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on

STATUS, IMPACTS, SOURCES AND REMEDIES OF ARSENIC CONTAMINATION IN FRESHWATER

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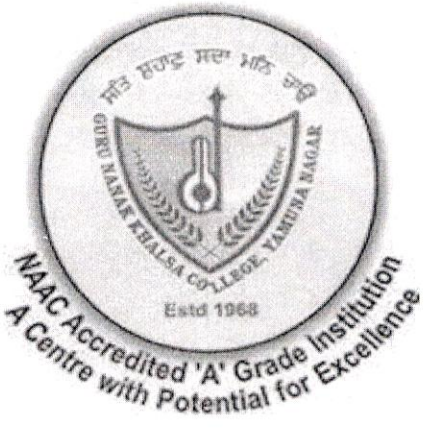

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CERTIFICATE

This is to certify that **Mr. MANJEET, M.Sc. (Environmental Science, 2nd Year; Registration No: - 12GNY974)** of the **Guru Nanak Khalsa College, Yamuna Nagar, Haryana,** has undergone Summer Training under my guidance and supervision from June 30 to August 30, 2016 and submitted his training report on **"STATUS, IMPACTS, SOURCES AND REMEDIES OF ARSENIC CONTAMINATION IN FRESHWATER"**.

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(MANJEET)

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1. ADVERSE IMPACTS OF ARSENIC CONTAMINATION

The symbol for arsenic is As. The atomic number of arsenic is 33, the atomic weight is 75. Arsenic occurs naturally in our environment in inorganic and organic forms. Inorganic arsenic occurs combined with metals in igneous and sedimentary rocks; and also combined with elements like oxygen, chlorine, and sulfur. Organic arsenic occurs combined with carbon and hydrogen. Both organic and inorganic arsenic compounds exist as white or silvery powder materials with no smell or unique taste.

Arsenic has been recognized since ancient times, from around 3000 BC, to be acutely poisonous. When dissolved in water it is colorless with no taste or smell. Anyone who drinks water at 60 milligrams per liter (60 mg/liter) or 60 parts per million (60 ppm) will soon die

Because it targets widely dispersed enzyme reactions, arsenic affects nearly all organ systems. The most sensitive endpoint from arsenic exposure is dermal effects. While several studies may identify effects on other endpoints at the same exposure level that produces dermal effects, the database for dermal effects is stronger than for effects on other endpoints [ATSDR 2007]. Key physiologic effects from arsenic exposure that have been covered in detail later on are:

- Patchy skin hyperpigmentation, small focal keratoses, and other skin lesions are common effects of heavy chronic exposure.
- It is difficult to establish strong associations between arsenic exposure and disease, as the prevalence and spectrum of diseases linked to chronic arsenic exposure differ not only between countries, but even within countries.
- Arsenic can cause lung and skin cancers and may cause other cancers.
- The association between chronic arsenic exposure and cancer is strongest for skin, lung, and bladder cancer. Liver (angiosarcoma), kidney, and other cancers have limited strength of association [IARC 2004; NRC 2000].
- The association between chronic arsenic exposure and cancer is strongest for skin, lung, and bladder cancer. Liver (angiosarcoma), kidney, and other cancers have limited strength of association [IARC 2004; NRC 2000].

1.1 Effects of Arsenic on Human Health

1.1.1 Gastrointestinal and hepatic effects

The gastrointestinal effects (GI) of arsenic are generally the result of ingestion; however, GI effects may also occur after heavy exposure by other routes.

- GI effects are seen acutely after arsenic ingestion, and less often after inhalation or dermal absorption.
- The fundamental GI lesion appears to be increased permeability of the small blood vessels, leading to fluid loss and hypotension.
- Extensive inflammation and necrosis of the mucosa and submucosa of the stomach and intestine may occur and progress to perforation of the gut wall.
- A hemorrhagic gastroenteritis may develop, with bloody diarrhea as a presenting effect.

Acute arsenic toxicity may be associated with hepatic necrosis and elevated levels of liver enzymes.

- Arsenic intoxication may also result in toxic hepatitis with elevated liver enzyme levels.
- Chronic arsenic ingestion may lead to cirrhotic portal hypertension [ATSDR 2007; Datta 1976].
- There is limited strength of association of chronic arsenic exposure and noncirrhotic portal hypertension [IARC 2004; NRC 2000].
- Case reports have also linked chronic high level arsenic exposure with hepatic angiosarcoma, a rare form of liver cancer [Popper et al. 1978; Zaldivar et al. 1981; ATSDR 2007].
- There is limited strength of association, however, between chronic arsenic exposure and angiosarcoma of the liver, as determined by International Agency for Research on Cancer (IARC) and National Research Council [IARC 2004; NRC 2000].

1.1.2 Renal effects

Arsenic is capable of causing renal effects.

- The systemic toxicity occurring in severe acute arsenic poisoning may include acute tubular necrosis with acute renal failure.
- Chronic renal insufficiency from cortical necrosis has also been reported.
- The precipitating cause of renal injury may be hypotensive shock, hemoglobinuric or myoglobinuric tubular injury, or direct effects of arsenic on tubule cells.
- Glomerular damage can result in proteinuria.
- The kidney is not the most sensitive target organ for chronic arsenic toxicity as other organ systems may be affected at lower doses.
- There is limited strength of association between chronic arsenic exposure and renal cancer [IARC 2004; NRC 2000].
- Arsine gas is more nephrotoxic than arsenic. However, both can cause acute tubular necrosis [Giberson et al. 1976].

1.1.3 Cardiovascular effects

Both acute and chronic exposure to high levels of arsenic may result in a wide range of adverse cardiovascular effects.

- There is limited strength of association between chronic arsenic exposure and peripheral vascular disease, hypertension, and cardiovascular disease [IARC 2004].
- Acute arsenic poisoning may cause both diffuse capillary leakage and cardiomyopathy, resulting in shock.
- The extent of cardiovascular injury may vary with
  - age,
  - arsenic dose, and
  - individual susceptibility.
- In acute arsenic poisoning (such as suicide attempts), diffuse capillary leakage may lead to
  - delayed cardiomyopathy,
  - hypotension,
  - shock,

- transudation of plasma, and
- vasodilation.

- Arsenic ingestion from contaminated beer has been reported to cause outbreaks of cardiomyopathy [Reynolds 1901; Aposhian 1989; Rosenman 2007].
- Other reports of arsenic poisoning have resulted in peripheral vascular disease, rather than congestive heart failure [Engel et al. 1994].
- Inhibition of endothelial nitric oxide synthase, changes in coagulation and inflammation due to arsenic exposure have been shown in experimental studies to contribute to atherosclerosis [Simeonova and Luster 2004].
- Hypertension has been reported with long-term exposure to arsenic [Chen et al. 1995].
- Ingesting arsenic levels of 0.8 to 1.82 ppm in drinking water (normal concentrations of arsenic in drinking water are lower than .01 ppm) as reported in Chile and Taiwan have resulted in an increased prevalence of peripheral vascular disease and cardiovascular mortality [Rosenman 2007].
- Myocardial damage can result in a variety of electrocardiographic findings, including
  - broadening of the QRS complex,
  - prolongation of the QT interval,
  - ST depression,
  - flattening of T waves, and
  - atypical, multifocal ventricular tachycardia.
- Gangrene of the extremities, known as "blackfoot disease", has been reported with drinking arsenic-contaminated well water in Taiwan, where the prevalence of the disease increased with increasing age and well water arsenic concentration (170–800 parts per billion) [ATSDR 2007].
  - Pathologically, blackfoot disease was due to arteriosclerotic or thromboangiitis obliterans. After the water supply was changed, the vascular and cardiovascular mortality reversed [Pi et al. 2005; Chang et al. 2004].
  - Persons with blackfoot disease also had a higher incidence of arsenic-induced skin cancers. However, investigators believe other vasoactive substances found in the water may have been contributory [ATSDR 2007].
- Vasospastic (Raynaud's) disease in arsenic-exposed smelter workers and German vineyard workers has been reported. Smelter workers had a total exposure of 4 to 9 grams of arsenic, compared to the 20 grams of arsenic exposure reported for those with blackfoot disease [Rosenman 2007].
- Drinking arsenic-contaminated water in Chile was associated with an increase of vasospastic changes (Raynaud's disease) and thickening of the small and medium sized arteries in autopsied children [Garcia-Vargas and Cebrian 1996].
- Arsenic ingestion affects the cardiovascular system, altering myocardial depolarization and causing cardiac arrhythmias and hypertension in some populations [Guha 2003].
- Inorganic arsenical pesticides are now generally not used by vineyard workers, and the organic arsenicals that are used have not been associated with vasospastic changes [Rosenman 2007].
- Increased cardiovascular mortality in occupationally exposed groups may be masked by the healthy worker effect [Hertz-Picciotto et al. 2000].

1.1.4 Neurologic Effects

In studies that support an association, arsenic-exposed patients may develop destruction of axonal cylinders, leading to peripheral neuropathy. This has been reported at acute high doses (>2 milligram (mg) arsenic (As)/kilogram (kg)/day) as well as from repeated exposures to lower levels (.03 – 0.1 mg As/kg/day) [Chakraborti et al. 2003a, 2003b; ATSDR 2007].

- Arsenic may cause encephalopathy at acute high doses (> 2mg As/Kg/day) [Uede and Furukawa 2003; Vantroyen et al. 2004; ATSDR 2007].

Arsenic poisoning can cause peripheral neuropathy. The lesion is a sensory-motor axonopathy.

- The classic finding is a peripheral neuropathy involving sensory greater than motor neurons in a symmetrical, stocking glove distribution [Murphy et al. 1981].
- In high-level arsenic exposures, onset of neuropathy may occur after 7 to 14 days, with intense
  - increased sweating in the distal lower extremities,
  - muscle cramps,
  - muscle tenderness,
  - numbness,
  - paresthesia, and
  - spontaneous pain [Bleecker 2007].
- Sensory effects, particularly painful dysesthesia, occur earlier and may predominate in moderate poisoning, whereas ascending weakness and paralysis may be evident in more severe poisoning.
- Those cases may at first seem indistinguishable from Guillain-Barré syndrome (acute inflammatory demyelinating polyneuropathy) [Donofrio et al. 1987].
- Cranial nerves are rarely affected, even in severe poisoning.
- The mechanism of arsenic neuropathy may be similar to the neuropathy of thiamine deficiency [Sexton and Gowdy 1963], whereby arsenic inhibits the conversion of pyruvate to acetyl coenzyme A and thus blocks the Krebs cycle.
- The neurotoxic forms of arsenic include inorganic trivalent (arsenite) and pentavalent (arsenate) and the methylated metabolites, monomethyl arsonic acid and dimethylarsenic acid [Foa et al. 1984].

Encephalopathy has been reported after both acute and chronic exposures.

- Onset may begin within 24 to 72 hours following acute poisoning, but it more often develops slowly as a result of chronic exposure [Beckett et al. 1986].
- The neuropathy is primarily sensory, with chronic exposure affecting vibration and positional sense to a greater extent than other modalities. Weakness of intrinsic muscles of the extremities is mild when present in chronic arsenic exposure [Bleecker 2007].
- The neuropathy is primarily due to destruction of axonal cylinders.
- Nerve conduction and electromyography studies most frequently show a sensory-motor axonopathy and can document severity and progression. A dose response effect has been reported between environmental exposure to arsenic-containing dust and vibrotactile threshold, tremor intensity, nerve conduction studies, and standing steadiness [Gerr and Letz 2000].

- Elevated vibration threshold has been associated with a cumulative arsenic index (drinking water arsenic exposure) and urinary arsenic levels [Hafeman et al. 2005].
- Subclinical neuropathy, defined by the presence of abnormal nerve conduction, but no clinical complaints or symptoms, has been described in chronically exposed individuals [Tseng 2003; ATSDR 2007].
- Recovery from neuropathy induced by chronic exposure to arsenic compounds is generally slow, sometimes taking years, and complete recovery may not occur.
- The prognosis for recovery in mild cases of neuropathy is excellent [Bleecker 2007].
- Follow-up studies of Japanese children who chronically consumed arsenic contaminated milk revealed an increased incidence of
  - cognitive deficits,
  - epilepsy,
  - other brain damage, and
  - severe hearing loss (ATSDR 2007).
- Hearing loss as a sequela of acute or chronic arsenic intoxication has not been confirmed by other case reports or epidemiologic studies [ATSDR 2007].
- There is limited strength of association between chronic arsenic exposure and neurologic effects, per the International Agency for Research on Cancer (IARC) and the National Research Council (NRC) [IARC 2004; NRC 2000].

1.1.5 Dermal effects

Pigment changes and palmoplantar hyperkeratoses are characteristic of chronic arsenic exposure.

- Benign arsenical keratoses may progress to malignancy.
- Delayed effects of acute or chronic exposure may be seen as Mee's lines in nails.
- Mees lines are horizontal lines in the nails of digits.

The skin lesions occurring most frequently in arsenic-exposed humans are

- hyperkeratosis,
- hyperpigmentation, and
- skin cancer.

Patchy hyperpigmentation, a pathologic hallmark of chronic exposure, may be found anywhere on the body.

- Patchy hyperpigmentation occurs particularly on the
  - axillae,
  - eyelids,
  - groin,
  - neck,
  - nipples, and
  - temples.
- The common appearance of the dark brown patches with scattered pale spots is sometimes described as "raindrops on a dusty road".
- In severe cases, the pigmentation extends broadly over the chest, back, and abdomen.
- Pigment changes have been observed in populations chronically consuming drinking water containing 400 ppb or more arsenic [ATSDR 2007].

Arsenical hyperkeratosis occurs most frequently on the palms and soles.

- Keratoses usually appear as small corn-like elevations, 0.4 to 1 centimeter (cm) in diameter.
- In most cases, arsenical keratoses show little cellular atypia and may remain morphologically benign for decades [ATSDR 2007].
- In other cases, cells develop marked atypia (precancerous) and appear indistinguishable from Bowen's disease, which is an in situ squamous cell carcinoma discussed in Carcinogenic Effects later in this section [ATSDR 2007].
- Basal cell carcinomas have also been reported [Cohen and Moore 2007].
- Confounding factors for arsenic-induced skin cancer may include exposure to sunlight, chronic liver disease, and nutritional status [Hsueh et al. 1995].

1.1.6 Respiratory effects

Inhalation of high concentrations of arsenic compounds produces irritation of the respiratory mucosa.

- Smelter workers experiencing prolonged exposures to high concentrations of airborne arsenic at levels rarely found today had inflammatory and erosive lesions of the respiratory mucosa, including nasal septum perforation.
- Lung cancer has been associated with chronic arsenic exposure in smelter workers and pesticide workers [ATSDR 2007].

1.1.7 Hematopoietic and hematologic effects

Bone marrow depression may result from acute or chronic arsenic intoxication and may initially manifest as pancytopenia.

- Both acute and chronic arsenic poisoning may affect the hematopoietic system.
- A reversible bone marrow depression with pancytopenia may occur.
- Anemia and leukopenia are common in chronic arsenic toxicity and are often accompanied by thrombocytopenia and mild eosinophilia.
- The anemia may be normocytic or macrocytic, and basophilic stippling may be noted on peripheral blood smears [Kyle and Pearse 1965; Selzer 1983].
- According to the NRC and IARC, there is a suggestive association between chronic arsenic exposure and immunosupression [NRC 2000; IARC 2004].
- Acute intoxication with arsine gas can cause fulminant intravascular hemolysis.

1.1.8 Reproductive effects

Increased frequency of spontaneous abortions and congenital malformations has been linked to arsenic exposure.

- Arsenic is a reproductive toxicant and a teratogen [Shalat 1996]. It is readily transferred across the placenta, and concentrations in cord blood are similar to those in maternal blood.
- A published case report described acute arsenic ingestion during the third trimester of pregnancy, leading to delivery of a live infant that died within 12 hours. Autopsy revealed intra alveolar hemorrhage and high levels of arsenic in the brain, liver, and kidneys [ATSDR 2007].

- A study of women working at or living near a copper smelter where ambient arsenic levels were elevated reported increased frequencies of spontaneous abortions and congenital malformations [Nordstrom et al. 1979].
  - The frequency of all malformations was twice the expected rate and the frequency of multiple malformations was increased fivefold [Nordstrom et. al. 1979].
  - However, a number of other chemicals, including lead, cadmium, and sulfur dioxide, were also present, and thus it is difficult to assess the role of arsenic in the etiology of these effects.

1.1.9 Carcinogenic effects

The carcinogenicity of arsenic in humans has been established.

Inorganic arsenic is a known human carcinogen [IARC 2004].		
Agency	**Carcinogenicity Classification**	**Description**
International Agency for Research on Cancer	1	Known human carcinogen
National Toxicology Program	--	Known human carcinogen
U.S. Environmental Protection Agency	Group A	Known human carcinogen

- In humans, chronic arsenic ingestion may cause cancers of the
  - bladder,
  - kidney,
  - liver,
  - lung,
  - prostate, and
  - skin (ATSDR 2007).
- Chronic inhalation of arsenicals has been associated with lung cancer and angiosarcoma (a rare form of liver cancer) has been reported [Falk et al. 1981].
- Several large-scale epidemiological studies of arsenic exposure have shown association and/or dose response trends for tumors of the
  - bladder,
  - kidney,
  - liver,
  - lung, and
  - prostate [ATSDR 2007].
- According to IARC and NRC, the association between chronic arsenic exposure and cancer is strongest for skin, lung, and bladder cancer. Liver (angiosarcoma), kidney, and other cancers have limited strength of association [IARC 2004; NRC, 2000].

Skin Cancer

Latency for skin cancer associated with ingestion of arsenic may be 3 to 4 decades, while the noncarcinogenic skin effects typically develop several years after exposure [ATSDR 2007].

- An increased risk of skin cancer in humans is associated with chronic exposure to inorganic arsenic in contaminated water and the workplace.
- Arsenic-induced skin cancer is frequently characterized by lesions over the entire body, mostly in unexposed areas such as the
  - palms,
  - soles, and
  - trunk.

More than one type of skin cancer may occur in a patient.

- Most of the Taiwanese who developed skin cancer in association with ingested arsenic-contaminated drinking water had multiple cancer types [ATSDR 2007]. The most commonly reported types, in order of decreasing frequency, were
  - intraepidermal carcinomas (Bowen's disease),
  - squamous cell carcinomas, and
  - basal cell carcinomas.
- Seventy-two percent of the Taiwanese with skin cancer also had hyperkeratosis, and 90% had hyperpigmentation.
- Some hyperkeratinized lesions can develop into intraepidermal carcinoma, which may ultimately become invasive. The lesions are sharply demarcated, round or irregular plaques that tend to enlarge; they may vary in size from 1 millimeter to more than 10 centimeters [ATSDR 2007].
- Arsenical basal cell carcinomas most often arise from normal tissue, are almost always multiple, and frequently occur on the trunk. The superficial spreading lesions are red, scaly, atrophic, and are often indistinguishable from Bowen's disease by clinical examination.
- Arsenic-associated squamous cell carcinomas are distinguished from ultraviolet-induced squamous cell carcinomas by their tendency to occur on the extremities (especially palms and soles) and trunk, rather than on sun-exposed areas such as the head and neck. However, it may be difficult to distinguish other arsenic-induced skin lesions from those induced by other causes.
- Epidemiologic studies indicate that a dose response relationship exists between the level of arsenic in drinking water and the prevalence of skin cancers in the exposed population [ATSDR 2007].

Excessive mortality rates due to arsenic-induced skin cancer have also been observed in vineyard workers with dermal and inhalation exposure [ATSDR 2007].

Lung Cancer

An association between lung cancer and occupational exposure to inorganic arsenic has been confirmed in several epidemiologic studies [Enterline et al. 1987], and arsenic is considered a cause of lung as well as skin cancer.

- In arsenic-exposed workers, there is a systematic gradient in lung cancer mortality rates, depending upon duration and intensity of exposure [ATSDR 2007].
- A higher risk of lung cancer was found among workers exposed predominantly to arsenic trioxide in smelters and to pentavalent arsenical pesticides in other settings.

Neither concomitant exposure to sulfur dioxide nor to cigarette smoke was determined to be an essential co-factor in these studies.

1.2 Effects of Arsenic on the Environment

Water and land-living plants and animals show a wide range of sensitivities to different chemical forms of arsenic. Their sensitivity is modified both by biological factors and by their surrounding physical and chemical environment. In general, inorganic forms of arsenic are more toxic to the environment than organic forms and, among inorganic forms, arsenite is more toxic than arsenate. This is probably because the way in which the various forms are taken up into the body differs and once taken up, they act in different ways in the body. The reason why arsenite is toxic is thought to be because it binds to particular chemical groups - sulfhydryl groups - found on proteins. Arsenate, on the other hand, affects the key energy producing process that take place in all cells.

Arsenic compounds cause short-term and long-term effects in individual plants and animals and in populations and communities of organisms. These effects are evident, for example, in aquatic species at concentrations ranging from a few micrograms to milligrams per litre. The nature of the effects depends on the species and time of exposure. The effects include death, inhibition of growth, photosynthesis and reproduction, and behavioral effects. Environments contaminated with arsenic contain only a few species and fewer numbers within species. If levels of arsenate are high enough, only resistant organisms, such as certain microbes, may be present.

1.3 Effects on Other Organisms in the Environment

Aquatic and terrestrial biota show a wide range of sensitivities to different arsenic species. Their sensitivity is modified by biological and abiotic factors. In general, inorganic arsenicals are more toxic than organoarsenicals and arsenite is more toxic than arsenate. The mode of toxicity and mechanism of uptake of arsenate by organisms differ considerably. This may explain why there are interspecies differences in organism response to arsenate and arsenite. The primary mechanism of arsenite toxicity is considered to result from its binding to protein sulfhydryl groups. Arsenate is known to affect oxidative phosphorylation by competition with phosphate. In environments where phosphate concentrations are high, arsenate toxicity to biota is generally reduced. As arsenate is a phosphate analogue, organisms living in elevated arsenate environments must acquire the nutrient phosphorous yet avoid arsenic toxicity.

Arsenic compounds cause acute and chronic effects in individuals, populations and communities at concentrations ranging from a few micrograms to milligrams per litre, depending on species, time of exposure and end-points measured. These effects include lethality, inhibition of growth, photosynthesis and reproduction, and behavioural effects. Arsenic-contaminated environments are characterized by limited species abundance and diversity. If levels of arsenate are high enough, only species which exhibit resistance may be present.

2. STATUS OF ARSENIC CONTAMINATION

- Arsenic is naturally present at high levels in the groundwater of a number of countries.
- Arsenic is highly toxic in its inorganic form.
- Contaminated water used for drinking, food preparation and irrigation of food crops poses the greatest threat to public health from arsenic.
- Long-term exposure to arsenic from drinking-water and food can cause cancer and skin lesions. It has also been associated with developmental effects, cardiovascular disease, neurotoxicity and diabetes.
- The most important action in affected communities is the prevention of further exposure to arsenic by provision of a safe water supply.

In India, since the groundwater arsenic contamination was first surfaced from West Bengal in 1983, a number of other States, namely; Jharkhand, Bihar, Uttar Pradesh in flood plain of the Ganga River; Assam and Manipur in flood plain of the Brahamaputra and Imphal rivers, and Rajnandgaon village in Chhattisgarh state have chronically been exposed to drinking arsenic contaminated hand tube-wells water above permissible limit of 50 µg/L. Many more NorthEastern Hill States in the flood plains are also suspected to have the possibility of arsenic in groundwater. Even with every additional survey, new arsenic affected villages and people suffering from arsenic related diseases are being reported. All the arsenic affected river plains have the river routes originated from the Himalayan region.

As of 2008, West Bengal, Jharkhand, Bihar, Uttar Pradesh in flood plain of Ganga River; Assam and Manipur in flood plain of Brahamaputra and Imphal rivers, and Rajnandgaon village in Chhattisgarh state have so far been exposed to drinking arsenic contaminated hand tube-wells water. The area and population of these states are 529674 km^2 & approx. 360 million respectively, in which 88688 km^2 and approximately 50 million people have been projected vulnerable to groundwater arsenic contamination. Almost all the identified arsenic affected areas in the Gangetic plains except areas in Chhattisgarh and 3 districts in Bihar namely, Darbhanga, Purnea and Kishanganj, are in a linear tract on either side of the River Ganga in UP, Bihar, and Jharkhand, and the River Bhaghirathi in West Bengal; while the areas in Assam and Manipur are in the flood plains of the Brahmaputra and Barack, respectively (Fig.1) . Analysis of 1,69,698 hand tube-well water samples from all these 7 states for arsenic detection by School of Environmental Studies, Jadavpur University (SOES, JU) reported presence of arsenic in 45.96% and 22.94% of the water samples more than 10 µg/L (WHO guideline value of arsenic in drinking water) and 50µg/L (Indian standard of arsenic in drinking water), respectively. Importantly, 3.3% of the analyzed tube-wells had been found arsenic concentrations above 300µg/L, the concentration predicting overt arsenical skin lesions. CGWB, State PHED, WB and Bihar, and other organizations also analyzed quite a large number of water samples.

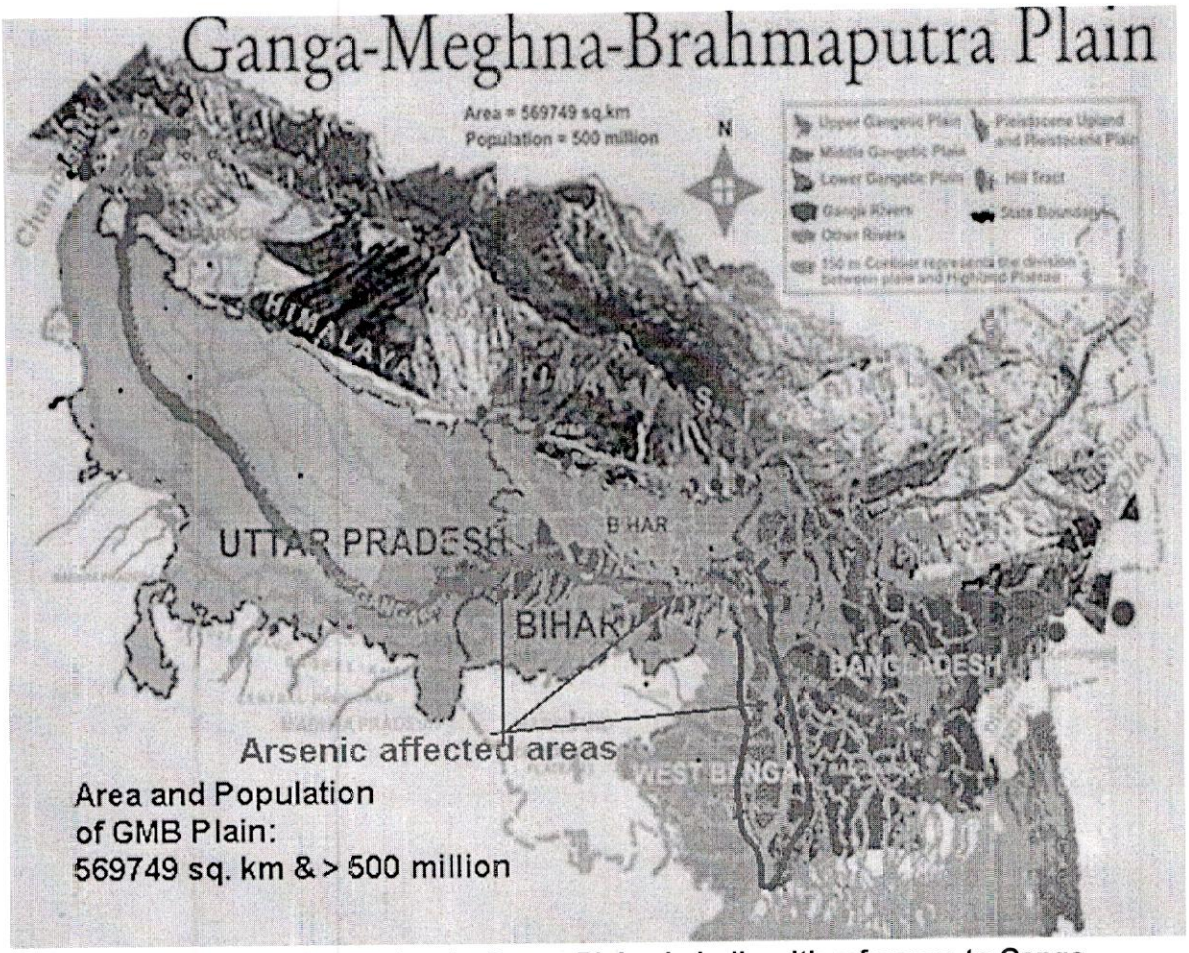

Fig. 1: Arsenic affected stretches in Ganga Plains in India with reference to Ganga-Meghna-Brahmaputra Plains

[Source: http://www.cgwb.gov.in/documents/papers/incidpapers/Paper%208%20-%20Ghosh.pdf]

3. SOURCES OF ARSENIC CONTAMINATION

3.1 Natural Sources of Arsenic Contamination

Arsenic is present in more than 200 mineral species, the most common of which is arsenopyrite. It has been estimated that about one-third of the atmospheric flux of arsenic is of natural origin. Volcanic action is the most important natural source of arsenic, followed by low-temperature volatilization.

Inorganic arsenic of geological origin is found in groundwater used as drinking-water in several parts of the world, for example Bangladesh.

Organic arsenic compounds such as arsenobetaine, arsenocholine, tetramethyl arsonium salts, arsenosugars and arsenic-containing lipids are mainly found in marine organisms although some of these compounds have also been found in terrestrial species.

3.2 Man-made Sources of Arsenic Contamination

Elemental arsenic is produced by reduction of arsenic trioxide (As_2O_3) with charcoal. As_2O_3 is produced as a by-product of metal smelting operations. It has been estimated that

70% of the world arsenic production is used in timber treatment as copper chromearsenate (CCA), 22% in agricultural chemicals, and the remainder in glass, pharmaceuticals and non-ferrous alloys.

Mining, smelting of non-ferrous metals and burning of fossil fuels are the major industrial processes that contribute to anthropogenic arsenic contamination of air, water and soil. Historically, use of arsenic-containing pesticides has left large tracts of agricultural land contaminated. The use of arsenic in the preservation of timber has also led to contamination of the environment

3.2.1 From food products

The greatest threat to public health from arsenic originates from contaminated groundwater. Inorganic arsenic is naturally present at high levels in the groundwater of a number of countries, including Argentina, Bangladesh, Chile, China, India, Mexico, and the United States of America. Drinking-water, crops irrigated with contaminated water and food prepared with contaminated water are the sources of exposure.

Fish, shellfish, meat, poultry, dairy products and cereals can also be dietary sources of arsenic, lthough exposure from these foods is generally much lower compared to exposure through contaminated groundwater. In seafood, arsenic is mainly found in its less toxic organic form.

3.2.2 From industrial processes

Arsenic is used industrially as an alloying agent, as well as in the processing of glass, pigments, textiles, paper, metal adhesives, wood preservatives and ammunition. Arsenic is also used in the hide tanning process and, to a limited extent, in pesticides, feed additives and pharmaceuticals.

3.2.3 From tobacco consumption

People who smoke tobacco can also be exposed to the natural inorganic arsenic content of tobacco because tobacco plants essentially take up arsenic naturally present in the soil. Also, in the past, the potential for elevated arsenic exposure was much greater when tobacco plants used to be treated with lead arsenate insecticide.

4. METHODS OF IDENTIFICATION OF ARSENIC IN WATER

This section provides information on a number of common analytical methods that may be used to accurately measure low concentrations (< 10 µg/L) of arsenic in drinking water. In the context of this report, "common analytical methods" refers to those methods that have been promulgated and/or published by various EPA offices or consensus organizations, i.e., the American Society for Testing and Materials (ASTM), and the American Public Health Association (APHA), the American Water Works Association (AWWA), and the Water Environment Federation (WEF). The common analytical methods published by EPA, ASTM, APHA, and AWWA are generally within the analytical capabilities of most EPA, state, utility, and commercial laboratories.

4.1 Methods Currently Approved by EPA for Measuring Arsenic

Eight methods are currently approved in 40 CFR 141.23 (l) for the analysis of arsenic in drinking water. Table 1 lists the approved methods and the method detection limits (MDLs) that are typical of the approved methodologies.

Table 1: Analytical methods currently approved for the analysis of arsenic in water

Method		Technique	MDL (µg/L)
Multi-Analyte Methods	EPA 200.8	Inductively coupled plasma/mass spectrometry (ICP/MS)	1.4
	EPA 200.7	Inductively coupled plasma/atomic emission spectrometry (ICP/AES)	8
	SM 3120 B	ICP/AES	50
Single-Analyte Methods	EPA 200.9	Graphite furnace atomic absorption spectrometry (GFAA)	0.5
	SM 3113 B	Graphite furnace atomic absorption spectrometry (GFAA)	1
	ASTM D 2972-93, Test Method C	Graphite furnace atomic absorption spectrometry (GFAA)	5
	SM 3114 B	Graphite furnace atomic absorption spectrometry (GFAA)	0.5
	ASTM D 2972-93, Test Method B	Graphite furnace atomic absorption spectrometry (GFAA)	1

Three of the approved methods are multi-element or multi-analyte, meaning other analytes besides arsenic can be measured during the analysis. Whereas two of the three methods (EPA 200.7 and SM 3120B) are Inductively Coupled Plasma-Atomic Emission Spectrometry (ICPAES) techniques, EPA 200.8 is an Inductively Coupled Plasma-Mass Spectrometry (ICP-MS) technique. The primary advantage of using a multi-analyte method is if the same method is approved for compliance monitoring of one or more other regulated analytes, compliance monitoring requirements for this metal will result in minimal additional analytical costs. The remaining five analytical methods approved by EPA for the measurement of arsenic in drinking water are all element-specific or single-analyte techniques (can only measure arsenic). These single-analyte techniques include EPA 200.9, SM 3113 B, SM 3114 B, ASTM D 2972-93 B and ASTM D 2972-93 C). Three of the five single-analyte methods, EPA 200.9, SM 3113 B, and ASTM 2972-93 C, are graphite furnace atomic absorption (GFAA) techniques that have traditionally been used to quantify arsenic in drinking water. Two of these GFAA methods, EPA 200.9 and SM 3113 B, employ the use of stabilized temperature platform graphite furnace atomic absorption (STP-GFAA) technology that significantly reduces interferences and improves analytical sensitivity. ASTM 2972-93 C employs regular hollow graphite tubes with off-the-wall atomization. In reality, any of the GFAA methods can be adapted to stabilized temperature platform (STP) technology, provided that the furnace and background correction system are compatible with STP graphite tubes. Users of GFAA should consult their instrument manufacturer for further guidance on this issue.

Two of the five single-analyte methods, SM 3114 B and ASTM 2972-93 B, utilize gaseous hydride atomic absorption (GHAA). These methods employ zinc in hydrochloric acid or sodium borohydride to convert arsenic to its volatile hydride. In ASTM 2972-93 B, the arsenic hydride is removed from the sample by a flow of nitrogen into an argon- or nitrogen-entrained hydrogen flame where it is determined by atomic absorption at 193.7 nm. In SM 3114 B, the volatile hydrides may also be swept into an entrained hydrogen flame, or alternatively, into a quartz atomization cell positioned in the optical path of an atomic spectrophotometer. Quartz atomization cells provide the most sensitive arsenic hydride determinations and minimize background noise associated with hydrogen flames. In both methods, the absorption of the light source is proportional to the arsenic concentration. Both hydride methods provide methodspecific sample digestion procedures that are required prior to analysis.

4.2 Inductively Coupled Plasma - Mass Spectrometry (ICP-MS) (EPA 200.8, SW-846 6020)

Inductively Coupled Plasma Mass Spectrometry methods provide for the multi-element determination of metals in solution. An aqueous solution of sample is introduced by pneumatic nebulization into a radio frequency plasma where energy transfer processes causes desolvation, atomization, and ionization. The ions are extracted from the plasma through a differentially pumped vacuum interface and separated on the basis of their mass-to-charge ratio by a quadrupole mass spectrometer. The ions transmitted through the mass spectrometer are detected by a continuous electron multiplier or Faraday detector. The mass spectrometer can be operated in two different modes, full-scan and selective ion monitoring. In the full-scan mode, the detector scans across a wide range of masses that encompass many target analytes. The selective ion monitoring mode restricts the detector to a few specific masses that are characteristic of the analyte of interest.

By selecting a narrow subset of all possible masses, the detector spends more time looking for those specific masses than in the full-scan mode, thereby increasing its sensitivity for those target analytes. Selective ion monitoring can be used to obtain an MDL as low as 0.4 µg/L for total recoverable arsenic and 0.1 µg/L arsenic for direct analysis (EPA Method 200.8,

4.3 Inductively Coupled Plasma-Atomic Emission Spectrometry (ICP-AES) (EPA 200.15, EPA 200.7 and SM 3120 B)

Inductively coupled plasma-atomic emission spectrometry methods provide for the multi-element determination of metals in solution using sequential or simultaneous instruments. The instruments measure characteristic atomic-line emission spectra by optical spectroscopy. Aqueous samples are introduced into the instrument via a peristaltic pump or by pneumatic nebulization. The resulting aerosol is desolvated before being transported to the plasma torch. Element-specific emission spectra are produced by a radio-frequency inductively coupled plasma and are dispersed by a grating spectrometer. The intensities of the line spectra are monitored at specific wavelengths by a photosensitive device. Quantitation of the target analyte(s) is performed by comparing the spectral intensity for each element to known calibration standards. Background correction is required to compensate for variable background contribution.

Two modifications to ICP-AES allow for greater sensitivity. These modifications include (i) the use of the axial torch configuration and/or (ii) the use of ultrasonic nebulization. Using the axial torch configuration, the torch is in a horizontal position, rather than in a vertical position. The source path length is increased by viewing axially or down the end of the torch. This longer path length increases analyte emission and improves sensitivity. "Trace ICP" as it is sometimes called, has detection limits typically ten times lower than standard ICP-AES.

Ultrasonic nebulization is a new sample introduction technique, which provides greater sensitivity due to the more efficient transport of the analyte(s) into the plasma torch. Ultrasonic nebulization ICP-AES (referred to as UNICP-AES) is the same as traditional ICP-AES except for the difference in the way the sample is introduced into the plasma torch. UNICP-AES employs an ultrasonic nebulizer to introduce the sample aerosol into the plasma, instead of pneumatic nebulization or a peristaltic pump. In reality, any ICP-AES instrument can be converted to UNICP by simply attaching a ultrasonic nebulizer. Stand-alone module kits are commercially available that can provide ultrasonic nebulization capabilities to any ICP. Ultrasonic nebulization can improve detection limits on any ICP-AES by a factor of 5 to 10, owing to the greater efficiency with which the sample is transported into the plasma. EPA method 200.15, which employs ultrasonic nebulization, has reported a method detection limit of 2 µg/L for direct analysis and 3 µg/L for a total recoverable digestion.

4.4 Gaseous Hydride/Atomic Absorption Spectrometry (GHAA) (SM 3114 B, ASTM 2972-93 B, SW-846 7062, EPA 1632)

The Gaseous Hydride/Atomic Absorption Spectroscopic methods operate on the following principal. Trivalent arsenic is converted to its volatile hydride (arsine) by reaction with zinc and hydrochloric acid or sodium borohydride. The volatile hydrides are swept into an argon-hydrogen flame or into a heated quartz cell positioned in the optical path of an atomic absorption spectrophotometer. A light beam from a hollow cathode lamp or electrodeless discharge lamp (EDL) containing the element of interest is directed through the flame or quartz cell, into a monochromator, and into a detector that measures the amount of light absorbed by the arsine. In this respect, GHAA is the same as flame or furnace atomic absorption. For total or total recoverable arsenic, the sample is first acid digested or placed in a special reaction vessel to destroy organic arsenic compounds and oxidize the arsenic to arsenic (V). The arsenic (V) is then reduced to arsenic (III) by reaction with potassium iodide or tin chloride, then converted to their volatile hydrides with zinc or borohydride.

Each of the methods cited above provide method-specific digestion procedures to prepare the sample for hydride analysis, except EPA Method 1632. Method 1632 is discussed in detail below. As noted in Technical Notes on Drinking Water Methods, October 1994, irrespective of the sample turbidity, drinking water samples analyzed for arsenic by gaseous hydride must be digested prior to analysis. SM 3114 B provides a persulfate digestion that is effective in converting organic arsenic compounds to the arsenic (V) oxidation state. The perchloric acid digestion provided in the method should be avoided due to safety concerns (Refer to Technical Notes on Drinking Water Methods, October 1994). ASTM 2972-93 B provides two sulfuric/nitric acid digestion procedures depending on whether zinc or sodium borohydride is used in the hydride generation step. SW-846 Method 7062 references SW-846 Method 3010A, a separate total metals hotplate digestion

procedure using nitric acid. SW-846 Method 7062 also employs an automated analysis system for automated sample analysis.

EPA 1632 is a GHAA method developed by the US EPA Office of Water in 1996, and provides for the direct analysis of dissolved and total arsenic in aqueous samples. This method does not require a separate preliminary digestion procedure, and in the context of the method, total arsenic and total recoverable arsenic are synonymous. An aliquot of sample is placed in a special reaction vessel and 6 M HCL is added. A 4% sodium borohydride solution is added to the sample to convert organic and inorganic arsenic to volatile arsines, which are purged into a cryogenic trap containing 15% OV-3 on Chromasorb. The trapped arsines are thermally desorbed from the trap and swept into a quartz furnace containing an air-hydrogen flame. The sample absorbance is recorded and compared with the response of known calibration standards. Use of a quartz furnace in conjunction with cryogenic trapping allows for the detection of arsenic down to 10 ng/L, a detection limit significantly lower than those provided by the other hydride methods.

4.5 Graphite Furnace Atomic Absorption Spectrometry (GFAA) (EPA 200.9, SM 3113 B, ASTM 2972-93 C, SW-846 7060A)

In the graphite furnace atomic absorption spectrometry technique, a small volume of sample (typically 5 to 50 µL) is injected into a graphite tube positioned in the optical path of an atomic absorption spectrophotometer. An electrical furnace is used to heat the tube sequentially through drying, charring, and finally, an atomization step. A light beam from a hollow cathode lamp or electrodeless discharge lamp (EDL) containing the element of interest is directed through the tube, into a monochromator, and into a detector that measures the amount of light absorbed by the free ground state atoms. The amount of light absorbed by the free ground state atoms is directly proportional to the concentration of the analyte in solution within the linear calibration range of the instrument. Because the greater percentage of analyte atoms are vaporized and dissociated within the light beam passing through the graphite tube, greater analytical sensitivity is obtained and lower detection limits are possible as compared with flame atomic absorption.

The limit of detection can be extended by increasing the injection volume or by using a multi-injection technique. These techniques effectively increase the total amount of analyte placed in the tube resulting in greater absorbance.

GFAA may be divided into two basic techniques, depending on the type of graphite tube used in the method. In standard GFAA, the sample is pipetted directly into a graphite tube and the tube is electrically heated to effect atomization of the analyte. This type of GFAA is known as "off-the-wall-atomization." ASTM 2972-93 C and SW-846 7060A utilize standard graphite tubes and "off-the-wall-atomization."

A newer technique utilizes stabilized temperature graphite furnace atomic absorption (STPGFAA). In this technique, a small platform (L'vov platform or similar device) is inserted into the graphite tube. The sample is pipetted directly onto the platform. As with plain graphite tube, the platform tube is serially heated to dry, char, and atomize the analyte. With the platform tube, atomization is more consistent and controlled, resulting in increased atomization efficiency.

Because the sample is more efficiently atomized on the stabilized platform than in the standard graphite tube, the detection of lower concentrations is possible, down to 0.5 µg/L. EPA 200.9 and SM 3113 B employ STP-GFAA, although any method can be adapted for this technology provided the background correction system and furnace are compatible with

the platform tube and the furnace temperature programs are adjusted appropriately. Users of GFAA should consult their instrument manufacturer for further guidance on this issue. As noted above, the detection limits for any of the GFAA methods, including EPA 200.9, can be lowered by employing multiple injections (thereby increasing the total sample volume injected into the graphite tube). Adjustment to the furnace heating program would be required to sequentially dry each injection aliquot prior to charring and atomization.

4.6 Anodic Stripping Voltammetry (ASV) (SW-846 Method 7063)

Anodic stripping voltammetry provides an alternative analytical technique for measuring free dissolved arsenic in drinking water. The ASV method is equally sensitive for As (III) and As (V) and is suitable for measuring low-levels of free arsenic from 0.1 to 300 µg/L. In this method, free dissolved arsenic [as As (III) and/or As (V)] is quantified by anodic stripping, at a potential of +145 mV with respect to the saturated calomel electrode, from a conditioned gold plated electrode. The analysis by ASV involves three major steps. First, a glassy carbon electrode (GCE) is prepared for use by plating on a thin film of gold onto the electrode. The plated electrode is then conditioned, and finally, the arsenic concentration in the sample is determined by comparing the sample response to external standards. Dissolved antimony and bismuth are positive interferences. Dissolved copper at a concentration > 1 mg/L is also a positive interference.

5. ENVIRONMENTAL STANDARDS OF ARSENIC IN WATER USED FOR DIFFERENT PURPOSES

The current drinking water standard or Maximum Contaminant Level (MCL) set by the U.S. Environmental Protection Agency (EPA) is 0.010 mg/L or parts per million (ppm). This is equivalent to 10 ug/L (micrograms per liter) or 10 ppb. In 2001, the U.S. Environmental Protection Agency (EPA) reduced the regulatory MCL from 50 ppb to 10 ppb on the basis on bladder and lung cancer risks. The MCL is based on the average individual consuming 2 liters of water a day for a lifetime. Long term exposure to drinking water containing arsenic at levels higher than 10 ppb increases the chances of getting cancer, while for lower arsenic water levels the chances are less.

If your water has arsenic levels above 10 ppb, you should obtain drinking water from another source or install a home treatment device. Concentrations above 10 ppb will increase the risk of long-term or chronic health problems, the higher the level and length of exposure, the greater the risk. It is especially important to reduce arsenic water concentrations if you have children or are pregnant. Children are at greater risk (to any agent in water) because of their greater water consumption on a per unit body weight basis. Pregnant women may wish to reduce their arsenic exposures because arsenic has been found at low levels in mother's milk and will cross the placenta, increasing exposures and risks for the fetus. If your water has arsenic levels above 200 ppb, you should immediately stop drinking the water until you can either obtain water from another source or install and maintain treatment.

6. REMEDIAL MEASURES

Technological options to combat arsenic menace, in groundwater, to ensure supply of arsenic free water, in the affected areas, can be one of the followings or a combination of all:

(i). In-situ remediation of arsenic from aquifer system,
(ii). Ex-situ remediation of arsenic from tapped groundwater by arsenic removal technologies,
(iii). Use of surface water source as an alternative to the contaminated groundwater source,
(iv). Tapping alternate safe aquifers for supply of arsenic free groundwater.

In-situ remediation of arsenic from aquifer system or decontamination of aquifer is the best technological option. However, in-situ remediation of arsenic contaminated aquifer would not only be an exercise of throwing stone in the dark but would also be very expensive and a difficult task because of the size of the plan and the absence of complete understanding of the physico-chemical and geochemical processes and behavior of aquifer system.

Ex-situ remediation of arsenic from tapped groundwater, by suitable removal technologies, seems to be a short-term option to provide potable arsenic free groundwater for domestic use only. But this would prove expensive and unsustainable for supply of irrigation water. Nevertheless, ex-situ technologies can only remove the arsenic from tapped groundwater but not from the aquifer system. The advantage of this approach is that it can be located on site.

Although the use of surface water sources, as an alternative to the supply of treated contaminated groundwater, seems to be a logical proposition, it would require availability and supply of surface water flow and organized water supply system for ensuring supply of both drinking and irrigation water. To meet requirement of potable water in arsenic affected areas, this approach can prove to be a potential alternative in areas having thick populace. Tapping alternate safe aquifers, for supply of arsenic free groundwater, could also prove to be a logical proposition. This has also been explored in many areas on a local scale. However, this approach would require extensive studies and analyses for mapping of groundwater availability, freshwater reserves and to examine mobilization of arsenic in the aquifer, both on spatial and temporal scale, due to forcing perturbation.

Out of the above options, arsenic removal technologies and ex-situ treatment technique are being practiced widely both in India and Bangladesh, to provide potable water to the people in the arsenic affected areas after treatment of contaminated groundwater. Their large scale use in West Bengal, based on different operating principles, with various degrees of success and failure, has been reported.

6.1 Conventional Arsenic Removal Technologies

A variety of treatment technologies, based on oxidation, co-precipitation, adsorption, ion exchange and membrane process, has been developed and are available for removal of arsenic from contaminated water. However, question regarding the efficiency and applicability/ appropriateness of the technologies, remains, particularly because of low influent arsenic concentration and differences in source water composition. Some of these

methods are quite simple, but the disadvantage, associated with them, is that they produce large amounts of toxic sludge. This needs further treatment before disposal into the environment, besides the sustainability of these methods in terms of economic viability and social acceptability.

Despite having numerous arsenic removal devices, which have been developed, based on different working principles, very few plants could show satisfactory performance at the field level, both in terms of arsenic removal efficiency and in sustainable running. The major setbacks, with most of the devices, remain with the operation, maintenance, replacement and removal of used filters. The systems in O & M have been linked to the responsibility of suppliers, and they have shown satisfactory performance. In addition to the above devices, a number of other devices can be seen to be developed and applied in other countries. However, all the technologies are primarily based on five principles of arsenic removal: oxidation, co-precipitation, adsorption, ion exchange and membrane process. It is to be mentioned that the efficiency, effectiveness and sustainability of arsenic removal technologies depend on: (i) how simple the devise is in use, and operation & maintenance? (ii) what is its removal efficiency? (iii) how much is the outflow rate and cost? (iv) how eco-friendly the device is? and (v) what mechanism in operation and maintenance is devised ?

6.2 Innovative Technologies

Innovative technologies, such as permeable reactive barriers, phytoremediation, biological treatment and electrokinetic treatment, are also being used to treat arsenic contaminated water, waste water and soil. However, only a few applications of these technologies at full scale are available in the literature and additional treatment data are needed to determine their applicability and effectiveness in field condition. These technologies may be developed at full scale to treat arsenic contaminated aquifers.

6.3 Waste Disposal and Sludge Management

Waste disposal is an important consideration in the treatment selection process. Arsenic removal technologies produce several different types of waste, including sludge, brine streams, backwash slurries and spent media. These wastes have the potential for being classified as hazardous and can pose disposal problems.

Treatment of the slurry, obtained from arsenic removal process (from groundwater), is essential to make the slurry arsenic free so that it can be disposed without any hazard of the arsenic re-entering the aquifer system. The slurry may be transferred to plastic tanks and clear water from top drained off, further slurry added and top clear water drained off. The arsenic-rich sludge should be disposed in a controlled manner. The slurry can be dissolved in hydrochloric and/or sulphuric acid. Then it can be treated with metal scraps and/or other suitable reducing agents to convert arsenic of the slurry solution into arsine gas, which can be allowed to escape in the atmosphere (as a primary tentative measure). As a future research, depending on the total amount of arsenic to be treated and availability of fund, the arsenic generated may be absorbed in oxidative alkaline medium to produce sodium arsenate or calcium arsenate. The compounds may be consumed by glass industries.

According to the study conducted by AIIH&PH, arsenic rich sludge may be disposed by the following method:

- Disposal in on-site sanitation pits,
- Mixing with concrete in a controlled ratio,
- Mixing with clay for burning for brick manufacturing.

Apparently sludge disposal, management and detoxification have not received due priority in the plan of actions, initiated along with the device installation by the ARP manufacturers. Even no discernible programme is seen for the backwash which contains high level of As in media-washed water. It needs high priority in the installation programmes. Both the raw water, pumped out for ordinary use, and back washed water; require to be passing through a soak pit type of arrangement, to avoid surface contamination.

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