 41 blocks in 6 districts of West Bengal. The committee analysed about 1200 water samples for arsenic and other common water quality parameters from these 6 districts, and the highest arsenic concentration reported was 3200 μg/l. The committee recommended alternative water supplies, an arsenic removal plant, an epidemiological study and a survey to discover the magnitude of contamination.

A report by the School of Environmental Studies was published in December 1994 narrating the severity and magnitude of the problem (Das et al., 1994). In this document, it was reported that 312 villages from 37 blocks/police stations in 6 districts were affected by arsenic groundwater contamination, and from the extrapolation of the data it was predicted that more than 800,000 people are drinking arsenic-contaminated water from these affected districts and about 175,000 people may suffer from arsenical skin lesions. The average ratio of arsenite to arsenate in water samples was 1:1.

The groundwater arsenic contamination problem of West Bengal came to limelight after the international conference held in Calcutta during 1995 (International Conference on Arsenic in Groundwater, 1995) and the opinions of experts about the arsenic contamination of West Bengal (Post Conference Report, 1995). Most of the organizations working on arsenic problem in West Bengal reported their findings in this conference. Reported abstracts on West Bengal were on epidemiology (Das Gupta et al., 1995), pathology (Bhattacharyya et al., 1995), health hazard induced by chronic arsenicosis (Guha Mazumder et al., 1995; Saha, 1995a), geology (Saha and Chakrabarti, 1995), hydrogeology (Sinha Ray, 1995), geochemistry (Das et al., 1995a), removal of arsenic from water (Bagchi and Bagchi, 1995; Das et al., 1995b; Nath et al., 1995), analysis of arsenic in biological samples (Samanta et al., 1995) and watershed management (Das et al., 1995c).

The severity and magnitude of the arsenic groundwater contamination in West Bengal were reported by various arsenic experts who attended the conference, in the Post Conference Report published in May 1995. Chappell (1995) narrated the problem as "The chronic arsenic poisonings occurring in the West Bengal area represent the single largest environmental health problem I know of other than that associated with the Chernobyl disaster". Epidemiologist Smith (1995) writes, "The problems are very serious and warrant a very high priority for solutions and further investigations". Seriousness of the problem and the need for its solution have been highlighted by various other experts (Guha Mazumder, 1995; Hering, 1995; Redekopp, 1995; Saha, 1995b).

In a report, Saha (1995c) reported 1214 cases of chronic arsenical dermatosis from drinking arsenic-contaminated tubewell water in 61 villages of 6 districts of West Bengal during 1983–1987. Cutaneous malignancy (SCC) was detected as complication in 6 cases. Liver histology showed NCPF. The author reported that treatment with BAL is superior to penicillamine. The duration of drinking arsenic-contaminated water for symptoms to develop varied from 6 months to 2 years or more depending on the arsenic concentration in the tubewell water and the period of drinking. In describing the arsenic calamity of West Bengal, it was reported (Pearce, 1995) that hunger was an old enemy of poor villagers in West Bengal. But irrigation schemes brought a new and more insidious killer – arsenic poisoning.

Chatterjee et al. (1995) reported on the analysis of a few thousand water samples from six arsenic-affected districts of West Bengal and the study showed that groundwater contains two arsenic species, arsenate and arsenite in the ratio (approximately) 1:1. The highest arsenic concentration in a hand tubewell sample from Ramnagar village of South 24-Parganas district was 3700 μg/l. Urine samples from affected villages were also analysed for As(III), As(V), MMAA and DMAA. DMAA and MMAA were the predominant species. XRF analysis of solid residue after roto-evaporating 5 l of water from contaminated tubewells showed that high concentrations of Fe and Mn were present in the sample along with arsenic. Das et al. (1995d) reported arsenic in the hair, nail, urine, skin-scale and a few liver tissues (biopsy) of people from arsenic-affected villages who had arsenical skin lesions. Results showed elevated levels of arsenic in biological samples. The liver tissue analysis showed high arsenic, but non-detectable selenium suggesting that selenium deficiency might have a relation to arsenic toxicity.

On the basis of analysis of 20,000 water samples from arsenic-affected areas of West Bengal, Mandal et al. (1996) reported that 7 districts (North 24-Parganas, South 24-Parganas, Nadia, Bardhaman, Murshidabad, Malda, Hooghly) are arsenic-affected. Around 45% of these samples have arsenic concentration above 50 μg/l. The average concentration of arsenic in contaminated water was 200 μg/l. Many people have arsenical skin lesions as diffuse melanosis, spotted melanosis, leucomelanosis, keratosis, hyperkeratosis, dorsum, non-pitting oedema, gangrene, skin cancer. In addition, there were reports of arsenic patients suffering from internal cancers, such as bladder, lung etc. A study also reported (Nag et al., 1996) that arsenic is present in the form of arsenite and arsenate in groundwater with low concentration of antimony 0.03–0.9 μg/l. The first report of chronic neuropathy in arsenic patients from West Bengal was in 1996 (Basu et al., 1996). Eight out of 46 patients having arsenical skin lesions and a high arsenic exposure also demonstrated features of chronic peripheral neuropathy. They had features of sensory ataxia due to posterior column affection (87%), distal sensory features (50%) and two patients showed distal motor affection. Water sources were changed and a follow-up study after 5 years found moderate improvements for sensory features and minimal for motor features.

Bagla and Kaiser (1996) reported the magnitude and severity of the problem and the opinion of international experts on the arsenic calamity of West Bengal – India.

More and more information on the groundwater contamination and suffering of people surfaced with time (Roy Chowdhury et al., 1997; Mandal et al., 1997). Guha Mazumder et al. (1997) reported non-cancer effects of chronic arsenicosis with special reference to liver damage. The same report also discussed the features of peripheral vascular disease

among some of the patients. A comparative study of the arsenic calamity of Bangladesh with West Bengal – India was also reported by Dhar et al. (1997). World Health Organization consultants visited some of the arsenic-affected districts of West Bengal from 19th to 30th August 1996 and submitted a report (Consultants' Report to the World Health Organization, 1997). Their recommendations were (1) to assess the extent of contamination throughout the state; (2) to develop a master registry of water quality data; (3) to develop an analytical laboratory infrastructure; (4) to replace contaminated sources with safe sources and encourage surface water sources; (5) to develop a programme to ascertain the extent of health problem; (6) to develop local and regional medical programmes to assist in the diagnosis, screening and suggestive treatment; (7) to promote public and professional education on arsenic related health problem; and (8) to promote epidemiological study. World Health Organization also arranged a meeting with international experts to discuss the arsenic problem of West Bengal – India and Bangladesh (Consultation on Arsenic in Drinking Water, 1997). A recommendation for action (Recommendations for Action, 1997) and report of regional consultation were published (Report of a Regional Consultation, 1997).

Clinical and various laboratory investigations were carried out (Guha Mazumder et al., 1998a) on 156 patients to ascertain the nature and degree of morbidity and mortality that occurred due to chronic arsenic toxicity in some affected villages of West Bengal. All the patients studied had arsenical skin lesions. Other features included weakness, gastro-intestinal symptoms, involvement of the respiratory system and the nervous system. Lung function tests showed restrictive lung disease, abnormal electromyography, enlargement of the liver and portal hypertension. Liver biopsy reports of 45 patients showed NCPF in 41, cirrhosis in 2 and normal histology in 2 cases. A prospective, double blind, randomised placebo-controlled trial was carried out on 11 patients (Guha Mazumder et al., 1998b) to evaluate the efficiency and safety of the chelating agent meso-2,3-dimercaptosuccinic acid (DMSA) for chronic arsenicosis due to drinking arsenic-contaminated (≥ 50 μg/l) subsoil water in West Bengal. The other 10 patients were given placebo capsules. The clinical features were evaluated by an objective scoring system before and after treatment. It was concluded from the study that DMSA was not effective in producing any clinical or bio-chemical benefit or any histopathological improvement of skin lesions in patients with chronic arsenicosis. Guha Mazumder et al. (1998c) also reported high arsenic levels in drinking water and the prevalence of skin lesions in West Bengal. The study demonstrates clear exposure–response relationship between prevalence of skin lesions and both arsenic water levels and dose per body weight, with males showing greater prevalence to both keratosis and hyperpigmentation. Based on limited exposure assessment, some cases appear to be occurring at surprisingly low levels of exposure. There is evidence that the risks were somewhat greater for those who might be malnourished. Subramanian and Kosnett (1998) visited the state of West Bengal in August 1996 as consultants to the World Health Organization and reported their overview of the arsenic contamination problem in West Bengal on the basis of field visits, meeting with Indian and West Bengal government officials as well as scientists, engineers and physicians studying various arsenic problems of West Bengal. They made a recommendation for the development of a comprehensive infrastructure and plan of action. Mandal et al. (1998) reported the input of safe water used for drinking and cooking on five arsenic-affected families (17 members) for 2 years in West Bengal. Eight of them with arsenical skin lesions did not recover completely

after 2 years of drinking safe water, indicating a long-lasting damage. The investigation also showed that despite having safe water for drinking and cooking, the study group could not avoid an intake of arsenic time and again through edible herbs grown in contaminated water, food materials contaminated through washing and the occasional drinking of contaminated water. After minimizing the level of contamination, a noteworthy declining trend was observed in urine, hair, nail in all cases but not to that level observed in a normal population. Biswas et al. (1998) reported similar results on the village level.

In the international conference on arsenic in Dhaka, Bangladesh in 1998, Dutta, Saha and Guha Mazumder presented their findings on histopathology (Dutta et al., 1998), diagnosis of arsenicosis (Saha, 1998) and clinical manifestation of chronic arsenic toxicity (Guha Mazumder et al., 1998d) from the arsenic-affected population of West Bengal. Samanta et al. (1999) reported analysis of 47,000 and 9640 water samples for arsenic from 8 arsenic-affected districts of West Bengal and 64 districts of Bangladesh. On the basis of about 30,000 biological sample analyses (urine, hair, nail, skin-scale, blood) finding elevated levels of arsenic even in those who have no arsenical skin lesions, it was concluded that many villagers might be subclinically affected. A comparative study was reported (Mandal et al., 1999) between arsenic-affected villages of West Bengal and Bangladesh. Guha Mazumder et al. (1999a) reported about an epidemiological study in West Bengal and treatment with a chelating agent. It was mentioned that chelating agent DMSA was not found to be superior to a placebo, but drinking arsenic safe water, rest, nutritious diet and symptomatic treatment could reduce nearly 40% of the patients' symptoms significantly. While describing the treatment related to arsenic toxicity by Guha Mazumder et al. (1999b), it was reported that chelating agents, like D-penicillamine, DMSA and 2,3-dimercapto-1-propanesulfonate (DMPS), appear to be the rational mode of therapy for chronic arsenicosis, however, their usefulness is yet to be established. In a bird's eye view on the arsenical calamity in West Bengal, Saha (1999) reported that more than 200,000 people in 1206 rural areas of 76 blocks of 9 districts of West Bengal have been found to be affected with arsenicosis.

While presenting a review of arsenic poisoning and its health effect, Saha (1999) described major dermatological signs using photographs of the patients from arsenic-affected districts of West Bengal. Hepatic manifestation in chronic arsenic toxicity from arsenic-affected villages of West Bengal was described by Santra et al. (1999).

Chowdhury et al. (2000a,b) reported groundwater contamination in 985 villages from 69 police stations/blocks in 9 affected districts of West Bengal on the basis of 58,166 water sample analyses. Thousands of hair, nail and urine samples from people living in arsenic-affected villages were analysed and 77% of the samples on the average contained arsenic above normal/toxic levels. From the affected villages at random 29,035 people had been examined and 15% of those examined had skin lesions. Out of the total 6695 children examined 1.7% had arsenical skin lesions. Arsenical neuropathy was found in 37.2% of 413 arsenicosis patients from a few villages. Electrophysiologic studies on 20 patients showed an affliction of the sensory nerves in nine patients (95%) and an affliction of the motor nerves in four patients (25%). After extrapolation of the water analysis data, screening of villagers for arsenical skin lesions and a detailed study of a block, it is estimated that about 5 million people are drinking arsenic-contaminated water above 50 μg/l and around 300,000 people may have arsenical skin lesions. The total population in 9 arsenic-affected districts of West Bengal is about 43 million. This does not mean that

all the individuals are drinking arsenic-contaminated water and will suffer from arsenic toxicity, but undoubtedly they are at risk. Guha Mazumder et al. (2000a) made a cross-sectional survey involving 7683 participants of all ages in some arsenic-affected villages of South 24-Parganas between April 1995 and March 1996. The study reported that arsenic ingestion also causes pulmonary effects. Paul et al. (2000) studied skin biopsies of 42 patients suffering from chronic arsenic toxicity. Histological studies of H/E stained sections showed hyperkeratosis in 13, para keratosis in 13, acanthosis in 12, papillomatosis in 24, elongation of reteridges in 21, increased basal pigmentation in 27 and dysplastic changes in 8 cases. Squamous cell carcinoma was present in 2, basisquamous in 1 and basal cell carcinoma in 1 case. Changes of skin lesions after drug DMSA and DMPS therapy compared to placebo were studied. The results were inconclusive. Proliferative activity of skin lesions in patients with chronic arsenic toxicity was studied with AgNOR (argyrophillic proteins of the nuclear organiser region) stain to assess the biological behaviour.

Samanta et al. (2000) reported high performance liquid chromatography inductively coupled plasma mass spectrometry for speciation of arsenic compounds in urine from some arsenic-affected villages of West Bengal, This study would relate to recent inorganic arsenic exposure. From this study it was concluded that those living in arsenic-affected villages may use safe water from their tubewell but they cannot avoid, from time to time, arsenic contamination as many water sources in the surrounding areas are arsenic contaminated. In the international workshop on control of arsenic contamination in groundwater, Calcutta, India the 5th and 6th January 2000, De (2000) described the global arsenic scenario with particular reference to West Bengal; Pandey and Raut (2000) described epidemiological study of arsenic contamination in West Bengal; Saha (2000) on malignancy in arsenicosis with respect to West Bengal and Guha Mazumder et al. (2000b) described clinical features and dose related clinical effect. Chappell (2000) described the future danger in West Bengal and Bangladesh about arsenic in food chain as many crops are being irrigated with tubewell water containing elevated level of arsenic. Saha reported arsenicosis and the spread of arsenicosis in West Bengal (Saha, 2001a) and cutaneous malignancy in arsenicosis highlighting West Bengal problem (Saha, 2001b). Saha and Chakraborti (2001) reported their 17 years experience of arsenicosis in West Bengal.

Mazumder (2001) reported clinical aspects of chronic arsenic toxicity with respect to affected areas of West Bengal. A hospital-based study on arsenic and liver disease (Guha Mazumder, 2001) on 248 patients suffering from chronic arsenic toxicity was reported. NCPF is a predominant lesion in this population. An epidemiological study on various non-carcinomatous manifestation of chronic arsenic toxicity in the population of districts of West Bengal was described (Guha Mazumder et al., 2001a). It was reported that chronic arsenic toxicity in man produces protean non-carcinogenic manifestations such as weakness, liver enlargement, chronic lung disease and peripheral neuropathy. Guha Mazumder et al. (2001b) investigated the clinical use of DMPS in some arsenic patients in a randomised placebo-controlled trial. It was concluded from the study that DMPS treatment caused significant improvement in the clinical score of patients suffering from chronic arsenic toxicity. It was further reported that increased urinary excretion of arsenic during the period of therapy is the possible cause of improvement.

Rahman et al. (2001) reported on the basis of 101,934 hand tubewells, approximately 25,000 biological samples analysis and the screening of 86,000 persons in affected