Arsenic contamination and its toxicity in algae

Shefali Sharma

Department of Bioscience and Biotechnology, Banasthali University, Banasthali, 304022, Rajasthan, India shefali.micro@gmail.com

Abstract: Arsenic, a metalloid, is considered nonessential for human beings. However, excess amount of As is extremely toxic, leading to many pathological conditions that are consistent with oxidative damage, carcinogenic and mutagenic properties. Arsenic compounds widely enter in environment through food chain and water supply and excess of them producing worldwide pollution threat. Due to this, millions of people around the world are suffering from arsenic toxicity which leads to major health problems including liver damage, cancer, diabetes, skin lesions and hyperkeratosis. In water bodies the high amount of arsenic compounds enter in the algal cells. After accumulation arsenic interferes with algal metabolic processes which lead to impairment of photosynthesis, respiration, depletion in protein, carbohydrate, lipid and cell viability. Higher amount of arsenic also induced the generation of reactive oxygen species (ROS). These ROS can easily oxidize different macromolecules present in the algal cells. To counteract arsenic toxicity, algae have evolved complex protective mechanisms to mitigate the deleterious effects and repair the damage caused by ROS.

[Sharma S. Arsenic contamination and its toxicity in algae. *Researcher* 2015;7(4):1-6]. (ISSN: 1553-9865). http://www.sciencepub.net/researcher. 1

Key words: algae; arsenic; oxidative stress

1. Introduction

Arsenic is a ubiquitous metalloid which is found in air, soil, and water bodies (Duker et al., 2005). Due to its potential toxicity and carcinogenic properties, it poses the greatest threat to living organisms including human beings (Abernathy et al., 1999). The long term exposure to arsenic may cause severe diseases like diabetes, skin cancer, lung cancer, bladder cancer, hepatocellular carcinoma, hyperpigmentation, black foot disease, and melanokeratosis (Brown and Ross, 2002; Liu et al., 2001; Mukherjee et al., 2003; Saha, 2003; Wai et al., 2003). Arsenic contamination in water bodies mainly occurs due to natural processes and anthropogenic activities. The major sources of arsenic contaminants are parent rock, volcanic eruption, industrial and household waste discharge and fuel combustion (Adriano, 2001). Excessive use of arsenic compound in agriculture and forestry practices (as insecticide, herbicide, rodenticide and fungicide) is additional source of arsenic contamination to soil and water (Hathaway et al., 1991). Various sources of arsenic contamination are summarized in figure 1.

Arsenic concentration in natural water ranges from 0.5 to 5,000 μ gL⁻¹ (Smedley and Kinniburgh, 2002). Environmental arsenic exists in both organic and inorganic form. Inorganic arsenic forms are generally toxic whereas the organic forms are considered as non toxic (Gochfield, 1995). Inorganic arsenic usually exists in two forms namely, arsenite, As(III), and arsenate, As(V), which are interconverted through redox and methylation reactions (Duker et al., 2005; Eisler, 2004). Several studies have been conducted to understand the interaction of arsenic with various plants e.g. rice, fern, beans, spinach, tomato and red clover (Chakrabarty et al., 2009; Shri et al., 2009; Srivastava et al., 2005; Stoeva et al., 2003; Shaibur and Kawai, 2010; Barrachina et al., 1995; Mascher et al., 2002). However, studies concerning the impact of arsenic stress on algal system are relatively few. Unfortunately, limited efforts have been made to understand the response of microalgae to arsenic stress. Therefore, the present article summarizes the responses of microalgae to arsenic. **Arsenic contamination in natural water**

Arsenic contamination in natural water Arsenic contamination in drinking water is a

global concern particularly in South East Asia. In recent years arsenic level indiscriminately increases due to anthropogenic activities. In contaminated water high amount of inorganic arsenic is found, which mainly exist in As(III) and As(V) forms however small amount of DMA (dimethylarsenic acid), MMA (monomethylarsonic acid) and methylated forms also found. Different forms of arsenic found in aqueous system are given in table 1. As(III) is generally considered as most mobile and biologically toxic form. As(III) dominates under anaerobic condition whereas As(V)found stable under aerobic condition but more common than As(III) species (Duker et al., 2005). Arsenic accumulation in water bodies generally take adsorption/desorption place due to or oxidation/reduction process. In water sediments under oxidized conditions, arsenic may have precipitated by iron and manganese oxyhydroxide thus remain unavailable or available in less amount in water (Smedley and Kiniburgh, 2002; Kneebone et al., 2002; Nicholas et al., 2003). On the onset of reducing

condition, the reduction of As rich oxyhydroxide takes place that can leads to the release of arsenic, reduced oxyhydroxide ion, iron and manganese. In water ecosystem, microorganisms play active role in transformation of As into several biological forms. Oxidation of As(III) to As(V) also catalyzed by microoraganisms like bacteria and algae (Johnson, 1972; Myers et al., 1973; Andreae and Klumpp, 1979; Sanders and Windom, 1980; Sanders, 1983).

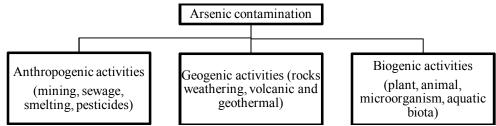


Figure 1. Sources of arsenic contamination in soil and water.

Effect of As on algae

Algae, the key primary producers found in an array of habitats ranging from fresh water to saline water, from hot spring to iceberg (Richmond et al., 2003; Leon-Banares, 2004). Any change due to the discharge of chemicals, such as, As and other metals in the aquatic environment is first noticed and encountered by these tiny organisms. Therefore, in water ecosystem, algae may serve as a useful biomarker of arsenic exposure and for stress and metabolic studies (Rachlin and Grosso, 1993; Lustigman et al., 1995).

Previously the interaction of algae with As has been reviewed by several researchers, including Rai et al. (1981), Yamaoka et al. (1992), Genter (1996), Abd-El-Monem et al. (1998), Mishra et al. (2008) and Bhattacharya and Pal (2010). As(V) is chemically similar to phosphate (PO_4^{3-}) , and readily taken up by phytoplankton by phosphate transporters (Sanders and Windom, 1980). It competes with phosphate in the formation of organic esters and upset the metabolic activities that require phosphorylation reactions. Plants and algae have ability to uptake more arsenate rather than PO₄ under PO₄ limited conditions (Knauer and Hemond, 2000). However, at high PO₄ concentration, the transport of As become reduced (Farhadi et al., 2013). The binding of As(III) to sulfhydryl groups of enzymes causes disruption of enzyme structure leading to enzyme inhibition (Cox, 1995). Additionally, arsenic toxicity causes lipid peroxidation, protein and enzyme oxidation, GSH

depletion, DNA oxidation and further generates reactive oxygen species (ROS) like, superoxide radical, hydroxyl radical, singlet oxygen etc (Wang et al., 1996; Lynn et al., 1997; Sharma et al., 2007). These ROS can easily oxidize various macromolecule of the cell (Mascher et al., 2002) and hence disrupts the dynamic equilibrium between the prooxidants and antioxidants and develop the condition known as oxidative stress (Scandalios, 1993).

It has been suggested that high level of arsenic can inhibit the growth rate of algae (Rai et al., 1981; Rana and Kumar, 1974; Whitton, 1970; Stauber and Florence, 1989; Genter et al., 1987, 1988; Genter, 1996). Arsenate induces a fluidization of liposome membrane of algal cell thus enhances the transport of toxicant across the membrane of algal cell and finally resulted into cell death (Tuan et al., 2008). Studies concerning the impact of As on algae found that the higher concentrations of As in water have been shown to sharply reduce the cell viability (Tuan et al., 2008) and to interfere with pentose phosphate pathway which leads to impairment in photosynthesis of alga (Srivastava et al., 2009; Zutshi et al., 2014). In addition to this, a significant reduction in protein and carbohydrate content of Phormidium laminosum and Scenedesmus acutus treated with different of concentrations As was also noticed (Abd-El-Monem et al., 1998; Bhattacharya and Pal, 2010). Moreover, the alleviation in MDA and H_2O_2 level of algal cells has been reported (Srivastava et al., 2009; Bhattacharya and Pal, 2010).

Table 1. Different forms of arsenic in aquatic environment.

Form	Arsenic compounds
Inorganic trivalent	Sodium arsenite, arsenic trioxide, arsenic trichloride
Inorganic pentavalent	Arsenic pentaoxide, arsenic acid, arsenates (calcium arsenate, lead arsenate, sodium
	arsenate)
Organic	Methylarsenic acid, arsanilic acid, dimethylarsinic acid and arsenobetaine.

To overcome the harmful effects of ROS, algal cells have developed a highly complex and intertwined antioxidant defense system which includes both enzymatic (superoxide dismutases, catalases, peroxidases) and non-enzymatic (ascorbate, carotenoids glutathione, and α -tocopherol) antioxidants (Pinto et al., 2003; Tripathi and Gaur, 2004; Tripathi et al., 2006). These components help in the re-establishment of redox-homeostasis by direct scavenging of ROS.

Enzymatic antioxidants

Enzymatic antioxidants actively work against ROS and neutralize their effect by converting them into less harmful products. These include superoxide dismutase, catalase and ascorbate peroxidase.

Superoxide dismutase

Superoxide dismutase (SOD) is one of the important antioxidant enzymes which act as first line of defense against ROS mediated damage (Bannister et al., 1987). SOD is a multimeric metalloenzyme that catalyze the conversion of superoxide radical (O2--) to dioxygen and H₂O₂ (Hassan, 1989). SOD enzymes contain metal ion cofactors that, depending on the isozyme, can be copper, zinc, manganese or iron. SODs have been categorized into three main types found: copper zinc superoxide dismutase (CuZnSOD), manganese superoxide dismutase (MnSOD) and iron superoxide dismutase (FeSOD). The CuZnSOD is found in cytosol, plastid, chloroplast and peroxisome. In plant, chloroplasts CuZnSOD is known to be the major form. However some plants also contain FeSOD in the chloroplasts (Kurepa et al., 1997). MnSOD is found in the mitochondria however its activity has also been reported in the chloroplast (Allen et al., 2007).

Catalase

Catalase (CAT) is a tetrameric heme containing enzyme that catalyze the dismutation of two H_2O_2 molecules to water and molecular oxygen (Hunt et al., 1998). CAT is found in all aerobic organisms and known to be localized in peroxisomes, glyoxisome, cytosol and in mitochondria. Catalase has low affinity for its substrate and it is found in the millimolar concentration range (Foyer and Noctor, 2000). This enzyme does not consume cellular reducing equivalents therefore known to be unique among H_2O_2 scavenging enzymes (Mallick and Mohn, 2000).

Ascorbate peroxidase

Ascorbate peroxidase (APX) are the main enzymes in the chloroplast that scavenges H_2O_2 , because catalase is absent in chloroplasts. A micromolar range of APX is found in the mitochondria, chloroplasts, cytosol, peroxisomes and apoplast. APX shows high affinity to its substrate and utilize ascorbate as an e- donor (Asada, 1992). APX neutralize ROS by reducing H_2O_2 into H_2O and monodehydroascorbate (MDA) (Noctor and Foyer, 1998). In chloroplast two types of APX, stroma-localized forms (sAPX) and thylakoid-bound (tAPX), are found. Along with PSI-associated SOD, tAPX acts as the first defense against ROS (Asada, 2006).

Non-enzymatic antioxidants

Non-enzymatic antioxidants also play a vital role to counteract the damage caused ROS. These non-enzymatic antioxidants scavenge the ROS to protect the algal cell. These include glutathione reductase, carotenoids and ascorbic acid.

Glutathione reductase

Glutathione reductase (GR) is a potential antioxidant, predominantly located in chloroplast however trace amount has also been located in mitochondria, cytosol and other plastids. The enzyme glutathione reductase catalyzes the reduction of glutathione disulfide (GSSG) back to glutathione (GSH) by utilizing electron from NADPH (Noctor and Foyer, 1998; Filomeni et al., 2002). GR play an important role against oxidative stress in almost all organisms (Pinto et al., 2003). Under oxidative stress, glutathionylation helps in preventing proteolysis, regulating gene transcription and cellular redox state, and changing protein turnover (Foyer and Noctor, 2000; Rouhier et al., 2008).

Carotenoids

Carotenoids are derivatives of geranylgeranyl diphosphate found in chloroplast. Carotenes (α -carotene, β -carotene) and xanthophylls (zeaxanthin, violaxanthin, neoxanthin) are the two types of carotenoids. Carotenes consist of linear or cyclic (β -ionone or ϵ -ionone rings) hydrocarbons whereas xanthophylls are derived from carotenes (Siefermann-Harms, 1987). Carotenoids, known to play vital role in photosynthesis and photoprotection, are synthesized by plants, algae, some bacteria and fungi. In algae and plants, most of the carotenoids are found in the chlorophyll binding proteins embedded in the thylakoid membrane (Baroli and Nivogi, 2000). They function in stabilizing the membrane, harvesting light energy, inhibiting lipid peroxidation, and quenching a triplet sensitizer (chl³) and singlet oxygen (Baroli and Niyogi, 2000).

Ascorbate (Ascorbic acid)

Ascorbic acid or vitamin C is an important, hydrophillic antioxidant mainly located in chloroplast but its small amount is also present in mitochondria, cytosol and in nucleus (Asada, 1999; Foyer and Nocter, 2005). It is normally produced outside the chloroplast but stored in chloroplast. Ascorbic acid has ability to scavenge H_2O_2 and free radicals (hydroxyl radicals, singlet oxygen) (Smirnoff and Wheeler, 2000). Ascorbic acid generally works as substrate for APX and finally converted into dehydroascorbic acid. In this process, APX utilize two molecules of Ascorbic acid as e- donor and catalyze the reduction of H_2O_2 into H_2O (Noctor and Foyer, 1998).

Conclusion

This review has provided a insight into the processes taking place when algal cells confront with arsenic stress. It also explained the role of antioxidant defense system in algae against arsenic. Algae have been used as biomarker tool against metal stress in several studies. Interaction of arsenic with algae leads to oxidative damage to algal cells. Antioxidant enzymes such as SOD and APX remove the superoxides and peroxides, so that they remain unavailable for reaction with As. Non enzymatic antioxidants help in scavenging the remaining reactive species that escaped enzymatic degradation. Since fewer studies were conducted to understand the effect of arsenic on algae therefore further studies will help to deeply understand the mechanisms of tolerance.

Acknowledgment:

I am thankful to Dr. Bhumi Nath Tripathi for encouragement.

Correspondence to:

Shefali Sharma

Stress Biotechnology Laboratory Department of Bioscience and Biotechnology,

Banasthali University, - 304022, Rajasthan, India Email: shefali.micro@gmail.com

References

- Abd-EL-Monem HM, Corradi MG and Gorbi G. Toxicity of copper and zinc to two strains of *Scenedesmus acutus* having different sensitivity to chromium. Environ Exp Bot 1998; 40:59-66.
- Abernathy CO, Liu YP, Longfellow D, Aposhian HV, Beck B, Fowler B, Goyer R, Menzer R, Rossman T, Thompson C and Waalkes M. Arsenic: health effects, mechanisms of actions, and research issues. Environ Health Perspect 1999; 107:593-597.
- 3. Adriano DC. Trace elements in terrestrial environments: biogeochemistry, bioavailability and risks of metals. Springer, New York 2001; p. 867.
- Allen MD, Kropat J, Tottey S, Del Campo JA and Merchant SS. Manganese deficiency in *Chlamydomonas* results in loss of Photosystem II and MnSOD function, sensitivity to peroxides, and secondary phosphorus and iron deficiency. Plant Physiol 2007; 143:263-277.
- 5. Andreae MO and Klumpp D. Biosynthesis and release of organoarsenic compounds by marine algae. Environ Sci Technol 1979; 13:738-741.

- Asada K. Ascorbate peroxidase a hydrogen peroxide scavenging enzyme in plants. Plant Physiol 1992; 85:235-241.
- 7. Asada K. Production and scavenging of reactive oxygen species in chloroplasts and their functions. Plant Physiol 2006; 141:391-396.
- Asada K. The water--water cycle in chloroplasts: Scavenging of active oxygens and dissipation of excess photons. Ann Rev Plant Physiol Plant Mol Biol 1999; 50:601-639.
- 9. Bannister JV, Bannister WH and Rotils G. Aspects of the structure, function and applications of superoxide dismutase. CRC Crit Rev Biochem 1987; 22:111-180.
- Baroli I and Niyogi KK. Molecular genetics of xanthophylls-dependent photoprotection in green algae and plants. Phil Trans R Soc Lond B Biol Sci 2000; 355:1385-1394.
- 11. Barrachina AC, Carbonell FB, Beneyto JM. Arsenic uptake, distribution, and accumulation in tomato plants-effect of arsenite on plant growth and yield. J Plant Nutrit 1995; 18:1237-1250.
- 12. Bhattacharya P and Pal R. Response of cyanobacteria to arsenic toxicity. J Appl Phycol 2010; 23:293-299.
- 13. Brown KG and Ross GL. Arsenic, drinking water, and health: A position paper of the American Council on Science and Health. Regul Toxicol Pharmacol 2002; 36:162-174.
- 14. Chakrabarty D, Trivedi PK, Misra P, Tiwari M, Shri M, Shukla D, Kumar S, Rai A, Pandey A, Nigam D, Tripathi RD and Tuli R. Comparative transcriptome analysis of arsenate and arsenite stresses in rice seedlings. Chemosphere 2009; 74:688-702.
- Cox MC. Arsenic characterization in soil and arsenic effects on canola growth. Ph.D. Dissertation, Louisiana State University, Baton Rouge, LA 1995.
- Duker AA, Carranza EJM and Hale M. Arsenic geochemistry and health. Environ Int 2005; 31:631-641.
- 17. Eisler R. Arsenic hazards to humans, plants, and animals from gold mining. Rev Environ Contam Toxicol 2004; 180:133-165.
- Farhadi R, Mohammad R, Salehibalashahri M and Gholami H. Effect of phosphorous on arsenic accumulation in two basil cultivars. J American Sci 2013; 9:64-66.
- 19. Filomeni G, Rotilio G and Ciriolo MR. Cell signaling and the glutathione redox system. Biochem Pharmacol 2002; 64:1057-1064.
- 20. Foyer CH and Noctor G. Oxygen processing in photosynthesis: regulation and signalling. New Phytol 2000; 146:359-388.
- 21. Foyer CH and Noctor G. Redox homeostis and

- 22. Genter RB, Cherry DS, Smith EP and, Cairns J Jr. Algal periphyton and community changes from arsenic stress in stream mesocosms. Hydrobiol 1987; 153:261-275.
- 23. Genter RB, Cherry DS, Smith P and Cairns J Jr. Attached-algal abundance altered by individual and treatments arsenic and pH. Environ Toxicol Chem 1988; 7:723-733.
- 24. Genter RB. Ecotoxicology of inorganic stresses. In: Algal Ecology: Freshwater Benthic Ecosystems, Stevenson RJ, Bothwell ML and Lowe RL (Eds., Academic Press, San Diego, 1996; pp. 403-468.
- 25. Gochfeld M. Chemical agents. In: Brooks S, Gochfeld M, Herzstein J, Schenker MJ (Eds.) Environ med: 1995; pp. 592-614.
- 26. Hassan HM. Microbial superoxide dismutases. Adv Genet 1989; 26:65-97.
- 27. Hathaway GJ, Proctor NH, Hughes JP and Fischman ML. Arsenic and arsine. In: Proctor NH, Hughes JP (Eds., Chemical hazards of the workplace. New York, Van Nostrand Reinhold, 1991; pp. 92-96.
- Hunt CR, Sim JE, Sullivan SJ, Featherstone T, Golden W, Von Kapp- Herr C, Hock RA, Gomez RA, Parsian AJ and Spitz DR. Genomic instability and catalase gene amplification induced by chronic exposure to oxidative stress. Cancer Res 1998; 58:3986-3992.
- 29. Johnson, DL. Bacterial reduction of arsenate in sea water. Nature 1972; 240:44-45.
- 30. Knauer K and Hemond H. Accumulation and reduction of arsenate by the freshwater green alga *Chlorella* sp. (chlorophyta). J Phycol 2000; 36:506-509.
- 31. Kneebone PE, O'Day PA, Jone N and Hering J. Deposition and fate of arsenic in iron- and arsenic-enriched reservoir sediments. Environ Sci & Tech 2002; 36:381-386.
- 32. Kurepa J, Hérouart D, Van Montagu M and Inzé D. Differential expression of CuZn- and Fe-superoxide dismutase genes of tobacco during development, oxidative stress, and hormonal treatments. Plant Cell Physiol 1997; 38:463-470.
- Leon-Banares R, Gonzdlez-Ballester D, Galvan A and Fernandez E. Transgenic microalgae as green cell-factories. Trends Biotechnol 2004; 22:45-52.
- 34. Liu T, Liu J, LeCluyse EL, Zhou YS, Cheng ML and Waalkes MP. Application of cDNA microarray to the study of arsenic-induced liver diseases in the population of Guizhou, China. Toxicol Sci 2001; 59:185-192.

- Lustigman B, Lee LH and Khalil A. Effects of nickel and pH on the growth of *Chlorella vulgaris*. Bull Environ Contam Toxicol 1995; 55:73-80.
- Lynn S, Lai HT, Gurr JR and Jan KY. Arsenite retards DNA break rejoining by inhibiting DNA ligation. Mutagen 1997; 12:353-358.
- Mallick M and Mohn FH. Reactive oxygen species response of algal cells. J Plant Physiol 2000; 157:183-193.
- Mascher R, Lippmann B, Holzinger S and Bergmann H. Arsenate toxicity: effects on oxidative stress response molecules and enzymes in red clover plants. Plant Sci 2002; 163:961-969.
- Mishra S, Srivastava S, Tripathi RD and Trivedi PK. Thiol metabolism and antioxidant systems complement each other during arsenate detoxification in *Ceratophyllum demersum* L. Aqua Toxicol 2008; 86:205-215.
- 40. Mukherjee SC, Rahman MM, Chowdhury UK, Sengupta MK, Lodh D, Chanda CR, Saha KC and Chakraborti D. Neuropathy in arsenic toxicity from groundwater arsenic contamination in West Bengal, India. J Environ Sci Health A Tox Hazard Subst Environ Eng 2003; 38:165-183.
- 41. Myers DJ, Heimbrook ME, Osteryoung J and Morrison SM. Arsenic oxidation state in the presence of micro-organism by differential pulse polarography. Environ Lett 1973; 5: 53-61.
- 42. Nicholas DR, Ramamoorthy S, Palace V, Spring S, Moore JN and Rosenzweig RF. Biogeochemical transformations of arsenic in circumneutral freshwater sediments. Biodegradation 2003; 14:123-137.
- 43. Noctor G and Foyer CH. Ascorbate and glutathione: Keeping active oxygen under control. Annu Rev Plant Physiol Plant Mol Biol 1998; 49:249-279.
- 44. Pinto E, Sigaud-Kutner TCS, Leitão, MAS, Okamoto OK, Morse D and Colepicolo P. Heavy metal-induced oxidative stress in algae. J Phycol 2003; 39:1-11.
- 45. Rachlin JW and Grosso A. The growth response of the green alga *Chlorella vulgaris* to combined cation exposure. Arch Environ Contamin Toxicol 1993; 24:16-20.
- 46. Rai LC, Gaur JP and Kumar HD. Phycology and heavy-metal pollution. Biol Rev Cambridge Phil Soc 1981; 56:99-151.
- 47. Rana BC and Kumar HD. The toxicity of arsenic to *Chlorella vulgaris* and *Plectonema boryanum* and its protection by phosphate. Phylcos 1974; 13:60-66.
- 48. Richmond A, Zhang CW and Zarmi Y. Efficient use of strong light for high photosynthetic

productivity: interrelationships between the optical path, the optimal population density and cell-growth inhibition. Biomol Eng 2003; 20:229-236.

- 49. Rouhier N, Lemaire SD and Jacquot JP. The role of glutathione in photosynthetic organisms: emerging functions for glutaredoxins and glutathionylation. Ann Rev Plant Biol 2008; 59:143-166.
- 50. Saha KC. Review of arsenicosis in West Bengal, India: A clinical perspective. Crit Rev Environ Sci Technol 2003; 33:127-163.
- 51. Sanders JG and Windom HL. The uptake and reduction of arsenic species by marine algae. Estuar Coast Mar Sci 1980; 10:555-567.
- 52. Sanders JG. Role of marine phytoplankton in determining the chemical speciation and biogeochemical cycling of arsenic. Can J Fish Aquat Sci 1983; 40:192-196.
- 53. Scandalios JG. Oxygen stress and superoxide dismutases. Plant Physiol 1993; 101:7-12.
- 54. Shaibur MR and Kawai S. Effect of arsenic on nutritional composition of japanese mustard spinach: An ill Effect of arsenic on nutritional quality of a green leafy vegetable. Nature and Sci 2010; 8:186-194.
- 55. Sharma I, Singh R and Tripathi BN. Biochemistry of arsenic toxicity and tolerance in plants. Biochem Cell Arch 2007; 7:165-170.
- 56. Shri M, Kumar S, Chakrabarty D, Trivedi PK, Malick S, Mishra P, Shukla D, Mishra S, Srivastava S, Tripathi RD and Tuli R. Effect of arsenic on growth, oxidative stress, and antioxidant system in rice seedling. Ecotoxicol Environ Safety 2009; 72:1102-1110.
- 57. Siefermann-Harms D. The light-harvesting and protective functions of carotenoids in photosynthetic membranes. Physiol Plantarum 1987; 69:561-568.
- 58. Smedley PL and Kinniburgh DG. A review of the source, behavior and distribution of arsenic in natural waters. Appl Geochem 2002; 17:517-568.
- 59. Smirnoff N and Wheeler GL. Ascorbic acid in plants: Biosynthesis and function. Crit Rev Plant Sci 2000; 19:267-290.
- 60. Srivastava AK, Bhargava P, Thapar R and Rai LC. Differential response of antioxidative

defense system of *Anabaena doliolum* under arsenite and arsenate stress. J Basic Microbiol 2009; 49:S63-72.

- 61. Srivastava M, Ma LQ and Singh N. Antioxidant responses of hyper- accumulator and sensitive fern species to arsenic. J Exp Bot 2005; 56:335-1342.
- 62. Stauber JL and Florence TM. Mechanism of toxicity of arsenic to the marine diatom *Nitzschia closterium*. Mar Biol 1989; 105:519-524.
- 63. Stoeva N and Bineva T. Oxidative changes and photosynthesis in oat plants grown in As-contaminated soil. Bulg. J Plant Phsiol 2003; 29:87-95.
- 64. Tripathi BN and Gaur, JP. Relationship between copper- and zinc-induced oxidative stress and proline accumulation in *Scenedesmus* sp. Planta 2004; 219:397-404.
- 65. Tripathi BN, Mehta SK, Amar A and Gaur JP. Oxidative stress in *Scenedesmus* sp. during shortand long-term exposure to Cu²⁺ and Zn²⁺. Chemosphere 2006; 62:538-544.
- 66. Tuan Le Q, Huong TT, Hong PT, Kawakami T, Shimanouchi T, Umakoshi H and Kuboi R. As(V) induces a fluidization of algal cell and liposome membrane. Toxicol In Vitro 2008; 22:1632-1638.
- 67. Wai CM, Wang JS and Yang MH. Arsenic contamination of groundwater, black foot disease, and other related health problems. Biogeochem. Environ Imp Trace Ele ACS Symp Ser 2003; 835:210-231.
- 68. Wang TS, Kuo CF, Jan KY and Huang H. Arsenite induces apoptosis in Chinese hamster ovary cells by generation of reactive oxygen species. J Cell Physiol 1996; 169:256-268.
- 69. Whitton BA. Toxicity of heavy metals of Chlorophyta from running waters. Arch Microbiol 1970; 72:353-360.
- 70. Yamaoka Y, Takimura O, Fuse H and Kamimura K. Effects of arsenic on the organic component of the alga *Dunaliella salina*. Appl Organomet Chem 1992; 6:357-362.
- Zutshi S, Bano F, Ningthoujam M, Habib K and Fatma T. Metabolic adaptations to arsenic-induced oxidative stress in *Hapalosiphon fontinalis*-339. Int J Innov Research Sci Eng Tech 2014; 3:9386-9394.

3/21/2015