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Arsenic Groundwater Contamination in Middle Ganga Plain, Bihar, India: A Future Danger?

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Arsenic Groundwater Contamination in Middle Ganga Plain, Bihar, India: A Future Danger? Dipankar Chakraborti, ¹ Subhash C. Mukherjee, ² Shyamapada Pati, ³ Mrinal K. Sengupta, ¹ Mohammad M. Rahman, Uttam K. Chowdhury, Dilip Lodh, Chitta R. Chanda, Anil K. Chakraborti, and Gautam K. Basu¹ ¹School of Environmental Studies, Jadavpur University, Kolkata 700 032, India; ²Department of Neurology, Medical College, Kolkata 700 073, India; ³Department of Obstetrics and Gynaecology, Institute of Post Graduate Medical Education and Research, S.S.K.M. Hospital, Kolkata 700 020, India. Address correspondence to D. Chakraborti, School of Environmental Studies, Jadavpur University, Kolkata 700 032, India. Telephone: 91 33 24146233; Fax: 91 33 24146266; E-mail: dcsoesju@vsnl.com

- 18 **Running Title:** Arsenic in the Middle Ganga Plain.
- 19 **Key Words:** arsenic poisoning; Ganga plain; Semria Ojha Patti village; childhood poisoning;
- 20 neurotoxicity; reproductive toxicity.
- 21 List of abbreviations: PMB = Padma Meghna Bramhaputra, BGS = British Geological
- 22 Survey, FI-HG-AAS = Flow injection hydride generation atomic absorption spectrometry,
- 23 SCM = Subhas Chandra Mukherjee, WHO = World Health Organization, SMP = Spotted
- 24 melanosis on palm, DMP = Diffuse melanosis on palm, SMT = Spotted melanosis on trunk,
- 25 DMT = Diffuse melanosis on trunk, LEU = Leuco melanosis, WBM = Whole body
- 26 melanosis, SKP = Spotted keratosis on palm, DKP = Diffuse keratosis on palm, SKS =
- 27 Spotted keratosis on sole, DKS = Diffuse keratosis on sole, DOR = Dorsal keratosis, CC =
- 28 Conjunctival congestion.

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59 ABSTRACT

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The pandemic of arsenic poisoning due to contaminated groundwater in West Bengal. India and all of Bangladesh has been thought limited to the Ganges Delta (the Lower Ganga Plain) despite early survey reports of arsenic contamination in groundwater in the Union Territory of Chandigarh and its surroundings in the northwestern Upper Ganga Plain and recent findings in the Terai area of Nepal. Anecdotal reports of arsenical skin lesions in villagers led us to evaluate arsenic exposure and sequelae in the Semria Ojha Patti village in the Middle Ganga Plain, Bihar, where tube wells replaced dug wells about 20 years ago. Analyses of the arsenic content of 206 tube wells (95% of the total) showed 56.8% to exceed arsenic concentrations of 50 μg/L with 19.9% >300 μg/L, the concentration predicting overt arsenical skin lesions. On medical examination of a self-selected sample of 550 (390 adults: 160 children), 13% of the adults and 6.3% of the children had typical skin lesions, an unusually high involvement for children, except in extreme exposures combined with malnutrition. The urine, hair, and nail concentrations of arsenic correlated significantly (r=0.72–0.77) with drinking water arsenic concentrations up to 1654 μg/L. On neurological examination, arsenic-typical neuropathy was diagnosed in 63% of the adults, a prevalence previously seen only in severe, subacute exposures. We also observed an apparent increase in fetal loss and premature delivery in the women with the highest drinking water arsenic. The possibility of contaminated groundwater at other sites in the Middle and Upper Ganga plain merits investigation.

INTRODUCTION

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Groundwater arsenic contamination in Lower Ganga Plain of West Bengal, India was first identified (Saha 1983) in July 1983. Garai et al. (1984) reported 16 patients in 3 families from one village of 24 Parganas district. Saha (1984) further reported 127 patients with arsenical skin lesions from 25 families of 5 villages in 3 districts. Over the last 15 years, as of July 2002, we have analyzed >125,000 water samples, >30,000 urine/hair/nail/skin scale samples, screened ~ 100,000 people in West Bengal for arsenical skin lesions and have registered 8500 people with arsenical skin lesions from 255 affected villages out of 306 screened. We have identified tube wells with arsenic concentrations >50 µg/L in over 3000 villages. Our overall study indicates that more than 6 million people from 9 affected districts (population ~ 50 million) of 18 total districts (total population ~ 80 million) are drinking water containing ≥50 µg/L arsenic and >300,000 people may have visible arsenical skin lesions (Chakraborti et al. 2002). The arsenic content of the biologic samples indicates that many more may be subclinically affected. In 1995, we identified 3 villages in 2 districts of the Padma-Meghna-Bramhaputra (PMB) delta of Bangladesh (Post Conference Report 1995), where groundwater contained ≥50 µg/L arsenic. The present situation is that in 2000 villages, in 50 of the total 64 districts of Bangladesh, groundwater contains arsenic >50 µg/L; and the British Geological Survey (BGS) estimates that more than 35 million people are drinking water containing arsenic ≥50 µg/L (BGS Technical Report 2001). In the combined areas of West Bengal and Bangladesh around 150 million people are at risk from groundwater arsenic contamination (Rahman et al. 2001). Despite years of research in West Bengal and Bangladesh, additional affected villages are identified by virtually every new survey. We feel our present research may be only the tip of the iceberg representing the full extent of arsenic contamination.

Although West Bengal's arsenic problem reached public concern almost 20 years ago, there are still few concrete plans, much less achievements, to solve the problem. Villagers are, usually, more severely affected than 20 years ago. Even now, many drinking arsenic contaminated water are not even aware of this fact and its consequences.

The source of arsenic in deltaic plain of West Bengal is considered to be the arsenic rich sediments transported from the Chotonagpur Rajmahal Highlands (Acharya et al. 2000; Saha et al. 1997) and deposited in sluggish meander streams under reducing conditions. It has been reported by Acharya et al. (1999) that the groundwater of Uttar Pradesh and Bihar has low concentrations of iron (0 to 700 μ g/L) and on this basis Archarya et al commented "the relatively low value of dissolved iron upstream of the Ganges delta indicates that the environment may not be sufficiently reducing to mobilize iron and arsenic". No detailed groundwater analysis for arsenic is available for the Middle and Upper Ganga Plains.

The Upper, Middle, and Lower Ganga Plains (Figure 1) are the most thickly populated areas of India. The fertile land and surplus in food production of the Gangetic Plain feeds India. The primary states of the Upper and Middle Ganga Plains are Uttar Pradesh (238,000 sq. km area; 166 million population) in the Upper Plain, and Bihar (94,163 sq. km area; 83 million population) in the Middle Ganga Plain and partly in the Upper.

Our studies since 1988 have centered on the severe arsenic contamination of groundwater in the Lower Ganga Plain of West Bengal and Bangladesh. We recently found severe groundwater arsenic contamination in the Bhojpur district, Bihar, which is in the Middle Ganga Plain. In 1976 there was a preliminary report of groundwater arsenic contamination from the Union Territory of Chandigarh and its surroundings (Datta 1976a, 1976b) in the northwestern Upper Ganga Plain. A recent report (Tandukar et al. 2001) shows groundwater in the Lower Plain area (Terai) of Nepal to be arsenic contaminated. The data from the Terai area together with our findings in the Bhojpur district of Bihar, about 200 km

south of Nepal, support further investigation of groundwater arsenic in the Middle and Upper Ganga Plains. Our available information has excluded the possibility of an anthropogenic source of groundwater arsenic in the area of Bhojpur, the subject of this report.

The present communication describes the groundwater arsenic contamination and an initial evaluation of the prevalence of arsenic toxicity in Semria Ojha Patti village in the Middle Ganga Plain of Bihar. The arsenical dermatosis, arsenical neuropathy, and arsenic toxicity among children are quite similar to that observed in West Bengal and Bangladesh (Biswas et al. 1998; Chowdhury et al. 1999, 2000a, 2000b; Mandal et al. 1996; Rahman et al. 2001; Roy Chowdhury et al. 1997). Our preliminary observations of an unusual reproductive toxicity indicate a particularly severe exposure.

METHODS

Location

A primary school teacher in Calcutta whose permanent address is Semria Ojha Patti village, Bhojpur district, Bihar, India, submitted a water sample to our laboratory because of his concerns over a possible toxic cause of the liver disease and skin lesions of his family in Bihar. The water sample contained 814 µg/L of arsenic. We showed him photographs of arsenical skin lesions and he noted that his family and neighbors have similar lesions, as did his first wife, who had died of cancer. The school teacher, who lived in Calcutta and visited his family every 6 months for 2–3 weeks, had no skin lesions. Preliminary analysis of 159 samples from the village showed such high concentrations of arsenic that a study was initiated.

The area studied was the Semria Ojha Patti village of Ara in the Bhojpur district of Bihar. Ara, the district's headquarter of Bhojpur district is between two important cities, Patna and Buxer, in the Middle Gangetic Plain, Bihar. The river Ganga is 8 km north of the village; the bordering state of Uttar Pradesh is a few kilometers to the west.

Figure 1 shows the position of Upper, Middle and Lower Plains of the Ganges, the groundwater arsenic contaminated area of Chandighar, arsenic affected areas of Terai region, Nepal; arsenic affected areas of West Bengal and Bangladesh in Lower Ganga Plain and the study village and its surroundings in Bhojpur district in the Middle Ganga Plain of Bihar.

Semria Ojha Patti, 4 sq. km in area with about 5000 inhabitants, is a remote, agricultural village. There are no factories on the periphery. Many of the adult males work outside Bihar to earn a living for their families. About 20 years ago the large-bore dug wells were abandoned and replaced by hand tube wells as the primary water source. The villagers denied any skin lesions prior to the tube wells. The aged villagers told us that at least 100 villagers who had arsenic skin lesions died during the last 10 years and some of them from cancer. Many died at a very young age. The villagers were unaware of any arsenic problem and believed that God's wrath was on the affected families.

Subjects

The 550 subjects examined were self-selected volunteers, 390 adults and 160 children, 6–11 years old, recruited by loudspeaker announcements at six central sites. All subjects consented, for themselves and their minor children, to medical evaluation and photography and provided samples of urine, hair, and nails. There was a low representation of women who feared stigmatization, of children attending school, and of men working outside the village.

Arsenical Skin Lesions. Of the 550 subjects examined, 60 (10.9%) had arsenical skin lesions (adults 13% and children 6.3%).

Neurological Examination. A convenience sample of 40 of the 60 subjects with arsenical skin lesions, (25 males and 15 females) underwent a detailed neurological examination.

Pregnancy Outcome. All 16 adult females in the group of 390 adults were examined clinically and their obstetric history was analyzed in detail. Of these 16 women, 12 were pregnant during our survey and 5 had arsenical skin lesions.

Arsenic Analysis

Water, hair, nail, and urine samples were analyzed for arsenic by flow injection hydride generation atomic absorption spectrometry (FI-HG-AAS). For urine samples, only inorganic arsenic and its metabolites together [arsenite, As (III), aresenate, As (V), Monomethyl arsonic acid, MMA (V), and Dimethyl arsinic acid, DMA (V)] were measured with no chemical treatment. Under the experimental conditions of FI-HG-AAS, arsenobetaine and arsenocholine do not produce a signal (Chatterjee et al. 1995). The modes of sample collection, the digestion procedures for hair and nails, analytical procedures, and the details of the instrument and flow injection system were as reported earlier (Chatterjee et al. 1995; Das et al. 1995; Samanta et al. 1999).

Iron Analysis

1, 10-phenanthroline method with UV-visible spectrophotometer was used for iron analysis of water samples (Fries and Getrost 1975).

RESULTS

Groundwater Arsenic Contamination in Semria Ojha Patti Village

The 206 water samples from Semria Ojha Patti represented 95% of the total tube wells of the village. We also analyzed 118 water samples from 5 villages within 3 km of Semria Ojha Patti (Figure 1) but none of the inhabitants were subjects. Figure 2 shows the relatively greater prevalence of highly contaminated hand tube wells compared to the arsenic contaminated areas of West Bengal and Bangladesh. The distribution indicates that, of the 5000 residents of Semria Ojha Patti, 18.4% used safe water (<10 μ g/L), 24.7% between 10 and 50 μ g/L, 56.8% \geq 50 μ g/L, and 19.9% \geq 300 μ g/L of arsenic. Our experience in West

Bengal and Bangladesh indicates the probability of skin lesions in a subject drinking water contaminated with ≥300 μg/L of arsenic. A comparative water analysis data for arsenic presented in Table 1 shows one village from West Bengal, India and one from Bangladesh, which are highly arsenic contaminated with Semria Ojha Patti village of Bihar. Table 1 shows that arsenic contamination of groundwater in Semria Ojha Patti village is comparable with highly arsenic contaminated villages of West Bengal and Bangladesh. The recommended value of arsenic in drinking water in India and Bangladesh is 50 μg/L.

Iron Concentration in Tube Well Water

Samples from 225 tube wells were analyzed for iron from Semria Ojha Patti and the surrounding 5 villages. The result (mean 2482 μ g/L, minimum 145 μ g/L, and maximum 8624 μ g/L) shows the iron concentrations to be higher than previously reported (0–700 μ g/L) for the Middle Plain (Acharya et al. 1999). The correlation between concentrations of iron and arsenic in water is poor (r=0.478).

Clinical Observations

Arsenical Skin Lesions. In this preliminary survey of 550 self-selected volunteers from the total 5000 villagers, 60 individuals (10.9% of the total and 6.3% of children) with arsenical skin lesions were registered. Figure 3 shows one subject with the full range of arsenical skin lesions including hyperkeratosis, Bowen's (suspected), and nonhealing ulcer (suspected cancer). The skin lesions observed in the village were similar to those noted in West Bengal and Bangladesh, but the relative prevalence of each type cannot be compared because of the inherent bias in self-selected volunteers with women particularly reluctant to be examined. Figure 4 tabulates the type of skin involvement of adults and children, the latter an unusual finding compared to West Bengal and Bangladesh (Biswas et al. 1998; Chowdhury et al. 1999, 2000b; Rahman et al. 2001; Roy Chowdhury et al. 1997).

Inorganic Arsenic and Its Metabolites in Urine. Analyses of 51 urine samples, including the mean, median, minimum, and maximum are given in Figure 5, along with a plot of the significant correlation of urine arsenic with drinking water arsenic (r=0.774; p<0.05).

Of the 51 urine samples analyzed, 98% have arsenic concentrations above the normal excretion level of arsenic in urine (Farmer and Johnson 1990), with 47% >500 μg/L, 33.3% >1000 μg/L, and 5.9% >3000 μg/L. The comparison of the urine arsenic of Semria Ojha Patti village with that of two highly arsenic contaminated villages described in our earlier work (Chowdhury et al. 2001) and cited in Table 1 shows a higher burden for Semria Ojha Patti village, Bihar: n=51, mean 798 μg/L, median 387 μg/L, range 24–3696 μg/L, than for Fakirpara village, West Bengal: n=325, mean 528 μg/L, median 318 μg/L, range 7–2911 μg/L, or Samta village, Bangladesh: n=300, mean 538 μg/L, median 289 μg/L, range 24–3085 μg/L). The urine arsenic of control populations (Chowdhury et al. 2003) with drinking water arsenic <3 μg/L was low in West Bengal (n=75, mean 16, median 15, range 10–41) and Bangladesh (n=62, mean 31, range 6–94, median 29). Village adults drink an estimated 4 liters of water per day and children 2 liters. Contaminated water is utilized for food preparation. In West Bengal, we attributed (Chowdhury et al. 2001) about 20–30% of the arsenic body burden to rice and vegetables grown in paddies irrigated by contaminated water; agricultural practices appeared similar in this village.

Total Arsenic in Hair and Nails. A total of 59 hair samples (34 samples from those with arsenical skin lesions and 25 without) and 38 nail samples (23 samples from those with arsenical skin lesions and 15 without) were analyzed for total arsenic. We found 57.6% of hair samples and 76.3% of nail samples to be above the normal range with a similar correlation of drinking water arsenic with the concentration in the hair (r=0.733; p<0.05; Figure 6) and the nails (r=0.719; p<0.05; Figure 7), similar to the findings in our West Bengal and Bangladesh studies (Biswas et al. 1998; Mandal 1998).

Arsenic Affected Children (6–11 years). In our field studies over the last 15 years in West Bengal and 7 years in Bangladesh, we have observed skin manifestations in exposed children under 11 years of age only under conditions of extreme exposure coupled with malnutrition (Chowdhury et al. 2000b; Rahman et al. 2001).

In the southern area of Semria Ojha Patti we identified a group of children (n=8) with skin involvement. All were drinking water from the same tube well, arsenic concentration 749 μ g/L. Table 2 lists their dermatological features and the concentrations of arsenic in their urine (inorganic arsenic and its metabolites), hair, and nails. The biological samples from village children with skin lesions are compared with those of children with arsenical skin lesions from the reference villages cited in Table 1. It is found that the Semria Ojha Patti village children have higher concentrations of arsenic in their biological samples compared to the Samta village, Bangladesh (Biswas et al. 1998) and Fakirpara village, West Bengal (Mandal et al. 1998). The arsenic concentrations at all 3 sites exceed those of control populations reported in our earlier work (Chowdhury et al. 2003).

NEUROLOGICAL INVOLVEMENT IN PATIENTS OF ARSENICOSIS

The obvious frequency of disabling neurologic signs initiated a more detailed examination and comparison with neuropathy found in arsenic affected areas of West Bengal (Chakraborti et al. 1999a; Chowdhury et al. 2000a, 2000b; Rahman et al. 2001). Of the 60 index subjects with skin lesions, a convenience sample of 40 (32 adults: 20 M, 12 F; 8 children 8–15 years: 5 M, 3 F) underwent a detailed neurological examination by the same neurologist (SCM) of earlier studies (Chakraborti et al. 1999a; Chowdhury et al. 2000a; 2000b; Rahman et al. 2001). Observations were recorded for items considered consistent with peripheral motor and sensory neuropathy and for other neurologic observations [as modified from Feldman et al. (1979), Galer (1998), and Kreiss et al. (1983)]. Items included to characterize neuropathy were (i) pain and paraesthesias (e.g. burning) in a stocking and glove

distribution, (ii) numbness, (iii) hyperpathia/allodynia, (iv) distal hypesthesias (reduced perception of sensation to pinprick/reduced or absent vibratory perception/affected joint position sensation/affected touch sensation), (v) calf tenderness, (vi) weakness/atrophy of distal limb muscles or gait disorder, (vii) reduction or absence of tendon reflexes.

Neurologic Findings

Arsenic neuropathy was clinically diagnosed in 21 (52.5%) of the 40 cases examined based on our previously defined criteria (Feldman et al. 1979; Galer 1998; Kreiss et al. 1983; Rahman et al. 2001). They all had arsenical skin lesions and elevated levels of arsenic in the hair, nail, and urine and in the drinking water (range 202 to 1654 µg/L). Table 3 shows arsenic concentration in urine, hair and nail of some patients and non-patients from Semria Ojha Patti village. The normal range of arsenic in biological samples is as cited in Table 3. Alternative causes excluded were inflammatory (Guillain Barre Syndrome), metabolic, nutritional, infectious, malignancy associated, hereditary, physical agents, entrapment, alcoholic, other toxins, and drugs. Two cases of arsenicosis who had mononeuritis multiplex due to leprosy were excluded.

The major presenting features are shown in Table 4. Most of the cases presented with distal paresthesias (40%) and distal hypoesthesias (35%) in stocking and glove distribution followed by limb pains and diminished or absent tendon reflexes (each 12.5%). Muscle weakness and atrophy affected only 3 patients (7.5%). Obvious signs of autonomic instability, cranial nerve involvement, headache, vertigo, sleep disorder, and mental changes were conspicuous by their absence. One 60-year-old woman had developed paranoid psychosis requiring treatment following the appearance of florid arsenical skin lesions, but this was not included in the tabulation.

Frequency of Neuropathy

The prevalence of neuropathy in this sample was 21/40 or 52.5% (Table 4), with males less affected (10/25; 40%) than females (11/15; 73.3%). Only 1 of 8 children (6–15 years) was affected (12.5%). The prevalence in males over 15 years of age was 62.5% and in females over 15 was 84.6%.

Type and Severity of Neuropathy

Table 4 lists 18 cases (45%) of sensory neuropathy while 3 cases (7.5%) had motor components as well (sensorimotor type). Moderate neuropathy was evident in 4 (10%). This was based on rigorous criteria of neuropathy (Kreiss et al. 1983) and included cases with impairment of at least 2 sensory modalities and reduced deep tendon reflexes. The remaining 17 cases (42.5%) had mild (predominantly sensory) neuropathy.

Magnitude of Neurological Involvement and Comparative Analysis

The reported prevalence of neuropathy in arsenic toxicity from chronic low dose exposure to arsenic contaminated water or occupational sources ranged from as low as 8.8% to 32% (Kreiss et al. 1983; Hotta 1989). Our own studies of large numbers of arsenicosis patients in West Bengal disclosed neuropathy in 34–37% (Chakraborti et al. 1999a; Chowdhury et al. 2000a, 2000b; Mukherjee et al. 2003; Rahman et al. 2001) except for a small population of subacute as opposed to chronic exposure where we found 86.8% (Rahman et al. 2001).

Relationship of Neuropathy and Arsenic Consumption

The 4 patients with moderate and sensorimotor neuropathy utilized water with arsenic 750 μ g/L and above; the 13 patients with mild and predominantly sensory neuropathy consumed water with arsenic 207 μ g/L to 637 μ g/L.

ARSENIC IN DRINKING WATER AND OBSTETRIC OUTCOME

The sample of 550 subjects included 16 adult females who were examined clinically and had their obstetric history analyzed in detail. Twelve women were pregnant when we

examined them. Table 5 summarizes the reproductive history of the 16 women categorized by the drinking water arsenic. The 5 subjects exposed to 463–1025 μg/L had an excess of miscarriage, stillbirths, preterm birth, and low birth weight infants. Data on the 3 women with the most adverse histories are given in Table 6; all 3 had severe skin lesions and were exposed to drinking water arsenic 1025 μg/L. The normal first pregnancy of all 3 women is noted. In this area, it is a social taboo to remain in the parent's home after first conception and it is possible that they drank arsenic safe water until the first conception (all three women reported that skin lesions similar to theirs were not observed in their native villages).

DISCUSSION

The manifestations of arsenicosis after exposure to contaminated groundwater in this small village at the western border of the Middle Ganga Plain are remarkably similar to our initial studies of the index villages in the Ganga Delta of West Bengal and Bangladesh where the finding of an intensely afflicted population led to the recognition of a pandemic. In retrospect the first case of arsenicosis was recognized in West Bengal in the 1980s (Chakraborti et al. 2002; Chakraborty et al. 1987; Garai et al. 1984; Saha 1983; Saha 1984) but widespread contamination was not defined until 1995. A similar pattern attended the evolving recognition of the groundwater contamination in the eastern Ganga delta of Bangladesh.

Understanding of the processes controlling the transfer of arsenic between aquifer sediments and groundwater is incomplete (Acharya et al. 1999, 2000; Akai et al. 1998; Bhattacharya et al. 1997; Chakraborti et al. 2001; Das et al. 1996; Nickson et al. 1998, 2000; Roy Chowdhury et al. 1999). According to Nickson et al. (1998) the primary source of arsenic is in association with iron oxyhydroxide in aquifer sediment and the key process of arsenic mobilization is desorption and dissolution of iron-oxides due to the reducing conditions of the aquifer and low hydraulic gradients. This theory does not explain the

increasing arsenic concentration in existing tube wells, previously safe but now progressively contaminated (Chakraborti et al. 2001). Das et al. (1996), Roy Chowdhury et al. (1999), and Chakraborti et al. (2001) proposed, on the basis of sediment analysis, that oxygen entering the aquifer due to heavy groundwater withdrawal for irrigation favors the oxidation of arsenic rich iron sulfide and mobilization of arsenic to the aquifer. The source of arsenic for West Bengal was considered by Acharya et al (2000), Saha et al. (1997) as the Rajmahal and Chotonagpur plateau of West Bengal. However, it appears the source of arsenic for Chandigarh, West Bengal, Bangladesh and Terai, Nepal is Himalaya (Chakraborti et al. 2001; Foster et al. 2000) and for Bihar, the source should also be the Himalaya.

Although it was reported (Acharya et al. 1999) that groundwater of Uttar Pradesh and Bihar has low concentrations of iron (0–700 μ g/L), our study of iron in groundwater of Semria Ojha Patti and its surrounding 5 villages of Bihar shows elevated concentrations of iron (145–8624 μ g/L).

Arsenic rich sediments derived from the Himalaya Mountains and foot hills of Shillong Plateau are deposited in Gangetic Plain, PMB delta of Bangladesh, Terai region of Nepal, Chandigarh area and, now, Bihar. Most of the arsenic contaminated tubewells are in the depth range 20–55m, similar to that of the West Bengal and Bangladesh. The deposition is expected to be in the Holocene type deposits. The meandering pattern of the river is responsible for the localized depositions of arsenic rich sediment in selected areas along the course of the river Ganga. Whether the huge groundwater withdrawal, pivotal to the green revolution, allows oxygen to enter into the aquifer initiating microbial activities, or has any relation to localized increases in arsenic mobilization is yet to be understood. As we reported (Chakraborti et al. 1999b; Rahman et al. 2001) on the basis of around 125,000 tube well analyses, some portions of Bangladesh and West Bengal are geologically free of arsenic. Similarly, the entire Ganga Plain, home of 449 million may not be uniformly affected despite

our expectations that groundwater will be arsenic contaminated over a wide region. Other toxic metals/metalloids in groundwater will also vary with the geological conditions and sedimentary deposits.

The extreme severity of the exposure in Semria Ojha Patti is typical of index villages with lesser exposures defined later. This preliminary study has the obvious deficits of a volunteer study population lacking full demographic representation. We captured relatively few women and missed many of the men working outside the village. We have no assurance that the childhood population was appropriately represented. The unverified obstetric histories were obtained from an extremely small sample with no control population. It is only by comparison with similar preliminary studies in West Bengal and Bangladesh that we can infer the severity of the exposure.

Those suffering from arsenical skin lesions (n=60) in Semria Ojha Patti village were drinking water with high concentrations of arsenic (mean 475 µg/L, median 431 µg/L, range 202–1654 µg/L). The World Health Organization (WHO) recommended maximum for arsenic in drinking water is 10 µg/L and the Indian standard is 50 µg/L. The finding of skin lesions in 13% of the adults group and a surprising 6.3% of children support severe exposure beginning with the transition to tube wells. The comparably high concentrations of arsenic in urine, hair and nails of the subjects (Table 3) are consistent with studies from West Bengal and Bangladesh (Biswas et al. 1998; Chowdhury et al. 1999, 2000b, 2003; Mandal et al. 1996; Rahman et al. 2001; Roy Chowdhury et al. 1997).

The particularly high prevalence of neuropathy in women is consistent with their more continuous exposure since many men work outside the home or village. As in our other studies (Mukherjee et al. 2003, Rahman et al. 2001) the extent and severity of the neuropathy increased with the arsenic concentrations in the drinking water. Although relatively few children had overt neuropathy they need to be tested for neurobehavioral and cognitive

effects. The effects of arsenic on the developing brain and nervous system may begin *in utero*, perinatally, or later and the severity is also dependent on other factors such as prematurity, intrauterine growth retardation, malnutrition and infection.

The anecdotal obstetric histories, suggesting reproductive toxicity at exposures sufficient to cause maternal toxicity, are highly provocative and consistent with the limited human data. An increase in spontaneous abortion, still birth, and perinatal mortality was reported from Karcag, Hungary, due to drinking water arsenic (Rudnai and Gulyas 1998). High perinatal and neonatal mortality have been reported from the mining area of northern Chile in association with arsenic contaminated water (Hopenhayn-Rich et al. 1998). In Bangladesh, Ahmad et al. (2001) reported a significant increase in spontaneous abortion, stillbirth and preterm birth. Increased arsenic in the cord blood and placental arsenic was reported for Argentine women drinking water with arsenic 200 µg/L (Concha et al. 1998). Studies implicating arsenic as a teratogen as well as a reproductive toxin are still inconclusive (Golub et al. 1998).

CONCLUSION

Groundwater arsenic contamination in West Bengal, India, surfaced during 1983 and that of Bangladesh in 1995 (Post Conference Report 1995). International attention focused on the arsenic problem in West Bengal and Bangladesh after the International Conference on Arsenic in Groundwater held in Calcutta, 1995 and the International Conference on Arsenic Pollution of Groundwater held in Dhaka, Bangladesh, 1998. The arsenic calamity of Bangladesh is considered to be world's biggest mass poisoning with millions of people exposed (Smith et al. 2000) and that of West Bengal has been compared with the Chernobyl disaster (Post Conference Report 1995).

The question of how much of Bihar and Uttar Pradesh are affected by groundwater arsenic contamination can be answered only by detailed surveys and water analyses. It is

relevant to recall that in 1984, only one village in West Bengal was known as arsenic affected; the present count is more than 3000 villages. For Bangladesh, it was 3 villages in 2 districts in 1995 and at present it is more than 2000 villages in 50 districts. Even after 15 years in West Bengal and 7 years in Bangladesh additional villages are identified by virtually every new survey. The geologic similarities of the Middle and Upper Ganga Plains support a test of the hypothesis that the risk may involve the entire Gangetic Plain. Twenty years ago and 7 years ago when the West Bengal government and Bangladesh were first informed of arsenic contamination it was considered a sporadic, easily remedied matter with little realization of the magnitude of the problem (Chakraborti et al. 2002). Even international aid agencies working in the subcontinent simply did not consider that arsenic could be present in groundwater (Chakraborti et al. 2002). The arsenic problem of West Bengal and Bangladesh intensified during a long period of neglect. Bihar's arsenic issue may not be a localized contamination. The magnitude of the problem should be assessed. Our earlier mistakes should not be repeated.

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586 587	Table 1 Distribution of Tubewell Arsenic Concentrations [ranges (μg/L)] in Fakirpara Village of West Bengal-India, Samta Village of Bangladesh and Semria Ojha Patti Village of Bihar-India											
588 589 590 591												
592 593 594	Area	Name of the village and district	Total Water samples analyzed	<10	Distribution 10-50	of total sa 51-99	mples in eac 100-299	ch range of ars 300-499	enic concer 500-699	ntration (μg/I 700-1000	L) >1000	
595 596 597 598	West Bengal India	Fakirpara North 24 Parganas	100% n=46	2 (4.35%)	3 (6.52%)	6 (13.04%)	12 (26.09%)	10 (21.74%)	8 (17.39%)	5 (10.87%)	-	
599 600 601 602	Bangladesh	Samta, Jessore	96% n=265	5 (1.89%)	18 (6.79%)	104 (39.25%)	93 (35.09%)	13 (4.91%)	21 (7.92%)	11 (4.15%)	-	
603 604 605 606	Bihar, India	Semria Ojha Patti, Bhojpur	95% n=206	38 (18.45%)	51 (24.76%)	26 (12.62%)	49 (23.79%)	22 (10.68%)	12 (5.82%)	6 (2.91%)	2 (0.97%)	

607 Table 2 608 609 Dermatological Features of a Group of Children and Arsenic Concentration in Their Drinking Water, Urine, Hair, and Nail 610 611 Sex & Melanosis Keratosis CB CC Arsenic Arsenic Arsenic Arsenic 612 concentration concentration concentration Age (yrs) Leu WB 613 Trunk Palm Sole in hair in nail Palm in water in urine 614 S D S D S D S D $(\mu g/L)$ $(\mu g/L)$ $(\mu g/kg)$ $(\mu g/kg)$ 615 F/7 749 1248 8471 7923 616 749 1259 5135 5121 M/6617 F/8 749 1333 3533 618 F/9 749 671 2710 749 619 M/11620 M/11749 2349 5414 621 M/9749 570 1935 2844 622 M/10749 2020 6833

S=Spotted, D=Diffuse, Leu=Leuco, WB=Whole Body; +=Mild, ++=Moderate, +++=Severe, CB=Chronic Bronchitis, CC=Conjunctival Congestion

625 Table 3 626 627 Arsenic Concentrations in Biological Samples of Patients and Non-patients in Semria Ojha Patti Village, Bhojpur, Bihar 628 629 Arsenic in hair^b Arsenic in urine^a Arsenic in nail^c 630 **Parameters** 631 $(\mu g/kg)$ $(\mu g/L)$ $(\mu g/kg)$ 632 633 38 No. of samples 51 59 634 Mean 798.6 2773.8 6976.9 635 Maximum 3696 12404 35790 636 Minimum 24 257 453 387 637 Median 1470 36015 638 ^aNormal urine arsenic ranges from 5–40 μg/d (1.5 L) (Farmer et al. 1990) 639 ^bNormal hair arsenic in hair ranges from 80–250 μg/kg with 1000 μg/kg an index of toxicity (Arnold et al. 1990). 640 641 ^cNormal arsenic content in nails is 430–1080 μg/kg (Ioanid et al. 1961).

642 643 644	Table Presenting Features, Incidence, Type Peripheral Neuropathy in S	and Severity of Arse	
645		Number of patients	Percentage
646	Presenting features (n=40)		
647	Distal paresthesias	16	40
648	Limb pains	5	12.5
649	Hyperpathia/allodynia	4	10
650	Distal hypesthesias	14	35
651	Calf tenderness	4	10
652	Distal limb weakness/atrophy	3	7.5
653	Diminshed or absent tendon reflexes	5	12.5
654	Tremor	3	7.5
655	Abnormal sweating	2	5
656	Overall incidence of neuropathy (n=40)	21	52.5
657	Type of neuropathy (n=21)		
658	Sensory	18	45
659	Sensorimotor	3	7.5
660	Severity of neuropathy (n=21)		
661	Mild	17	42.5
662	Moderate	4	10

663 Table 5 664 Arsenic in Drinking Water and Obstetric Outcome 665 Group A Group B Group C 666 667 (n=5)(n=4)(n=7)668 Skin lesions Positive 669 Range of arsenic concentration in water (µg/L) 463-1025 174-459 7–39 670 No. of pregnancies 24 14 26 671 Spontaneous abortion 12.5% 21.3% 672 Still birth 12.5% 7.1% 8% 673 Preterm birth 25% 7.1% 8% 674 Low birth weight 20.5% 7.1% 675 Neonatal death 4.1% 7.1% 676 Congenital anomaly 4.1% 7.1% 677

677 678 679	Table 6 Three Women of Group A Suffering from Chronic Arsenic Toxicity with Obstetric Outcome														
680 681 682	Case No.	Age	Pal	Melar m	nosis Trunk	Leuco	Whole body		Kera lm			No. of regnancies	Details of previous pregnancies	Arsenic in water (µg/L)	Arsenic in hair (µg/kg)
683 684			S	D	S D)	00 4 j	S	D	S	D			(108/2)	(100,220)
685 686 687 688	1	21	_	+++	++ ++		++	++	++	++	+	4	1 st pregnancy – FTND 2 nd Pregnancy – Stillbirth 3 rd Pregnancy – Preterm birth 4 th Pregnancy – FTND	1025	9764
689 690 691 692 693 694 695 696	2	28	-	++	++ ++		+	+	+	+	+	6	1 st Pregnancy – FTND 2 nd Pregnancy – Spontaneous abortic – 4 months 3 rd Pregnancy – Spontaneous abortic – 3 months 4 th Pregnancy – Still birth 5 th Pregnancy – Lowbirth weight 6 th Pregnancy – FTND		4497
697 698 699 700 701 702 703	3	25	_ D_D;	++ ***********************************	++ ++	Eull torm	++	++	++	++	++	6	1 st Pregnancy – Preterm birth 2 nd Pregnancy – Preterm birth 3 rd Pregnancy – Preterm birth 4 th Pregnancy – Spontaneous abortio 5 th Pregnancy – Neonatal death 6 th Pregnancy – Preterm birth	1025 on	6203
704	S=Spotted, D=Diffuse, FTND=Full term normal delivery														

- 705 Legends
- 706 Figure 1. Shows the position of Upper, Middle and Lower plains of Ganges, the
- 707 groundwater arsenic contaminated area of Chandighar, affected areas of Nepal, affected areas
- of West Bengal and Bangladesh in Lower Ganga Plain and the study site, Semria Ojha Patti
- village and its surroundings in Bhojpur district in Middle Ganga Plain of Bihar.
- 710 Figure 2. Arsenic concentrations in the tube wells of Semria Ojha Patty village
- 711 compared with the arsenic affected areas of West Bengal and Bangladesh.
- 712 Figure 3. A subject from Semria Ojha Patti village with the full panoply of arsenical
- skin lesions including hyper-keratosis, suspected Bowen's, and non-healing ulcer (suspected
- 714 cancer).
- 715 Figure 4. Comparative prevalence of dermatological involvement manifested by the
- arsenic affected adults and children of Semria Ojha Patti village. SMP=Spotted melanosis on
- 717 palm, DMP=Diffuse melanosis on palm, SMT=Spotted melanosis on trunk, DMT=Diffuse
- 718 melanosis on trunk, LEU=Leuco melanosis, WBM=Whole body melanosis, SKP=Spotted
- 719 keratosis on palm, DKP=Diffuse keratosis on palm, SKS=Spotted keratosis on sole,
- 720 DKS=Diffuse keratosis on sole, DOR=Dorsal keratosis, CC=Conjunctival congestion.
- 721 Figure 5. Correlation between arsenic concentrations in urine and drinking water.
- Figure 6. Correlation between arsenic concentrations in hair and drinking water.
- 723 Figure 7. Correlation between arsenic concentrations in nails and drinking water.

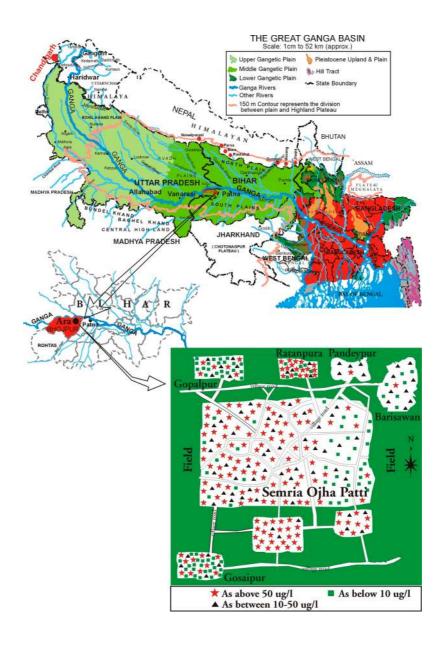


Fig. 1

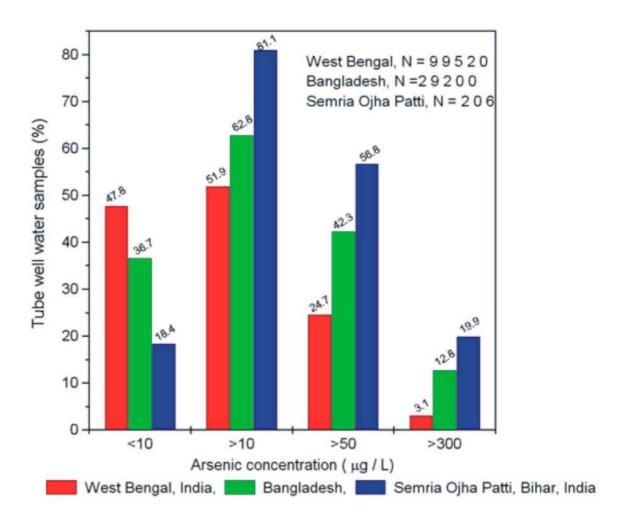
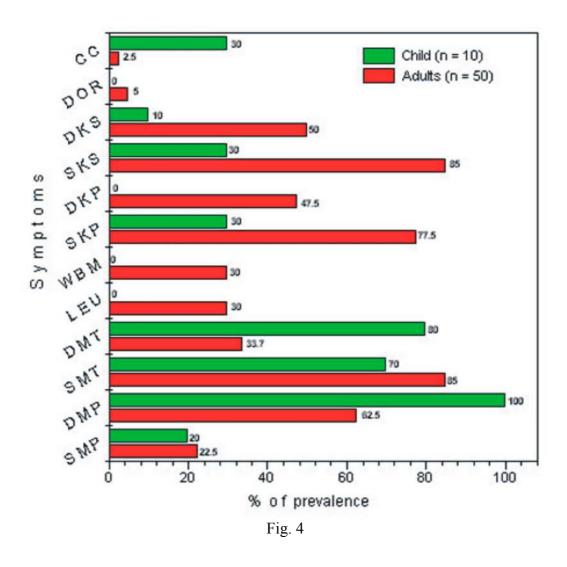


Fig 2





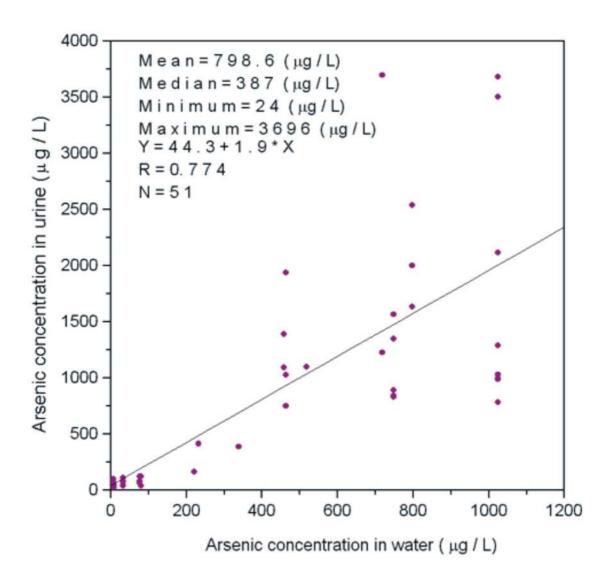


Fig. 5

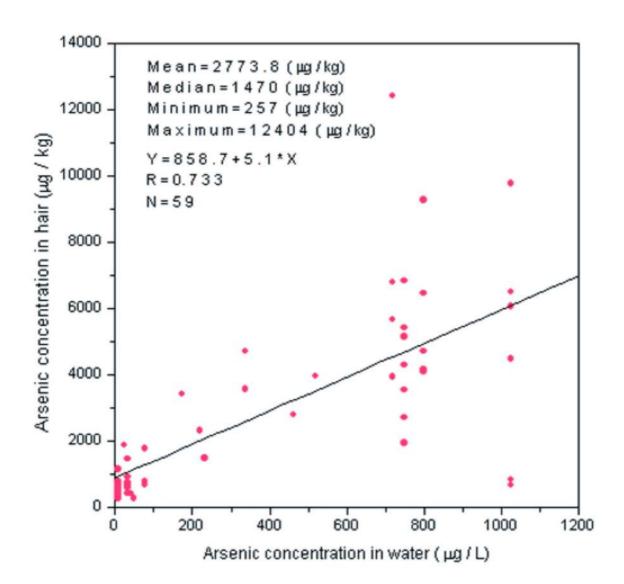


Fig. 6

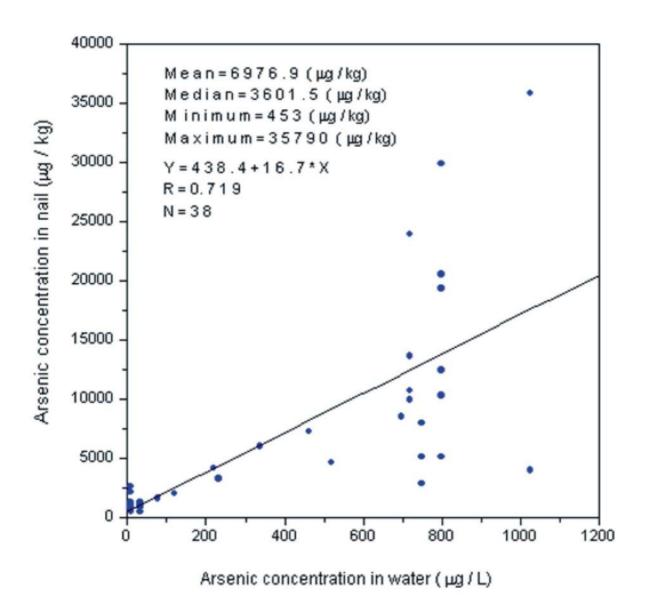


Fig. 7