



Arsenic: Source, Distribution, Toxicity and Bioremediation

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Ghanshyam Kumar Satyapal and Nitish Kumar

Abstract

Arsenic is ubiquitous in nature and a well-known toxic metalloid. There are four oxidation states (-3 , 0 , $+3$ and $+5$) of arsenic found in nature and most common forms are $+3$ and $+5$. The main sources of arsenic in nature are anthropogenic and natural activities. The natural sources include rocks, soils, seawater, arsenic-bearing minerals, volcanic emission and river originating from Himalaya. The anthropogenic activities include mining, smelting, use in herbicides and combustion of fossil fuels. The exposure to arsenic occurs mainly by consumption of arsenic contaminated drinking water or food. Arsenic is distributed all around the world beyond permissible limits in drinking water. Such type of contamination was reported in India, Thailand, Mexico, Chile, Argentina, China, Taiwan, USA Hungary and Bangladesh. The arsenic toxicity largely depends on its physical state and chemical form of the arsenic compound. Arsenic toxicity causes bladder, prostate, lung and skin cancer, rhagades, skin lesions, oxidative stress, mitochondrial damage and may interfere with the DNA methylation or DNA repair system. The ubiquitous nature of arsenic leads microorganism to evolve several plan of action for their survival in stressed environments. These strategies include arsenic oxidation, reduction, intracellular bioaccumulation and methylation. These strategies can be used in mitigation of the environmental arsenic from contaminated sites. In bacteria, the uptake of As(III) is mediated by GIpF whereas the A(V) uptake is facilitated by Pst and Pit membrane proteins. The oxidation of arsenic occurs in the periplasm of the bacteria and is regulated by arsenite oxidase (AoxAB) enzyme. The arsenate As(V) reduction occurs either in cytoplasm or in periplasm of the bacteria by arsenate reductase, ArsC or by arrA and arrB,

G. K. Satyapal · N. Kumar (✉)

Department of Biotechnology, Central University of South Bihar, Gaya, Bihar, India
e-mail: nitish@cub.ac.in

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respectively. The bioremediation is a low-cost and eco-friendly technique for the treatment of arsenic contaminated sites.

Keywords

Arsenic · Arsenic distribution · Anthropogenic · Arsenic toxicity · Arsenite oxidation · Arsenate reduction · Bioremediation

6.1 Arsenic

Arsenic (atom. no. 33) is a poisonous semi-metallic element and is broadly distributed all around the world. In the earth's crust, arsenic was ranked 20th for its abundance (Bahar et al. 2012; Zhang et al. 2002). There are four oxidation states (-3 , 0 , $+3$ and $+5$) of arsenic found in nature and most common forms are trivalent arsenite [$+3$, As(III)] and pentavalent arsenate [$+5$, As(V)] (Bahar et al. 2012; Mateos et al. 2006). The -3 species of arsenic, arsines and methylarsines, are generally unstable when present in air (Adriano 2001). As(III) is highly toxic in comparison with As(V) and has a high magnitude solubility which makes it difficult to remove from water (Bahar et al. 2012). More than 200 minerals occur in nature which contain arsenic, most of these minerals are in close association with metals such as Ni, Cu, Fe, Co, Ag, Cd and Pb. Most of the arsenic occur in minerals are sulphur conjugated for example, orpiment (As_2S_3), enargite (Cu_3AsS_4), realgar (As_4S_4) and arsenopyrite (FeAsS) (Drewniak and Sklodowska 2013). The anionic forms of arsenous acid and arsenic acid are the most common compounds of arsenite and arsenate, respectively. MMAs(V) (monomethylarsonic acid) and DMAs(V) (dimethylarsinic acid) are stable methylated form of inorganic arsenic in mammalian metabolites and are excreted in the urine. DMAs(V) and the sodium salts of MMAs(V) have been used as herbicides. For long time, DMAs(III) (dimethylarsinous acid) and MMAs(III) (monomethylarsonous acid) have been proposed intermediates in the arsenic metabolism (Hughes 2002).

6.2 Source of Arsenic

Arsenic is found everywhere in natural surroundings. It is a well-known toxic element for all forms of life (Banerjee et al. 2011; Tripathi et al. 2007; Villadangos et al. 2012). The main sources of arsenic in nature are anthropogenic and natural activities (Mandal and Suzuki 2002). Arsenic is released in the environment, primarily by natural activities like volcanic emission, weathering of minerals containing arsenic, etc. and due to anthropogenic activities such as burning of fossil fuels, smelting and mining (Bahar et al. 2012).

6.2.1 Natural

Arsenic is widely distributed in nature and is a rare crystal element. In rocks, the concentration of arsenic depends on the rock type, as higher concentration of arsenic is present in sedimentary rocks than igneous rocks (Mandal and Suzuki 2002). Generally, in sedimentary rocks, the range of mean value of arsenic concentrations is varied from 0.3 to 500 ppm and from 1.5 to 3.0 ppm in igneous rocks (Adriano 2001). The concentration of arsenic in rocks ranges from 0.5 to 2.5 mg kg⁻¹ (Kabata-Pendias 2010); however, higher concentrations were present in phosphorites and finer-grained argillaceous sediments (Mandal and Suzuki 2002). More than 200 minerals occur in nature which contain arsenic (Drewniak and Sklodowska 2013), of which approximately 20% are sulphosalts and sulphides, 20% comprises silicates, arsenides, oxides and elemental arsenic and remaining 60% are arsenates (Mandal and Suzuki 2002). In soil, the arsenic concentration in various countries varies considerably among the geographic regions, and the range of arsenic concentration are 0.1–40 mg kg⁻¹ and 1 to 50 mg kg⁻¹ (Mandal and Suzuki 2002). In seawater, the arsenic concentration ordinarily found in the range of 0.001–0.008 mg l⁻¹ (Johnson 1972). The high level of arsenic concentration was observed from an area nearby Alaska in well water samples after performing the arsenic speciation (Harrington et al. 1978), it shows inorganic As(III) comprises 3 to 39% and remaining were inorganic As(V) (Mandal and Suzuki 2002).

In unpolluted freshwater, arsenic concentrations range from 1 to 10 g l⁻¹; however, in the area of sulphide mineralization and mining it ranges from 100 to 5000 g l⁻¹ (Smedley et al. 1996). In air, the concentration of arsenic ranges from 0.4 to 30 ng m⁻³, thus human exposure to arsenic from air is generally very low (Mandal and Suzuki 2002).

6.2.2 Anthropogenic

Arsenic is released in the environment, primarily by natural activities like volcanic emission, weathering of minerals containing arsenic, etc. and due to anthropogenic activities such as burning of fossil fuels, smelting and mining (Bahar et al. 2012). Arsenic is extensively spread in water, land, and air through water run-off and wind-blown dust (Mateos et al. 2006). There are some more primary anthropogenic input derives from burning of fossil fuels in power plants based on oil- and coal-fire, discharge from metal smelters, combustion of solid waste from municipals and use of herbicides containing arsenic directly in agriculture and by industry (Zhang et al. 2002). Arsenic is naturally available in ores of copper, gold, lead and zinc and during the smelting process it can be released in the environment. The neighbouring ecosystem may become polluted by particulates and flue gases released from smelters (Adriano 2001).

The arsenic concentration in coal combustion residues and fly ash varies in the range from 100 to 1000 ppm (Adriano 2001). Therefore, generating power from combustion of coal and disposal of its fly ash may play a role in arsenic input in the

surroundings. Metal forms of arsenic are used in copper and lead alloys as an additive. Mainly compounds containing arsenic are being used in forestry and agriculture as silvicides, herbicides and pesticides. As(III) is a raw material for arsenical pesticides which includes sodium arsenite, calcium arsenate, lead arsenate, and organic arsenicals. These arsenicals are being used in the production of wood preservatives, algicides, fungicides, herbicides, insecticides and ship dips (Adriano 2001). For some animals, arsenic is an essential trace element and thus used as additive in the animal feed. Aluminium gallium arsenide or gallium arsenide crystals are used as a component of laser, light emitting diodes, semiconductors and in different types of transistors (Ratnaik 2003). The anthropogenic sources of arsenic are playing a crucial role for elevation of arsenic level in environment. The consequences occur by repeated use of arsenic in agricultural and industrial areas which results in increased levels of arsenic.

6.2.3 Source of Exposer to Arsenic

Humans can be exposed to arsenic by several ways. Probably, the most common way is ingestion of food or drinking water contaminated with arsenic (Ratnaik 2003; Zhang et al. 2002). The concentration of arsenic in worldwide aquifers ranges from <5 to 5000 µg/l and this leads it to become a human health concern globally due to its subsequent contamination in drinking water and food (Mandal and Suzuki 2002; Suttigarn and Wang 2005). According to the guidelines established by WHO, the permissible concentration of arsenic is 10 µg/l in drinking water but due to the economic reasons most of the developing countries accepted 50 µg/l, including Bangladesh (Bahar et al. 2012). Peoples are ingesting arsenic from contaminated water by agrochemical or industrial waste or from wells drilled in arsenic-rich geographic area (Adriano 2001). Arsenic contamination in fruit and vegetable crops occurs by uptake of arsenic from roots, soil or through spraying application. Seafood consumption from different sources may be another reason of arsenic intake by humans (Bishop and Chisholm 1966). Fish, algae and seafood are the sources of richest organic supplement (Edmonds and Francesconi 1987). Humans consuming seafood in their supplementary diet from different sources may be a reason for arsenic intake. The amount of arsenic in fish differs with their location and their species. The high level of arsenic is found in marine seafood products available commercially than terrestrial animals (Adriano 2001).

6.3 Arsenic Distribution

The arsenic level beyond permissible limits in drinking water around the world is the chief reason of arsenic toxicity. Such type of contamination was reported in India, Thailand, Mexico, Chile, Argentina, China, Taiwan, USA, Hungary and Bangladesh. In Nepal (2001), it came to notice that the groundwater of lower Plain area (Terai) is contaminated with arsenic (Chaurasia et al. 2012). In the Asian

countries, arsenic contamination was recorded from the Red River delta and from the Hanoi city. In addition, they reported high loads due to flooding and from delta plains of the Irrawaddy delta of Myanmar, in the Indus basin and Mekong valley in Vietnam and Cambodia. This indicates that the arsenic groundwater contamination is prone in lower flood plains and delta regions of south-eastern Asia (Saha 2009). The chronic mass toxicity of arsenic contamination in groundwater is a reason of large scale threat and there are more than 20 countries in the midst of it, including India (Chaurasia et al. 2012). However, due to drinking of arsenic contamination groundwater, the largest population affected by chronic arsenic toxicity in the world belongs to China, Bangladesh and India (Chaurasia et al. 2012; Saha 2009). In 1983, the first arsenic groundwater contamination was reported in West Bengal (Chakraborti et al. 2003; Ghosh and Singh 2009). In India, contamination of arsenic was first identified in Punjab, Haryana, Uttar Pradesh and Himachal Pradesh (Chaurasia et al. 2012). The groundwater contamination of arsenic and its effects on health were noticed in 1999 in Rajnandgaon district (Chhattisgarh, India). In 2002, in the western part of Bihar (India), the two villages of Bhojpur districts, Semaria Ojha Patti and Brisban, were reported for exceeding permissible level of 50 $\mu\text{g/l}$ arsenic contamination in groundwater (Chaurasia et al. 2012; Ghosh and Singh 2009; Nath et al. 2015). States of India, like Jharkhand, Uttar Pradesh, Bihar, West Bengal come under the flood plain of River Ganga; Manipur and Assam come under the flood plains of rivers Imphal and Brahmaputra, are reported above 50 $\mu\text{g/l}$ of arsenic contamination level in groundwater (Ghosh and Singh 2009). The availability of arsenic in India and Bangladesh depends geologically on nature. The sediment deposition took place 25,000 to 80,000 years ago, i.e., the Quaternary Period, in the arsenic affected areas. These sediments almost cover entire region of the river Ganga and it contains arsenic-rich pyrite (Adriano 2001).

6.4 Arsenic Toxicity

Arsenic is well-recognized to cause cancer in humans (Hughes 2002; Shi et al. 2004). The species of arsenic are toxic and bioactive (Zhang et al. 2002). Arsenic toxicity largely depends on its physical state and chemical form of the arsenic compound. Inorganic As(III) is highly toxic in comparison with the inorganic As(V), however inorganic As(V) is more toxic than the methylated form of arsenic (Adriano 2001). The exposure to arsenic in drinking water even in low concentrations can result in many types of cancer like prostate, bladder, lung and skin. The ingestion of arsenic in low levels can also result in non-cancerous effects such as diabetes, anaemia and developmental, cardiovascular, reproductive, neurological and immunological. The exposure to high dose of arsenic for short-term may result in many adverse health problems (Zhang et al. 2002). The arsenic toxicity also leads to skin lesions, rhagades, and damage to digestive, circulatory, respiratory and mucous membrane (Rehman et al. 2010). The most prevalent form of arsenic in oxic condition is As(V) and its toxicity depends on its tetrahedral oxyanion structure which resembles phosphate, thus As(V) is involved in uncoupling the intermediary

metabolic conversions, for example in oxidative phosphorylation (Kruger et al. 2013; Villadangos et al. 2014). As(V) may interfere in the methylation state or repair system of DNA, oxidative stress, promotion of cell proliferation, telomerase activities and inhibition of p53 inhibit activation of transcription factors by interfering with signal transduction pathways (Butt and Rehman 2011; Shakoori et al. 2010). As(III) is highly toxic in comparison with As(V), as it can react with free thiols which results in disturbing the redox homeostasis. Additionally, As(III) stimulates the production of reactive oxygen species (ROS), subsequently ROS damages the DNA as well as proteins (Villadangos et al. 2014). The presence of arsenic during DNA replication shown to induce sister chromatid exchanges and chromosomal aberrations, and arsenic is mutagenic according to the results of genotoxicity studies (Shakoori et al. 2010). 200 enzymes can be inactivated by arsenic toxicity, mostly the enzymes that are involved in cellular energy pathways. It generates ROS which in turn exerts its toxicity causing DNA damage and lipid peroxidation (Ratnaik 2003). Arsenic also exerts its toxicity on plants involving in their metabolisms. Plants can easily take up As(III) and As(V) by their root cells. The roots tissue of plants is the first one to interact with arsenic, which in turn results in inhibition of root proliferation and extension. After taken up from the roots, the arsenic is translocated to the shoots, where it arrests or slows down the biomass accumulation and expansion by inhibiting the plant growth. Arsenic also leads plants to compromise in their reproductive capacity by losing fruit production, yield as well as plant fertility (Garg and Singla 2011). Both, As(III) and As(V), play role in disruption of plant metabolism by distinct mechanism of action. As(V) is structurally resembles phosphate, it can disrupt phosphate dependent metabolisms. It competes with phosphate uptake in plants leading to imbalance of phosphate supply. As (V) forms unstable and short-lived adducts by competing with phosphate in phosphorylation reactions. The enzymes that contain closely spaced dithiol co-factors or cysteine residues are inactivated by As(III) as it binds with the thiols group of the enzymes, as As(III) is a dithiol reactive compound (Finnegan and Chen 2012).

6.5 Bioremediation of Arsenic

Bioremediation is the process of eliminating toxic waste from environment by exploiting biological agents (Ahemad 2012). The metals present in industrial effluents can be removed by conventional methods like chemical reduction or oxidation, chemical precipitation, ion exchange, filtration, evaporation recovery, membrane technologies, electrochemical treatment and reverse osmosis (Ahluwalia and Goyal 2007). When the concentration of metals in solutions is 1–100 mg/l then these conventional methods are ineffective or extremely expensive. So, there is an urgent need to develop an eco-friendly, cost-effective and innovative technique for elimination of the metals from contaminated water (Rehman et al. 2010). The microorganisms can transform the oxidation state of arsenic having different solubility properties, thus performing an important role in biochemical cycle of arsenic (Silver and Phung 2005). In metal-stressed environment, the bacteria have

developed several mechanisms for their survival to reduce the uptake of heavy metals (Nies 1999). Bioremediation of arsenic by microorganisms involves their intracellular bioaccumulation, methylation, reduction and oxidation (Satyapal et al. 2016). The arsenic can be utilized in metabolism of bacterial strains which are arsenic resistant for producing energy by chemoautotrophic As(III) oxidation (Santini et al. 2000). In aerobic respiration, bacteria can utilize As(V) as terminal electron acceptor (Ahmann et al. 1994; Stolz and Oremland 1999).

6.5.1 Arsenic Uptake and Extrusion System in Bacteria

Numerous mechanisms have been adopted by bacteria to survive in metal stress, to tolerate the heavy metal uptake and to protect themselves from cell homeostasis caused by heavy metals. These mechanisms include the efflux of metal ions, reduction of heavy metals and metal ion complexation and accumulation inside the cell (Ahemad 2012). In prokaryotes, the uptake of arsenic is due to its molecular similarity with the substrates of membrane transporter proteins. In aqueous solutions, at optimum pH, As(III) is structurally similar to glycerol and exists as As(OH)₃. However, As(V) is taken up by phosphate transporter proteins of membrane as it is a structural analogue of phosphate (Maciaszczyk-Dziubinska et al. 2012). The GlpF, an aquaglyceroprotein, involves in the transport of As(III) across the cell membrane. Pst and Pit are phosphate transporter proteins facilitating AsV uptake in bacteria (Kruger et al. 2013). In bacteria, arsenic extrusion is done by an arsenite-translocating ATPase. The three gene operon, *arsRBC*, present in bacteria encoding ArsB, involve in arsenite extrusion. Majority of the bacteria use ArsB alone to extrude arsenite. Some bacteria have the five gene operon, *arsRDABC*, encoding for ArsA and ArsB as components of ArsAB ATPase complex (Rosen 2002; Satyapal et al. 2016). The arsenite permease Acr3, an arsenic resistance transporter, is a member of the bile/arsenite/riboflavin transporter (BART) superfamily. BART includes members of archaea, fungi, and bacteria. Acr3 are more widely available than ArsB and are small-sized proteins (Villadangos et al. 2012).

6.5.2 Bacterial Arsenite Oxidation

The As(III) can be oxidized by bacteria into As(V), a less toxic form of arsenic. The *Centibacterium arsenoxidans* contains *aoxABCD* operons consisting four adjoining genes encodes for arsenite oxidase, which is involve in arsenite oxidation (Satyapal et al. 2016). The larger subunit of arsenite oxidase is also known as *aoxB/asoA/aioA* and the small subunits are called as *aoxA/asoB/aioB* (Silver and Phung 2005; Van Lis et al. 2013). In *H. arsenoxidans*, *aox* operon is regulated by *aoxR* gene product and *aoxS* gene product is a sensor kinase, both are involved in quorum sensing (Koechler et al. 2010). The expression of *aox* operon leads to the synthesis of arsenate oxidase, AoxAB complex, which is then exported to the periplasm of the bacteria by a Tat (Twin-Arginine Translocation) protein. In periplasm of the bacteria

the AoxAB complex involves in oxidation of As(III) to As(V) (Silver and Phung 2005).

6.5.3 Bacterial Arsenate Reduction

The *ars* operon is involved in the reduction of As(V) to As(III). The *arsRBC*, a three gene operon, is present in *E. coli* genome, whereas a five gene operon, *arsRDABC*, is reported in *S. aureus* (Rosen 2002). The transcriptional regulator (*arsR*) is encoded by *arsR*, whereas *arsA* and *arsB* encode *arsA* and *arsB*, respectively. These are components of ArsAB, ATPase, an arsenite efflux pump and *arsC* encodes for an arsenate reductase (*arsC*) (Arsène-Ploetze et al. 2010). The ArsD encoded by *ArsD* acts as arsenic chaperone. The *ars* operons, *arsRBC* and *arsRDABC*, may present in a single strain, as observed in *T. arsenitoxidans* 3As (Anderson and Cook 2004). The arsenate reduction in prokaryotes is of two types: periplasmic and cytoplasmic arsenate reduction. Pst and Pit are membrane transporter proteins involve in the uptake of As(V). The As(V) is then reduced to As(III) by ArsC, arsenate reductase (Kruger et al. 2013). The As(III) is then transferred by arsenic chaperon, ArsD, from the glutathione-bound complex to the small subunit, ArsA, of the ArsAB complex which in turn activates the ArsAB pump. The As(III) is then extruded out through the ArsAB pump (Satyapal et al. 2016). The As(V) reduction in periplasm of bacteria is mediated by the components of respiratory arsenate reductase, i.e., *arrA* and *arrB* encoded by the *arr* operon (Kruger et al. 2013).

6.5.4 Arsenic Methylation in Bacteria

Arsenic methylation is very less known in bacterial system; however, it was considered as a detoxification process. In methylation process, the intermediate compounds or methylated products are more toxic for the eukaryotic cell lines in comparison with the inorganic forms of arsenic (Kruger et al. 2013; Stolz et al. 2006). In methylation process, the methylated arsenicals are more toxic because of increase in their volatility (Kruger et al. 2013). The bacteria can perform enzymatic activity for the methylation of arsenic by involving a *S*-adenosylmethionine (SAM) and methyltransferase enzyme. The methyltransferase, ArsM/AS3MT, is an *arsM* gene product which methylates As(III) into a MMAs(III) (monomethyl arsenite/MMA³⁺) (Obinaju 2009; Satyapal et al. 2016). This MMAs(III) is then methylated to form DMAs(III) (dimethyl arsenite/DMA³⁺) followed by a final product as TMAs(III) (trimethyl arsine). These arsenicals can be extruded out from the cell through the process of diffusion (Hughes 2002). Though, the demethylation mechanism of arsenic in prokaryotes is not well understood. The demethylation of arsenic compounds (mono and dimethyl) is observed in some microorganisms, e.g., *Alcaligenes*, *Pseudomonas* and *Mycobacterium* species (Stolz et al. 2006).

6.6 Conclusion

Arsenic is found worldwide and is one of the most toxic pollutants found in nature. It is naturally found and distributed in rocks, rivers, soils, sea, air and water. The anthropogenic activities are the big reason in distribution of arsenic at large scale. Nevertheless, there are some bacterial systems that involve in detoxification of toxic arsenic in present nature. The bacteria have developed tolerance against arsenic by different mechanisms. These mechanisms of oxidation, reduction and methylation may play a great role in bioremediation of arsenic. The genes regulating these mechanisms may play a great role in developing a cheap and cost-effective model for arsenic bioremediation.

References

- Adriano DC (2001) Arsenic trace elements in terrestrial environments. Springer, New York, pp 219–261
- Ahemad M (2012) Implications of bacterial resistance against heavy metals in bioremediation: a review. *J Inst Integr Omics Appl Biotechnol* 3(3):39–46
- Ahluwalia SS, Goyal D (2007) Microbial and plant derived biomass for removal of heavy metals from wastewater. *Bioresour Technol* 98(12):2243–2257
- Ahmann D, Roberts AL, Krumholz LR, Morel FM (1994) Microbe grows by reducing arsenic. *Nature* 371(6500):750–750
- Anderson CR, Cook GM (2004) Isolation and characterization of arsenate-reducing bacteria from arsenic-contaminated sites in New Zealand. *Curr Microbiol* 48(5):341–347
- Arsène-Pløetze F, Koechler S, Marchal M, Coppée J-Y, Chandler M, Bonnefoy V et al (2010) Structure, function, and evolution of the *Thiomonas* spp. genome. *PLoS Genet* 6(2):e1000859
- Bahar MM, Megharaj M, Naidu R (2012) Arsenic bioremediation potential of a new arsenite-oxidizing bacterium *Stenotrophomonas* sp. MM-7 isolated from soil. *Biodegradation* 23(6):803–812
- Banerjee S, Datta S, Chattopadhyay D, Sarkar P (2011) Arsenic accumulating and transforming bacteria isolated from contaminated soil for potential use in bioremediation. *J Environ Sci Health A* 46(14):1736–1747
- Bishop R, Chisholm D (1966) Arsenical spray residues on apples and in some apple products. *Can J Plant Sci* 46(3):225–231
- Butt AS, Rehman A (2011) Isolation of arsenite-oxidizing bacteria from industrial effluents and their potential use in wastewater treatment. *World J Microbiol Biotechnol* 27(10):2435–2441
- Chakraborti D, Mukherjee SC, Pati S, Sengupta MK, Rahman MM, Chowdhury UK et al (2003) Arsenic groundwater contamination in middle ganega plain, Bihar, India: a future danger? *Environ Health Perspect* 111(9):1194–1201
- Chaurasia N, Mishra A, Pandey S (2012) Finger print of arsenic contaminated water in India—a review. *J Forensic Res* 3(10):1–4
- Drewniak L, Sklodowska A (2013) Arsenic-transforming microbes and their role in biomining processes. *Environ Sci Pollut Res* 20(11):7728–7739
- Edmonds J, Francesconi K (1987) Transformations of arsenic in the marine environment. *Experientia* 43(5):553–557
- Finnegan P, Chen W (2012) Arsenic toxicity: the effects on plant metabolism. *Front Physiol* 3:182
- Garg N, Singla P (2011) Arsenic toxicity in crop plants: physiological effects and tolerance mechanisms. *Environ Chem Lett* 9(3):303–321
- Ghosh N, Singh R (2009) Groundwater arsenic contamination in India: vulnerability and scope for remedy

- Harrington JM, Middaugh JP, Morse DL, Housworth J (1978) A survey of a population exposed to high concentrations of arsenic in well water in Fairbanks, Alaska. *Am J Epidemiol* 108 (5):377–385
- Hughes MF (2002) Arsenic toxicity and potential mechanisms of action. *Toxicol Lett* 133(1):1–16
- Johnson DL (1972) Bacterial reduction of arsenate in sea water. *Nature* 240(5375):44–45
- Kabata-Pendias A (2010) Trace elements in soils and plants. CRC Press, Boca Raton
- Koehler S, Cleiss-Arnold J, Proux C, Sismeiro O, Dillies M-A, Goulhen-Chollet F et al (2010) Multiple controls affect arsenite oxidase gene expression in *Herminiimonas arsenicoxydans*. *BMC Microbiol* 10(1):53
- Kruger MC, Bertin PN, Heipieper HJ, Arsène-Ploetze F (2013) Bacterial metabolism of environmental arsenic—mechanisms and biotechnological applications. *Appl Microbiol Biotechnol* 97 (9):3827–3841
- Maciaszczyk-Dziubinska E, Wawrzycka D, Wysocki R (2012) Arsenic and antimony transporters in eukaryotes. *Int J Mol Sci* 13(3):3527–3548
- Mandal BK, Suzuki KT (2002) Arsenic round the world: a review. *Talanta* 58(1):201–235
- Mateos LM, Ordóñez E, Letek M, Gil JA (2006) *Corynebacterium glutamicum* as a model bacterium for the bioremediation of arsenic. *Int Microbiol* 9(3):207–215
- Nath A, Shailendra K, Priyanka S, Anshu A, Singh M (2015) Arsenic in tube well water in six blocks of Supaul District, Bihar. *IOSR J Environ Sci Toxicol Food Technol* 9(1):05–08. <https://doi.org/10.9790/2402-09110508>
- Nies DH (1999) Microbial heavy-metal resistance. *Appl Microbiol Biotechnol* 51(6):730–750
- Obinaju BE (2009) Mechanisms of arsenic toxicity and carcinogenesis. *Afr J Biochem Res* 3 (5):232–237
- Ratnaik RN (2003) Acute and chronic arsenic toxicity. *Postgrad Med J* 79(933):391–396
- Rehman A, Butt SA, Hasnain S (2010) Isolation and characterization of arsenite oxidizing *Pseudomonas lubricans* and its potential use in bioremediation of wastewater. *Afr J Biotechnol* 9 (10):1493–1498
- Rosen BP (2002) Biochemistry of arsenic detoxification. *FEBS Lett* 529(1):86–92
- Saha D (2009) Arsenic groundwater contamination in parts of middle Ganga plain, Bihar. *Curr Sci* 97(6):753–755
- Santini JM, Sly LI, Schnagl RD, Macy JM (2000) A new chemolithoautotrophic arsenite-oxidizing bacterium isolated from a gold mine: phylogenetic, physiological, and preliminary biochemical studies. *Appl Environ Microbiol* 66(1):92–97
- Satyapal G, Rani S, Kumar M, Kumar N (2016) Potential role of arsenic resistant bacteria in bioremediation: current status and future prospects. *J Microb Biochem Technol* 8(3):256–258
- Shakoori FR, Aziz I, Rehman A, Shakoori A (2010) Isolation and characterization of arsenic reducing bacteria from industrial effluents and their potential use in bioremediation of wastewater. *Pak J Zool* 42(3):331
- Shi H, Shi X, Liu KJ (2004) Oxidative mechanism of arsenic toxicity and carcinogenesis. *Mol Cell Biochem* 255(1–2):67–78
- Silver S, Phung LT (2005) Genes and enzymes involved in bacterial oxidation and reduction of inorganic arsenic. *Appl Environ Microbiol* 71(2):599–608
- Smedley P, Edmunds W, Pelig-Ba K (1996) Mobility of arsenic in groundwater in the Obuasi gold-mining area of Ghana: some implications for human health. *Geol Soc Lond Spec Publ* 113 (1):163–181
- Stolz JF, Oremland RS (1999) Bacterial respiration of arsenic and selenium. *FEMS Microbiol Rev* 23(5):615–627
- Stolz JF, Basu P, Santini JM, Oremland RS (2006) Arsenic and selenium in microbial metabolism. *Annu Rev Microbiol* 60:107–130
- Suttigarn A, Wang Y-T (2005) Arsenite oxidation by *Alcaligenes faecalis* strain O1201. *J Environ Eng* 131(9):1293–1301
- Tripathi RD, Srivastava S, Mishra S, Singh N, Tuli R, Gupta DK, Maathuis FJ (2007) Arsenic hazards: strategies for tolerance and remediation by plants. *Trends Biotechnol* 25(4):158–165

- Van Lis R, Nitschke W, Duval S, Schoep-Cothenet B (2013) Arsenics as bioenergetic substrates. *Biochimica et Biophysica Acta* 1827(2):176–188
- Villadangos AF, Fu H-L, Gil JA, Messens J, Rosen BP, Mateos LM (2012) Efflux permease CgAcr3-1 of *Corynebacterium glutamicum* is an arsenite-specific antiporter. *J Biol Chem* 287(1):723–735
- Villadangos AF, Ordóñez E, Pedre B, Messens J, Gil JA, Mateos LM (2014) Engineered coryneform bacteria as a bio-tool for arsenic remediation. *Appl Microbiol Biotechnol* 98(24):10143–10152
- Zhang W, Cai Y, Tu C, Ma LQ (2002) Arsenic speciation and distribution in an arsenic hyperaccumulating plant. *Sci Total Environ* 300(1–3):167–177