1. **Introduction:**

Arsenic (atomic number, 33; relative atomic mass 74.92) has some important chemical and physical. It is an element between a metal intermediate between a metal and non-metal, that is why it is called a metalloid or semi- metal. However, sometimes it is regarded as a metal from a toxicological point of view. It is also known as heavy metal substances (Mandal and Suzuki 2002). It belongs to group VA of the periodic table, below nitrogen and phosphorus. It can exist in four oxidation states: -3, 0, +3, and +5. Arsenite, As^{III}, and arsenates, As^{V}, are predominant oxidation states of arsenic, reducing and oxygenated condition (WHO, 2001; IARC, 2004). Arsenic is a ubiquitous metalloid element present in rock, soil, water and air

Table 1: Nomenclature and formulas of selected arsenic species in environment.

Compound	*Chemical Abstracts Service No.*	*Molecular formula*
Arsenic	7440-38-2	As
Arsenic trioxide	1327-53-3	As_2O_3
Arsenic pentoxide	1303-28-2	As_2O_5
Arsenic sulfide	1303-33-9	As_2S_3
Dimethylarsinic acid (DMA)	75-60-5	$(CH_3)_2AsO(OH)$
Monomethylarsonic acid (MMA)	124-58-3	$(CH_3)AsO(OH)_2$
Lead arsenate	10102-48-4	$PbHAsO_4$
Potassium arsenate	7784-41-0	KH_2AsO_4
Potassium arsenite	10124-50-2	$KAsO_2HAsO_2$

earth crusts. It is also abundant in the other biospheres regions (Sumi et al., 2011). Arsenic ranked as the 20th in abundance of all elements present in the Earth's crust and 14th in sea water. Arsenic compounds can be categorized as inorganic compounds, that means it is present without an arsenic-carbon bond. Arsenic form compounds when these are an arsenic-carbon bond. Numerous studies have suggested that inorganic arsenic in contaminated

drinking water is more toxic than their organic form (Balakumar and Kaur, 2009; Das et al., 2010). Inorganic arsenic remains in two valence states, trivalent (As^{III}), and pentavalent (As^{V}) form. In general, arsenite is more toxic than the arsenate (Landrigan, 1982; Manna et al., 2008).

Arsenic (As) is contaminated into soil and groundwater during weathering of and minerals followed by subsequent leaching and runoff. It can also be contaminated into soil and groundwater from anthropogenic sources. Arsenic pollution via the drinking ground-water, used for drinking purposes, has been experienced as a problem of global concern.

Table 2: physical and chemical properties of arsenic species (IARC, 1980)

Compound	*Melting point (°C)*	*Boiling point (°C)*	*Density (g/cm³)*	*Water solubility (g/l)*
As	613	–	5.727 at 14 °C	insoluble
As_2O_3	312.3	465	3.738	37 at 20 °C
As_2O_5	315 (decomposes)	–	4.32	1500 at 16 °C
As_2S_3	300	707	3.43	5×10^{-4} at 18 °C
$(CH_3)_2AsO(OH)$	200	–	–	829 at 22 °C
$CH_3AsO(OH)_2$	–	–	–	–
$PbHAsO_4$	720 (decomposes)	–	5.79	very slightly soluble
KH_2AsO_4	288	–	2.867	190 at 6 °C
$KAsO_2HAsO_2$	–	–	–	soluble

Arsenic contamination in drinking water has been reported from many countries like Taiwan, China, Argentina, Chile, Mexico, Cambodia, Thailand, Myanmar, Nepal, USA, (IARC., 2004). In India, a significant arsenic contamination in groundwater was detected in the year 1983 in the state West Bengal, In relation to this incidence some villagers of the concerned area were diagnosed to be suffering from arsenicosis due to drinking of arsenic contaminated water for a long time. (IARC, 2004) A large number of ground water sources in eastern India contain more than 50µg/L and some of those are above even 300µg/L arsenic. The acceptable

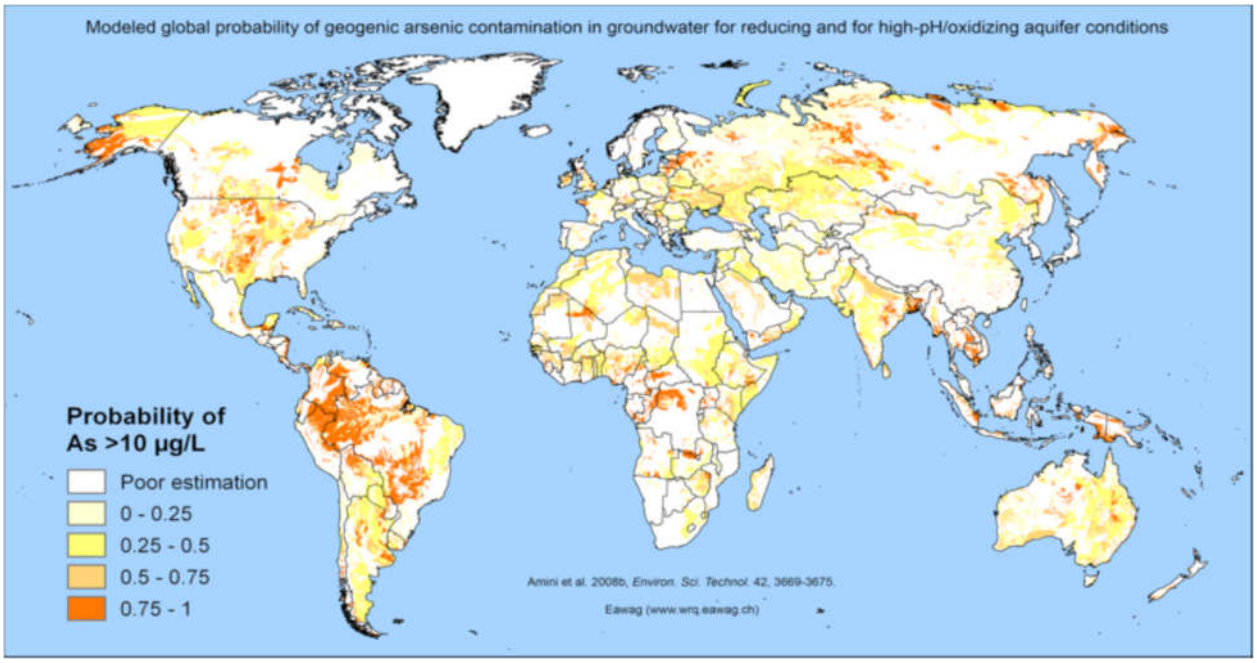

Fig 1: Arsenic contaminated region pointed in the world (Amini et al., 2008b)

level as defined by WHO for maximum concentrations of arsenic in safe drinking water is 0.01 mg/L. As because in arsenic contamination is more severe Bangladesh, the standard rate is slightly higher 0.05 mg/L (WHO., 2007). Only in West Bengal state, 26 million people are at risk due to drinking of arsenic-contaminated water (above 10µg/L) (Chakraborti et al., 2009). A total 83 blocks under 8 districts of West Bengal are affected with Arsenic contamination above permissible limit (> 0.05 mg/l). Similarly 43 4locks under 7 districts of West Bengal are affected with Arsenic Contamination above permissible limit (> 1.5 mg/l). The eastern part of India, Bangladesh and certain part of China are the most exposed areas in South-East Asia (Fatmi et al., 2009). Arsenic exposure also occurs from inhalation, absorption of arsenicals through the skin. Sometime arsenic in food occurs as relatively non-toxic organic compounds (arsenobentaine and arsenocholine). Seafood, fish, and algae are the richest organic sources of arseniz (Edmonds and Francesconi, 1987). Arsenic intake is higher

from solid foods than from liquids including drinking water (Thomas et al., 1997) (Tripathi et al.,1999). Organic and inorganic arsenic compounds may enter into the plant food-chain from agricultural products or from soil irrigated with arsenic contaminated water (Tamaki and Frankenberger, 1992). Arsenic is extensively reported as carcinogen which causes cancers of several organs including skin, lung, and bladder, prostate and liver. Information regarding arsenic-associated cardiovascular diseases is also available. The liver is a major target tissue of arsenic toxicity and hepatotoxicity may restrict. its chemotherapeutic efficacy of other drugs. It causes oxidative damage to normal cells leading to death or malignant transformation (soria et al., 2010). As (III) is thought to be the most toxic and act as a tumour promoter in the carcinogenic process (Cavigelli et al., 1996). Arsenic disrupts ATP production through several mechanisms. Arsenic inhibits several responsible metabolic enzymes i.e. pyruvate dehydrogenase (PDH) complex which catalyzes the oxidation of pyruvate to acetyl-CoA by NAD^+, causing cellular apoptotic events. Arsenic exposure results in the generation of reactive oxygen species (ROS) and oxidative stress. This oxidative stress may be of different types like cellular oxidative stress, where cellular substances and organelle materials are damaged by different free radicals. Sometime this stress may create systemic oxidative stress, where major organs become affected. The main reactive oxygen species are superoxide radical ($O_2^{\cdot-}$), hydroxyl radical ($OH^{\cdot}$), hydrogen peroxide (H_2O_2), hypochlorous acid (HOCl), singlet oxygen ($^1O_2$) and different lipid- peroxides. Out of these reactive oxygen species hydroxyl radical is regarded as more reactive than the others. Arsenic in cells clearly stimulates the production of hydrogen peroxide (H_2O_2) which might form reactive oxygen species and oxidative stress when it reacts with several biomolecules of multivalent metal ions. These metabolic interferences lead to a death from multi-system organ failure probably from necrotic cell death (Klaassen et al., 1993). Metal ions are

abundant in biological systems. When the H_2O_2 reacts with Fe^{+2}, produces a highly reactive hydroxyl radical that is more damaging for the organic molecules. Hydrogen peroxide acts not only as a damaging oxidant but also as a signalling molecule. This signalling process performs some essential physiological events. Hydrogen peroxide signalling can be either direct or indirect. The formation of lipid peroxides results in the destruction of the original lipid, leading to the loss of integrity of the membranes. Lipid peroxides are 100-1000 times more effective than hydrogen peroxide. A number of reactive lipid aldehydes, such as 4-hydroxy-2-alkenals and malondialdehyde, have been recognized as causative agents in cytotoxic processes (Uchida, 1999). Arsenic also inhibits some enzymes which are very much useful in hydrogen peroxide metabolism. Such enzymes are catalase (CAT), glutathione peroxidase (GPx), myeloperoxidase (MPO), xanthine oxidase (XO), superoxide dismutase (SOD), Peroxidase etc. The CAT activity, mRNA expression and protein levels were decreased by an exposure 5–20 μmol/l of sodium arsenite (Sun et al., 2006). Arsenic depressed GPx activity and the stable selenium content in liver (Pederson, 1991). A repast reveals that superoxide dismutase and xanthine oxidase activity decreased significantly on arsenic exposure. Although certain level of arsenic inhibits the peroxide metabolizing enzymes, catalase and glutathione peroxidase are shown to be involved the in defence against arsenite genotoxicity. Increasing the intracellular antioxidant level may have preventive or therapeutic effects in arsenic associated poisoning.

A few drugs like British Anti Lewisite (BAL), dimercaptosuccinic acid (DMSA) are available in the market as arsenic chelating agents. however, all of these therapeutic agents have appreciable side effects (Inns et al., 1990). Several components of herbal origin like quercetin (a flavonol, IUPAC name: 2-(3,4- dihydroxyphenyl)-3,5,7-trihydroxy-4*H*-chromen-4-one), combination of monoisoamyl dimercaptosuccinic acid (MiADMSA) and *Moringa*

oleifera Lam (family Moringaceae), *Hippophae rhamnoides L*., curcumin ((a diarylheptanoid, IUPAC name: (1E,6E)-1,7-Bis(4- hydroxy-3-methoxyphenyl)-1,6-heptadiene-3,5-dione)), *Emblica officinalis* Gaertn (a deciduous tree of the family Phyllanthaceae and synonym of *Phyllanthus emblica* L.) and some other phytochemicals have been Shown with their varied protection effects against arsenic-induced DNA breakage, hepatic damage, fibrosis and carcinogenesis (Chattopadhyay et al., 2011; Flora et al., 2012; Maiti et al., 2014; Srivastava et al., 2014). Recent studies in our laboratory elucidate the protective role of Green tea flavonoids (*Camellia sinensis,* family Theaceae) and vitamins (B12; cyanocobalamin+folate) against arsenic-induced gastro-intestinal damages (Acharyya et al., 2014a; Acharyya et al., 2015). Though an appreciable amount of phytotherapeutic research has been performed against arsenic to date, report is scanty on the effect of small organism/animal tissue extract against this toxicity.