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# Arsenic In Drinking Water: A Public Health Hazard

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Arsenic contamination of drinking water has been reported from Asia and many other parts of the globe. Besides India and Bangladesh, the presence of arsenic in drinking water has been reported from Taiwan, Japan, Latin America, Hungary, Greece, Russia, Inner Mongolia, various parts of the USA and Chile[1,2]. The first and second international conferences held in India and Bangladesh during 1995 and 1997, respectively, on 'Arsenic In Ground Water: Cause, Effect And Remedy', was a revelation of the magnitude of the problem around the globe. Severe and irreversible health problems in six districts of West Bengal and neighbouring Bangladesh have been recently attributed to arsenic in drinking water. The term 'arsenicosis' denoting arsenic poisoning is being used in recent years. It was earlier referred to as 'arsenic toxicology' or 'arsenism'. Some of the steps required to combat arsenicosis are briefly discussed here.

## PERMISSIBLE LIMITS

According to the Bureau of Indian Standards (BIS), the permissible upper limit of arsenic in drinking water should not exceed 0.05 mg/l (50 μg/litre). The WHO guidelines have been revised during the recent past and the permissible limits have been reduced from 0.05 mg/l to 0.02 mg/l (20 μg/litre) due to adverse health reports arising from different parts of the world where arsenic is causing severe health problems. The Canadian Regulatory Agency has reduced the permissible limit of arsenic to 25 μg/litre. The United States Environmental Protection Agency is reviewing the current allowable level for arsenic with a view to lowering it significantly[3]. It is likely that in the course of time, the BIS may also amend the present standards, as there is a necessity to do so.

## CONTAMINATION

Arsenic occurs in the earth's crust along with sulphides and iron pyrites. There are endemic areas in West Bengal in India, and in Inner Mongolia in China, where arsenic and fluoride have been co-existing in the earth's crust[4]. Selenium is considered an antagonist to arsenic[5].

Arsenic may be released into natural water either due to oxidation of iron pyrites/arsenopyrites or due to the reduction of the iron coating on sand grains.

Arsenic is released into the human environment, including drinking water, through coal mining, coal burning, copper smelters and industrial effluents[6,7]. Rivers flowing through the coal fields of Bihar have been reported to carry large amounts of arsenic responsible for arsenic poisoning in downstream areas of West Bengal[8]. The coal fields of Bachara and Piprawar areas of South Bihar have contaminated the waters of the Damodar and its tributary, the Safi, causing problems in West Bengal. According to Nitish Priyadarshi[8], arsenic contamination arises mainly due to the dumping of waste from the coal mines along the river bed.

Arsenic, unlike other chemicals, exists in soluble and insoluble forms; organic and inorganic forms; and tri- and pentavalent forms in which the trivalent arsenite is highly toxic compared to the pentavalent arsenate.

Arsenic toxicity depends upon the oxidation state, the chemical form and the solubility of the chemical. Both organic and inorganic arsenic are found in natural water in the ratio of 1:1.

Pentavalent arsenate species are found mostly in oxygen-rich aerobic environment, whereas the highly toxic trivalent arsenite species are found mostly in anaerobic environments such as ground water.

Arsenic, in view of its potential to participate in various chemical reactions, is commonly used in industries. Industries that use arsenic in their applications include the metallurgical industry; glassware and ceramic industries; dye and pesticide manufacturing industries; petroleum refineries; and rare earth industries.

Arsenic containing chemicals are

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also used in the manufacture of herbicides and pesticides; wood and hide preservation; lead short manufacturing; phosphate detergent; and presoaks used in fertilisers[9]. In West Bengal, a factory manufacturing aceto-copper arsenite (an insecticide) existed and polluted the drinking water in the southern part of Calcutta[10]. But the list is by no means complete as there may be other industries which use arsenic, as a raw material, or cause it to arise as a bi- or an end-product.

## ARSENIC IN PLANTS

Arsenic, a phytotoxin, is a constituent of most plants. Upon absorption, it is translocated to different parts of the plant and high concentrations are found in roots and leaves. Natural arsenic levels in plants seldom exceed 1 $\mu$g/g dry weight[7].

In an exercise to evaluate the arsenic levels in crop plants, 32 vegetable gardens in Cornwall in the UK were surveyed, and it was found that the soil arsenic content ranged from 144-892 $\mu$g/g and arsenic concentration exceeding 1 $\mu$g/g was detected in several vegetables, such as lettuce, onion, beetroot, carrot, peas and beans. It was observed that the vegetable arsenic content increased with the increase in soil arsenic content[7].

## ARSENIC IN MARINE FOODS

Foods of marine origin are very rich in arsenic. Many species of bony fish contain 2 to 8 ppm, oysters 3 to 10 ppm and mussels as high as 120 ppm of arsenic. Chemical analyses of coastal waters along the UK and the USA have revealed high concentrations of arsenic in marine animals; 174.0 ppm in prawns in coastal waters of the UK and 42.0 ppm in shrimps of coastal waters of the USA. However, there is no information available on marine edible animals in the Bay of Bengal or the Arabian Sea[5].

Normal cow's milk has arsenic in the range of 0.03 to 0.06 ppm. However, in cows grazing on grass contaminated with arsenic in New Zealand, arsenic levels as high as 1.5 ppm/unit have been reported in milk. It has also been reported that milk of lactating women undergoing treatment with arsenic-containing drugs for syphillis had high levels of arsenic in breast milk[11].

In earlier years, arsenic was used to treat a variety of diseases, such as syphillis, 'liver spots', amoebiasis, and in dentistry.

## ACTION ON BIOLOGICAL SYSTEM

- Trivalent toxic arsenite is known to inhibit enzymes by reacting with biological ligands containing sulphydryl (SH) groups. Pyruvic dehydrogenase is highly sensitive to arsenic because of its interaction with two 'SH' groups of lipoic acid. Arsenic inhibits pyruvate dehydrogenase, leading to pyruvate accumulation[11].

- Arsenic readily crosses the placental barrier and foetal damage has been reported. Arsenic content in human umbilical cord blood has been found to be the same as in the maternal blood.

- Small doses of inorganic arsenic induce mild vasodilatation and result in slight oedema. This had been misinterpreted as weight gain and was recommended as 'tonic' in earlier times.

Arsenic, when absorbed into the biological system, can undergo biotransformation under *in vivo* conditions. Less toxic pentavalent arsenate could be reduced to the highly toxic trivalent arsenite. The half-life of this bio-transformed highly toxic arsenite is three to five days, when it can cause harmful effects to the system. The lethal dose of arsenic is 1-4 mg/kg body weight[12].

It has been reported that arsenic levels in skin (0.12 ppm), nail (0.36 ppm) and hair (0.65 ppm) are relatively higher because of its affinity for SH groups. Blood arsenic level ranges from 0.01 to 0.92 ppm/unit[13].

The estimation of arsenic content in hair and nail is considered useful for diagnostic purposes and the early detection of arsenic poisoning. It is also known that the hair of males has a higher arsenic content as compared to the hair of females (0.62 ppm/unit in males and 0.37 ppm/unit in females). Historical documents reveal that Emperor Napoleon's hair, analysed after his death, revealed 10.00 ppm/unit of arsenic. Hair containing arsenic levels greater than 3.0 ppm could denote poisoning; 2-3 ppm calls for further investigation and less than 2.0 should not be dismissed.

The four reliable methods used for arsenic estimation are: Spectrophotometric; Atomic Absorption Spectrometer (AAS); Spectrofluorometric; and Neutron Activation Analysis (NAA).

By using NAA – a highly sophisticated technology which estimates arsenic with high precision – the arsenic content of a single hair can also be determined in as little as 0.3 $\mu$g of hair sample.

## ARSENICOSIS IN WEST BENGAL

The increased arsenic content of drinking water has been causing a major public health issue in six districts of West Bengal, where an estimated 30 million people dependent on tubewell water sources have been adversely affected[14]. Tubewells were popularised in the country in the 1970s. The switching over for drinking water from shallow dug wells, rivers and ponds to tubewells, was mainly to protect the community from cholera, typhoid and other water-borne diseases. However, while there was relief from the above diseases, disastrous consequences due to arsenic poisoning of drinking water followed.

The six districts endemic for arsenicosis are from the eastern sector of West Bengal, extending 450 km from north to south and bordering Bangladesh. The districts severely affected by arsenicosis are Malda, Murshidabad, Nadhia, Bardhaman, 24 Parganas (North) and 24 Parganas (South)[15]. As many as 434 villages in the six districts where the arsenic content in ground water ranges from 10 to 590 $\mu$g/l are known to be affected. The tubewells in this area have been dug to a depth of 12 to 150 feet[16].

Arsenicosis was not known as an endemic problem in West Bengal before the mid-1970s. The reasons for the present aggravation of this problem, particularly in West Bengal, require careful elucidation. The mid-1970s witnessed the large-scale exploitation of ground water resources for irrigation. Further, as a result of the war in Bangladesh in 1971, the migration of 80 to 90 million people from Bangladesh to West Bengal increased the number of settlements in the bordering districts of West Bengal, thereby significantly increasing the demand for water. Adverse health effects caused by the consumption of arsenic-contaminated water were manifested among the population in

a timespan of eight to 10 years[17,18,19].

Arsenical dermatosis was first detected by Saha in 1983, in patients attending the Skin OPD of the School of the Tropical Medicine, Calcutta. The first patient came from village Gangapur, District 24 Parganas (North). A clinical diagnosis was made on the basis of the salient features, that is, melanosis (diffused and spotted) and keratosis (spotted) after excluding genetic and other environmental causes of pigmentation and keratosis. Tubewell water as a vehicle of arsenic was suspected by excluding other causes such as arsenic-containing drugs and factory effluents. Upon testing the water, it was found that the arsenic content exceeded the permissible limits of 50 µg/l. Poisoning was further proved by the high arsenic levels in skin scales, nails and hair. Hepatic damage, found in some patients, was confirmed by liver function tests and biopsy study. Out of the 1,214 patients studied in 36 villages in six districts, six cases had developed squamous cell carcinoma in the skin in the extremities. Mild cases improved on withdrawal of further intake of arsenic contaminated water[20].

Dipankar Chakraborty carried out a survey of six arsenic affected districts in West Bengal in 1994. The survey covered 37 arsenic affected blocks with a total population of 7,456,250. It showed that 200,630 subjects living in 405 villages in these blocks were affected[21]. Arsenic concentration in hair, nail and urine samples of 300 affected individuals investigated by Mr Chakraborty are reported in Table 1.

Poisoning due to drinking water contaminated with arsenic has thus become a serious health issue in West Bengal.

In Bangladesh, arsenicosis is widespread[22]. According to Hiroshi Yokota, a geo-engineering professor in a Japanese university, there are fast flowing underground 'rivers of Arsenic' in Samta village, in the Jessore district of Bangladesh. In this village only five out of 282 wells were safe. 330 people were poisoned with arsenic and were living with amputated legs, gangrenous toes and gruesome melanomas[23]. Arsenical dermatosis and hepatomegaly were seen among 92.5 per cent of the population exposed to arsenic in the concentration of 0.2 to 2.0 mg/l[24].

## EARLY SIGNS

The early manifestations of arsenic poisoning are: muscle weakness and aching; skin pigmentation in eyelid, nipples, chest and axilla; skin oedema; garlic odour of breath and perspiration; excessive salivation and sweating; generalised itching; sore throat; numbness; liver enlargement; and kidney dysfunction.

**TABLE 1**
**Arsenic Content in Hair, Nails and Urine of Patients from West Bengal**

| District | Hair mg/ $(kg^{-1})$* | Nails $(mg/kg^{-1})$** | Urine (total) (µg/ml)*** |
|---|---|---|---|
| 24 Parganas (S) | 1.32 – 28.38 | 1.47 – 38.77 | 0.05 – 0.71 |
| 24 Parganas (N) | 2.16 – 31.05 | 3.12 – 52.03 | 0.14 – 2.0 |
| Nadhia | 2.05 – 10.33 | 6.88 – 26.27 | 0.11 – 1.42 |
| Bardhaman | 1.18 – 7.20 | 1.50 – 33.42 | 0.12 – 0.47 |
| Murshidabad | 1.84 – 16.30 | 1.78 – 29.52 | 0.06 – 1.44 |
| Malda | 1.70 – 13.50 | 7.19 – 32.33 | 0.12 – 1.05 |

*Source: Dipankar Chakraborty,* Arsenic Contamination of Six Districts, West Bengal, 1995. *The background 1995 International Conference on Arsenic in Ground Water Contamination, Calcutta.*
** The normal amount of arsenic in hair is about 0.08 to 0.25 mg $kg^{-1}$ with 1.0 mg $kg^{-1}$ being an indication of the presence of excess arsenic.*
*** The normal arsenic content of nails is 0.43 – 1.08 ppm*
**** Normal excretion levels of arsenic in urine range from 5 to 40 µg per day; the total arsenic amount will depend on the amount of urine per day.*

## REMOVAL OF ARSENIC

The procedures known to remove arsenic are:

- Absorption with co-precipitation using iron and aluminium salts
- Absorption on activated alumina, carbon and bauxite
- Reverse osmosis
- Ion exchange
- Oxidation with potassium permanganate ($KMnO_4$) followed by filtration using manganese green sand and iron oxide-coated sand.

In endemic areas for arsenicosis in India, until such a time as an economically viable system acceptable for community operations emerges, the traditional sources, such as surface water sources after sand filtration and clarification, rainwater harvesting structures are being promoted. The government has also invested substantial sums in bringing water from distant safe sources through pipelines for supply in endemic areas in West Bengal.

- In 1984, two projects for improving the quality of drinking water for a population of 100,000 were completed in a region in China which is endemic for arsenic toxicosis. A follow-up study of the area showed significant improvement in health[25].

## POINTS FOR ATTENTION

- Suspect arsenic contamination of drinking water in endemic areas where excess iron is a contaminant of drinking water. Drinking water should be tested for arsenic besides other chemical parameters in such areas.
- In coal mining areas, drinking water may be contaminated with arsenic, and testing for arsenic is necessary.
- Arsenic testing requires sophisticated technology and instrumentation. On a regional basis, some of the existing water testing laboratories should be equipped and personnel trained to meet with the requirements of arsenic testing.
- Cancer hospitals in the country may consider including testing of hair, nails and blood for arsenic as a

**TABLE 2**
**Arsenic Content in Drinking Water in West Bengal[14]**

| Arsenic content (mg/litre) | Percentage of drinking water sources contaminated with arsenic |
|---|---|
| 0.05 – 0.1 | 20 |
| 0.10 – 0.5 | 60 |
| 0.50 – 1.0 | 15 |
| > 1 | 5 |

routine, irrespective of the type of cancer the patient suffers from.

- It may be appropriate to introduce arsenic testing in biological samples in hospital laboratories located in endemic areas.

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## NUTRITION NEWS

- **Diet, Nutrition and Chronic Disease – An Asian Perspective,** published by Smith Gordon, UK, and edited by P.S. Shetty and C. Gopalan, has been released. Its copies are available at NFI and with Dr P.S. Shetty, London School of Hygiene and Tropical Medicine.

- **Nutrition in Major Metabolic Diseases,** edited by C. Gopalan and Kamala Krishnaswamy, is available at the Oxford University Press, YMCA Library Building, Jai Singh Road, New Delhi 110 001.

# Folic Acid For Prevention Of Neural Tube Defects

S.S. Agarwal

One of the most significant developments in the field of micronutrients and teratology during the last two decades has been the demonstration of the efficacy of periconceptional folic acid supplementation in the prevention of occurrence and recurrence of neural tube defects (NTD)[1,2]. The success has been so remarkable that several countries have either already approved, or are in the process of approving, fortification of food with folic acid (FA) to enable minimal dietary intake of 0.4 mg folic acid per day by all women in the reproductive age group[3]. Incidentally, folic acid supplementation has also been recommended to reduce the risk of coronary artery disease[4].

## NEURAL TUBE DEFECT

The failure of the closure of the neural tube leads to a group of congenital anomalies which are collectively called neural tube defects. These include anencephaly, encephalocele, meningocele and meningomyelocele. The latter two conditions are also referred to as open spina bifida, or spina bifida cystica. In more severe cases, the entire neural canal may remain open, giving rise to craniospinalrachisis. Iniencephaly is also included under the category of NTD. The open spina bifida is often associated with hydrocephalous. However, isolated hydrocephalous without spina bifida is not included under NTD. Although technically closed spina bifida is a defect of neural tube closure, it is generally not considered with other neural tube defects because of differences in its clinical presentation, diagnosis, need for treatment and prognosis. The role of folic acid has been mainly studied in context of open NTDs.

## PREVALENCE

There is significant geo-ethnic variation in the prevalence of NTD worldwide.

One of the highest prevalence has been reported from Northern Ireland and Wales (6.38-10.92 per 1,000 births)[5]. The prevalence of NTD in India too is considerably high but appears to vary in different parts of the country[6,7]. In general, the prevalence from Punjab (Amritsar and Chandigarh) and other places in northern India (Delhi, Rohtak, Jaipur, Lucknow and others) has been reported to be higher than from the eastern (Calcutta). Southern (Pondicherry, Chennai and Mysore) and western (Mumbai) parts of the country. One exception to the above is the report from Davangere in Karnataka where the prevalence was reported to be 11.4/1,000 births[8].

In Lucknow, at a teaching hospital, we have found the prevalence of NTD to be 4.7/1,000 births[9]. This has remained unchanged over a period of 10 years[10]. One reason for the high prevalence of NTD in Punjab could be its high prevalence amongst Sikhs[11,12,13]. And high prevalence of NTD at Devangere has been attributed to high rate of consanguinity in south India[14]. However, its prevalence has not been reported to be high from Chennai, Pondicherry and Mysore. There are several areas in the country from where the prevalence of NTD is not known. For monitoring the impact of any intervention, a network of surveillance centres may need to be established.

## EMBRYOGENESIS OF NEURAL TUBE

Neural tube is one of the first structures to develop during embryogenesis. It starts as a flat neural plate at day 17-19 post-ovulation (since the gestational age is counted from the first day of LMP, it is approximately 15 days more than the post-ovulation age at all points), corresponding to stage number 8 of embryonic development when the embryo is approximately 1.0-1.5 mm in length. The next stage is the formation of neural folds (day 19-21), followed by the fusion of these folds in the midline (day 23-26) giving rise to a neural canal. Subsequently, the anterior (rostral) and posterior (caudal) neuropores of the canal are closed, leading to the formation of the neural tube. This completes by developmental stage 12 when the embryonal length is approximately 5 mm. Failure of fusion of neural folds gives rise to craniospinalrachisis; and that of anterior and posterior neuropores to anencephaly and spina bifida cystica, respectively. Thus, the critical period for neural tube development is day 17-30 post-ovulation. Any exposure to a teratogen at this stage which interferes with the expression or function of genes connected with neural tube development, or a de-novo defect of these genes by itself, could give rise to NTD. This period is also the optimal window for any therapeutic intervention.

According to classical teaching the fusion of neural folds starts in the dorsolumbar area, which then spreads both rostrally and caudally. However, it fails to explain the co-existence of multiple non-contiguous NTDs. Alternatively, it has been proposed that fusion of the neural folds may start at multiple sites simultaneously, and that each site may be differentially sensitive to different teratogens[15,16]. However, this hypothesis needs to be verified.

## AETIOPATHOLOGY OF NTD

Aetiopathologically the NTD is a heterogenous entity. Although it can occur in association with chromosomal anomaly (for example, trisomy 13, 18, 22q-, 2q-), and single gene disorders (for example, Meckel Gruber syndrome, etc) the great majority of isolated NTD cases are sporadic, and are considered to be of polygenic-multifactorial origin. The evidence for the role of genes in the causation of sporadic NTDs is illustrated by the risk of recurrence of this disorder. In first degree relatives, the risk is 3-4 per cent with one affected individual, 5-10 per cent with two affected individuals and higher with more than two affected individuals. The risk is comparatively much less for second and third degree relatives[17,18].

Although the exact genes that predispose to NTD in humans are not known, more than 40 candidate genes have been identified in experimental animals[19].

Amongst the environmental factors, the best known evidence points towards the role of folates in the diet. NTD may also be associated with