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

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The pandemic of arsenic poisoning due to contaminated groundwater in West Bengal, India, and all of Bangladesh has been thought to be 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 that 56.8% exceeded 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 and 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 1,654 µg/L. On neurologic 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 concentrations of arsenic in their drinking water. The possibility of contaminated groundwater at other sites in the Middle and Upper Ganga Plain merits investigation. *Key words:* **arsenic poisoning, childhood poisoning, Ganga Plain, neurotoxicity, reproductive toxicity, Semria Ojha Patti village.** *Environ Health Perspect* **111:1194–1201 (2003). doi:10.1289/ehp.5966 available via** *http://dx.doi.org/* **[Online 5 February 2003]**

Groundwater arsenic contamination in the Lower Ganga Plain of West Bengal, India, was first identified in July 1983 (Saha KC. Unpublished data). Garai et al. (1984) reported 16 patients in three families from one village of 24 Parganas District. Saha (1984) further reported 127 patients with arsenical skin lesions from 25 families of five villages in three districts. Over the last 15 years, as of July 2002, we have analyzed > 125,000 water samples and > 30,000 urine/hair/nail/skin scale samples, screened approximately 100,000 people in West Bengal for arsenical skin lesions, and registered 8,500 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 more than 3,000 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 three villages in two districts of the Padma–Meghna–Bramhaputra delta of Bangladesh (Post Conference Report 1995), where groundwater contained ≥ 50 µg/L arsenic. Presently, in 2,000 villages in 50 of the total 64 districts of Bangladesh, groundwater contains arsenic concentrations ≥ 50 µg/L, and the British Geological Survey (BGS) has estimated that > 35 million people are drinking water containing concentrations of arsenic ≥ 50 µg/L (BGS 2001). In the combined areas of West Bengal and Bangladesh, around 150 million people are at risk from arsenic-contaminated groundwater (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 that 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 they were 20 years ago. Even now, many who are 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 meandering streams under reducing conditions. Acharya et al. (1999) has reported that the groundwater of Uttar Pradesh and Bihar has low concentrations of iron (0–700 µg/L) and, on this basis, commented that

> 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 food production of the Gangetic Plain feeds India. The primary states of the Upper and Middle Ganga Plains are Uttar Pradesh (238,000 km^2 area, 166 million population) in the Upper Plain, and Bihar (94,163 km^2 area, 83 million population) in the Middle and partly in the Upper Ganga Plain.

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. A preliminary report of groundwater arsenic contamination from the Union Territory of Chandigarh and its surroundings in the northwestern Upper Ganga Plain was published in 1976 (Datta 1976; Datta and Kaul 1976). A recent report (Tandukar et al. 2001) shows groundwater in the Lower Plain area (Terai) of Nepal to be contaminated with arsenic. The data from Tandukar et al. (2001) together with our present 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.

In this article we describe the groundwater arsenic contamination and an initial evaluation

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

Methods

Location. A primary school teacher in Kolkata, India, 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 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 Kolkata 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 headquarters, 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 the Upper, Middle, and Lower Plains of the Ganges; the area of arsenic-contaminated groundwater in Chandighar; arsenic-affected areas of the Terai region of Nepal; arsenic-affected areas of West Bengal and Bangladesh in the Lower Ganga Plain; and the study village and its surroundings in Bhojpur District in the Middle Ganga Plain of Bihar.

Semria Ojha Patti, 4 km^2 in area with about 5,000 inhabitants, is a remote agricultural village. Because 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-diameter dug wells (~3 m) were abandoned and replaced by hand tube wells (in which subsurface water is withdrawn by a hand pump) as the primary water source. The villagers denied any skin lesions before the installation of the tube wells. The older villagers told us that at least 100 villagers who had arsenical skin lesions died during the last 10 years, 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 of age, 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), children who attended school, and men who worked outside the village.

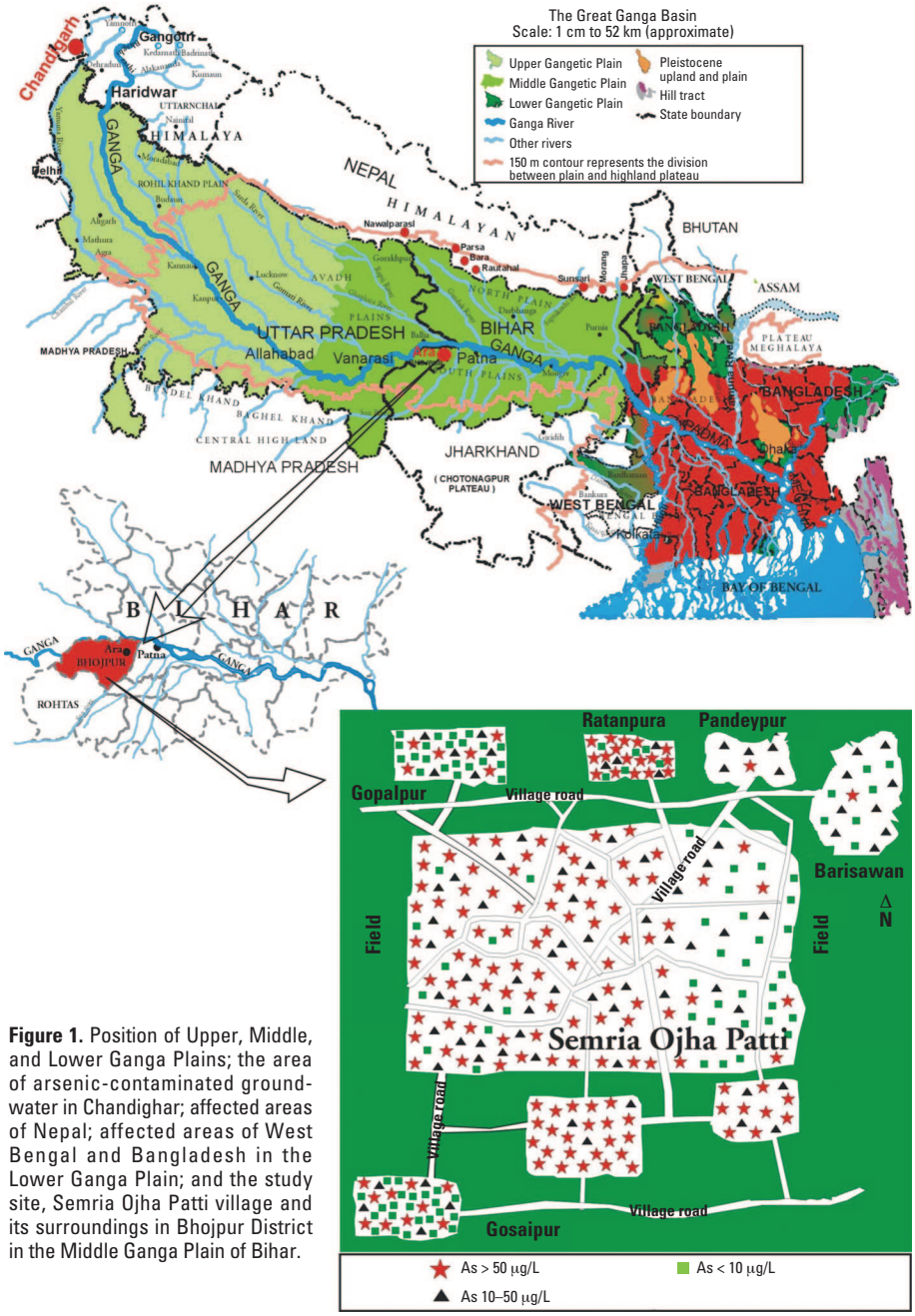

Figure 1. Position of Upper, Middle, and Lower Ganga Plains; the area of arsenic-contaminated groundwater in Chandighar; affected areas of Nepal; affected areas of West Bengal and Bangladesh in the Lower Ganga Plain; and the study site, Semria Ojha Patti village and its surroundings in Bhojpur District in the Middle Ganga Plain of Bihar.

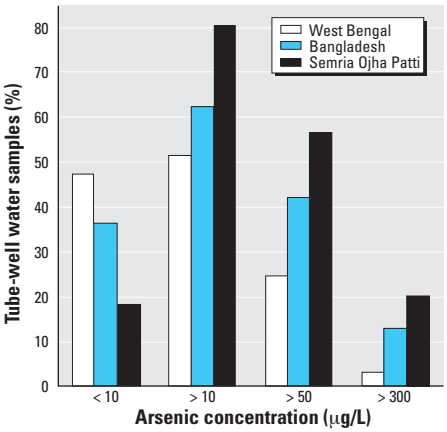

Figure 2. Arsenic concentrations in the tube wells of Semria Ojha Patti village (n = 206) compared with the arsenic-affected areas of West Bengal (n = 99,520) and Bangladesh (n = 29,200).

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

Neurologic examination. A convenience sample of 40 of the 60 subjects with arsenical skin lesions (25 males and 15 females) underwent a detailed neurologic 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. We analyzed water, hair, nail, and urine samples 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); arsenate, 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 described previously (Chatterjee et al. 1995; Das et al. 1995; Samanta et al. 1999).

Iron analysis. We used the 1,10-phenanthroline method with an ultraviolet-visible spectrophotometer 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 five villages within 3 km of Semria Ojha Patti (Figure 1), but none of their inhabitants were subjects. Figure 2 shows the relatively greater prevalence of highly contaminated tube wells compared with the arsenic-contaminated areas of West Bengal and Bangladesh. The distribution indicates that, of the 5,000 residents of Semria Ojha Patti, 18.4% used safe water (< 10 µg/L arsenic), 24.7% used water with 10–50 µg/L arsenic, 56.8% with ≥ 50 µg/L, and 19.9% ≥ 300 µg/L. Our experience in West Bengal and Bangladesh indicates the probability of skin lesions in a subject drinking water contaminated with ≥ 300 µg/L arsenic. Table 1 shows water analysis data for arsenic from a village in West Bengal, India, and one in Bangladesh, with water that is highly contaminated with arsenic, and data from Semria Ojha Patti village of Bihar. The arsenic contamination of groundwater in Semria Ojha Patti village is comparable with that in the 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 concentrations in tube-well water. Samples from 225 tube wells from Semria Ojha Patti and the surrounding five villages were analyzed for iron. Iron concentrations in these samples (mean, 2,482 µg/L; minimum, 145 µg/L; maximum, 8,624 µg/L) were higher than previously reported (0–700 µg/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 of 5,000 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 disease (suspected), and nonhealing ulcers (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 with West Bengal and Bangladesh (Biswas et al. 1998; Chowdhury TR et al. 1997; Chowdhury UK et al. 1999, 2000b; Rahman et al. 2001).

Inorganic arsenic and its metabolites in urine. Analyses of 51 urine samples, including the mean, median, minimum, and maximum, are presented 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% had arsenic concentrations above the normal excretion level of arsenic in urine (Farmer and Johnson 1990), with 47% > 500 µg/L, 33.3% > 1,000 µg/L, and 5.9% > 3,000 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 UK et al. 2001) and detailed in Table 1 shows a higher burden for Semria Ojha Patti village in Bihar ($n = 51$; mean, 798 µg/L; median, 387 µg/L; range, 24–3,696 µg/L) than for Fakirpara village, West Bengal ($n = 325$; mean, 528 µg/L;

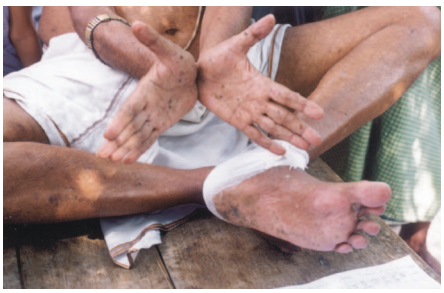

Figure 3. Subject from Semria Ojha Patti village with the full panoply of arsenical skin lesions, including hyperkeratosis, suspected Bowen's disease, and nonhealing ulcers (suspected cancer).

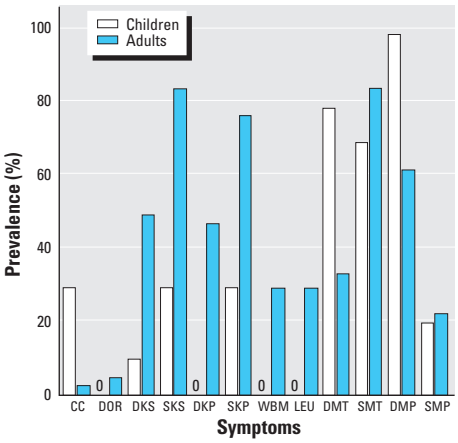

Figure 4. Comparative prevalence of dermatologic involvement manifested by the arsenic-affected adults ($n = 50$) and children ($n = 10$) of Semria Ojha Patti village. Abbreviations: CC, conjunctival congestion; DKP, diffuse keratosis on palm; DKS, diffuse keratosis on sole; DMP, diffuse melanosis on palm; DMT, diffuse melanosis on trunk; DOR, dorsal keratosis; LEU, leuco-melanosis (white spots with some black); SKP, spotted keratosis on palm; SKS, spotted keratosis on sole; SMP, spotted melanosis on palm; SMT, spotted melanosis on trunk; WBM, whole-body melanosis.

Table 1. Distribution [number (percent)] of tube wells by arsenic concentration range (µg/L) in Fakirpara village, Samta village, and Semria Ojha Patti village.

Location	Village and district	Percent water samples analyzed	Arsenic concentration range (µg/L)							
			< 10	10–50	51–99	100–299	300–499	500–699	700–1,000	> 1,000
West Bengal	Fakirpara, North 24 Parganas, India ($n = 46$)	100	2 (4.35)	3 (6.52)	6 (13.04)	12 (26.09)	10 (21.74)	8 (17.39)	5 (10.87)	—
Bangladesh	Samta, Jessore ($n = 265$)	96	5 (1.89)	18 (6.79)	104 (39.25)	93 (35.09)	13 (4.91)	21 (7.92)	11 (4.15)	—
Bihar	Semria Ojha Patti, Bhojpur, India ($n = 206$)	95	38 (18.45)	51 (24.76)	26 (12.62)	49 (23.79)	22 (10.68)	12 (5.82)	6 (2.91)	2 (0.97)

median, 318 µg/L; range, 7–2,911 µg/L), or Samta village, Bangladesh (n = 300; mean, 538 µg/L; median, 289 µg/L; range, 24–3,085 µg/L). The urine arsenic of control populations (Chowdhury UK 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 L water per day, and children 2 L/day. Contaminated water is used for food preparation. In West Bengal, we attributed about 20–30% of the arsenic body burden to rice and vegetables grown in paddies irrigated by contaminated water (Chowdhury UK et al. 2001); agricultural practices appeared similar in this village.

Total arsenic in hair and nails. We analyzed total arsenic in 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). 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 of age). 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 younger than 11 years of age only under conditions of extreme exposure coupled with malnutrition (Chowdhury UK 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, which had an arsenic concentration of 749 µg/L. Table 2 lists their dermatologic features and the concentrations of arsenic in their urine (inorganic arsenic and its metabolites), hair, and nails. The biologic samples from village children with skin lesions are compared in Table 3 with those of children with arsenical skin lesions from the reference villages cited in Table 1. As defined in Table 3, the Semria Ojha Patti village children have higher concentrations of arsenic in their biologic samples. The arsenic concentrations at all three sites exceed those of control populations reported in our earlier work (Chowdhury UK et al. 2003).

Neurologic 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. 1999b; Chowdhury UK et al. 2000a, 2000b; Rahman et al. 2001). Of the 60 index subjects with skin lesions, a convenience sample of 40 (32 adults, 20 male and 12 female; 8 children 8–15 years of age, 5 male and 3 female) underwent a detailed neurologic examination by the same neurologist (S.C.M.) who performed examinations in earlier studies (Chakraborti et al. 1999b; Chowdhury UK 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 *a*) pain and paresthesias (e.g., burning) in a stocking and glove distribution, *b*) numbness, *c*) hyperpathia/allodynia, *d*) distal hypesthesias (reduced perception of sensation to pinprick, reduced or absent vibratory perception, affected joint position sensation, affected touch sensation), *e*) calf tenderness,

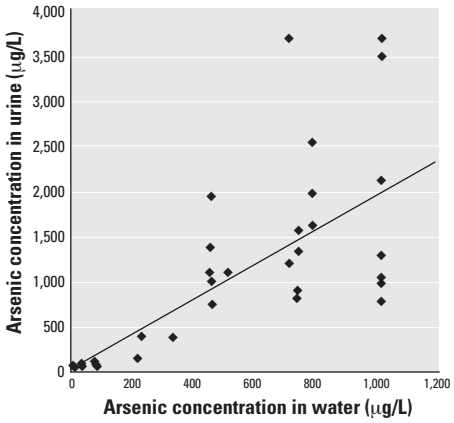

Figure 5. Correlation between arsenic concentrations in urine and drinking water. Mean = 798.6 µg/L; median = 387 µg/L; minimum = 24 µg/L; maximum = 3,696 µg/L; Y = 44.3 + (1.9 × X); R = 0.774; n = 51.

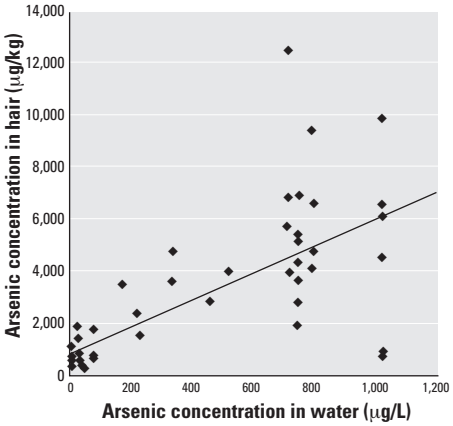

Figure 6. Correlation between arsenic concentrations in hair and drinking water. Mean = 2773.8 µg/kg; median = 1,470 µg/kg; minimum = 257 µg/kg; maximum = 12,404 µg/kg; Y = 858.7 + (5.1 × X); R = 0.773; n = 59.

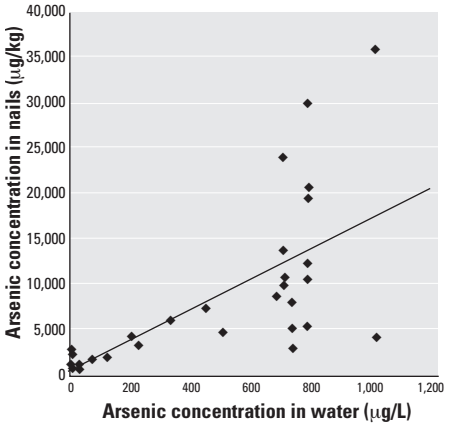

Figure 7. Correlation between arsenic concentrations in nails and drinking water. Mean = 6976.9 µg/kg; median = 3601.5 µg/kg; minimum = 453 µg/kg; maximum = 35,790 µg/kg; Y = 438.4 + (16.7 × X); R = 0.719; n = 38.

Table 2. Dermatologic features of a group of children and arsenic concentrations in their drinking water, urine, hair, and nails.

	Melanosis						Keratosis						Arsenic concentration			
	Palm		Trunk				Palm		Sole		CB		Water	Urine	Hair	Nails
Sex/age	S	D	S	D	Leu	WB	S	D	S	D	(years)	CC	(µg/L)	(µg/L)	(µg/kg)	(µg/kg)
F/7 years	–	+	++	++	–	–	–	–	–	–	–	–	749	1,248	8,471	7,923
M/6 years	–	+	+	++	–	–	–	–	–	–	–	+	749	1,259	5,135	5,121
F/8 years	–	+	+	+	–	–	–	–	–	–	–	–	749	1,333	3,533	–
F/9 years	–	+	+	+	–	–	–	–	–	–	2	–	749	671	2,710	–
M/11 years	–	+	+	+	–	–	–	–	–	–	2	+	749	–	–	–
M/11 years	–	+	+	+	–	–	+	–	+	–	4	–	749	2,349	5,414	–
M/9 years	+	+	+	+	–	–	+	–	+	+	–	+	749	570	1,935	2,844
M/10 years	+	+	+	+	–	–	–	–	–	–	–	–	749	2,020	6,833	–

Abbreviations: +, mild; ++, moderate; +++, severe; –, not detected; CB, chronic bronchitis; CC, conjunctival congestion; D, diffuse; F, female; Leu, leuco-melanosis; M, male; S, spotted; WB, whole body.

f) weakness/atrophy of distal limb muscles or gait disorder, and *g*) 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 their hair, nails, and urine (Table 4) and in the drinking water (range, 202–1,654 µg/L). The normal range of arsenic in biologic samples is shown in Table 3. Alternative causes excluded were inflammatory (Guillain-Barré syndrome), metabolic, nutritional, infectious, malignancy-associated, and hereditary factors and physical agents, entrapment, alcohol, other toxins, and drugs. Two subjects with arsenicosis who had mononeuritis multiplex due to leprosy were excluded.

The major presenting features are shown in Table 5. Most of the cases presented with distal paresthesias (40%) and distal hypesthesias (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 three 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, which required treatment after 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 of 40, or 52.5% (Table 5), with males less affected (10 of 25; 40%) than females (11 of 15; 73.3%). Only one of eight children (6–15 years of age) was affected (12.5%). In children more than 15 years of age, the prevalence in males was 62.5%, and that in females was 84.6%.

Type and severity of neuropathy. Table 5 lists 18 cases (45%) of sensory neuropathy; 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 two sensory modalities and reduced deep tendon reflexes. The remaining 17 cases (42.5%) had mild (predominantly sensory) neuropathy.

Magnitude of neurologic 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% (Hotta 1989; Kreiss et al. 1983). Our own studies of large numbers of arsenicosis patients in West Bengal disclosed neuropathy in 34–37% (Chakraborti et al. 1999b; Chowdhury UK 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 four patients with moderate and sensorimotor neuropathy used water with arsenic concentrations of ≥ 750 µg/L; the 13 patients with mild and predominantly sensory neuropathy consumed water containing 207–637 µg/L arsenic.

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. The reproductive histories of the 16 women categorized by drinking water arsenic are presented in Table 6. The 5 subjects exposed to 463–1,025 µg/L had an excess of miscarriages, stillbirths, preterm births, and infants with low birth weights. Data on the 3 women with the most adverse histories are presented in Table 7; all 3 had severe skin lesions (Figure 8) and were exposed to drinking water containing 1,025 µg/L arsenic. The normal first pregnancy is noted for 2 of 3 women. 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 low-arsenic or arsenic-safe (< 10 µg/L) water until the first conception (all 3 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 and Saha 1987; Garai et al. 1984; Saha 1984; Saha KC. Unpublished data), but widespread contamination was not defined until 1995. A similar pattern attended the evolving recognition of groundwater contamination in the eastern Ganga Delta of Bangladesh.

The processes controlling the transfer of arsenic between aquifer sediments and groundwater is not completely understood (Acharya et al. 1999, 2000; Akai et al. 1998; Bhattacharya et al. 1997; Chakraborti et al. 2001; Chowdhury TR et al. 1999; Das et al. 1996; Nickson et al. 1998, 2000). 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

Table 3. Arsenic concentrations in biologic samples from children of Semria Ojha Patti village and from Fakirpara village, West Bengal, and Samta village, Bangladesh.

	Fakirpara			Samta			Semria Ojha Patti		
Parameters	In urine[a] (µg/L)	In hair[b] (µg/kg)	In nail[c] (µg/kg)	In urine[a] (µg/L)	In hair[b] (µg/kg)	In nail[c] (µg/kg)	In urine[a] (µg/L)	In hair[b] (µg/kg)	In nail[c] (µg/kg)
No. of samples	13	13	13	24	20	19	7	7	3
Mean	598.3	4,370	7,910	764	2,200	8,300	1,350	4861.5	5,296
Maximum	1473.6	13,260	15,790	3,085	4,880	16,660	2,349	8,471	7,923
Minimum	278.8	1,200	1,700	110	630	2,720	570	1,935	2,844
Median	415.6	4,230	7,010	385	2,025	6,900	1,259	5,137	5,121

[a]Normal urine arsenic ranges from 5 to 40 µg/day (1.5 L) (Farmer and Johnson 1990). [b]Normal hair arsenic is 80–250 µg/kg, with 1,000 µg/kg an index of toxicity (Arnold et al. 1990). [c]Normal arsenic content of nails is 430–1,080 µg/kg (Ioanid et al. 1961).

Table 4. Arsenic concentrations in biologic samples of patients and nonpatients in Semria Ojha Patti village.

Parameters	Urine[a] (µg/L)	Hair[b] (µg/kg)	Nails[c] (µg/kg)
No. of samples	51	59	38
Mean	798.6	2773.8	6976.9
Maximum	3,696	12,404	35,790
Minimum	24	257	453
Median	387	1,470	3601.5

[a]Normal urine arsenic ranges from 5 to 40 µg/day (1.5 L) (Farmer and Johnson 1990). [b]Normal arsenic in hair ranges from 80–250 µg/kg, with 1,000 µg/kg being an index of toxicity (Arnold et al. 1990). [c]Normal arsenic content in nails is 430–1,080 µg/kg (Ioanid et al. 1961).

Table 5. Presenting features, incidence, type, and severity of arsenic-induced peripheral neuropathy in Semria Ojha Patti village.

	No. of patients (%)
Presenting features (*n* = 40)	
Distal paresthesias	16 (40)
Limb pains	5 (12.5)
Hyperpathia/allodynia	4 (10)
Distal hypesthesias	14 (35)
Calf tenderness	4 (10)
Distal limb weakness/atrophy	3 (7.5)
Diminished or absent tendon reflexes	5 (12.5)
Tremor	3 (7.5)
Abnormal sweating	2 (5)
Overall incidence of neuropathy (*n* = 40)	21 (52.5)
Type of neuropathy (*n* = 21)	
Sensory	18 (45)
Sensorimotor	3 (7.5)
Severity of neuropathy (*n* = 21)	
Mild	17 (42.5)
Moderate	4 (10)

explain the increasing arsenic concentration in existing tube wells, previously safe but now progressively contaminated (Chakraborti et al. 2001). Das et al. (1996), Chowdhury TR 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) and Saha et al. (1997) to be the Rajmahal and Chotonagpur plateau of West Bengal. However, it appears that the source of arsenic for Chandigarh, West Bengal; Bangladesh; and Terai, Nepal, is the Himalayas (Chakraborti et al. 2001; Foster et al. 2000), and for Bihar, the source should also be the Himalayas.

Although it has been reported that groundwater of Uttar Pradesh and Bihar has low concentrations of iron (0–700 µg/L) (Acharya et al. 1999), our study of iron in groundwater of Semria Ojha Patti and its surrounding five villages in Bihar shows elevated concentrations of iron (145–8,624 µg/L).

Arsenic-rich sediments derived from the Himalayan mountains and the foothills of the Shillong Plateau are deposited in the Gangetic Plain, Padma–Meghna–Bramhaputra delta of Bangladesh, Terai region of Nepal, Chandigarh area, and now Bihar. Most of the arsenic-contaminated tube wells are in the depth range of 20–55 m, similar to that of West Bengal and Bangladesh tube wells. 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 reported in our analyses of approximately 125,000 tube wells (Chakraborti et al. 1999a; Rahman et al. 2001), some portions of Bangladesh and West Bengal are geologically free of arsenic. Similarly, the entire Ganga Plain, home of 449 million people, 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 geologic conditions and sedimentary deposits.

The extreme severity of the exposure in Semria Ojha Patti is typical of index villages. This preliminary study has the obvious deficits of a volunteer study population lacking full demographic representation. We were able to include relatively few women, and we missed many of the men who work 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 consumed drinking water with high concentrations of arsenic (mean, 475 µg/L; median, 431 µg/L; range, 202–1654 µg/L). The World Health Organization–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 adult group and a surprising 6.3% of children supports severe exposure beginning with the transition to tube wells. The comparably high concentrations of arsenic in urine, hair, and nails of the subjects (Table 4) are consistent with studies from West Bengal and Bangladesh (Biswas et al. 1998; Chowdhury TR et al. 1997; Chowdhury UK et al. 1999, 2000b, 2003; Mandal et al. 1996; Rahman et al. 2001).

The particularly high prevalence of neuropathy in women is consistent with their more continuous exposure, because 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 arsenic concentrations in the drinking water. Although relatively few children had overt neuropathy, they should be tested for neurobehavioral and

Table 6. Arsenic in drinking water and obstetric outcome.

	Arsenic concentration in water		
	463–1,025 µg/L ($n = 5$)	174–459 µg/L ($n = 4$)	7–39 µg/L ($n = 7$)
Skin lesions	Positive	—	—
No. of pregnancies	24	14	26
Spontaneous abortion (%)	12.5	21.3	—
Stillbirth (%)	12.5	7.1	8
Preterm birth (%)	25	7.1	8
Low birth weight (%)	20.5	7.1	—
Neonatal death (%)	4.1	7.1	—
Congenital anomaly (%)	4.1	7.1	—

Table 7. Characteristics of three women suffering from chronic arsenic toxicity with obstetric outcome.

Case No.	Age (years)	Melanosis						Keratosis				No. of pregnancies	Previous pregnancies	Arsenic concentration	
		Palm		Trunk				Palm		Sole				Water (µg/L)	Hair (µg/kg)
		S	D	S	D	LEU	WB	S	D	S	D				
1	21	–	+++	++	++	–	++	++	++	++	+	4	1st pregnancy, FTND 2nd pregnancy, stillbirth 3rd pregnancy, PT 4th pregnancy, FTND	1,025	9,764
2	28	–	++	++	++	–	+	+	+	+	+	6	1st pregnancy, FTND 2nd pregnancy, SA at 4 months 3rd pregnancy, SA at 3 months 4th pregnancy, stillbirth 5th pregnancy, low BW 6th pregnancy, FTND	1,025	4,497
3	25	–	++	++	+++	–	++	++	++	++	++	6	1st pregnancy, PT 2nd pregnancy, PT 3rd pregnancy, PT 4th pregnancy, SA 5th pregnancy, Neo death 6th pregnancy, PT	1,025	6,203

Abbreviations: +, mild; ++, moderate; +++, severe; –, not detected; BW, birth weight; D, diffuse; FTND, full-term normal delivery; LEU, leuco-malanosis; Neo, neonatal; PT, preterm birth; S, spotted; SA, spontaneous abortion; WB, whole body.

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, which suggest reproductive toxicity at exposures sufficient to cause maternal toxicity, are highly provocative and consistent with the limited human data. Rudnai and Gulyas (1998) reported an increase in spontaneous abortions, stillbirths, and perinatal mortality in Karcag, Hungary, due to arsenic in drinking water. Hopenhayn-Rich et al. (1998) reported high perinatal and neonatal mortality in the mining area of northern Chile in association with arsenic-contaminated water. In Bangladesh, Ahmad et al. (2001) reported a significant increase in spontaneous abortions, stillbirths, and preterm births. Increased arsenic in cord blood and the placenta was reported in Argentine women who drank water containing 200 µg/L arsenic (Concha et al. 1998). Studies implicating arsenic as a teratogen as well as a reproductive toxin are still inconclusive (Golub et al. 1998).

Conclusions

Groundwater arsenic contamination in West Bengal, India, surfaced during 1983 and that in 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 6–8 February 1995 and the International Conference on Arsenic Pollution of Groundwater held in Dhaka, Bangladesh, 8–12 February 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. In 1984, only one village in West Bengal was known to be contaminated with arsenic; the present count is more than 3,000 villages. For Bangladesh, three villages in two districts were identified in 1995, and at present it is more than 2,000 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 and Bangladesh governments, repectively, were first informed of the arsenic contamination, it was considered a sporadic, easily remedied matter; few people realized the magnitude of the problem (Chakraborti et al. 2002). Even international aid agencies working in the subcontinent 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. The arsenic in Bihar may not be a localized contamination. The magnitude of the problem should be assessed; we do not want to repeat our earlier mistakes.

Figure 8. Three women exposed to arsenic-contaminated water (1,025 µg/L) and studied for pregnancy outcome (Table 7).

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