Chapter 29
Mechanisms of Arsenic Hyperaccumulation by Plants

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Abstract Arsenic (As) which is a heavy metal is ubiquitously present in soil as well as in water. As has been ranked as a potent carcinogen and is found to be very harmful to all the living beings ranging from bacteria to plant to animals as well as humans. All the organisms possess various defense mechanisms to combat such types of stresses. However, if it remains detoxified in plants, it may lead to oxidative stress, misfolded proteins thus disrupting the functioning of the proteins, mutations in the genetic material which ultimately results in the inhibition of the growth, disruption of photosynthe-

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sis, and loss in crop yield. Plants are sessile creatures of nature so they are more vulnerable to any type of stress. However, they possess a very strong defense system that fights against these stresses. There are various mechanisms responsible for defense against As stress such as phytochelatin (PC)-dependent defense in which As forms complex with PCs and these complexes are sequestered inside the vacuole. The antioxidant defense system is a very basic and strong player in this defense system. One of the interesting parts of this system is the hyperaccumulation of As. However, hyperaccumulation is not common to all the plants. This is a trait of some specific plant species which had gained a very high capacity of accumulation of As in the aboveground part without suffering phytotoxic effects during evolution. Hyperaccumulator plants differ from normal or non-accumulator plants in various ways. Among them, very fast translocation of As from root to aboveground part, much higher detoxification ability, and higher sequestration capacity of As in aboveground part are the main mechanism which differentiates hyperaccumulator plants to non-accumulator plants. In particular, a determinant role in driving the uptake, translocation to leaves, and, finally, sequestration in vacuoles is played in hyperaccumulators by constitutive overexpression of genes encoding transmembrane transporters, such as members of arsenical compound resistance 3 (ACR3). In this chapter, we will discuss mainly the As toxicity in the plants along with the mechanisms that are involved in hyperaccumulator plants, detoxification of As in plants, as well as the tolerance of As in plants.

Keywords Arsenic · Hyperaccumulator · Heavy metal toxicity · Oxidative stress · NOS · Signaling molecules

29.1 Introduction

Soil and water are primarily contaminated by arsenic due to volcanic eruption, weathering of arsenic-rich rocks, and various anthropogenic activities, viz., mining; use of pesticides, herbicides, fertilizers, and preservatives; etc. Arsenic exists in both organic and inorganic forms. The inorganic forms of arsenic are found either in the oxidized form, i.e., arsenate (AsV), or in the reduced form, i.e., arsenite (AsIII); they are profusely found in water and soil in comparison to the organic forms, i.e., arsenobetaine, monomethylarsonic acid (MMA), dimethylarsinic acid (DMA), and arsenosugars (Panda et al. 2010). However, the solubility of As depends upon the pH and ionic milieu. AsV is found abundant in aerobic soils, while AsIII is more toxic and mobile, found profusely in anaerobic conditions like paddy soil at pH < 8 in general (Souri et al. 2017). The methylated forms of As were earlier used as pesticides and herbicides consequently accumulating more MMA and DMA in soil, which is now reported to be more toxic than inorganic forms. The DMA was found to be more toxic than AsV in wheat. Duncan et al. (2017) reported that the germination rates of wheat are 80% and 30% under AsV and DMA stress, respectively. In the same report,

the grain yield also decreased by 20–50% when compared to AsV. Similarly, Naranmandura et al. (2011) reported MMA to be almost 18 times more toxic than arsenite especially in animal cells. The problem of As toxicity is alarming in South and Southeast Asian countries, as the groundwater as well as soil in these countries are highly contaminated. Apart from consuming As-contaminated water, rice has been identified as the second major source of arsenic to humans (Meharg et al. 2009). As arsenic-contaminated groundwater is also used for irrigation purposes, it further loads As into the soil and leads to its buildup in vegetables/agriculture products, eventually posing a significant health risk to humans as well as animals because fodder offers an alternate route for arsenic entry in the food chain. Populations of As-contaminated areas suffer from several deformities and diseases (Meharg et al. 2009).

Arsenic is non-essential and highly phytotoxic, with threshold concentrations usually varying from 5 to100 mg kg^{-r} DW (dry weight). The roots are the first tissue for As exposure, where it inhibits the root extension as well as its proliferation. Further, As translocation to shoot severely reduces or arrests growth, biomass reduction, fertility loss, loss in fruit production, and yield loss (Garg and Singla 2011). An interesting paradox of As toxicity is that the low concentrations of As resulted in growth enhancement under axenic conditions (Garg and Singla 2011). It has been reported by Chen et al. (2010) and further suggested that the growth enhancement observed in *Arabidopsis thaliana* is possibly due to the As participation with plant metabolism and with other plant nutrients. However the mechanism remains unclear; another explanation was given by Tu and Ma (2003) that the growth enhancement by arsenic is due to activation of Pi uptake as well.

Arsenic removal mainly relies on the chemical process of excavation and treatment of soil; however, this method of remediation is costly and cannot be used regularly (Wuana and Okieimen 2011). On the other hand, phytoremediation uses hyperaccumulator plants for remediating toxic metal compounds from soil and water in a very cost-effective and environmentally safe manner (Jiang et al. 2015). The accumulation of > 1000 mg As kg^{-1} DW in above ground parts, without any symptoms of phytotoxicity, is rare in terrestrial plants and considered as hyperaccumulation. Hyperaccumulation is rare in terrestrial plants, and they exhibit elevated growth rates, higher tolerance, and greater accumulation of heavy metals (Ghori et al. 2016). Same report mentions that plant hyperaccumulators entail both root-to-shoot and a root-to-soil heavy metal concentration ratio greater than one. Numerous plant species are known for their capability of detoxifying and hyperaccumulating heavy metals. However, few species of *Pteris* genus, viz., *Pteris vittata*, *Pteris criteca*, *Pteris longifolia*, *Pteris umbrosa*, *Pityrogramma calomelanos*, and *Isatis cappadocica*, are found to be As hyperaccumulators (Xie et al. 2009; Karimi et al. 2009).

Among these, *P. vittata* (Xie et al. 2009) and *I. cappadocica* (Karimi et al. 2009) are the most studied and will help elucidate mechanistic features in the arsenic hyperaccumulation and tolerance. Increase in thiol synthesis and the chelation of arsenic with glutathione (GSH) and phytochelatins (PCs) are the two important strategy of Isatis cappadocica which provide As tolerance to this hyperaccumulating plant (Karimi et al. 2009). However, the fern *Pteris vittata* utilizes the efficient system of

AsV/AsIII sequestration in vacuoles (Xie et al. 2009). In this chapter, we will discuss the mechanism utilized by As hyperaccumulator plants specially *Pteris vittata*, how it can accumulate such high concentration of As, and the various factors which are responsible for this trait. Also, the involvement of reactive oxygen species (ROS), nitric oxide (NO), and antioxidants in arsenic detoxification and tolerance will be discussed. Arsenic uptake, its transport, and translocation from root to the aboveground parts and detoxification mechanism in plants emphasizing on hyperaccumulator plant will also be discussed here.

29.2 Arsenic Toxicity in Plants

Hydroponic experiments have helped to identify the strength of exogenously applied arsenic on the overall growth of the plant; they eliminate the intricate and difficult factors occurring due to the variation in the mobility of several arsenic species. The arsenic uptake by plants is in the respective order, AsIII>AsV>MMAV>DMAV, while further translocation to the aboveground parts has the order DMAV>MMAV>AsV≥AsIII (Raab et al. 2007). The order of arsenic phytotoxicity is inconsistent; this is possibly due to differences in chemical reactivity of As species with the available nutrients.

29.2.1 By Substituting Phosphate

As and arsenate possess structural similarities with Pi; AsV could be substituted for Pi in biochemical reactions, possibly disturbing the basic cellular processes. Potentially, AsV-sensitive reactions would interfere with the pathways requiring Pi as key factors, like in glycolysis, phosphorylation/dephosphorylation, phospholipid metabolism, and DNA and RNA metabolism. Plants are generally equipped with both low- and high-affinity Pi transport systems. As soon as AsV enters the root and comes in contact with cell surface, the Pi transporter is probably the first site where AsV competes with Pi. Competition between AsV with Pi has been observed in monocots as well as dicots and also in both As hyperaccumulators and non-hyperaccumulators (Clark et al. 2000; Wang et al. 2002; Bleeker et al. 2003). It has also been reported by Guo et al. (2008) that AsV can pass the *Arabidopsis* AtPHT4 (phosphate transporter) which is localized in the plastid and Golgi complex.

29.2.2 Reacting with Thiols

AsIII, MMAIII, and DMAIII are highly reactive toward thiol groups. AsIII binds to the thiol groups of proteins with enzymatic properties of enzyme co-factors altering or inhibiting their activity or structural properties. AsIII can crosslink by binding up to three monothiol molecules, like GSH (Kitchin and Wallace 2006), or it can bind a single molecule like PCs with polythiol properties. Arsenite toxicity is aggravated due to its binding capacity to other thiol-containing proteins and co-factors (Gupta et al. 2011). The stability of arsenite-thiol complexes depends upon the number of bonds and the preferential binding of AsIII to the proteins possessing three or more Cys residues in their zinc-finger motifs (Zhou et al. 2011). Also, the binding of AsIII to proteins alters their folding pattern and thus their function (Cline et al. 2003). These proteins include proteolytic, metabolic, and redox-regulatory enzymes, structural proteins, transcription factors, and signal-transducing proteins. Styblo et al. (1997) reported that MMAIII acts as a potent inhibitor like glutathione reductase. A similar report was published for thioredoxin reductase by Lin et al. (1999). In comparison to inorganic arsenic (AsIII and AsV) the methylated form of arsenic can displace Zn^{+2} from Zn finger protein at a low micromlar concentration so there is a need of understanding the plants ability to chnage less toxic form of As to more toxic methylated form (Schwerdtle et al. 2003).

29.2.3 Oxidative Stress

Arsenic toxicity further imposes oxidative stress (Zhao et al. 2010). By definition, during the oxidative stress, the cells are exposed to excessive molecular oxygen or the reduced form, i.e., reactive oxygen species (ROS). The overproduction of ROS such as superoxide radicals ($O_2^{\bullet -}$), hydroxyl radicals (OH•), peroxyl radicals (ROO•), and alkoxyl radicals (RO•) and also some non-radical compounds such as hydrogen peroxide (H_2O_2), the singlet oxygen ($^1O_2$), ozone (O_3), and hypochlorous acid ($HOCl^-$) (Halliwell and Gutteridge 2015). ROS production can also be triggered by heavy metals (Sandalio et al. 2012). The regulation and expression of these proteins change depending upon varied oxidation states of different heavy metals (Cuypers et al. 2011). ROS are the intermediates of basal metabolism including mitochondrial respiration, photosynthesis in chloroplast, and photorespiration in peroxisomes and by NADPH oxidase (NOX) associated with the plasma membrane (Torres and Dangl 2005).

In general, overproduction of ROS oxidizes proteins, lipids, and nucleic acids, modifying their activity or function. Their levels are tightly regulated byenzymatic and non-enzymatic ROS-scavenging mechanisms (Apel and Hirt 2004). The enzymatic ROS scavengers include superoxide dismutases (SOD), catalase (CAT), and components of the ascorbate-glutathione (ASC-GSH) cycle. ASC-GSH comprises of ascorbate peroxidase (APX), monodehydroascorbate reductase (MDHAR), dehydroascorbate reductase

(DHAR), and glutathione reductase (GR). The non-enzymatic antioxidants include glutathione (GSH) and ascorbic acid (AsA), involved in maintaining H_2O_2 levels and also helping in maintaining the cellular redox state (Apel and Hirt 2004).

ROS overproduction by arsenic leads to (a) generation of intermediate arsenic with different valencies, (b) perturbations in mitochondrial electron-transport chain, (c) overexpression of NOX, and (d) altering the levels of GSH and GSH/GSSG ratio and affecting the redox pool.

The overexpression of PCs has also been observed during As toxicity (Singh et al. 2006). Ha et al. (1999) reported that PC has significant role in regulating As toxicity, as the mutant of PC synthase gene (PCS; *cad1–3*) in exhibited enhanced sensitivity to arsenate as compared to the control *Arabidopsis* plant. Overexpression of PCS in *Arabidopsis*, *Brassica juncea*, and *Nicotiana tabacum* resulted in enhanced tolerance toward arsenic toxicity (Li et al. 2004; Gasic and Korban 2007; Wojas et al. 2010). Conversely, PC overexpression can also diminish cellular GSH levels, disturbing the cellular redox pool and the overall antioxidant capability (Hartley-Whitaker et al. 2001).

Box 29.1 Mechanism of As Hyperaccumulation in Plants

- Higher affinity of As with their transporter
- Increased uptake and translocation of As
- Efficient reduction of AsV to AsIII
- Higher accumulation in aboveground part with root to shoot translocation of >1
- Decreased efflux of As to external medium
- Efficient loading of As to xylem

29.3 Mechanisms Irresponsible for As Hyperaccumulation

As hyperaccumulator plants behave differently with other non-hyperaccumulator plants in various traits. They can withstand such a higher concentration of As which is very much toxic to other plants, and toxicity is so much high that the non-accumulator plants are even not able to survive. Higher uptake of As, efficient translocation, and accumulation are the key features of hyperaccumulator plants. Along with these, the mechanism which is responsible for withstanding such a higher concentration of As without showing any toxic effects is also important. *Pteris vittata* member of the Pteridaceae family is the first As hyperaccumulator fern identified by Ma et al. (2001). This plant is very efficient in accumulating the As in its fronds without showing any phytotoxic effects (Lombi et al. 2002; Tu and Ma 2002). If we compare this plant with the normal or non-accumulator plant, we can say that it possesses a very high rate of As translocation from root to aboveground parts (Caille et al. 2005). Box 29.1 is showing important mechanisms of As hyperaccumulation in

hyperaccumulator plants, while the major difference in As hyperaccumulator and non-accumulator plants has been listed in Table 29.1.

29.3.1 Arsenic Uptake, Transport, and Translocation

There are various factors such as soil characteristics, soil pH, As speciation (AsV, AsIII, and methylated As), plant species, water content, etc. that are responsible for the rate of uptake of As from soil (Zhao et al. 2009). As previously discussed, arsenic exists in the environment in organic and inorganic forms. These different forms of As are differentially taken up by the plants via their respective transporters (Farooq et al. 2016). AsV which is a phosphate analog is transported with the help of high-affinity Pi transporters (Fig. 29.1) following Michaelis-Menten kinetics in *P. vittata* as well as other higher plants (Karimi and Souri 2015). There are various evidences available in the literature for the transport of AsV via phosphate transporter. The two independent types of research had shown that a competitive inhibition exists for the uptake of AsV by Pi with the help of radiotracer 73 AsV (Abedin et al. 2002; Karimi et al. 2009). *Arabidopsis thaliana* mutants who are defective for phosphate transporters were also defective for AsV transport (Catarecha et al. 2007). Pi is very much important for the plant so its uptake is highly regulated. However, this transporter is also responsible for AsV transport; the uptake of AsV must also be under higher regulation with the same mechanism (Sun et al. 2012).

AsIII at neutral pH remains as arsenous acid, and this is mainly uptaken with the help of aquaporins which transport water, urea, as well a neutral molecule, and the inhibition studies had confirmed that glycerol competitively inhibits AsIII uptake. This arsenous acid shows structural similarity with silicic acid, and thus it shares the transporters which are transporting and uptaking silicic acid. Nodulin 26-like intrinsic proteins (NIPs), an important subclass of aquaporins, have been shown to transport AsIII from soil to roots of plants (Zhao et al. 2009; Chen et al. 2016). In *Arabidopsis* also the uptake and transport of arsenous acid occur via various members of NIPs such as

Table 29.1 Difference between hyperaccumulator and non-accumulator plants

S. no.	Mechanism	Hyperaccumulator plants	Non-accumulator plants
1	Uptake of As	High	Low to medium
2	Efflux of As	Low	High
3	Reduction of AsV to AsIII	Very efficient	Moderate to low
4	Sequestration in root vacuole	Very less	Efficient
5	Accumulation in root	Low	High
6	Accumulation in above ground part	Very high	Low
7.	Translocation from soil to root to shoot	>1	<1
8	Volatilization of As	Less	Moderate to high

NIP1;1, NIP1;2, NIP3;1, NIP5;1, NIP6;1, and NIP7;1 (Bienert et al. 2008; Xu et al. 2015). Not only in *Arabidopsis* the same family of transporters is also involved in AsIII transport in other plants also such as HvNIP1;2 in *Hordeum vulgare* and OsNIP3;3 (Katsuhara et al. 2014) Lsi1, and Lsi2, in rice roots (Ma et al. 2008). However, in *P. vittata* hyperaccumulator plant, neither glycerol nor silicic acid affects AsIII uptake suggesting that AsIII uptake and transport differ a lot in this plant in comparison to other plant species (He et al. 2016). In *P. vittata*, tonoplast intrinsic protein (TIP), a member of aquaporins called PvTIP4, is involved in AsIII uptake (He et al. 2016). The second difference between hyperaccumulator and non-accumulator plants is the affinity of transporters with AsIII. Hyperaccumulator plants show higher affinity in comparison to non-accumulator. The affinity of AsIII transports in *P. vittata* is much higher for

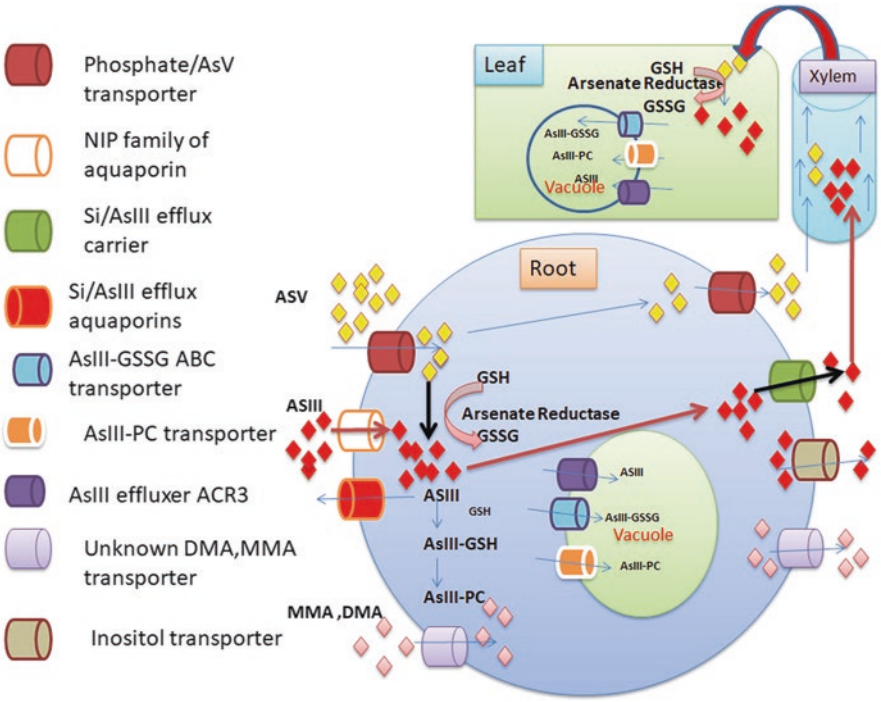

Fig. 29.1 Diagrammatic representation of As uptake, transport, translocation, and detoxification mechanism in plants. AsV being phosphate analogue enters the cell via phosphate transporters. As a detoxification mechanism, arsenate is reduced to arsenite via arsenate reductase using GSH as reducing agent. AsIII uptake is taken place via nodulin 26-like intrinsic (NIP) aquaglyceroporin channels. Transporters of methylated species (DMA/MMA) are either unknown or taken up by NIP. AsIII forms complexes either with phytochelatins (PCs) or GSH, and these complexes are sequestered in vacuole via ABC transporter. In *P. vittata*, AsIII is sequestered inside the vacuole via ACR3 effluxer. As a hyperaccumulation strategy, loading of AsIII to xylem must be efficient and is mediated by the Si/arsenite efflux transporters or inositol transporters (INT). Efficient translocation of As from root to shoot and sequestration capacity in vacuole of aboveground part makes hyperaccumulator plants to accumulate higher As in aboveground part. Dark lines represent efficient pathways of As transport and translocation in hyperaccumulator plants

AsIII in comparison to other plants explaining its extraordinary capacity for AsIII uptake (Chen et al. 2016). In *Arabidopsis* INT transporters (At INT) which are responsible for inositol uptake and transport are also responsible for AsIII distribution in phloem siliques and seeds (Duan et al. 2016). However, the presence of the same inositol transporter in hyperaccumulator plants is still doubtful. Another difference between the hyperaccumulator and non-accumulator plants is the site of accumulation of As. In hyperaccumulator plants, As is accumulated more in the aboveground part, while in non-accumulator plants, it is restricted in roots only, and translocation is also reduced from root to shoot and thus accumulation more As in roots. As hyperaccumulators, such as *I. cappadocica* and *P. vittata*, accumulate a higher amount of As (approximately 60–80%) in aboveground parts. In these plants, the As shoot-to-root ratio was found to be greater than 1 (Karimi et al. 2013; Chen et al. 2016). On one side, the As hyperaccumulator plants are accumulating approximately 60–80% of As in shoots. However, conversely, the non-accumulator plants, such as *Arabidopsis* (Isayenkov and Maathuis 2008) and *P. tremula* fern (Caille et al. 2005), accumulate comparatively very less proportion of As in shoots (5–10% of total As). Once As enters the plants, its efflux to root or volatilization can reduce As translocation and thus As accumulation to aboveground parts. Efflux of As to external media is also a very important strategy of non-accumulator plants for reducing As accumulation. In contrast to non-accumulator plant, translocation rate of As from root to shoots in hyperaccumulator plants is very efficient, and the efflux rate of As to external media is very less (Chen et al. 2016). As a detoxification mechanism as early as AsV enters the cell it is converted to AsIII (either enzymatically or GSH dependent) and this AsIII then forms complexes with either PCs or GSH and sequestered inside the vacuole and thus further reduces translocation of As to shoot and above-ground parts (Fig. 29.1). In hyperaccumulator plants, reduction of Asv to AsIII is very efficient; however their sequestration inside the vacuole is compromised for the efficient translocation of As from root to aboveground parts. Reports suggest AsIII is the main form of As transported from root to shoot through xylem sap, regardless of any inorganic form which is supplied to the plants (Su et al. 2008). For translocation and As accumulation in shoots, it is very important to have efficient loading of As to the xylem which is an important feature of As hyperaccumulator plants. Organic forms of As are also taken up by the plants. Some of the methylated forms of As are more cytotoxic in comparison to inorganic forms, yet their translocation in xylem and phloem is much higher in comparison to AsIII and AsV. However, in As hyperaccumulator plants, the key transporters for methylated species have not been identified in these tissues (Huang et al. 2008; Li et al. 2009). Uptake of methylated form of As in rice roots occurs via Lsi1 (Li et al. 2009).

29.3.2 *Accumulation of Arsenic in* Pteris vittata

Arsenic hyperaccumulator accumulates As in a different manner in comparison to non-accumulator plants. In *P. vittata*, most of the As is accumulated in aboveground biomass of the fern. There is a report stating that in these plants most of the As (almost 93%) was found to be accumulated in the fronds of the plant (Ma et al. 2001). Even in fronds, accumulation of As was found to be higher in the lamina of pinnae in comparison to midribs and spores. If we talk about the pinnae, it is the base of midrib which has a significantly higher amount and accumulation of As than those that are accumulated in the central or apical part of the pinnae. This can be explained based on the development of pinnae as the base of pinnae develops earlier than the central part while the apical parts develop recently. Accumulation of As was found to be least in spores. At the cellular level, accumulation of As in the pinnae of *P. vittata* also follows the same pattern as other hyperaccumulator plants; As was majorly accumulated in the upper and lower epidermal cell. The accumulation and distribution pattern of As differ a lot from other elements such as Mg, S, and Ca. The distribution pattern of As and K was found to be positively correlated in upper epidermis cells of *P. vittata*. A probable explanation of this fact could be the counterbalance of negative charge of As by positive charge of potassium ion. However, at acidic pH of vacuole, AsIII has to be undissociated from the complexes (either with PCs or with GSH) and requires locations (other than the undissociated protons) for balancing charge, unless it forms negatively charged complexes. It is observed that the restriction of As in the central part of the cell which is the vacuole was reported by X-ray microanalyses. This observation for the accumulation of As inside the vacuole is in agreement with the tentative distribution of heavy metals and metalloids in hyperaccumulating plants. It depicts the accumulation of both metals and metalloids inside the vacuoles which play a key role in providing tolerance against these heavy metals/metalloids. The XANES analyses showed AsIII is the predominant form of As accumulated inside the fronds (75%) while the rest was AsV.

29.3.3 *Regulation of Arsenic Accumulation in Roots of Hyperaccumulator Plant* Pteris vittata *at the Transcript Level*

Potdukhe et al. (2018) had done a root transcriptome analysis of *P. vittata* and identified 554,973 transcripts associated with As uptake transport translocation and accumulation. Various transcription factors, transporters of metals along with genes responsible for the biosynthesis of GSH and PCs, were found to be crucial in this study. Some of the genes which were found to be highly regulated in that study such as cysteine-rich receptor-like kinases and G family of ABC transporter further need validation.

In root transcriptome data, the transcripts having a role in As uptake and transporters have been identified. As previously in this chapter we had discussed the uptake and accumulation of As in hyperaccumulator plants, this study also confirms the previous facts that the AsV transport pathways share transport pathways of inorganic phosphate. Transcripts of the transporter that were associated with inorganic phosphate (PiT) and a large number of members of aquaporins which transports AsIII are upregulated. The higher modulation of the transcripts associated with AsIII transport and translocation might be accountable for the direct influx and accumulation of AsIIIe (Abedin et al. 2002). Arsenic further results in oxidative stress to the plant and activate various stress-responsive genes and molecules via kinase signaling (Rao et al. 2011). Oxidative phosphorylation also plays a very crucial role in activating stress-responsive genes.

Cysteine-rich RLK (receptor-like protein kinase) is induced by Cr stress (Trinh et al. 2014) which has also been upregulated by As stress in *P. vittata* indicating its role in heavy metal stress. This kinase has also been shown to be affected by oxidative stress (ROS) (Burdiak et al. 2015). This cascade further may induce various transcription factors such as asWRKY, bZIP, and MYB families which will further regulate the expression of various genes responsible for providing the tolerance against As stress (Thapa et al. 2012).

29.4 Arsenic Detoxification

29.4.1 Chelation of Arsenite

As detoxification is carried by chelating arsenate to arsenite, this chelation leads to a subsequent loss of toxicity. Generally, chelation is done by glutathione and PCs, and their biosynthesis is common in plants. However, As exposure leads to a substantial increase in the biosynthesis of both glutathione and PCs (Srivastava et al. 2007) indicating its importance as a key detoxifying mechanism. Similarly, upregulation of cysteine, GSH, and PC biosynthesis genes leads to tolerance toward arsenic toxicity (Shri et al. 2014; Tripathi et al. 2007; Wojas et al. 2008). However, the augmented PC biosynthesis has been observed constitutively in both hypertolerant and non-tolerant and in non-accumulator as well as in hyperaccumulator plants (Tripathi et al. 2007; Gupta et al. 2011). Hypertolerance and amassing of As in *I. cappadocica* due to >50% PC complexation indicate the presence of systematic tolerance machinery (Karimi et al. 2009). Above reports indicate that PCs are instrumental in arsenic detoxification. On the other hand, exceptionally low concentration of phytochelatins was perceived in hyperaccumulator *P. vittata* and *P. cretica*, indicating a minor or no role in direct As detoxification (Zhao et al. 2009). Interestingly several reports are indicating that PCs shave little or no role in As tolerance in certain hypertolerants (*H. lanatus* and *Silene paradoxa*; Raab et al. 2007). Phytochelatin-dependent As detoxification system has also been observed in certain non-accumulator plants like

Helianthus annuus, *Ceratophyllum demersum*, and *Hydrilla verticillata* (Raab et al. 2004; Srivastava et al. 2007, 2011; Mishra et al. 2008. In *Arabidopsis* root, Liu et al. (2010) reported that binding of AsIII with phytochelatins withheld the further translocation of As to shoot and also its efflux to the external environment.

29.4.2 Arsenate Reductase

Arsenate reductase (AR) reduces arsenate to arsenite by deploying glutathione as a reducing power (Finnegan and Chen 2012). Several AR genes from *Arabidopsis*, rice, and *H. lanatus* were found to regulate arsenic accumulation, viz., *AtHAC1/ATQ1*, OsHAC1;1, OsHAC1;2, and *HlAsr*, respectively) (from rice) (Chao et al. 2014; Shi et al. 2016; Bleeker et al. 2006). Hyperaccumulator *P. vittata* showed almost sevenfold greater AR activity when compared to As-susceptible *Oryza sativa* and *A. thaliana* (Danh et al. 2014). The expression analysis of AR and AsIII transporters in *P. vittata* indicated higher expression, thus possibly explaining the mechanism for hyperaccumulation by deploying vacuolar transporters in sequestering arsenic (Song et al. 2010).

29.4.3 Sequestration of Arsenic to Vacuoles

The sequestration of complexes As-GSH-PCs in the vacuoles is critical in arsenic detoxification (Gupta et al. 2011). Raab et al. (2004) reported that the vacuoles of hyperaccumulating and tolerant plants like *P. vittata* and *H. lanatus* contain a large amount of non-complexed arsenite, thus suggesting that hyperaccumulators don't save As in complexed forms and also indicating the role of vacuolar arsenite transporters (similar to bacterial extrusion pumps). A study demonstrated the role of arsenical compound resistance 3 (AsIII effluxer, ACR3), found on vacuolar membrane, in arsenic hyperaccumulation in *P. vittata* (Indriolo et al. 2010). Knockdown studies in yeast were performed to analyze the role of ACR in the gametophyte; it ensued arsenite-susceptible phenotype. PvACR3 imparted tolerance to ACR3-deficient yeast, signifying the importance of ACR3. Nevertheless, the absence of these transporters in flowering plants probably is the reason that As hyperaccumulators discovered till date are from Pteridaceae.

For the first time, Song et al. (2010) identified vacuolar PC transporters AtABCC1 and AtABCC2 in *A. thaliana*; they reported that they are vital for As detoxification. Further, double knockout of *atabcc1 atabcc2* of comparable characteristics and *cad1* mutant, lacking phytochelatin biosynthesis, implies the importance of vacuolar sequestration of phytochelatin-arsenite complexes and is as important as the synthesis of PCs for arsenic detoxification. Song et al. (2014) reported similar transporter OsABCC1 with an identical function in rice. Localization of OsABCC1was reported

on tonoplast, and it enhanced As resistance in yeast cells expressing PC synthase, while the knockout of OsABCC1 resulted in significantly higher As hypersensitivity.

29.5 Arsenic Tolerance

29.5.1 ROS

Initially, ROS were considered toxic; during the last decade, ROS has been established to play an important role in the signal transduction pathways (Verma et al. 2019). In the last decade, research has been done on the heavy metal toxicity and its role in elevating ROS; however, lacunae regarding their effect on the regulation of cellular responses as well as signaling cascades remain to be done. Kidwai et al. (2019) reported that arsenic can elevate ROS production, viz., $O_2^{\bullet-}$, OH•, H_2O_2, and singlet oxygen (). ROS, primarily H_2O_2, has been established as a signaling molecule playing an important role in cellular response to arsenic stress (Sharma 2012). A proteomic analysis performed by Requejo and Tena (2005) in *Zea mays* roots also indicates that oxidative stress is associated with As toxicity as well; Gupta et al. (2017) reported that by generating ROS, NADPH oxidase C (NOXC) helps in regulating both the translocation and uptake of arsenic in *Arabidopsis thaliana*. H_2O_2 also activates mitogen-activated protein kinase (MAPK) signal transduction cascade under arsenic toxicity (Huang et al. 2012). During arsenate treatment in rice, out of the 11 reported MAPK kinases (MAPKKKs), one MAPK and ten phosphatase genes were upregulated. Recently, Kidwai et al. (2019) also documented the importance of class III peroxidase in arsenic tolerance and eventually low accumulation in rice. Another important report is by Dixit et al. (2015), who reported that high-sulfur treatment enhances the activities of antioxidant enzymes and mitigates the effect of oxidative stress due to As stress (Dixit et al. 2015).

29.5.2 Nitric Oxide

Nitric oxide (NO) is a hydrophobic, diffusible, and gaseous free radical molecule and plays an effective role as a signaling molecule in the plant system. NO can pass through the biological membranes, without requiring any specific membrane transporter, making it a suitable candidate for a signaling molecule. NO, a free radical, acts as a ROS scavenger, and being a redox-related signaling compound, it can alleviate oxidative stress by inducing antioxidant machinery, and it also exhibits immune responses against pathogen (Romero-Puertas and Sandalio 2016a, b; Bellin et al. 2013). NO derivatives are named reactive nitrogen species (RNS); nitrosonium ion (NO^+) and nitroxyl radical (NO^-) are the result of losing and gaining electron by NO, respectively. Reactive nitrogen species (RNS) are generated by the reaction between

NO and ROS, such as peroxynitrite and the NOx compounds ((ONOO$^-$, NO_2, N_2O_3, and N_2O_4; Romero-Puertas and Sandalio 2016a). NO can interact with different molecules resulting in modulation of gene expression and protein function. This NO interaction brings the post-translational modification (PTM), which leads to *S*-nitrosylation, nitration, and nitrosylation of proteins. It has been reported that *NO*-dependent *S*-nitrosylation can change the levels of H_2O_2 either by modifying the ROS scavenging (or perturbing the ROS-generating enzymes (GOX and NOX; Romero-Puertas and Sandalio 2016a, b)). Similarly, APX and SODs are regulated by ONOO$^-$ (Romero-Puertas and Sandalio 2016a). The emerging data from the past few years suggest that NO is instrumental in heavy metal tolerance (Fancy et al. 2016). In a study, SNP treatment (a NO donor) suppressed O_2^- levels during metal toxicity (Kopyra and Gwóźdź 2003). Exogenously supplied NO provides protection against AsIII toxicity by attenuating oxidative stress in rice (Singh et al. 2009). Similarly, in *Vigna radiata*, arsenic toxicity is prevented by mitigating ROS generation and accumulation and preventing membrane damage by lowering malondialdehyde (MDA) content (Ismail 2012). Besides, NO can also regulate arsenic accumulation by modulating silicone (*OsLis1* and *OsLis2*; Singh et al. 2016) and ABC transporters (Hussain et al. 2016) in rice. NO can help in sulfate uptake (Farnese et al. 2013) and PC synthesis (Singh et al. 2016). Like H_2O_2, it can also activate MAPK signaling pathway which is also instrumental in combating metal and As stress (Ye et al. 2013). According to Dixit et al. (2015), AsV supplementation significantly hampers thiol metabolism, reduces GSH content and GSH/GSSG ratio, and also enhances the PC level. NO co-treatment with AsV helped in maintaining the GSH/GSSG ratio and reduce PCs.

29.6 Conclusions

In nature, few plants are equipped to survive extremely high levels of arsenic by either accumulating or detoxifying it. Research performed on arsenic metabolism in plants has helped in answering basic questions like how As is taken up and translocated in the plant, how it is chelated and sequestered in the fairly less toxic state, why it is transported through phosphate transporters, and how arsenite binds to sulfhydryl groups and aggravates oxidative stress. With the work done in a couple of decades, the mechanism of As hyperaccumulation without poisoning the plant has almost been decoded. As mentioned above, these hyperaccumulators have evolved well-synchronized approaches for As uptake, efficient transport, and translocation to aboveground parts. Another important mechanism is followed by *P. vittata*, where rapid arsenic uptake and chelation are done either with GSH and/or with PCs, and then this complex is further sequestered to vacuoles; a higher antioxidant capacity has also been reported in this hyperaccumulator plant. However, few important questions remain unanswered like how the cells can mask AsIII and prevent any interference with various metabolic targets while reaching the vacuole for its sequestration or how the AsIII concentrations are kept under check to prevent its harmful effect while translocation. A comprehensive study of other hyperaccumulator plants is also required so

that the exact mechanism can be deciphered which will help us to utilize them for phytoremediation. However, in the future, the detailed physiological, biochemical, genomic, metabolomic, and proteomic approaches will help to achieve better insight into the contrasting features of As hyperaccumulators and non-accumulators and fill the lacunae in our understanding of the overall mechanism.

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