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CONSEQUENCES OF SELENIUM UPTAKE IN PLANTS – A REVIEW

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Selenium (Se) is an essential micronutrient for all life forms. However, excessive quantity of Se and its subsequent bioaccumulation lead to toxicity effects. The element is commonly found in sedimentary rocks and exists in four different inorganic species. The concentration of selenium in soils is highly variable and reflects the nature and intensity of leaching processes on the parent material. The vertical distribution of selenium in soil profiles relates closely to the content of organic matter in the surface horizons and soil type. Although plants demonstrate the ability to accumulate selenium, but information related to the essentiality of this element and the potential response to its impact within the organs of plant is still evolving. Being chemically similar to sulfur (S), Se is taken up by the plants using sulfur transporters and accumulated in their root system. The uptake and distribution of selenium depends on phases of plant growth, plant species, concentration and form of selenium in soil and translocation mechanisms of specific plant. Overburden of selenium leads to oxidative stress in plants. However, specific knowledge of mechanisms on the Se toxicity and deficiency may help the scientist to design effective selenium remediation or fortification strategies using specific plants. The paper analyses the complex behavior of selenium in the environment which is determined by a range of chemical and biological factors as well as modern methods of analysis that providing ever more reliable insights into the physicochemical states of selenium in environmental media and the processes of this element speciation. Moreover, this review delivers an overview of Se uptake, its transportation, metabolism and possible toxic impacts on general physiology and biochemistry of plants. *Key words:* selenium, selenite, toxicity, plants, phytoremediation, hyperaccumulator.

Наслідки накопичення селену рослинами – огляд. Мітра А., Чаттерджі С., Дубчак С.В., Мудгал Ш., Гаур А., Шрівастава К., Гупта Д.К.

Селен (Se) є важливим мікроелементом для всіх форм життя. Однак надмірна кількість селену та його подальше біонакопичення можуть призвести до виникнення ефектів токсичності. Цей елемент зазвичай зустрічається в осадових породах та існує в чотирьох різних неорганічних формах. Концентрація селену в грунтах дуже неоднорідна і відображає характер та інтенсивність процесів вимивання вихідного матеріалу. Вертикальний розподіл селену в грунтових профілях тісно пов'язаний з вмістом органічної речовини в поверхневих горизонтах та типом грунту. Хоча рослини демонструють здатність накопичувати селен, але інформація щодо важливості цього елементу та потенційної реакції на його вплив органами рослин все ще є недостатньо повно вивченою. Будучи хімічно подібним до сірки (S), селен поглинається рослинами за допомогою S-транспортерів та накопичується в їх кореневій системі. Поглинання та розподіл селену залежать від фази росту та виду рослин, концентрації та форми селену в ґрунті і механізмів транслокації в окремій рослині. Надлишок селену призводить до окислювального стресу в рослинах. Докладні знання механізмів токсичності та дефіциту селену можуть допомогти в розробці ефективних стратегій ремедіації або акумуляції цього елементу з використанням конкретних рослин. У статті проаналізовано комплексну поведінку селену в навколишньому середовищі, яка визначається низкою хімічних та біологічних факторів, а також сучасні методи аналізу, які дають все більш повноцінне уявлення про фізико-хімічний стан селену в довкіллі та процеси його формоутворення. В статті також розглянуто процеси накопичення селену рослинами, його транслокацію, метаболізм та можливий токсичний вплив на загальну фізіологію та біохімію рослин. *Ключові слова:* селен, селеніт, токсичність, рослини, фіторемедіація, гіперакумулятор.

Introduction. Selenium (symbol Se; atomic number 34) is a naturally found non-metal component of sedimentary rocks. Four inorganic Se species are known to exist in soil, namely selenate (SeO_4^{2-}), selenite (SeO_3^{2-}), elemental Se (Se^0) and selenide (Se^2) [1]. Soluble property of selenate (VI) renders it as a dominant species in soil and which can be taken up by plants easily in neutral or alkaline soil conditions. Insoluble Se^0 is predominant in reducing soil having a very low toxicity; but, Se^{2-} is a highly toxic which can rapidly be oxidized

into non-toxic elemental Se in aerobic condition. Selenite (IV) has higher adsorbent affinity to the soil particles compared to selenate (VI). The occurrence of Se in the soil depends upon Se content in parent rock, soil type, poor percolation through soil, Se-loaded water irrigation, application of phosphate fertilizer, organic compound present, mining activities or volcanic eruption and percentage of rainfall in that area [2, 3]. Volatile organic Se species (DMSe, DMDSe, methane selenol) predominates in atmosphere. Inorganic species SeO_2 present in the air is not stable forming selenious acid. In comparison to soil and water, atmospheric Se content is low and ranges between 1–10 ng/m^3.

Nearly forty countries worldwide have inadequate availability of selenium. The Se toxicity is also widely reported due to natural or man-made reasons [4]. Rocky areas (in Switzerland, Korea, Australia, New Zealand, Scotland, Finland, and Sweden) are found to have low soil Se concentration than the clay soils while arid areas containing higher Se. Different region of Chile, Peru, USA, Canada, China, Philippines, Zambia, Zaire, New Guinea and Australia are reported to represent around 80% of the total Se reserves of the world. In India, north-eastern part of Punjab is rich in Se [1]. Around the World, typical Se content in soils is limited to 0.4 mg·kg^{-1} but the concentration in selenium rich soils the levels may be ranging from > 2–5000 mg·kg^{-1}. WHO recommended that Se concentration in drinking water should be restricted to less than 10 µg·L^{-1} [5]. The reason behind increasing levels of Se in groundwater is the indiscriminate use of fertilizers having Se. For example, Se concentration in groundwater has been reached an estimated concentration from 2.4–40 µg·L^{-1} in France, and 341 µg·L^{-1} in Punjab, India [1]. Besides natural events (like erosions of soil, and forest fires) several anthropogenic activities like the combustion of fossil fuels (coal and petroleum), tiers and papers are responsible for appending Se into the atmosphere.

Se uptake, transport and metabolism in plants.

Selenium uptake and transport. Except the metal selenides, compounds like selenite, selenate and organoselenium (e.g., selenomethionine, selenocysteine etc.) those predominates in soil show easy uptake by plants. Due to its structural analogy with sulphur, Se is transported through high affinity root epidermal and cortical sulphate transporters (homologs to AtSULTR1;1 and AtSULTR1;2 transporters of *Arabidopsis*) [4]. Other transporters like phosphate transporter (rice OsPT2) and aquaporin channels (homologue to rice OsNIP2;1) have been reported to uptake $HSeO_3$ and H_2SeO_3 respectively [6]. The transporters channelizing cysteine and methionine inside the plant may co-transport SeMet and SeCys.

Selenate enters into the root cells through high affinity proton-sulphate symporters SULTR1;1 and SULTR1;2, that permit entry of selenate along with 3 protons within roots. Sulphur resource of the plant regulates transporter expression, as reduced rates of expression of SULTR1;1 transporter under S-scarcity, and higher rate of expression of SULTR1;2 was found during both S-abundance and S-insufficient environments [7]. Both SULTR1;1 and SULTR1;2 are known to facilitate Se transportation from soil to root, although diverse useful redundancy exists amongst them as evidenced from *Arabidopsis thaliana* sultr1;2 mutants, high selenate tolerant compared to wild type plants and sultr1;1, mutant plants, whereas, double mutant's plants for sultr1;1-sultr1;2 displayed the extreme selenate tolerance.

The observation validates the SULTR1;2 transporters as the key portal for selenate entry into the plant. Sulphate transporters were first described in *Arabidopsis thaliana*, which are clustered into four discrete assemblies of proteins encoded by 12 genes with complementary physiological activities [8]. Group 1 comprises high affinity sulphate transporters (SULTR1;1 and SULTR1;2) primarily located in the root. Group 2, S-transporters show reduced affinity for sulphate, are distributed all over the plant body playing key role sulphate translocation. Group 2 S-transporters in *A. thaliana* includes two isoforms SULTR2:1 and SULTR2:2, both are localized in roots and leaves but in different tissues. In leaves, AtSULTR2;1 expresses the xylem parenchyma, and in phloem cells while AtSULTR2;2 mostly found in the root and leaf bundle sheath; AtSULTR2;1 and AtSULTR2;2 are found to express in pericycle and in the phloem cells respectively.

Expression of Group 3 S-transporters like AtSULTR3;1 is restricted in chloroplast of leaves, and indifferent of the sulphur status of the plant [9], but sulphate uptake in chloroplast was greatly abridged in the absence of this transporter. Group 4 S-transporters (AtSULTR4;1 and AtSULTR4;2) clustered in tonoplastic cells and are low affinity S-transporters regulating vascular efflux of the sulphate, thus increasing sulphate availability for export. Experimental evidences suggest that other angiosperms as well as Se hyperaccumulators likely to have a comparable number of genes expressing S-transporters [8]. SULTR1;1 and SULTR1;2 gene expressions are upregulated in the roots of non-accumulating Se-indicator species in S-surplus condition or during higher tissue Se content. Whereas, in Se-hyperaccumulators these genes are constitutively expressed in higher rates resulted in greater Se uptake. It has been revealed that hyperaccumulators can accumulate Se at up to 1000 times higher concentrations than normal plant [10].

Plant uptake selenite by phosphate transporters in roots rather than through high affinity S-transporters. Up to 50% reduction in selenite uptake was observed in perennial ryegrass (*Lolium perenne*) and strawberry clover (*Trifolium fragiferrum*) when soil phosphate content was increased up to 10- fold. Further, K_m value of selenite entry was elevated in wheat (*Triticum aestivum*) for sustenance of phosphate. The outcomes pointed out that selenite and phosphate compete each other for the common transporter denoting that they share the

common transporter [11]. It was observed that increased expression of phosphate and sulphate transporters and improved root architecture may promote selenium uptake by high-Se accumulating rice cultivar. Further, the additional sulphur application has revealed a pronounced inhibition effect on the Se uptake by *Glycine max* grown in different soil types. Plants have the capability to uptake organic seleno-compounds (SeCys and SeMet) through a membrane localized amino acid permeases mediating transport of amino acids into the cell. Organic Se species are taken up by plants in higher rate than the inorganic Se compounds as evidenced from the studies in *Brassica napus* (spring canola) and *Triticum turgidum* (wheat) where the 20-fold increased rate of uptake was found in case of organic Se compounds as related to selenate and selenite (Fig. 1) [12].

Soil chemical, biological or physical properties regulate the rate of Se uptake by plants. In a field trial of UK based study irrigation promotes wheat grain production, but it reduced plant uptake and grain Se concentration. The presence of competing ions (e.g. arsenic) can also alter Se uptake by plants. For example, antagonistic role of arsenic (As) in Se uptake has been established as in higher soil Se levels (<2.5 mg·L^{-1}) inclusion of As

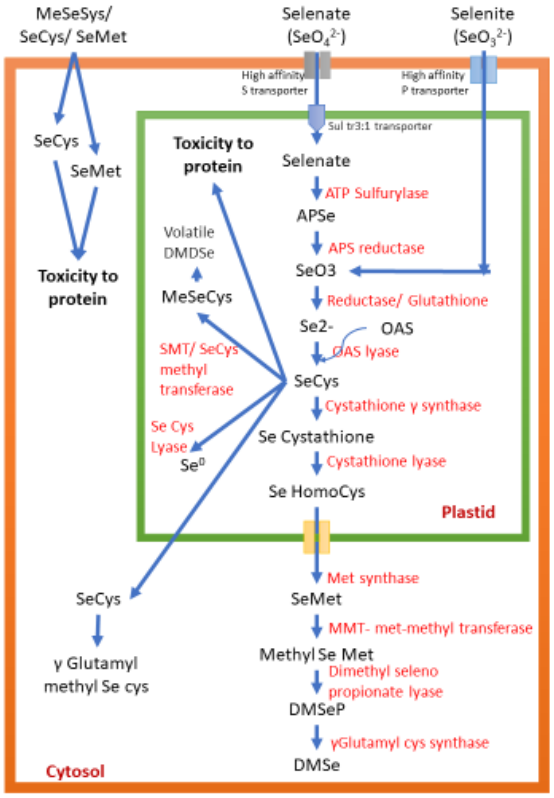

Fig. 1. Uptake and assimilation of inorganic and organic selenium species by plants (Sultr3:1 – sulphate/selenate cotransporters; APSe – adenosine phosphoselenate; OAS – O-acetylserine; SeCys – selenocysteine; SeMet – selenomethionine; DMSeP – imethylselenoproprionate; DMSe – dimethylselenide; DM(D)Se – dimethyl(di) selenide; SehomoCys – seleno-homocysteine).

supressed Se uptake in *Pteris vittata* but at low soil Se content As stimulates Se uptake [13].

Metabolism of Se. Phytotoxicity of Se is attributed following metabolic conversion of Se to biologically active molecule rather than the parent molecule itself as well to get assimilated within the plant. Following entry into the root cell, selenate enters into chloroplast through S-Se co-transporter and form adenosine 5′-phosphoselenate (APSe), a step which is rate limiting in Se assimilation can also occur in cytosol [14]. In *A. thaliana* plastid localized four isoforms of ATP sulfurylase (APS1–4) have been identified, although APS2 has been reported to be localized both in the cytosol and plastid. In the second step APS reductase (APR) transforms APSe in to selenite; this is entirely restricted in the plastids and being another rate-limiting stage in the assimilation of selenate. Role of APR, that is conversion of APSe into selenite, was confirmed from the observation of Apr2–1 Arabidopsis mutants, where selenate level in cell was enhanced, but reduced levels of selenite were found [15].

Further, in the reductive Se assimilation pathway, reduction of selenite to selenide takes place for getting embodied within amino acids, which occurs either through enzyme sulphite reductase (SiR) or by reduced glutathione, a non-enzyme component. In *A. thaliana* the gene encoding the enzyme SiR is represented as a single copy within plastids. SiR is mediating the transfer of sulphite to sulphide during the reductive sulphate assimilation process and also catalysing the reduction of selenite to selenide within the plastids [7]. The non-enzymatic pathway follows multiple steps involving reduced glutathione (GSH). In this pathway, selenite is first converted into organic selenodiglutathione (GSSeSG) and then to glutathioselenol (GSSeH) nonenzymatically and lastly to selenide by the catalytic action of glutathione reductase (GR), utilizing the reductant NADPH [16]. Although, alteration of selenite to selenide is nonenzymatic, the renewal of reduced glutathione is facilitated by the enzyme GR, a crucial cellular process for combating oxidative stress.

Formation of selenocysteine (SeCys) and methylselelenocysteine (MeSeCys). In the next step complexation of Se with amino acids occurs in chloroplasts as well as in cytosol and mitochondria through the catalytic action of the cysteine synthase (CS) enzyme complex, producing selenocysteine (SeCys) from selenide and O-acetylserine (OAS) [7, 16]. The cysteine synthase enzyme complex is functionally active by the cooperative action of enzymes, namely, OAS thiol-lyase (OAS-TL) and serine acetyltransferase (SAT). Toxicity of Se in plants is manifested because of the nonspecific insertion of SeCys into proteins leading to interrupted protein function. Therefore, to overcome Se toxicity plants have to inhibit the non-specific amalgamation of SeCys to proteins which is achieved by methylation of SeCys forming methyl-SeCys (MeSeCys) catalysed by SeCys methyltransferase (SMT) enzyme. Thus Se-

hyperaccumulator decrease the availability of SeCys for inclusion within proteins by yielding MeSeCys, the key pathway for reducing toxic effects of Se [17].

Presence of SMT was reported in both Se hyperaccumulator and non-accumulator species, but only the hyperaccumulators can efficiently produce MeSeCys. It is reported that, presence of Se promotes the expression of *SMT* gene in Se accumulator *Brassica oleracea* (Broccoli) [16]. Again, MeSeCys was the major species of Se found in hyperaccumulators *Stanleya pinnata* and *A. bisulcatus* [7]. In associated non-accumulator species selenate was the main form of Se [16,18]. The above findings signify the higher activity of SMT and its vital role for higher tolerance and amassment of Se in hyperaccumulators.

Conversion of selenocysteine to volatile dimethydilselenide (DMDSe). MeSeCys produced in plants is further processed into volatile compounds dimethyldiselenide (DMDSe) through oxidation and methylation process, thus making the plant highly tolerant to Se. Primarily, MeSeCys is transformed into methylselenocysteineselenideoxide (MeSeCysSeO) and then form methaneselenol (CH_3SeH) by the catalytic activity of the Cys sulfoxide lyase enzyme [19]. Production of DMDSe has been detected in the leaves, in the Se hyperaccumulator plant *Astragalus racemosus* (Fig. 1).

Formation of selenomethionine (SeMet) and volatile dimethylselenide (DMSe). SeMet is formed from SeCys through multistep enzymatic processes. By the condensation reaction, SeCys is changed to Se-cystathionine of O-phosphohomoserine (OPH) and SeCys by cystathionine-γ-synthase (CGS) [16, 31]. Se-cystathionine undergoes hydrolysis to form Se-homocysteine, through the enzymatic action of cystathionine beta-lyase. Finally, the SeMet is synthesized from Se-homocysteine by the enzymatic action of Met synthase, distinguished in a wide variety of angiosperms such as *Catharanthus roseus, A. thaliana, and Coleus blumei*.

Due to its greater toxicity, SeMet is subjected to further processing to produce less toxic dimethylselenide (DMSe) and reduces the chance of its incorporation into proteins. DMSe has 600 times less toxicity than inorganic Se. The Se-non-accumulators release DMSe as the main volatile forms of Se, while, DMDSe is the primary volatile product in hyperaccumulators [17]. Synthesis of volatile DMSe occurs sharing sulphur volatilization pathway preparing from SeMet. The enzyme CGS has been reported to apparently limiting the change of SeCys to volatile DMSe as faster volatilization is reported from SeMet than from SeCys. For this reason, CGS is selected for overexpression for phytoremediation of Se. Similarly, transgenic *B. juncea* that expressing constitutively *A. thaliana* CGS enzyme had 2–3-times more volatilization rates of Se. The transgenic plants were found to accumulate 20–40% lower and 50–70% lower Se in the shoot and root tissues, respectively, in comparison to wild type plants [20]. In the volatilization process, at first, by S-adenosyl-L-Met:Met-S-methyltransferase (MMT) mediated methylation SeMet forms methylselenomethionine (SeMM). SeMM is transformed into volatile DMSe either directly by the catalytic action of methylmethionine hydrolase or via intermediate product, 3-dimethylselenoniopropionate (DMSeP).

Toxicity of selenium. Plants vary in the Se accumulating capacity. Non-accumulator plants are those which accumulate less than 25 $mg \cdot kg^{-1}$ of Se and are incapable to tolerate higher Se in the soil and Se toxicity ensues following an accumulation of 10–100 $mg \cdot kg^{-1}$ of Se in dry matter, but it is also regulated by rhizospheric selenite and sulphate ratio [7]. It was suggested in [21] that, the Se-accumulator plants use a number of mechanisms to become Se-tolerant, such as (a) less amount of high concentration of Se become transported into the leaf cells; (b) excess Se become transformed into seleno-amino acids but, incorporation of these seleno-aminoacids into proteins is escaped; (c) sequestration of Se as selenate into vacuolar compartments thereby less interference into sensitive cytoplasmic reactions; (d) enhancing the activities of SeCys methyltransferase and ATP sulphurylase enzymes, thereby, decreasing inorganic Se into organic compound; (e) increasing antioxidant protective activities by conjugating with glutathione (GSH); (f) complexation of Se-binding polypeptides and proteins thus reducing inorganic Se; (g) maximizing the volatilization of organic Se to get rid of plant cells and tissues from Se.

Non-accumulator plants, can accumulate between 2 $mg \cdot kg^{-1}$ (eg. rice) to 330 $mg \cdot kg^{-1}$ in white clover while thousands milligram of Se was found in the tissue of *Astragalus bisulcatus*, a Se accumulator. Excess Se beyond the utilization of plant is accumulated within tissues lead to selenosis with the signs of underdeveloped growth, fading of the leaves and chlorosis [6]. Se toxicity or selenosis in plants is implemented either due to abnormal selenoproteins produced or by promoting oxidative stress, both are harmful in plants. As discussed in the previous section, aberrant seleno-proteins are synthesized due to the replacement of the Cys/Met with SeCys/SeMet in the protein chain.

Distortion of protein structure and function. In a protein chain, cysteine residues are indispensable for maintaining higher order structure of protein and function by forming enzyme catalysis, disulfide bridges, and provide binding sites for metals. Following substitution with SeCys, tertiary protein structure gets disrupted due to the formation of a bigger diselenide bridge with alteration in redox potential upsetting enzyme activities. Chloroplast proteins like NifS-like protein (Fe-S cluster proteins) and mitochondrial electron transport chain are susceptible to SeCys substitution [16, 17] as larger size of the Fe-Se cluster are unfit to apoproteins. Replacing Fe-S cluster with Fe-Se in *Klebsiella pneumonia*

nitrogenase enzyme, five-fold decrease in activity was reported. Therefore, SeCys replacing cysteine is more destructive to protein structure and functionality in comparison to substitution with SeMet. Larger size and higher deprotonation nature of SeCys made it more reactive than cysteine, as observed for methionine sulfoxide reductase enzyme, catalytic function of which got impaired following replacement with SeCys [22]. To avoid Se incorporation into the peptide chains, plants execute strategies either by reducing the production of selenoproteins or by promoting their degeneration and thus boosting Se tolerance. Formation of methylated Selenocysteine (MeSeCys) is one strategy for Se tolerance. e.g. *Arabidopsis and Brassica juncea* showing overexpression of SeCys-methyltransferase. Another chloroplastic enzyme (NifS-like protein), cystathionine gamma synthase has activity like that of selenocysteine lyase, overexpression of which in *Brassica juncea* showed higher Se tolerance in this plant by decreasing Se incorporation in protein [23].

Se toxicity and oxidative stress. Like other heavy metals, pro-oxidant action of both forms of inorganic Se has been reported at higher concentration causing oxidative stress generating reactive oxygen species (ROS). During Se stress higher glutathione activity was observed in Se tolerant plants while reduced glutathione activity was found in Se non-tolerant plants. Discrete observations from cad2-1 and apr2-1 mutant plants defective in glutathione synthesis displayed stress for Se. A number of studies indicated increased accumulation of ROS due to upsurge activities of antioxidant enzymes during Se stress [14] and adverse effects like augmented lipid peroxidation, cell mortality and faulty ascorbic acid biosynthetic pathway (as found in vtc^1 mutant plants) were found in some plants [24]. Accumulation of ROS leads to oxidative attack to proteins by oxidizing Tyr, Met, Cys, Trp amino acids in proteins, thereby; increase the susceptibility of proteins toward proteolysis. An upsurge of oxidized protein levels was found following Se exposure [23]. Selenite induced oxygen dependent DNA fragmentation has been previously reported, later supported by authors claiming base oxidation and DNA strand breaks for ROS generation [25].

Reactive nitrogen species (RNS), Se toxicity and protein modifications. Like ROS, nitric oxide (NO) also has the capability to react with several biological molecules known to yield nitrous acid (HNO_2), peroxynitrite ($ONOO^-$), S-nitrosoglutathione (GSNO), nitrogen dioxide radical ($NO_2^{\cdot-}$) or dinitrogen trioxide (N_2O_3), dinitrogen tetroxide (N_2O_4). Molecular NO and its products targeting definite groups of proteins results in their post-translational changes by metal nitrosylation, S-nitrosylation, and nitration based on the redox potential (PTMs). NO attacks the thiol groups of cysteine amino acids forming S-nitrosothiol in reversible S-nitrosylation that consequences to conformational changes in the proteins thereby affecting their function like protein-protein interactions or protein localization [26]. Besides, NO has high affinity to the metal centres (Cu^{2+}, Zn^{2+}, Fe^{2+} or Fe^{3+}) of some proteins like haemoglobin forming metal-nitrosyl complexes and peroxynitrite promotes irreversible nitration of tyrosine and tryptophan amino acids; all these conformational changes ultimately steer proteins dysfunction.

Reports are available on the disturbed equilibrium of RNS in Se stress in plant cells [27]. In *Arabidopsis* roots at seedling stage long term Se exposure leads to higher NO and GSNO levels and mediates Se toxicity. Biochemical evidences revealed that Se induces nitric oxide synthase and nitrate reductase activities to overproduce NO; also evidenced from the experiments with pea plants following selenite supplementation, where, augmented endogenous NO was found in their roots and leaves along with toxicity indications [28].

Se induced hormonal imbalance. Hormonal balance in plants is highly affected due to Se stress. Studies of [29] reported that some of the growth regulatory hormones such as cytokinins, auxins, and ethylene (ET) leads to Se induced growth alteration. Reports also exist about Se made upregulation of genes associated with the biosynthesis of ET ethylene-response factor (ERF) and jasmonic acid (JA) by selenium [23] supporting the role of these hormones in Se tolerance.

Se induced disruption in element homeostasis. Intervening action of Se in macro and microelement homeostasis has been revealed following Se uptake [30]. The intensified activity of nitrate reductase, nitrogen reserve and sulphur content in wheat following low level Se exposure was observed, which may play significant role in Se phytotoxicity.

Conclusion and future prospects. Se, an indispensable micronutrient for both animals and plants, is toxic when accrued in higher concentration. Substantial research work has been carried out for perceiving the mechanism of acquisition, metabolism and accumulation of Se by plants which have notable application in the field of agroindustry and medicine. A number of research works are carried out focusing on molecular machineries and ecological aspects of Se hyperaccumulation targeting the plant propagation with greater probability of Se accumulation. These can be exploited for remediation of seleniferous soil and also for Se biofortification.

Researchers have identified specific genes and proteins for Se uptake, metabolism, transport and accumulation in plants. The knowledge has been implemented for gene manipulation of the Se metabolism process to intensify the accumulation of Se in harvested tissues in plants and facilitate Se volatilization into the atmosphere. Further insight is essential regarding the specific receptor involved in instigating jasmonic acid, ethylene, and salicylic acid-mediated defensive pathways upregulating the Se acquirement mechanisms in plants.

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