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Arsenic in Plants: A synopsis of its Acquisition, Transport, Metabolism, Impact, and Detoxification

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ABSTRACT

Arsenic (As) is a ubiquitous element that ranks 20th in abundance in the earth's crust, 14th in seawater, and 12th in the human body. For centuries, this element has been at the heart of heated debates and controversies that have shaped human history. Its discovery by Albertus Magnus in 1250 A.D. marked a turning point in our understanding of the world and sparked a fascination that endures to this day. Arsenic, a notorious poison, that elicits a fearful response in most people, is now recognized to be one of the world's greatest environmental hazards, threatening the lives of several hundred million people. This article provides a brief overview of how plants acquire, transport, metabolize, and detoxify arsenic. It also explores the impact of arsenic on plants and their environment.

Key Words :- Arsenic; metabolism; oxidation states; toxicity

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INTRODUCTION

Arsenic toxicity is one of the major health issues throughout the world. Approximately 108 countries that account for more than 230 million people worldwide are at high risk of arsenic poisoning mainly through drinking water and diet (Banerjee *et al.*, 2023). Arsenic is a metalloid which is classified as a member of group VA in the periodic table. It exists in nature in the oxidation states +V (arsenate), +III (arsenite), 0 (arsenic), and -III (arsine) (Bissen and Frimmel, 2003). Soils contain both organic and inorganic arsenic species. Inorganic arsenic occurs in the environment as arsenite and arsenate, which are the two most common forms (Jimenez *et al.*, 2023).

Arsenic acquisition, transport, and metabolism in plants:

As an element, it can exist in the environment in both inorganic and organic forms. Of the two inorganic forms, the oxidized arsenate (AsV) predominates in aerobic environments, while the reduced arsenite (AsIII) is the predominant form in anaerobic environments, such as flooded rice paddy fields. As (V) is an analogue of inorganic phosphate (Pi) and is easily transported across the plasma-lemma by Pi transporter (PHT) proteins (Ullrich-Eberius *et al.*, 1989; Meharg and Macnair, 1990, 1991, 1992; Wu *et al.*, 2011). As (V) and Pi compete for uptake through the same transport systems in As hyper-accumulators (Wang *et al.*, 2002; Tu and Ma, 2003), As-tolerant non-hyper-accumulators (Meharg and Macnair, 1992; Bleeker *et al.*, 2003) and As-sensitive non-accumulators (Abedin *et al.*, 2002; Esteban *et al.*, 2003). Under low Pi conditions, As (V) may outcompete Pi for entry into the plant, amplifying Pi deprivation symptoms. Pi fertilization has been found to have

a protective effect on plants, including the hyper-accumulator *P. vittata*, against As (V) toxicity (Tu and Ma, 2003). Additionally, increasing or decreasing the amount or activity of the PHT protein at the plant's plasma membrane through genetic manipulation can respectively increase or decrease the uptake of Pi and As, thereby also increasing or decreasing the toxicity of As (V). (Shin *et al.*, 2004; González *et al.*, 2005; Catarecha *et al.*, 2007; Wu *et al.*, 2011).

Once inside the cell, As(V) can be readily converted to As (III), the more toxic of the two forms. The As(V) and As(III) both disturb plant metabolism, but through distinct mechanisms. The As(V) being a chemical analogue of phosphate, disrupts at least some phosphate-dependent aspects of metabolism. It can compete with phosphate during phosphorylation reactions, leading to the formation of As(V)-adducts that are often unstable and short-lived. The creation of As(V)-ADP and its quick auto-hydrolysis initiate a futile cycle that separates oxidative phosphorylation from photophosphorylation, reducing the cells' capacity to make ATP and carry out regular metabolism. As (V) can likely travel freely across cellular compartments in a plant cell by passing through different Pi transporters and internal membranes. As an illustration, As (V) has been shown to function as a co-substrate for three mitochondrial dicarboxylate transporters, which are proteins found in the inner mitochondrial membrane and are in charge of dicarboxylate exchange between the organelle matrix and the cytosol with co-substrates like Pi (Palmieri *et al.*, 2008). This quick mobility would cause As to quickly rebalance throughout the cell, exposing the toxicant to every aspect of cellular metabolism.

As (III) can enter the root cells through nodulin 26-like intrinsic proteins (NIPs, Meharg and Jardine, 2003; Bienert *et al.*, 2008; Isayenkov and Maathuis, 2008; Ma *et al.*, 2008). These proteins are a part of the aquaporin family, which consists of major intrinsic proteins. In rice roots, the major As(III) uptake protein is the OsNIP2;1/OsLsi1 silicon transporter, while As(III) efflux from root cells to xylem occurs through the OsLsi^2 silicon transporter (Ma *et al.*, 2008). In yeast, the transportation of As (III) into cells is facilitated by various types of proteins. However, the most prevalent mode of As (III) uptake in yeast is through hexose permeases, as reported in a study by Liu et al. in 2004. It is still unknown if plants have a way for As (III) to enter their cells through proteins. However, in species that hyper-accumulate As, such as *P. vittata*, As (III) is not stored in the roots but is quickly transported through the xylem to the fronds. In the fronds, As (III) is confined as free As (III) in the vacuole, where it accumulates to extremely high levels. Arsenic exposure can induce the production of reactive oxygen species, leading to the production of antioxidant metabolites and numerous enzymes involved in antioxidant defense mechanisms. Oxidative carbon metabolism, amino acid and protein relationships, and nitrogen and sulphur assimilation pathways are also impacted by As exposure.

According to several studies (Marin *et al.*, 1992; Quaghebeur and Rengel, 2003; Raab *et al.*, 2005, 2007b; Xu *et al.*, 2007), methylated As species are present in plants. Moreover, a variety of organisms can metabolize arsenic to produce arseno-choline, arseno-betaine, and arseno-sugars. Certain terrestrial plants have been shown to contain these chemicals (Dembitsky and Levitsky, 2004).

It has been reported that when plants are supplied with As (V), typically more than 90% of the As in the roots and in the shoots is found to be in the form of As (III) (Pickering *et al.*, 2000; Dhankher *et al.*, 2002; Xu *et al.*, 2007). This shows that As (V) is readily reduced to As (III) by plants. It is acknowledged that the primary As detoxification mechanism in plants begins with this reduction (Pickering *et al.*, 2000; Schmöger *et al.*, 2000). As (V) can be reduced both enzymatically and non-enzymatically to As (III). In the non-enzymatic pathway, two molecules of GSH are able to reduce As (V) to As (III) (Delnomdedieu *et al.*, 1994). The oxidation of GSH is *via* the formation of a disulphide bond, producing a GSH dimer (GSSG; Delnomdedieu *et al.*, 1994), which can be rapidly

recycled to two GSH molecules by GSH reductase (Foyer and Noctor, 2011). While As (V) reduction can occur non-enzymatically, the enzymatic rate is much higher (Duan *et al.*, 2005). Arsenate reductase (ACR) is an enzyme that was initially isolated from bacteria and yeast that can directly reduce As (V) to As (III). (Mukhopadhyay et al., 2000). In addition to being able to reduce As (V) to As (III), plants also appear to be able to reduce MMA^{V} taken into the roots to MMA^{III} (Finnegan and Chen, 2012). Inorganic As has only limited mobility in most plants, as demonstrated by the steep decline in As concentration from roots to stems to leaves to grain (Liu *et al.*, 2006; Zheng *et al.*, 2011; Zhao *et al.*, 2012).

Arsenic detoxification in plants

In non hyper-accumulator plants, As toxicity often occurs at a shoot As concentration varying between 1 and 100 mg kg^{-1} (Kabata-Pendias and Pendias, 1992), whereas the As hyper-accumulator *P. vittata* can withstand 5000–10,000 mg kg^{-1} of As in the frond tissue without suffering from toxicity (Lombi *et al.*, 2002; Tu and Ma, 2002). Plant species, which colonize As-contaminated soils, are able to restrict arsenate uptake through an adaptive suppression of high-affinity phosphate transporters. The As entering into cells has to be detoxified through complexation and/or vacuolar compartmentation. Another possible constitutive mechanism of detoxification in plants, suggested rather recently (Logoteta *et al.*, 2009), is the efflux of arsenite to the external medium. Arsenic is a strong inducer of phytochelatin (PC) synthesis (Schmoger *et al.*, 2000; Sneller *et al.,* 1999). PCs play a crucial role in As detoxification in As non-hyper-accumulators. The PC-arsenite complexes are likely to be stored in vacuoles (Ghosh *et al.,* 1999). In *P. vittata* fronds, As is stored in the vacuoles mainly as inorganic arsenite (Lombi *et al.*, 2002; Pickering *et al.*, 2006). Arsenite is most likely transported across the tonoplast by an energy-dependent active process due to the cytoplasm to vacuole's likely substantial concentration differential. The transporter(s) responsible for arsenite uptake into the vacuoles is not yet known but may be the key determinant of the hyper-tolerant phenotype in *P. vittata* and other hyper-accumulator plants (Fig. 1) (Zhao *et al.*, 2010).

Impact of arsenic toxicity on plants

The vegetables and cereals, especially rice that grows in arsenic contaminated soil, are vulnerable to the toxicity of arsenic. People unknowingly consuming become victims of arsenic toxicity. Abedin *et al.*, 2002 have reported accumulation of arsenic, and transformation of arsenic species in rice plant. Rice is enriched in arsenic as it grows in flooded paddy fields where the phytomobile As(III) predominates in the soil solution (Sanyal and Nasar, 2002; Takahashi *et al.*, 2004). Many reports have demonstrated the arsenic contamination in rice and vegetables from the agricultural field in arsenic contaminated areas of West Bengal. It has been observed that the root, shoot and leaf tissue of rice plant contain mainly inorganic As (III) and As (V), while the rice grain contains predominantly DMA (85 to 94%) and As (III) (Smith *et al.*, 2008, Liu *et al.*, 2006). In case of vegetables, the highest arsenic accumulation was observed in potato, brinjal, arum, amaranth, radish, okra, cauliflower whereas lower level of arsenic accumulation was observed in beans, green chili, tomato, bitter guard, lemon, turmeric, etc. Several greenhouse studies show that an increment in As in cultivated soils leads to an increment in the levels of As in edible crops and vegetables (Sanyal and Nasar, 2002; Sanyal *et al.*, 2012, 2015; Bhattacharya *et al.*, 2012; Burlo *et al.*, 1999, Carbonell- Barachina *et al.*, 1999). Arsenic accumulation by agricultural plants depends on the availability of arsenic and also on the physiological properties of the plant (Norra *et al.*, 2005, Huq *et al.*, 2001, Panda and Das, 2000, Lehoczky *et al.*, 2002). Tuberous vegetables accumulate higher amount of arsenic than do leafy vegetables, whereas leafy vegetables accumulate more arsenic than fruity vegetables (Samal, 2005, Samal *et al.*, 2011; Sanyal *et al.*, 2012, 2015).

Roots are usually the first tissue to be exposed to As, where the metalloid inhibits root extension and proliferation. As can seriously impede plant growth by delaying or stopping biomass building and

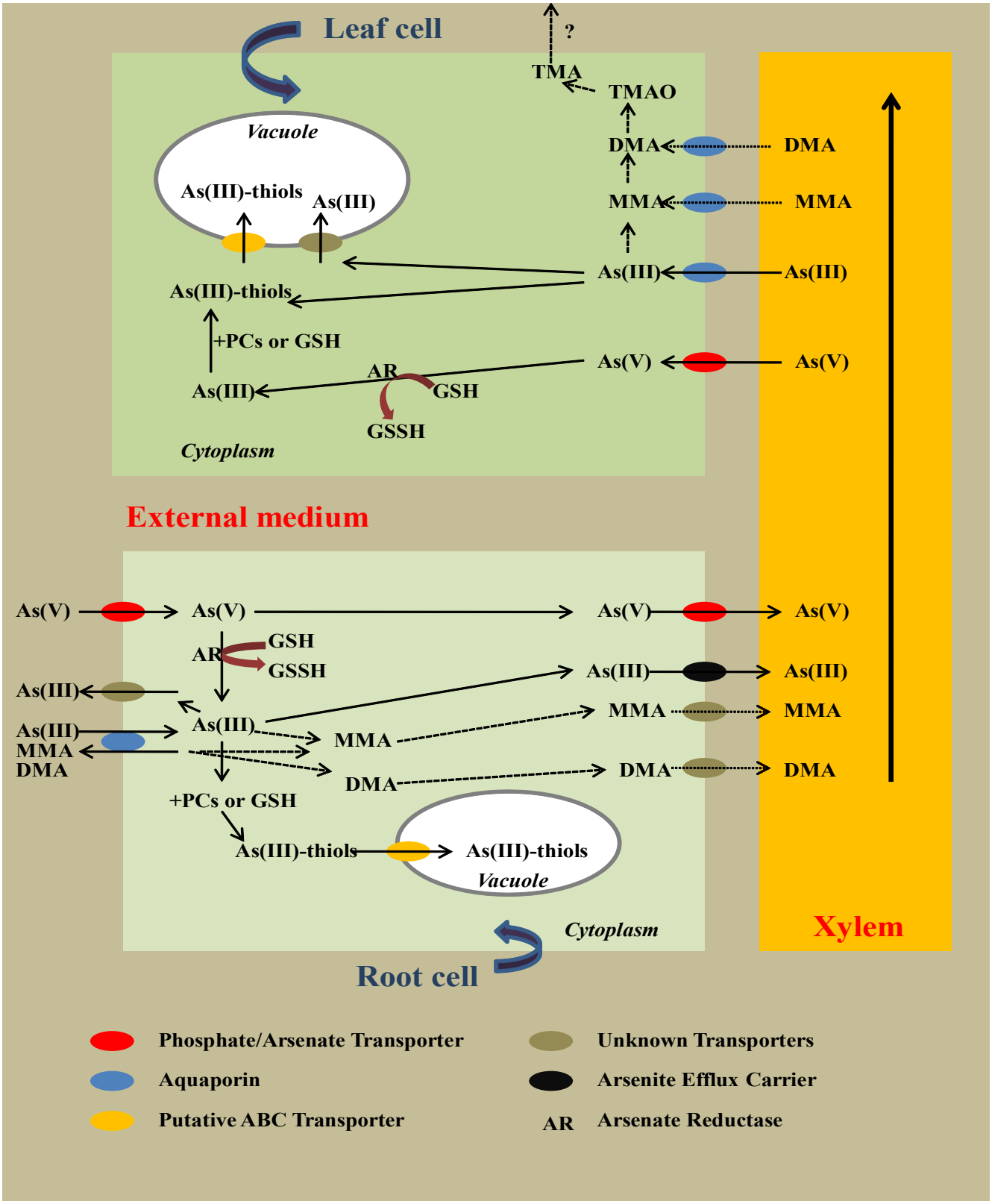

Fig. 1 A simplified schematic diagram of arsenic transport and metabolism in plants. The thickness of arrow lines is indicative of the relative flux. Transporters for As uptake into leaf cells are assumed to be similar to those in roots, but there is little knowledge of their identities. (Source: Zhao et al., 2010)

expansion upon translocation to the shoot. It can also jeopardize plant reproductive potential by reducing fertility, yield, and fruit production (Garg and Singla, 2011). As can cause death when it obstructs vital metabolic processes at high enough quantities. Most plants have systems in place to keep a large portion of their As load in the roots. Nonetheless, some As are translocated to the plant's shoot and other tissues in a genotype-dependent manner.

As poisoning can affect many physiological processes. When plants are exposed to As, cellular membranes are harmed, which results in electrolyte leakage (Singh *et al.*, 2006). Malondialdehyde, a byproduct of lipid peroxidation, frequently rises in response to membrane damage, suggesting the involvement of oxidative stress in As toxicity. Arsenic exposure induces antioxidant defense mechanisms. It is well documented that exposure of plants to As^{III} and As^{V} induces the production of reactive oxygen species (ROS), including superoxide ($O_2^{\bullet-}$), the hydroxyl radical ($^{\bullet}HO$), and H_2O_2 (Hartley-Whitaker *et al.*, 2001; Requejo and Tena, 2005; Singh *et al.*, 2006; Ahsan *et al.*, 2008; Mallick *et al.*, 2011). ROS can damage proteins, amino acids, purine nucleotides and nucleic acids and cause peroxidation of membrane lipids (Moller *et al.*, 2007). Lipid peroxidation not only compromises cellular function, but leads to the production of lipid-derived radicals (Van Breusegem and Dat, 2006; Moller *et al.*, 2007). AsV was also found to induce lipid peroxidation in the As hyper-accumulator *P. vittata* (Srivastava *et al.*, 2005; Singh *et al.*, 2006). This suggests that the production of reactive oxygen species (ROS) is a characteristic of the plant As response in general, and that the degree of redox imbalance within the cell may play a significant role in determining the toxicity of ROS. While the exact mechanism underlying the As-induced generation of ROS remains unclear, it has been suggested that As detoxification pathways, such as the conversion of AsV to AsIII and the activation of PC synthesis (Meharg and Hartley-Whitaker, 2002), may contribute to ROS generation. Several enzymes are involved in ROS defense strategies. Highly reactive superoxide can be converted to less active but longer-lasting H_2O_2 through the action of superoxide dismutase (SOD). With As treatment, the SOD activity in plants fluctuates quite a bit. The enzyme is triggered by low As exposure in some plants, such as *Zea mays*, As-sensitive clones of *H. lanatus,* and the As-hyper-accumulator *P. vittata.* At greater As levels, the enzyme either remains at the same level or decreases in activity (Mylona *et al.*, 1998; Hartley-Whitaker *et al.*, 2001, Cao *et al.*, 2004). The fact that SOD is a metallo-enzyme has been proposed as one explanation for this fluctuation in activity (Meharg and Hartley-Whitaker, 2002).

The ability of As^{V} to substitute for Pi, the propensity of As^{III}-based compounds to bind to and change the activity of enzymes and the damaging effects of ROS all have direct and important consequences for plant metabolism. The requirement of the plant for enough metabolite-based chemicals to counteract the ROS generated by exposure has more indirect effects on metabolism, although they are nonetheless significant. The plant response to these factors predominantly impacts on carbon, nitrogen, and sulphur metabolism (Finnegan and Chen, 2012). In general, As uptake by plants follows the order: As^{III}->As^{V}>MMA^{V}>DMA^{V}, while translocation from the roots to the rest of the plant has the order DMA^{V}>MMA^{V}>As^{V}e"As^{III}. But, the order for phytotoxicity in maize, a species with the typical order for uptake (Raab *et al.*, 2007a), was As^{V}>As^{III}>DMA^{V} (Abbas and Meharg, 2008). The different As species' varying orders of phytotoxicity may indicate that As has reacted differently with the available nutrients or that the phytotoxic form of As varies depending on the kind of plant. The precise mechanism underlying As toxicity is still unknown, though.

One plausible mechanism of action for AsV toxicity could include Pi's substitution in crucial biological pathways. Several biological activities have been shown to include Pi substitution by AsV, and any reaction that uses Pi or a Pi-ester as a substrate could be the target of an AsV disruption

(Gresser, 1981). Potential As^{V}-sensitive reactions would include those central to cellular metabolism (i.e., glycolysis, oxidative phosphorylation) and biosynthesis (i.e., phospholipid metabolism), information storage and retrieval (DNA, RNA metabolism), and cellular signaling (i.e., protein phosphorylation/ dephosphorylation). High Pi conditions reduce As^{V} toxicity because Pi competes with As^{V} for uptake. However, in low Pi situations, As^{V} may outcompete Pi for uptake, aggravating Pi deficiency (Tu and Ma, 2003).

As^{III}'s mechanism of action is very different from As^{V}'s. As^{III} is a thiol reactive compound that can bind up to three sulfhydryl groups (Kitchin and Wallace, 2006). Because of this, AsIII can bind up to three monothiol molecules, including the antioxidant GSH, and function as a cross-linking agent. As an alternative, it might attach itself to a single poly-thiol molecule, like PC, which is the Cys-rich polymerization result of GSH. AsIII has the ability to attach to co-factors and proteins that contain thiols. Dihydrolipoamide has been long thought to be an important cellular target for As^{III} binding (Bergquist *et al.*, 2009). As^{III} binding to proteins can significantly impact how those proteins fold (Cline *et al.*, 2003; Ramadan *et al.*, 2007). More than 100 enzyme activities that were sensitive to As compounds were identified by 1966 (Webb, 1966), and this number have grown considerably. Transcription factors, signal transduction proteins, proteolytic proteins, metabolic enzymes, redox regulatory enzymes, and structural proteins are among the proteins known to bind As^{III} in a variety of systems. It is well known that exposure of plants to As^{III} and As^{V} induces the production of ROS, which can damage proteins, amino acids, purine nucleotides and nucleic acids and cause peroxidation of membrane lipids (Moller *et al.*, 2007).

CONCLUSION

Researchers have studied arsenic uptake, acquisition, toxicity, and the oxidative state of plants exposed to high arsenic in great detail. Despite a perfect understanding of the interactions between arsenic and plants, nothing is known about pilot-scale trials on plant growth, yield, and the precise processes underlying arsenic toxicity, binding interactions, and oxidative stress in plants. Because arsenic poisoning of soil, water, and plant ecosystems is currently a global hazard for humanity, it requires the utmost care and attention.

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