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

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

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18

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**

60 The pandemic of arsenic poisoning due to contaminated groundwater in West Bengal,
61 India and all of Bangladesh has been thought limited to the Ganges Delta (the Lower Ganga
62 Plain) despite early survey reports of arsenic contamination in groundwater in the Union
63 Territory of Chandigarh and its surroundings in the northwestern Upper Ganga Plain and
64 recent findings in the Terai area of Nepal. Anecdotal reports of arsenical skin lesions in
65 villagers led us to evaluate arsenic exposure and sequelae in the Semria Ojha Patti village in
66 the Middle Ganga Plain, Bihar, where tube wells replaced dug wells about 20 years ago.
67 Analyses of the arsenic content of 206 tube wells (95% of the total) showed 56.8% to exceed
68 arsenic concentrations of 50 µg/L with 19.9% >300 µg/L, the concentration predicting overt
69 arsenical skin lesions. On medical examination of a self-selected sample of 550 (390 adults;
70 160 children), 13% of the adults and 6.3% of the children had typical skin lesions, an
71 unusually high involvement for children, except in extreme exposures combined with
72 malnutrition. The urine, hair, and nail concentrations of arsenic correlated significantly
73 ($r=0.72-0.77$) with drinking water arsenic concentrations up to 1654 µg/L. On neurological
74 examination, arsenic-typical neuropathy was diagnosed in 63% of the adults, a prevalence
75 previously seen only in severe, subacute exposures. We also observed an apparent increase
76 in fetal loss and premature delivery in the women with the highest drinking water arsenic.
77 The possibility of contaminated groundwater at other sites in the Middle and Upper Ganga
78 plain merits investigation.

79

79 INTRODUCTION

80 Groundwater arsenic contamination in Lower Ganga Plain of West Bengal, India was
81 first identified (Saha 1983) in July 1983. Garai et al. (1984) reported 16 patients in 3 families
82 from one village of 24 Parganas district. Saha (1984) further reported 127 patients with
83 arsenical skin lesions from 25 families of 5 villages in 3 districts. Over the last 15 years, as of
84 July 2002, we have analyzed >125,000 water samples, >30,000 urine/hair/nail/skin scale
85 samples, screened ~ 100,000 people in West Bengal for arsenical skin lesions and have
86 registered 8500 people with arsenical skin lesions from 255 affected villages out of 306
87 screened. We have identified tube wells with arsenic concentrations ≥ 50 $\mu\text{g/L}$ in over 3000
88 villages. Our overall study indicates that more than 6 million people from 9 affected districts
89 (population ~ 50 million) of 18 total districts (total population ~ 80 million) are drinking
90 water containing ≥ 50 $\mu\text{g/L}$ arsenic and >300,000 people may have visible arsenical skin
91 lesions (Chakraborti et al. 2002). The arsenic content of the biologic samples indicates that
92 many more may be subclinically affected. In 1995, we identified 3 villages in 2 districts of
93 the Padma–Meghna–Bramhaputra (PMB) delta of Bangladesh (Post Conference Report
94 1995), where groundwater contained ≥ 50 $\mu\text{g/L}$ arsenic. The present situation is that in 2000
95 villages, in 50 of the total 64 districts of Bangladesh, groundwater contains arsenic ≥ 50 $\mu\text{g/L}$;
96 and the British Geological Survey (BGS) estimates that more than 35 million people are
97 drinking water containing arsenic ≥ 50 $\mu\text{g/L}$ (BGS Technical Report 2001). In the combined
98 areas of West Bengal and Bangladesh around 150 million people are at risk from
99 groundwater arsenic contamination (Rahman et al. 2001). Despite years of research in West
100 Bengal and Bangladesh, additional affected villages are identified by virtually every new
101 survey. We feel our present research may be only the tip of the iceberg representing the full
102 extent of arsenic contamination.

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

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

115 The Upper, Middle, and Lower Ganga Plains (Figure 1) are the most thickly
116 populated areas of India. The fertile land and surplus in food production of the Gangetic
117 Plain feeds India. The primary states of the Upper and Middle Ganga Plains are Uttar Pradesh
118 (238,000 sq. km area; 166 million population) in the Upper Plain, and Bihar (94,163 sq. km
119 area; 83 million population) in the Middle Ganga Plain and partly in the Upper.

120 Our studies since 1988 have centered on the severe arsenic contamination of
121 groundwater in the Lower Ganga Plain of West Bengal and Bangladesh. We recently found
122 severe groundwater arsenic contamination in the Bhojpur district, Bihar, which is in the
123 Middle Ganga Plain. In 1976 there was a preliminary report of groundwater arsenic
124 contamination from the Union Territory of Chandigarh and its surroundings (Datta 1976a,
125 1976b) in the northwestern Upper Ganga Plain. A recent report (Tandukar et al. 2001) shows
126 groundwater in the Lower Plain area (Terai) of Nepal to be arsenic contaminated. The data
127 from the Terai area together with our findings in the Bhojpur district of Bihar, about 200 km

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

131 The present communication describes the groundwater arsenic contamination and an
132 initial evaluation of the prevalence of arsenic toxicity in Semria Ojha Patti village in the
133 Middle Ganga Plain of Bihar. The arsenical dermatosis, arsenical neuropathy, and arsenic
134 toxicity among children are quite similar to that observed in West Bengal and Bangladesh
135 (Biswas et al. 1998; Chowdhury et al. 1999, 2000a, 2000b; Mandal et al. 1996; Rahman et al.
136 2001; Roy Chowdhury et al. 1997). Our preliminary observations of an unusual reproductive
137 toxicity indicate a particularly severe exposure.

138 **METHODS**

139 **Location**

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

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

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

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

165 **Subjects**

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

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

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

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

180 **Arsenic Analysis**

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

190 **Iron Analysis**

191 1, 10-phenanthroline method with UV-visible spectrophotometer was used for iron
192 analysis of water samples (Fries and Getrost 1975).

193 **RESULTS**

194 **Groundwater Arsenic Contamination in Semria Ojha Patti Village**

195 The 206 water samples from Semria Ojha Patti represented 95% of the total tube
196 wells of the village. We also analyzed 118 water samples from 5 villages within 3 km of
197 Semria Ojha Patti (Figure 1) but none of the inhabitants were subjects. Figure 2 shows the
198 relatively greater prevalence of highly contaminated hand tube wells compared to the arsenic
199 contaminated areas of West Bengal and Bangladesh. The distribution indicates that, of the
200 5000 residents of Semria Ojha Patti, 18.4% used safe water ($<10 \mu\text{g/L}$), 24.7% between 10
201 and $50 \mu\text{g/L}$, 56.8% $\geq 50 \mu\text{g/L}$, and 19.9% $\geq 300 \mu\text{g/L}$ of arsenic. Our experience in West

202 Bengal and Bangladesh indicates the probability of skin lesions in a subject drinking water
203 contaminated with ≥ 300 $\mu\text{g/L}$ of arsenic. A comparative water analysis data for arsenic
204 presented in Table 1 shows one village from West Bengal, India and one from Bangladesh,
205 which are highly arsenic contaminated with Semria Ojha Patti village of Bihar. Table 1
206 shows that arsenic contamination of groundwater in Semria Ojha Patti village is comparable
207 with highly arsenic contaminated villages of West Bengal and Bangladesh. The
208 recommended value of arsenic in drinking water in India and Bangladesh is 50 $\mu\text{g/L}$.

209 **Iron Concentration in Tube Well Water**

210 Samples from 225 tube wells were analyzed for iron from Semria Ojha Patti and the
211 surrounding 5 villages. The result (mean 2482 $\mu\text{g/L}$, minimum 145 $\mu\text{g/L}$, and maximum 8624
212 $\mu\text{g/L}$) shows the iron concentrations to be higher than previously reported (0–700 $\mu\text{g/L}$) for
213 the Middle Plain (Acharya et al. 1999). The correlation between concentrations of iron and
214 arsenic in water is poor ($r=0.478$).

215 **Clinical Observations**

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

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

229 Of the 51 urine samples analyzed, 98% have arsenic concentrations above the normal
230 excretion level of arsenic in urine (Farmer and Johnson 1990), with 47% >500 $\mu\text{g/L}$, 33.3%
231 >1000 $\mu\text{g/L}$, and 5.9% >3000 $\mu\text{g/L}$. The comparison of the urine arsenic of Semria Ojha Patti
232 village with that of two highly arsenic contaminated villages described in our earlier work
233 (Chowdhury et al. 2001) and cited in Table 1 shows a higher burden for Semria Ojha Patti
234 village, Bihar: $n=51$, mean 798 $\mu\text{g/L}$, median 387 $\mu\text{g/L}$, range 24–3696 $\mu\text{g/L}$, than for
235 Fakirpara village, West Bengal: $n=325$, mean 528 $\mu\text{g/L}$, median 318 $\mu\text{g/L}$, range 7–2911
236 $\mu\text{g/L}$, or Samta village, Bangladesh: $n=300$, mean 538 $\mu\text{g/L}$, median 289 $\mu\text{g/L}$, range 24–
237 3085 $\mu\text{g/L}$). The urine arsenic of control populations (Chowdhury et al. 2003) with drinking
238 water arsenic <3 $\mu\text{g/L}$ was low in West Bengal ($n=75$, mean 16, median 15, range 10–41) and
239 Bangladesh ($n=62$, mean 31, range 6–94, median 29). Village adults drink an estimated 4
240 liters of water per day and children 2 liters. Contaminated water is utilized for food
241 preparation. In West Bengal, we attributed (Chowdhury et al. 2001) about 20–30% of the
242 arsenic body burden to rice and vegetables grown in paddies irrigated by contaminated water;
243 agricultural practices appeared similar in this village.

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

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

255 In the southern area of Semria Ojha Patti we identified a group of children (n=8) with
256 skin involvement. All were drinking water from the same tube well, arsenic concentration
257 749 µg/L. Table 2 lists their dermatological features and the concentrations of arsenic in their
258 urine (inorganic arsenic and its metabolites), hair, and nails. The biological samples from
259 village children with skin lesions are compared with those of children with arsenical skin
260 lesions from the reference villages cited in Table 1. It is found that the Semria Ojha Patti
261 village children have higher concentrations of arsenic in their biological samples compared to
262 the Samta village, Bangladesh (Biswas et al. 1998) and Fakirpara village, West Bengal
263 (Mandal et al. 1998). The arsenic concentrations at all 3 sites exceed those of control
264 populations reported in our earlier work (Chowdhury et al. 2003).

265 **NEUROLOGICAL INVOLVEMENT IN PATIENTS OF ARSENICOSIS**

266 The obvious frequency of disabling neurologic signs initiated a more detailed
267 examination and comparison with neuropathy found in arsenic affected areas of West Bengal
268 (Chakraborti et al. 1999a; Chowdhury et al. 2000a, 2000b; Rahman et al. 2001). Of the 60
269 index subjects with skin lesions, a convenience sample of 40 (32 adults: 20 M, 12 F; 8
270 children 8–15 years: 5 M, 3 F) underwent a detailed neurological examination by the same
271 neurologist (SCM) of earlier studies (Chakraborti et al. 1999a; Chowdhury et al. 2000a;
272 2000b; Rahman et al. 2001). Observations were recorded for items considered consistent with
273 peripheral motor and sensory neuropathy and for other neurologic observations [as modified
274 from Feldman et al. (1979), Galer (1998), and Kreiss et al. (1983)]. Items included to
275 characterize neuropathy were (i) pain and paraesthesias (e.g. burning) in a stocking and glove

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

280 **Neurologic Findings**

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

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

299 **Frequency of Neuropathy**

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

304 **Type and Severity of Neuropathy**

305 Table 4 lists 18 cases (45%) of sensory neuropathy while 3 cases (7.5%) had motor
306 components as well (sensorimotor type). Moderate neuropathy was evident in 4 (10%). This
307 was based on rigorous criteria of neuropathy (Kreiss et al. 1983) and included cases with
308 impairment of at least 2 sensory modalities and reduced deep tendon reflexes. The remaining
309 17 cases (42.5%) had mild (predominantly sensory) neuropathy.

310 **Magnitude of Neurological Involvement and Comparative Analysis**

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

318 **Relationship of Neuropathy and Arsenic Consumption**

319 The 4 patients with moderate and sensorimotor neuropathy utilized water with arsenic
320 750 µg/L and above; the 13 patients with mild and predominantly sensory neuropathy
321 consumed water with arsenic 207 µg/L to 637 µg/L.

322 **ARSENIC IN DRINKING WATER AND OBSTETRIC OUTCOME**

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

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

333 **DISCUSSION**

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

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

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

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

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

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

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

386 Those suffering from arsenical skin lesions (n=60) in Semria Ojha Patti village were
387 drinking water with high concentrations of arsenic (mean 475 $\mu\text{g/L}$, median 431 $\mu\text{g/L}$, range
388 202–1654 $\mu\text{g/L}$). The World Health Organization (WHO) recommended maximum for
389 arsenic in drinking water is 10 $\mu\text{g/L}$ and the Indian standard is 50 $\mu\text{g/L}$. The finding of skin
390 lesions in 13% of the adults group and a surprising 6.3% of children support severe exposure
391 beginning with the transition to tube wells. The comparably high concentrations of arsenic in
392 urine, hair and nails of the subjects (Table 3) are consistent with studies from West Bengal
393 and Bangladesh (Biswas et al. 1998; Chowdhury et al. 1999, 2000b, 2003; Mandal et al.
394 1996; Rahman et al. 2001; Roy Chowdhury et al. 1997).

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

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

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

414 **CONCLUSION**

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

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

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

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Table 1

Distribution of Tubewell Arsenic Concentrations [ranges ($\mu\text{g/L}$)] in Fakirpara Village of West Bengal-India, Samta Village of Bangladesh and Semria Ojha Patti Village of Bihar-India

Area	Name of the village and district	Total Water samples analyzed	Distribution of total samples in each range of arsenic concentration ($\mu\text{g/L}$)							
			<10	10-50	51-99	100-299	300-499	500-699	700-1000	>1000
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%)	-
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%)	-
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%)

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Table 2

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Dermatological Features of a Group of Children and Arsenic Concentration in Their Drinking Water, Urine, Hair, and Nail

Sex & Age	Melanosis				Keratosis				CB (yrs)	CC	Arsenic concentration in water (µg/L)	Arsenic concentration in urine (µg/L)	Arsenic concentration in hair (µg/kg)	Arsenic concentration in nail (µg/kg)		
	Palm		Trunk		Leu	WB	Palm								Sole	
	S	D	S	D			S	D							S	D
615 F/7	-	+	++	++	-	-	-	-	-	-	-	-	749	1248	8471	7923
616 M/6	-	+	+	++	-	-	-	-	-	-	-	+	749	1259	5135	5121
617 F/8	-	+	+	+	-	-	-	-	-	-	-	-	749	1333	3533	-
618 F/9	-	+	+	+	-	-	-	-	-	-	2	-	749	671	2710	-
619 M/11	-	+	+	+	-	-	-	-	-	-	2	+	749	-	-	-
620 M/11	-	+	+	+	-	-	+	-	+	-	4	-	749	2349	5414	-
621 M/9	+	+	+	+	-	-	+	-	+	+	-	+	749	570	1935	2844
622 M/10	+	+	+	+	-	-	-	-	-	-	-	-	749	2020	6833	-

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

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Table 3

Arsenic Concentrations in Biological Samples of Patients and Non-patients in Semria Ojha Patti Village, Bhojpur, Bihar

Parameters	Arsenic in urine ^a (µg/L)	Arsenic in hair ^b (µg/kg)	Arsenic in nail ^c (µg/kg)
No. of samples	51	59	38
Mean	798.6	2773.8	6976.9
Maximum	3696	12404	35790
Minimum	24	257	453
Median	387	1470	36015

^aNormal urine arsenic ranges from 5–40 µg/d (1.5 L) (Farmer et al. 1990)

^bNormal hair arsenic in hair ranges from 80–250 µg/kg with 1000 µg/kg an index of toxicity (Arnold et al. 1990).

^cNormal arsenic content in nails is 430–1080 µg/kg (Ioanid et al. 1961).

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Table 4
Presenting Features, Incidence, Type and Severity of Arsenic-Induced
Peripheral Neuropathy in Semria Ojha Patti Village

	Number of patients	Percentage
Presenting features (n=40)		
Distal paresthasias	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

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Table 5
Arsenic in Drinking Water and Obstetric Outcome

	Group A	Group B	Group C
	(n=5)	(n=4)	(n=7)
Skin lesions	Positive	–	–
Range of arsenic concentration in water (µg/L)	463–1025	174–459	7–39
No. of pregnancies	24	14	26
Spontaneous abortion	12.5%	21.3%	–
Still birth	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%	–

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Table 6

Three Women of Group A Suffering from Chronic Arsenic Toxicity with Obstetric Outcome

680	Case	Age	Melanosis				Leuco	Whole	Keratosis				No. of	Details of previous pregnancies	Arsenic	Arsenic
681	No.		Palm		Trunk			body	Palm		Sole		Pregnancies		in water	in hair
682			S	D	S	D			S	D	S	D		(µg/L)	(µg/kg)	
683																
684																
685	1	21	-	+++	++	++	-	++	++	++	++	+	4	1 st pregnancy – FTND	1025	9764
686														2 nd Pregnancy – Stillbirth		
687														3 rd Pregnancy – Preterm birth		
688														4 th Pregnancy – FTND		
689	2	28	-	++	++	++	-	+	+	+	+	+	6	1 st Pregnancy – FTND	1025	4497
690														2 nd Pregnancy – Spontaneous abortion		
691														– 4 months		
692														3 rd Pregnancy – Spontaneous abortion		
693														– 3 months		
694														4 th Pregnancy – Still birth		
695														5 th Pregnancy – Lowbirth weight		
696														6 th Pregnancy – FTND		
697	3	25	-	++	++	+++	-	++	++	++	++	++	6	1 st Pregnancy – Preterm birth	1025	6203
698														2 nd Pregnancy – Preterm birth		
699														3 rd Pregnancy – Preterm birth		
700														4 th Pregnancy – Spontaneous abortion		
701														5 th Pregnancy – Neonatal death		
702														6 th Pregnancy – Preterm birth		
703																

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 Chandigarh, affected areas of Nepal, affected areas
708 of West Bengal and Bangladesh in Lower Ganga Plain and the study site, Semria Ojha Patti
709 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 Patti 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
713 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
716 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.

722 Figure 6. Correlation between arsenic concentrations in hair and drinking water.

723 Figure 7. Correlation between arsenic concentrations in nails and drinking water.

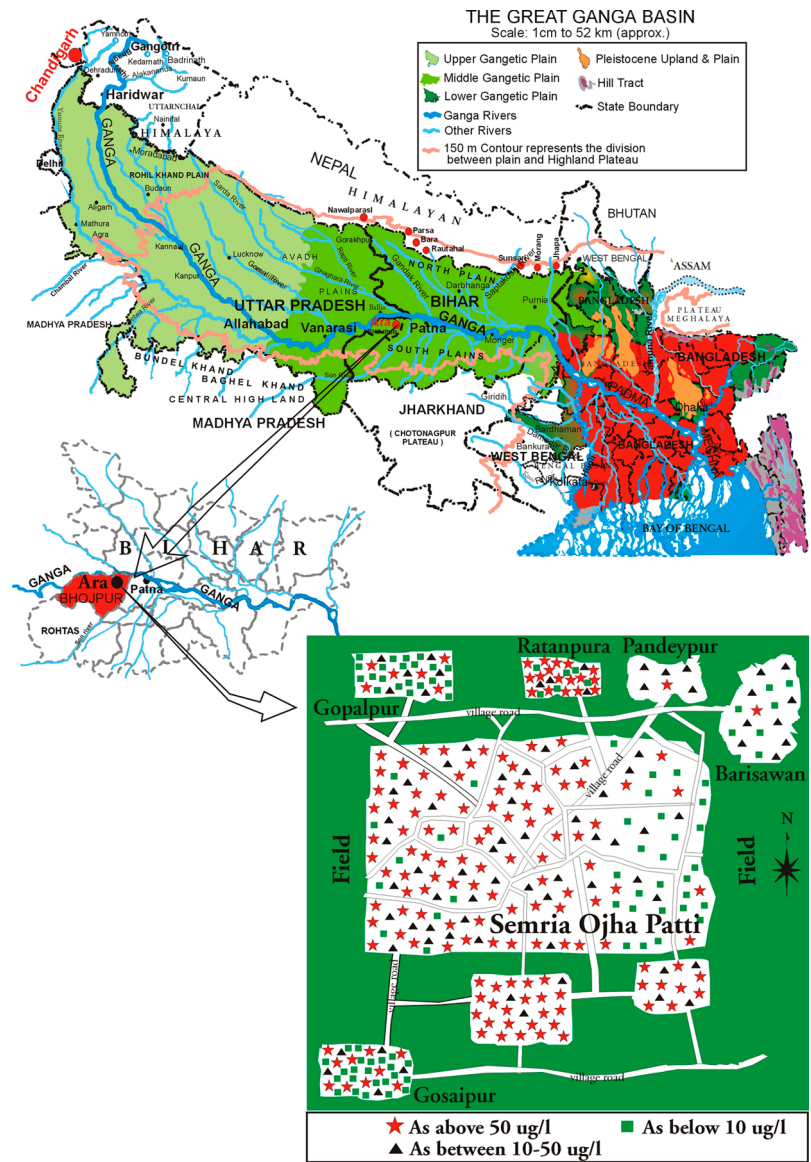


Fig. 1

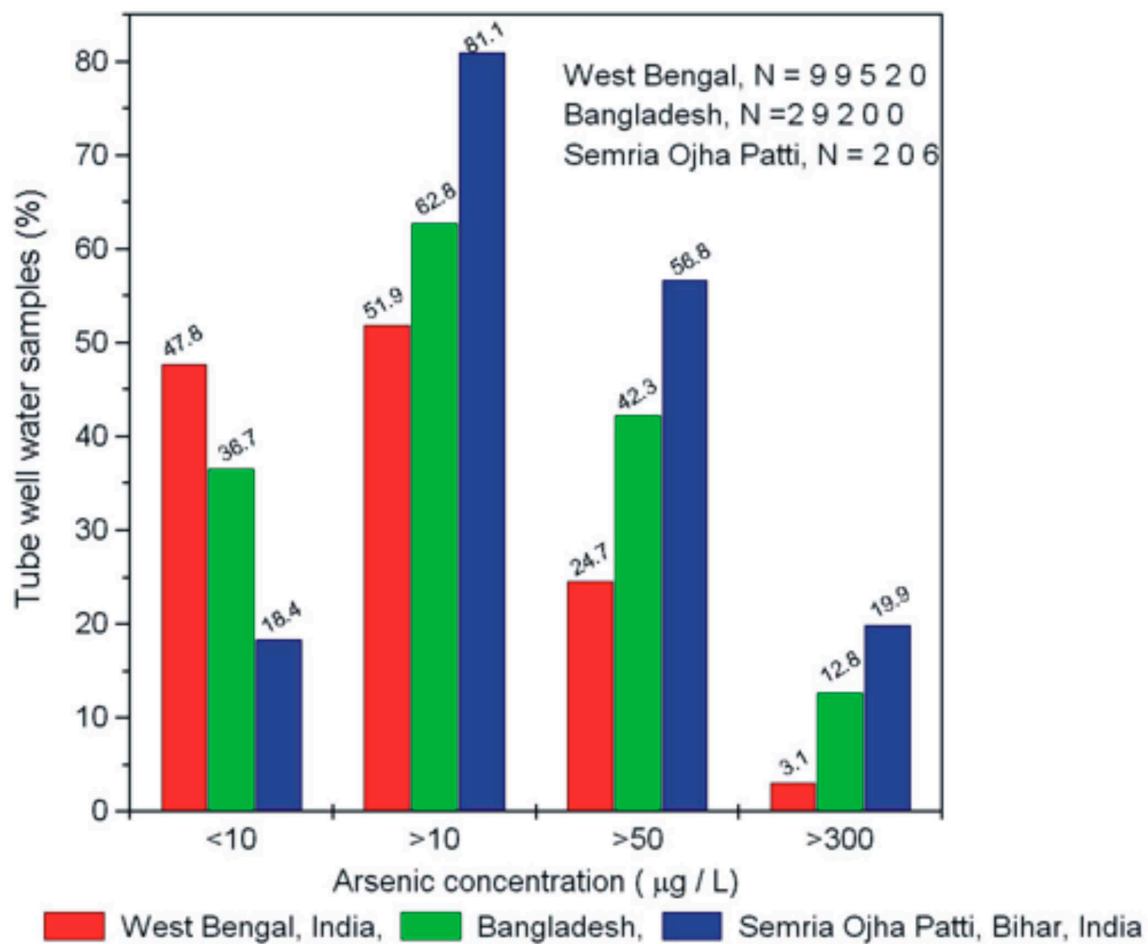


Fig 2



Fig. 3

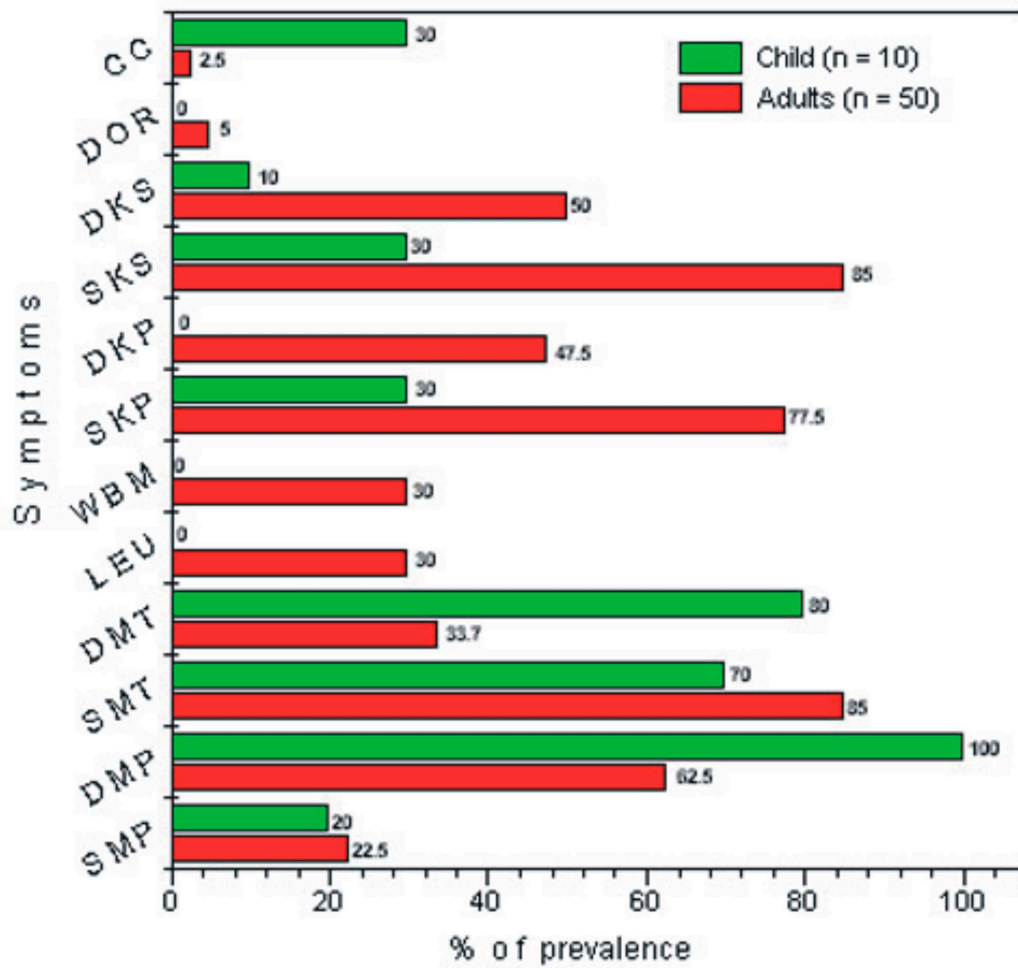


Fig. 4

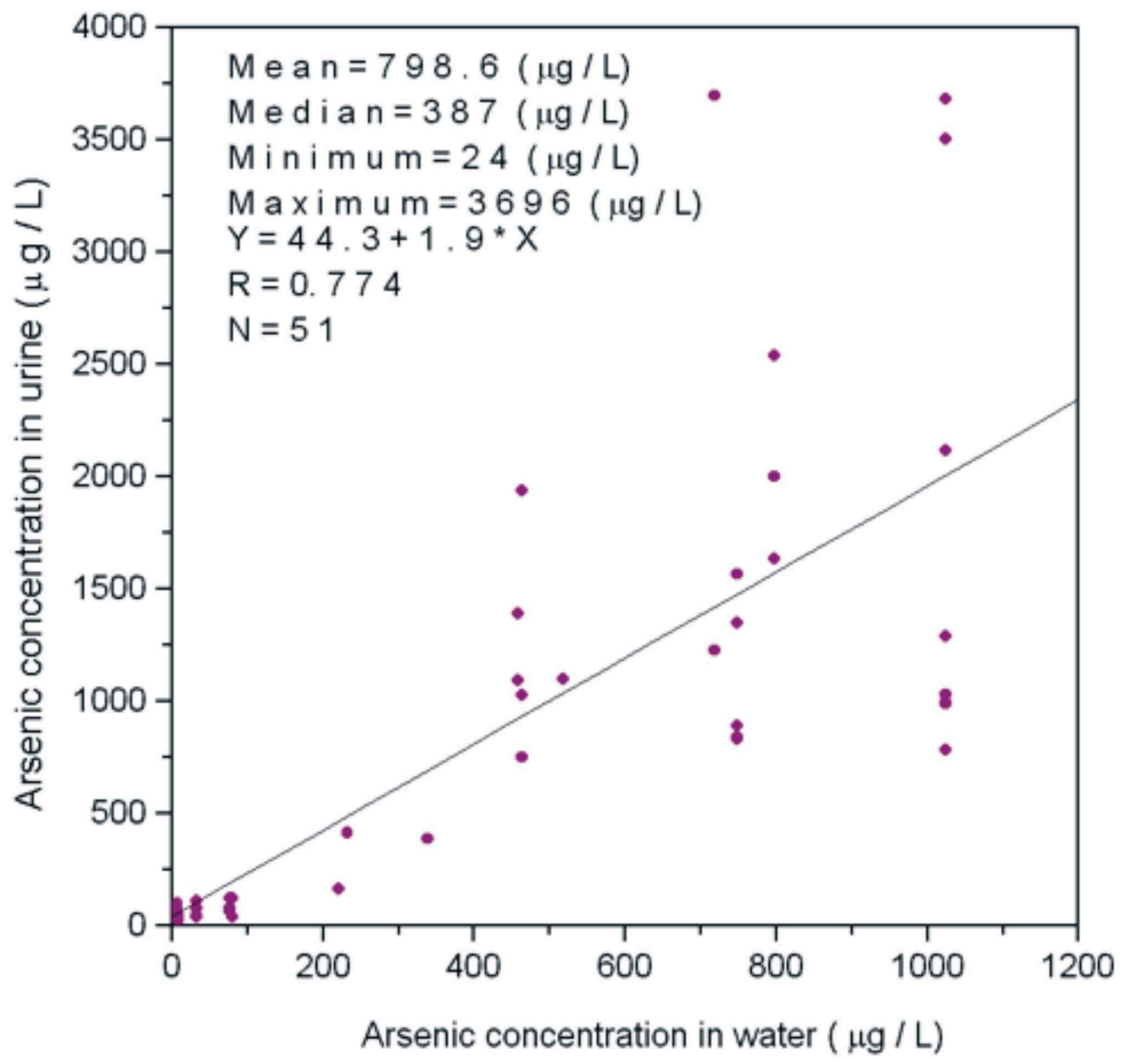


Fig. 5

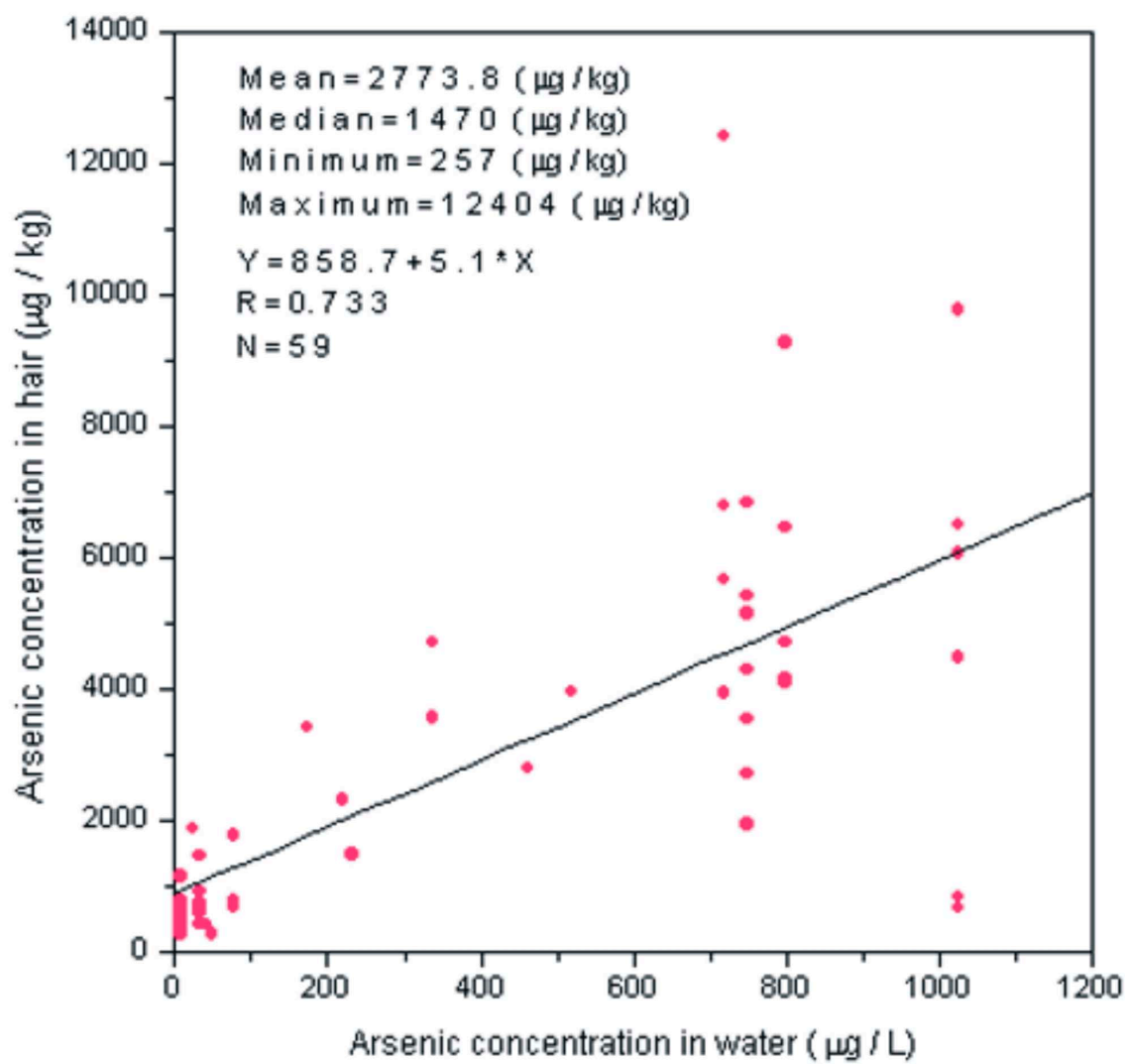


Fig. 6

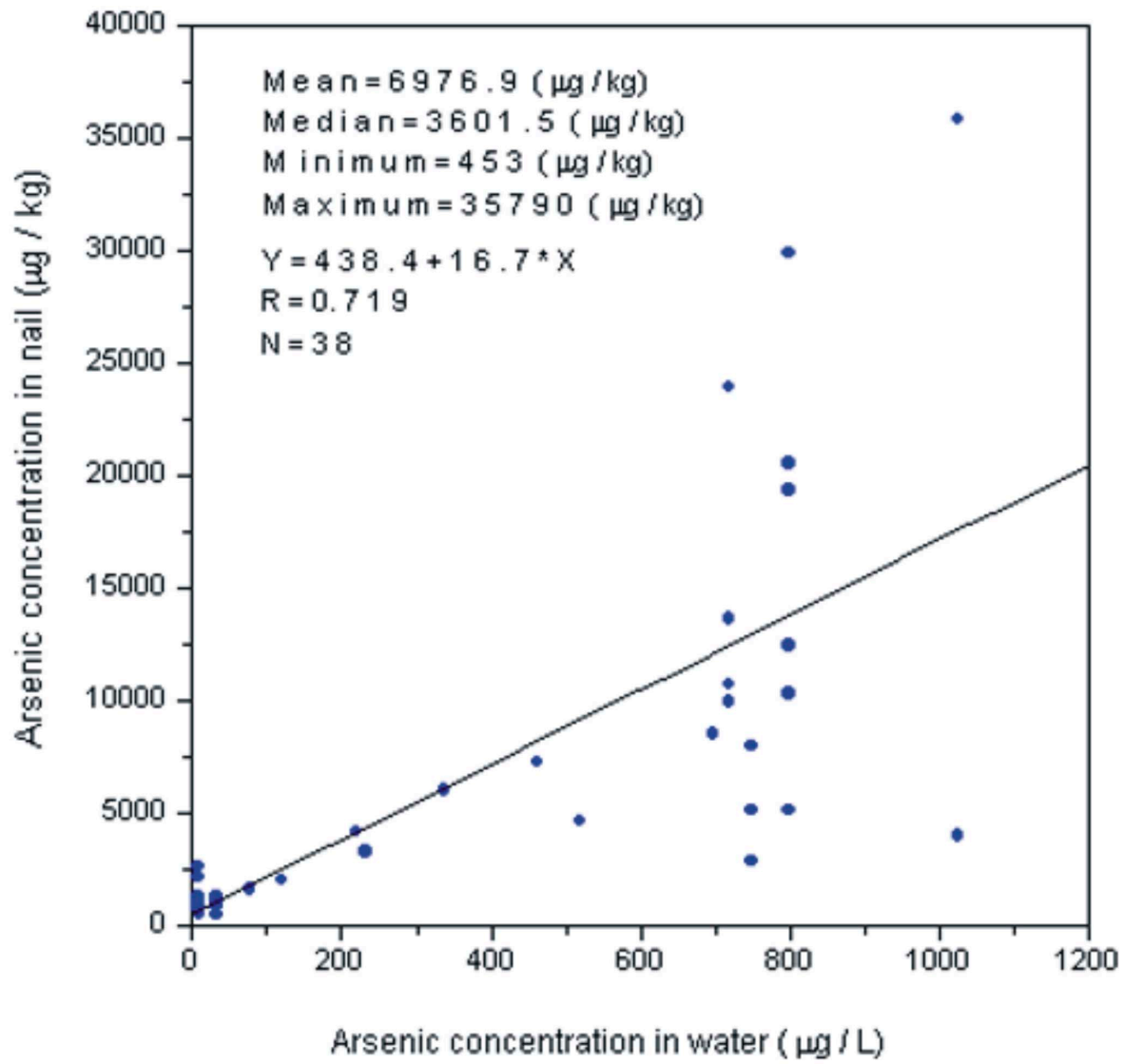


Fig. 7