

CORRESPONDENCE

ARSENIC IN GROUNDWATER, WEST BENGAL

[We reproduce below some more excerpts from the responses received on the above subject — Ed.]

“To understand a problem, is to partially solve it” - Chairman Mao of China

Statement of the problem: It has been reported that hyper-keratosis, hyper-pigmentation and painful skin blisters, caused by the drinking of water with high arsenic content (60 to 3700 $\mu\text{g L}^{-1}$, as against the maximum permissible limit of 50 $\mu\text{g L}^{-1}$ prescribed by WHO), affect about 600,000 people living in an area of about 37,500 km^2 near Calcutta in West Bengal. The note suggests ways and means of mitigating the problem through an understanding of the speciation-based toxicity, biogeochemical cycling, pathways to man, etc of arsenic (Fergusson, 1990).

Historical: Arsenic has been known in India for millenia, as an ingredient in Ayurvedic medicines, and in crime (legends of “*visha kanya*”, and murders). It was believed that intake of arsenic improved complexion.

Possible sources of arsenic: Any model for the source of arsenic has to take into account two key facts: (i) hyper-keratosis and hyper-pigmentation are endemic over a large area and (ii) the incidence of these diseases became more pronounced during the last decade (probably because people started drawing water from deeper bore-wells). The source of arsenic may be anthropogenic or natural. Anthropogenic emissions of arsenic arise from fuel combustion, mining, smelting of ores and the use of arsenic in fertilizers and pesticides. For instance, acidic mine waters could have solute arsenic content as high as 250 mg L^{-1} . Very high As contents (from 9 to 20%) can accumulate from such waters in the form of ferric arsenate and arsenate sulphate precipitates in rapidly growing bacteria made structures (Leblanc *et al.* 1996). These possibilities do exist in West Bengal, and the high arsenic content could be an artifact of the mining and smelting activities during the last couple of centuries. But there is no reason to believe that these emission rates could have been higher in the past than at present. This could imply that sediments at the depths from which drinking water is being presently drawn, could not have acquired its arsenic content from man-made sources. It is, therefore, possible that arsenic content is attributable to natural causes. There are two possibilities. It is known that estuaries act as sinks for heavy metals coming from rivers and atmosphere. The normal range of As in estuarine sediments (5-12 $\mu\text{g g}^{-1}$) cannot cause the kind of arsenic abnormality that we are dealing with in West Bengal. However, there are some instances of very high As content of Restronguet estuarine sediment with 900 $\mu\text{g g}^{-1}$ of As; Thornton *et al.* 1975). Volcanic dust (with its very high content of 55-850 ng m^{-3} of arsenic) is a good candidate (vide Fig.1, Biogeochemical cycling of arsenic; after Fergusson, 1990, p.158), but it remains to be established whether there has indeed been volcanism at the relevant time. Only the evidence of the bore-hole cores (mineralogy, geochemistry, age dating, etc.) can clinch the issue.

Geochemistry: Arsenic has four states of oxidation (+5, +3, 0 and -3). As^{5+} occurs in water at high Eh values and oxygenated systems, while As^{3+} occurs at lower Eh values and mildly reducing conditions. As^0 and As^{3-} are rare in aquatic environments. Fig. 2 (after Fergusson, 1990, p.72) gives the Eh-pH diagram of arsenic in aquatic systems. Arsenic (V) is more strongly absorbed onto sediments, but inter conversion between

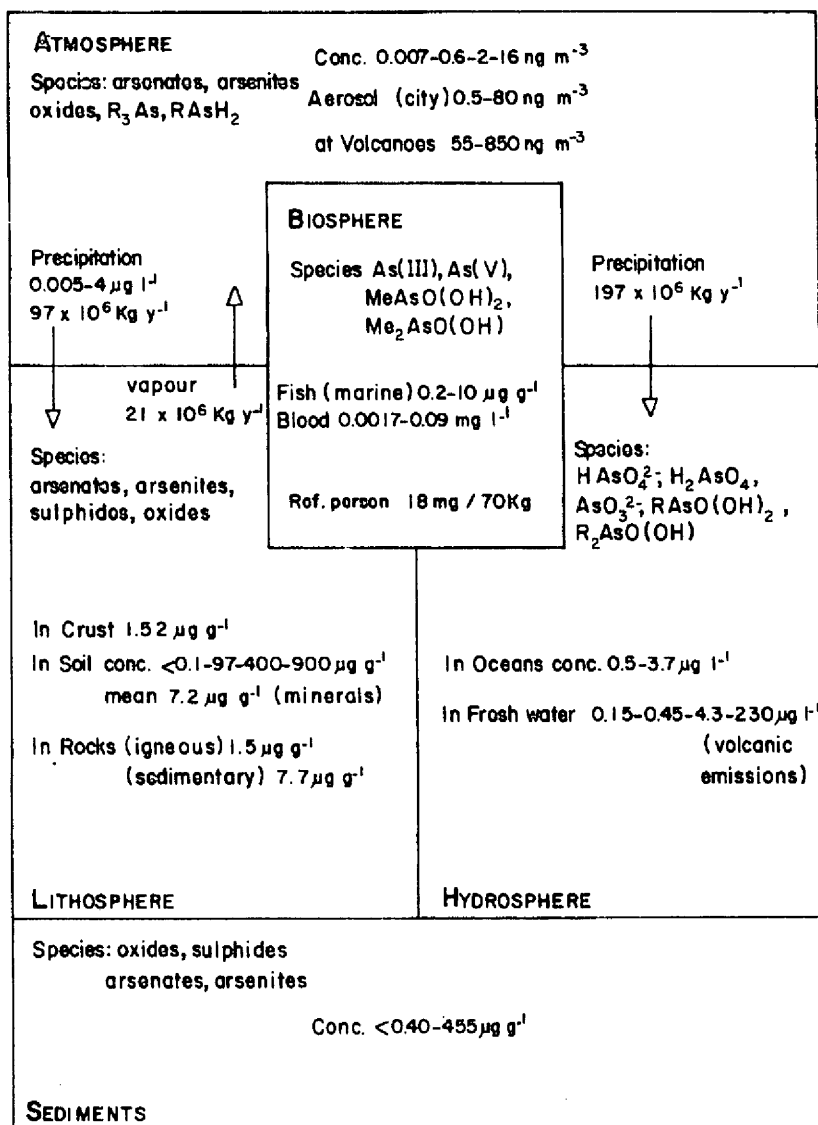


Fig.1. Biogeochemical cycling of arsenic (source: Fergusson, 1990, p.158). The pools, concentrations, fluxes and speciation of As in different spheres are given. The pool is the product of the mass of the material in the pool, and the average concentration of arsenic in the pool. Two levels of concentrations ("natural" and contaminated) of As are given for each pool. Flux is the movement of the material between the pools

between As(V) and As(III) does take place in the sediments, depending upon Eh. High arsenic levels are associated with high iron levels in sediments. As (III) is most mobile of

all As species in water. The absorption of arsenic in human body is high for anionic and soluble species, and low for insoluble species.

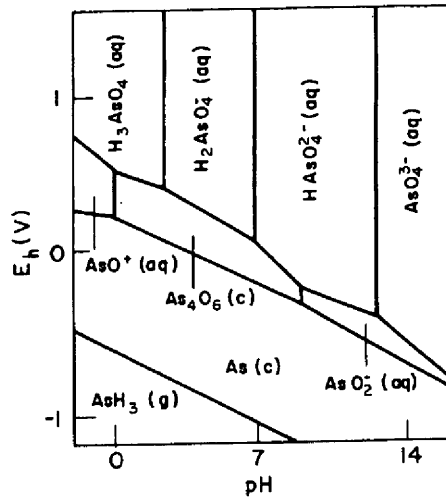


Fig.2. Eh-pH diagram of arsenic in aqueous systems (Source: Fergusson, 1990, p.72)

Arsenic in the form of arsenate, AsO_4^{3-} , is strongly sorbed onto clays, iron and manganese oxides/hydroxides and organic matter. In the acidic soils, arsenic occurs in the form of aluminium and iron arsenates (AlAsO_4 , FeAsO_4), whereas in alkaline and calcareous soils, arsenic occurs as Ca_3AsO_4 . Flooded soils, like the paddy soils in West Bengal, contain arsenite salts with As(III), which is 5-10 times more soluble and hence more mobile than the corresponding arsenates. It is also more toxic. Thus the kind of soil environment that prevails in the affected area, may have accentuated the problem.

Arsenic gets biomethylated (*i.e.* addition of CH_3 to arsenic through biological activity) in the soil-water, sediment-water interfaces through the activity of bacteria (such as, *Escherichia coli*, *Flavobacterium sp*, *Methanobacterium sp*) and fungi (such as, *Aspergillus glaucus*, *Candida humicola*). In the course of biomethylation, arsenic (III) is oxidized to arsenic (V), and CH_3+ is reduced to CH_3- , and stable arsenic oxy-species are formed (Fig.3, Biomethylation processes; after Fergusson, 1990, p.159). Arsenic in the form of organoarsenic compounds has been reported in water, fish, tissues of mammals, *etc.*

Toxicity: The speciation of arsenic determines its toxicity, and its pathways to man. The toxicity of arsenic decreases from As(III) to As(V) and organoarsenic. The gas arsine (AsH_3) is particularly toxic, as its absorption is close to 100% (the toxicity due to arsine inhalation is almost wholly confined to industrial accidents). But the body develops a defence mechanism to counteract the toxic effect – arsenic is alkylated, producing the less toxic alkylarsenic (V) compounds, such as $\text{CH}_3\text{AsO}(\text{OH})_2$ and $(\text{CH}_3)_2\text{AsO}(\text{OH})$, which are then excreted. The strong bond between As(III) and sulphur appears to be responsible for the accumulation of arsenic in hair and nails (the high arsenic content in the hair of Napoleon led the historians to suspect that the British murdered Napoleon by slow arsenic poisoning, when he was under British custody).

Health effects: Arsenic intoxication has a number of health effects depending upon

the route of intake. Inhalation leads to irritation in nose and throat and neurological consequences. Oral intake leads to intense abdominal pains, vomiting and coma. Arsenic ingestion can cause a variety of health effects, such as, cirrhosis of liver, motor sensory paralysis, spontaneous abortions, *etc.*

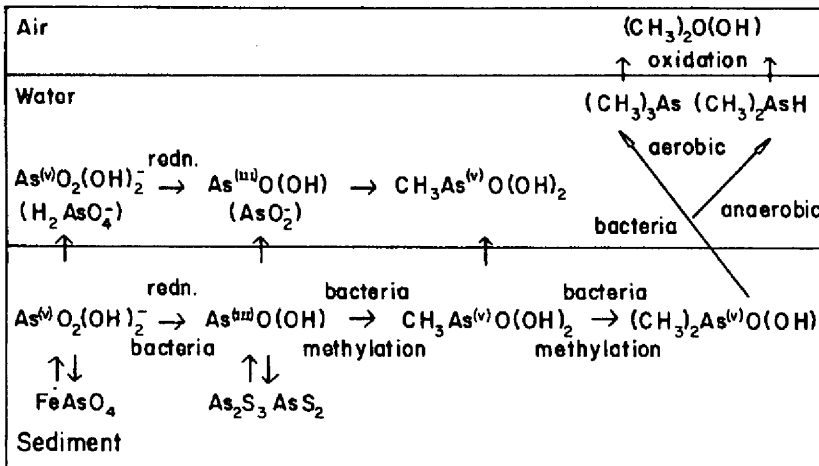


Fig.3. Biomethylation in the sediment-water interface, leading to the production of organoarsenic compounds (*source: Fergusson, 1990, p.159*)

Arsenic affects the skin; it causes hyperpigmentation in areas not exposed to the sun, and hyperkeratosis on the palms of hands or soles of feet. Prolonged exposure leads to the black foot disease (peripheral arteriosclerosis and gangrene) (the author recollects that *India Today* carried colour pictures of all these symptoms). Studies made in Taiwan and Chile (Nriagu, 1988) indicate that drinking for a long time of water containing more than $200 \mu g L^{-1}$ of arsenic, will cause skin cancer. Since the arsenic content of water in the affected area exceeds this threshold value in some instances, there is a real danger of skin cancer developing on a large scale in those areas.

Suggested methods of amelioration:

(i) *Public Health measures:* It is almost impossible to find immediately alternate sources of safe drinking water for 600,000 people at risk from arsenic intoxication. Some simple steps may, however, be taken to ease the situation. A survey may be made of the arsenic content of water from the tube-wells, and a colour coding may be provided for each well:

- < $50 \mu g L^{-1}$: Green (safe): Use it principally for drinking;
- $50 - 200 \mu g L^{-1}$: Yellow (unsafe, but may have to be presently tolerated),
- $200 \mu g L^{-1}$: Red: (hazardous, drinking to be avoided at all costs).

In the case of yellow and red coded wells, it is necessary to know the form of occurrence of arsenic. A particular level of arsenic content, where arsenic is in the form of organoarsenic is less hazardous than if the same arsenic is in the form of As(III).

(ii) *Geological:* As recommended by Dr. B.P. Radhakrishna (JGSI, v.48, p.227,

August 1996), it is necessary to drill an experimental bore hole, and subject the cores to detailed chemical analysis, in order to trace the sources of arsenic in the subsurface. If the arsenic content associated with different aquifers is known, an attempt can be made to seal off the "culprit" aquifers, if that is feasible.

(iii) *Hydrochemical*: An inexpensive, readily available and easy to use, compound has to be developed, either to absorb As(III) or precipitate it or convert it into a form (organoarsenic ?) which is readily excretable (on the analogy of defluoridating highly fluoruous water through the use of dead-burnt magnesite or charcoal made with coconut husk). Microbial remediation holds great potential. For instance, it has been found that bacterial cultures of *Thiobacillus acidophilus* precipitated in a month 75% of the initial solute As in acid mine waters (Leblanc *et al.* 1996). It should be possible to identify or develop through biotechnology, microbes capable of removing As from drinking water in West Bengal. When developed, such a microbial remediation would come as a boon to communities (say, in Bihar) which are faced with drinking water shortages in areas with lots of acid mine water.

(iv) *Treatment*: On the analogy of what has been done to remove lead from the human system (when injected, the reagent chelates lead in the extracellular fluid, and gets lead excreted through urine), it is necessary to identify a similar reagent to cure the victims of arsenic toxicity.

(v) *International cooperation*: Kenneth G. Brown Inc., P.O. Box 16608, Chapel Hill, NC 27516-6608, USA (Tf: +1- 919-408 8067; Fax: +1-919-408 8138; e-mail: <kbinc@mindspring.com>) is engaged in the study of the health effects arising from arsenic in drinking water in several countries, notably, Taiwan, Inner Mongolia, Chile (Antofagasta), Ghana (Obuasi), and Argentina (Corodoba). They welcome cooperation with individuals or institutions who are engaged in the study of the etiology and epidemiology of hyperkeratosis in West Bengal. Such a cooperation would be mutually beneficial. Mitigation measures which succeeded elsewhere could be evaluated for their cost-effectiveness and social acceptability, and adapted in the context of West Bengal.

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DELVING INTO ANCIENT HISTORY

The tribute to Prof. Sediypu Krishna Bhat by Dr. B.P. Radhakrishna (Editorial, JGSI, v.48, No.4, 1996, pp.373-376) is quite illuminating and aspects of his character like giving