9

Selenium Metabolism in Plants: Molecular Approaches

Özgür Çakır[1], Neslihan Turgut-Kara[1*] and Şule Arı[1,2]
[1]*Department of Molecular Biology and Genetics, Faculty of Science*
Istanbul University, Istanbul
[2]*Research and Application Center for Biotechnology and*
Genetic Engineering, Istanbul
Turkey

1. Introduction

Selenium (Se) is placed in Group VIA of the Periodic Table. Its chemistry is similar to sulfur (S). Practically all small organic selenium compounds are isologues of corresponding sulfur compounds. With a few exceptions, they are also isologues of sulfur amino acids or derivatives thereof. Selenium plays an indispensable role for humans, animals and microorganisms. It is beneficial for the metabolism at lower concentrations, whereas at higher concentrations it becomes toxic. In other words, the range between deficiency and toxicity is very narrow. Short-term consumption of high levels of Se by human and animals may cause nausea, vomiting, and diarrhea, whereas chronic consumption of high concentrations of Se compounds can result in a disease called selenosis (Goldhaber, 2003). Excess selenium in the environment can be the result of either natural geological processes or industrialization.

Selenium acts as a cancer preventative agent when given in pharmacological amounts. Numerous studies have demonstrated the efficacy of methylselenocysteine (MeSeCys) in preventing mammary cancer in mammalian model systems, and importantly, MeSeCys has been shown to be twice as active as Se-methionine (the primary component of Se-yeast supplements) in preventing the development of mammary tumors (Ip & Ganther, 1992; Lu et al., 1996; Ip et al., 2000; Finley and Davis 2001; Medina et al., 2001; McKenzie et al., 2009). This non-protein seleno amino acid is produced in certain plants including members of the *Astragalus, Allium* and *Brassica* genera (Cai et al., 1995; Clark et al., 1996). While the specific mechanism for the anticancer activity of Se has not been fully elucidated, researchers have speculated that the Se could be effect the cell cycle then induce apoptosis in cancer cell lines (Foster et al., 1986; Cai et al., 1995; Andreadou et al., 1996; Ganther & Lawrence, 1997; Combs & Gray, 1998; Ip 1998; Sinha et al., 1999; Lu & Jiang, 2001; Kim et al., 2001; Wang et al., 2002; Ip et al., 2000). There is also evidence that Se may inhibit tumor angiogenesis (Lu & Jiang 2001). The molecular mechanism of cancer prevention by selenium using the genomics approach was studied on the target organs breast, prostate, colon and lung. The results of the microarray analysis indicated that selenium, independent of its form and the target organ, alters several genes in a manner that can account for cancer prevention. Selenium can

* Corresponding Author

up regulate genes related to phase II detoxification enzymes, certain selenium-binding proteins and apoptotic genes, while down regulating those related to phase I activating enzymes, stress responsive genes, cytoskeletal and cell adhesion functions and cell proliferation (El-Bayoumy & Sinha, 2005; Goulet et al., 2007). Also, Goulet and her colleagues were demonstrated that an increase in the occupancy of phospho-histone H3 at selected promoters, which suggest that SeMet can influence gene expression by chromatin remodeling in a manner of epigenetic (Goulet et al., 2007).

Plant roots can take up Se from soil as selenate, selenite, or organoselenium compounds. The biosynthesis of most selenium compounds in nature follows the pathways leading to isologous sulfur compounds in plants (Table 1) as well as yeast, bacteria or animals. Roots

Compounds	Species
Selenocysteine	*Vigna radiata*
Selenocystathionine	*Astragalus praleongus*
	Astragalus pectinatus
	Neptunia amplexicaulis
	Morinda reticulate
	Brassica oleracea capitata
	Stanleya pinnata
	Lecythis ollaria
Se-Methylselenocysteine	*Astragalus crotalariae*
	Astragalus bisulcatus
	Astragalus praleongus
	Brassica oleracea capitata
	Brassica oleracea botrytis
	Allium sativum
	Allium cepa
	Allium tricoccum
	Melilotus indica
	Oonopsis condensata
	Phaseolus lunatus
γ-Glutamyl-*Se*-methylselenocysteine	*Astragalus bisulcatus*
	Allium sativum
	Allium cepa
	Phaseolus lunatus
Selenomethionine	*Brassica juncea*
	Brassica oleracea capitata
	Allium tricoccum
	Melilotus india
Se-Methylselenocysteine *Se*-oxide	*Brassica oleracea capitata*
Selenobiotin	*Phycomyces blakesleeanus*
γ-Glutamylselenocystathionine	*Astragalus pectinatus*
γ-Glutamylselenomethionine	*Allium sativum*
3-Butenyl isoselenocyanate	*Stanleya pinnata*
Selenosinigrins	*Armoracia lapathifolia*
	Stanleya pinnata
Selenosugars	*Astragalus racemosus*

Table 1. Low molecular weight selenium-containing compounds in plants (adapted from Birringer et al., 2002)

take up selenate faster than selenite at the same concentration but acquire organoselenium compounds, such as selenocysteine (SeCys) and selenomethionine (SeMet), most avidly (White et al., 2007). Thereafter it is metabolized (via sulfur assimilation pathway) in that selenocysteine, SeMet and other Se analogues of various S metabolites (Ellis and Salt, 2003). The nonspecific incorporation of seleno amino acids into proteins is thought to contribute to Se toxicity (Brown & Shrift, 1981). Plants differ in their ability to metabolize and tolerate Se, and divided into three groups according to Se accumulation capacity: primary accumulators (hyperaccumulators), secondary accumulators, and non-accumulators. One proposed mechanism of Se tolerance in accumulator plants is the specific conversion of potentially toxic seleno amino acids into nonprotein derivatives such as MeSeCys. Some *Allium* and *Brassica* species, when grown in Se enriched medium, can accumulate 0.1–2.8 μmol g^{-1} dry weight MeSeCys or its functional equivalent γ-glutamylmethylselenocysteine (γ-glutamyl-Se-MeSeCys). However, certain specialized Se accumulating plants, such as *Astragalus bisulcatus*, accumulate up to 68 μmol g^{-1} dry weight Se (6000 mg kg^{-1} dry weight), of which 90–95% is MeSeCys in young leaves. The seeds of these plants also accumulate Se as γ-glutamyl-Se-MeSeCys (Pickering et al., 2003). During to incorporation of the active seleno amino acid SeCys into essential selenoproteins some of the key enzymes play important roles as a regulatory manner. Mutation or overexpression analysis were showed that ATP sulphurylase, selenocystein methyltransferase (SMT), APS reductase, serine acetyltransferase, selenocysteine lyase, selenocysteine transferase, cystathionine-γ-synthase, and chloroplast selenocysteine lyase are important enzymes on the way Se tolerance and accumulation (Figure 1).

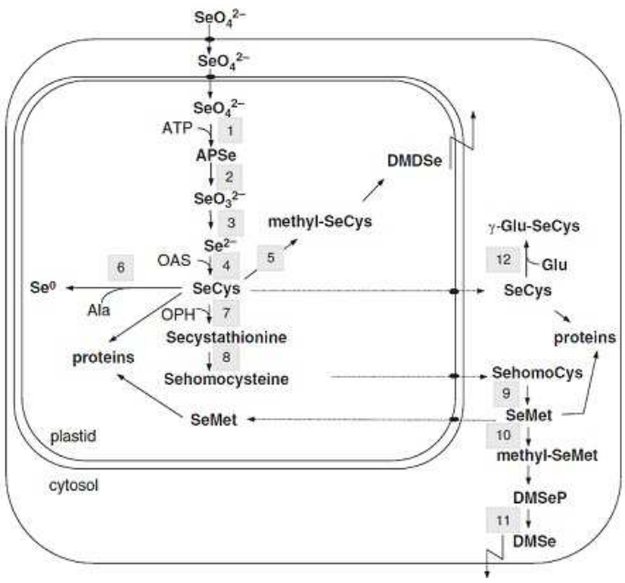


Fig. 1. Schematic overview of Se metabolism in plants. APSe adenosine phospho selenate, OAS O-acetylserine, OPH O-phosphohomoserine, SeCys selenocysteine, SeMet selenomethionine, DMSeP dimethylselenoproprionate, DMSe dimethylselenide, DMDSe dimethyldiselenide. Numbers denote known enzymes. (1) ATP sulfurylase, (2) adenosine phosphosulfate reductase, (3) sulfite reductase (or glutathione), (4) OAS thiol lyase, (5) SeCys methyltransferase, (6) SeCys lyase, (7) cysthathionine-γ-synthase, (8) cysthathionine-β-lyase, (9) methionine synthase, (10) methionine methyltransferase, (11) DMSP lyase, (12) γ-glutamylcysteine synthetase (from Pilon-Smits & Quinn 2010)

SMT is the most important enzyme in Se hyperaccumulating plants. SMT catalyses the methylation of SeCys to MeSeCys, and the gene firstly isolated from hyperaccumulator *A. bisulcatus* (Neuhierl & Bock, 1996; Neuhierl et al., 1999), then isolated some other accumulator and nonaccumulator plant species (Table 2). SMT is constitutively expressed in roots and leaves of *A. bisulcatus*, and does not induced by Se (Pickering et al., 2003). Heterologous expression of AbSMT in transgenic *Arabidopsis thaliana* results in the production of MeSeCys and its derivative γ-glutamyl-Se-MeSeCys, compounds not normally produced in *A. thaliana* (Ellis et al., 2004). Accumulation of MeSeCys was similarly observed in transgenic *Brassica juncea* expressing AbSMT (LeDuc et al., 2004). According to these results, only Se-hyperaccumulating species of *Astragalus* are capable of synthesizing MeSeCys compared with their non-accumulating relatives, and that SMT activity is closely linked with the capacity to hyperaccumulate Se (Sors et al., 2005), it can be hypothesized that Se non-accumulating species do not contain a functional SMT enzyme.

Plants that SMT gene isolated and characterized	**Accumulation capacity**	**Accession number**	**Reference**
Astragalus bisulcatus	Hyperaccumulator	AJ131433.1	Neuhierl et al., 1999
Astragalus chrysochlorus	Secondary accumulator	GQ844862.2	Çakır & Arı (Unpublished data, 2012)
Camellia sinensis	Secondary accumulator	DQ480337.1	Zhu et al., 2008
Brassica oleracea var. italica	Secondary accumulator	AY817737.1	Lyi et al., 2005
Plants that SMT gene isolated			
Astragalus racemosus	Accumulator	GQ398501.1	Sors et al., 2009
Astragalus pectinatus	Accumulator	GQ398502.1	
Astragalus ceramicus	Nonaccumulator	GQ398503.1	
Astragalus drummondii SMT-like	Nonaccumulator	GQ398504.1	
Astragalus leptocarpus	Nonaccumulator	GQ398505.1	

Table 2. Plants that SMT gene isolated and characterized so far

In general, accumulator species likely do not have any Se-specific pathways but take up and metabolize Se and S indiscriminately, also current knowledge demonstrates that Se essentiality in higher plants are still not definitive. The potential health benefits of some Se compounds when combined with the increased application of phytoremediation techniques in contaminated soil were augmented the study of Se biochemistry in plants. Thus, Se metabolism in plants has been reviewed by a number of authors (Terry et al., 2000; Briggers et al., 2002; Germ et al., 2007a; White et al., 2007; Pilon & Quinn, 2010; De Filippis 2010). The present Chapter will focus on basic aspects of molecular selenium metabolism in plants and future perspectives of phytoremediation techniques.

2. Selenium metabolism in plants

2.1 Uptake and transport

Selenium could be occurs in following oxidation states: –2 (selenide), 0 (elemental Se), +4 (selenite), and +6 (selenate). Selenate is accumulated in plant cells via the process of active transport (Brown & Shrift, 1982). Unlike selenate, there is no evidence that the uptake of selenite is mediated by membrane transporters. Alternatively, plants can take up organic forms of Se such as selenomethionine (SeMet) actively, but not effectively (Abrams et al., 1990) (Figure 2). Selenate directly competes with sulfate for uptake by plants. It has been proposed that both anions are taken up via a sulfate transporter in the root plasma membrane. Selenate uptake in other organisms, including *Escherichia coli* (Lindblow-Kull et al., 1985) and yeast (Cherest et al., 1997), is also mediated by a sulfate transporter.

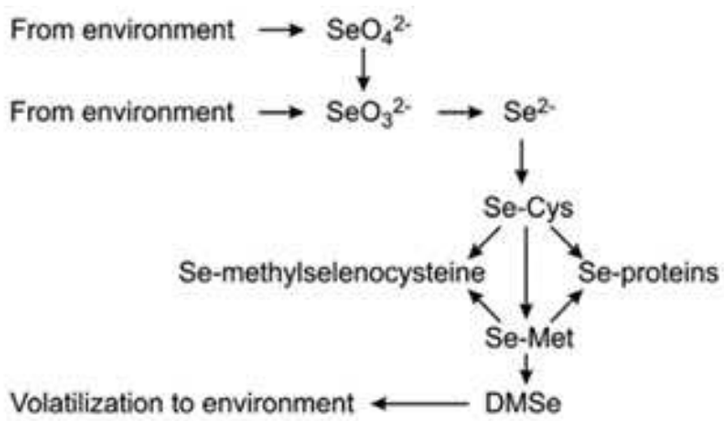


Fig. 2. Schematic representation of the main steps of Se metabolism in plants (from Germ et al., 2007b)

The expression of the sulfate transporter genes is regulated by the S status of the plant, as well as by the regulators, glutathione (GSH) and O-acetylserine. While high levels of sulfate and GSH decrease transcription, high levels of O-acetylserine increase transcription of the high-affinity transporter genes as well as sulfate uptake. Thus, increasing O-acetylserine levels can potentially increase selenate uptake (Davidian et al., 2000). Terry and colleagues reported that application of O-acetylserine increased selenate accumulation in Indian mustard almost two-fold compared to untreated plants, and they speculate that O-acetylserine, a precursor of cysteine (Cys) and a product of the nitrate assimilation pathway, might pivotal importance as a coregulator of the S and nitrogen metabolic pathways. Overexpression studies on the sulfate transporter genes increased selenate accumulation up to two-fold in transgenic plants compared to wild type. These information show that sulfate transporter is involved in selenate uptake (Terry et al., 2000).

Translocation of the Selenium in the plant parts depend on the form of how it is supplied. Zayed et al (1998) showed that the shoot/root ratio of the Se concentrations ranged from 1.4 to 17.2 when selenate was supplied but was only 0.6 to 1 for plants supplied with SeMet and less than 0.5 for plants supplied with selenite. Time-dependent kinetics of Se uptake by Indian mustard showed that only 10% of the selenite taken up was transported from root to

shoot, whereas selenate was rapidly transported into shoots (De Souza et al., 1998). Thus, plants transport and accumulate substantial amounts of selenate in leaves but much less selenite or SeMet. Selenite is rapidly converted to organic forms of Se such as SeMet which are retained in the roots (Zayed et al., 1998), this helps to explain why selenite is poorly translocated to shoots. In addition, partitioning of Se in various plant parts is species specific, also depends on the stage of development, and on physiological condition of the plant. In the accumulators, Se is gathered in young leaves during the early vegetative and reproductive stage of growth, and 3,5 fold high levels of Se are found in seeds while the Se content in leaves is drastically reduced. Non accumulating cereal crop plants, often show about the same Se content in grain and in roots, but smaller amounts in the stems and leaves. Distribution of Se in plants also depends on the form and concentration of Se supplied to the roots and on the nature and concentration of other substances, especially sulfates, accompanying the Se (Zayed et al., 1998). Plants can also absorb volatile Se from the atmosphere via the leaf surface. The Se absorbed by the leaves is accumulated in roots as inorganic selenite, selenoglutathione (SeGSH), SeMet, and protein-bound SeMet..

2.2 Accumulation in plants

Hyperaccumulation is the ability of certain plants to accumulate extraordinarily high concentrations of metals and trace elements, even when grown in soil with low concentrations (Baker & Brooks, 1989). This ability for certain elements gives some selective advantage to the hyperaccumulators plants. A selective benefit of hyperaccumulation is predominant occurrence of this kind of plant species on soils that are enriched in the elements. Hypothesis for the ecological significance of hyperaccumulation include drought tolerance, allelopathy, and chemical defense against herbivores and/or pathogens (Boyd & Martens, 1993; Jhee et al., 1999, Galeas et al., 2007). Some plant species are known to hyperaccumulate more than one metals or trace elements. At least 400 plant species in 45 plant families are hyperaccumulators, and these have been found in many different geographic locations (Reeves & Baker, 2000). Hyperaccumulation of Se has been observed in the plant families *Asteraceae, Brassicaceae, Chenopodiaceae, Lecythidaceae, Fabaceae, Rubiaceae* and *Scrophulariaceae*, are only found on seleniferous soils (Beath et al., 1934; Cannon, 1960; Reeves & Baker, 2000). The Se accumulator species can tolerate Se in the field up to 10.000 mg kg^{-1} DW (Pilon-Smits & Quinn, 2010).

Plants differ in their ability to accumulate Se when they grow on seleniferous soils, and divided into three groups according to Se accumulation capacity: primary accumulators (hyperaccumulators), secondary accumulators, and non-accumulators. Accumulator plants can accumulate from hundreds to several thousand milligrams of Se kg^{-1} dry weight in their tissues, without any negative effects. That ability is mainly due to the reduction of the intracellular Se concentration of Se–Cys and Se–Met which are normally incorporated into proteins (Pilon-Smits & Quinn, 2010).

Special attention should be paid to *A. bisulcatus*, since this is the best-characterized Se accumulator. This species grows on naturally Se-rich soils in the southwestern part of the USA. Typical for these plants is their strong Se (sweet) odor. In its natural habitat, that species can take up to 0.65% Se dry weight in their shoots (Dumont et al., 2006). When the plants are grown on a selenate rich soil, the older leaves contain mainly inorganic Se (91%),

whereas in the young leaves 90–95% of the Se is organic. The roots show the lowest Se level when compared with others tissues. Although, at root level, the Se is mainly organic (92%). There is a presumption that the MeSeCys in the young leaves is metabolized and that the Se is reoxidized to form selenate as the leaves become older. Another explanation would be that the MeSeCys is exported from the young shoots as it ages and accumulates in the even younger shoots. An alternative explanation is the metabolization of MeSeCys to DMSe, which would explain the malodorous nature of the plant used for protection against insect attack. In these plants, the main compound found is MeSeCys, one of the common species found in Se accumulators (Dumont et al., 2006; Pickering et al., 2003).

On the nonaccumulators side, most forage and crop plants, as well as grasses, contain less than 25 mg Se kg^{-1} dry weight and do not accumulate Se much (Brown & Shrift, 1982). Although Se accumulators grow on seleniferous soils, not all plant species on seleniferous soils are Se accumulators: some plants accumulate only a few milligrams of Se kg^{-1} dry weight. For example, the genus *Astragalus* contains both Se accumulating species and nonaccumulating species, and they can grow next to each other on the same soil (Duckart et al., 1992).

Primary accumulators have discrimination coefficients (DC_i= $[Se/S]_{plant}/[Se/S]_{solution}$) of more than one in solution culture, and have concentrations of Se in the range of thousands of mg per kg dry weight (Ellis & Salt, 2003). Primary accumulators include various *Astragalus* species, which are members of the Fabaceae, as well as *Stanleya pinnata*, a member of the *Brassicaceae* (Feist & Parker, 2001). Secondary accumulators take up Se in proportion to the amount of Se available in the soil, they have a DC_i of less than one, and tissue concentrations of Se in the hundreds of mg kg^{-1} (Bell et al., 1992). Members of this group include species of *Astragalus*, *Aster, Atriplex* and *Melilotus*, as well as *Brassica juncea* (Indian mustard) (Banuelos & Meek, 1990; Guo & Wu, 1998; Ellis & Salt, 2003). Recently, Ari and her colleagues has identified an *Astragalus* species, *A. chrysochlorus*, as a new secondary Se-accumulator plant with a typical Se concentration of more than several hundred milligrams of Se kg^{-1} dry weight in tissues (DC_i=0.95) when grown on tissue culture media containing sodium selenate (Ari et al., 2010).

Selenium hyperaccumulations may increase the surrounding soil Se concentrations (phytoenrichment). The enhanced soil Se contents around hyperaccumulators can impair the growth of Se-sensitive plant species, pointing to a possible role of Se hyperaccumulation in elemental allelopathy (El Mehdawi et al., 2011). Selenium also may increase the tolerance of the plants to drought-induced oxidative damage and high temparature stress by enhancing their antioxidant defense and methylglyoxal detoxification system (Hasanuzzaman & Fujita, 2011; Djanaguiraman et al., 2010).

2.3 Incorporation of Se into protein

In most of the selenoproteins discovered so far, selenium is present as a selenocysteine residue that is integrated into the main chain of amino acids, as was first demonstrated for formate dehydrogenase and glutathione peroxidase (Birrigger et al., 2002). Whenever investigated, the selenocysteine residue was shown to be of pivotal importance for the catalytic efficiency of such proteins. The incorporation of selenocysteine into these selenoproteins is directed by a specific tRNA that recognizes a UGA codon. Normally, the UGA codon acts to terminate translation. In combination with a selenocysteine insertion sequence (SECIS), however, the

UGA codon is recognized by the selenocysteine tRNA, which directs the insertion of selenocysteine (Gladyshev & Kryukov, 1999; Low and Berry, 1996; Ellis & Salt, 2003).

Organisms that require Se for normal cellular function contain essential selenoproteins, such as formate dehydrogenase, glutathione peroxidase, and selenophosphate synthase. Reports have suggested the presence of selenoproteins in plants, but there is no direct evidence for the specific incorporation of selenocysteine in vascular plants. However, plants are thought to assimilate SeCys, where SeCys is metabolized to SeMet, which are both nonspecifically incorporated into proteins. SeCys is formed by the action of Cys synthase, which couples selenide with O-acetylserine (Ng & Anderson, 1978) (Figure 3). GS-Se^- may be the physiological substrate of Cys synthase rather than free Se^{2-} (Tsang & Schiff, 1978). Kinetic studies of *in vitro* enzymes were showed that cystathionine- synthase exhibited a preference for SeCys: It had a higher affinity for SeCys (K_m=70 μM) than for Cys (K_m=240 μM) (Dawson & Anderson, 1988). Cystathionine-lyase did not differentiate between the Se and S forms of cystathionine, since the enzyme had a similar affinity for cystathionine (K_m=0.31 mM) and selenocystathionine (K_m=0.35 mM) (McCluskey et al., 1986). The most likely enzyme for the synthesis of SeMet from SeHomoCys is the cytosolic enzyme, Met synthase (Figure 3). Selenium is readily incorporated into proteins in nonaccumulator plants treated with Se (Brown & Shrift, 1982). The incorporation into proteins occurs through the nonspecific substitution of SeCys and SeMet in place of Cys and Met, respectively (Figure 3). Studies showed that both Met and SeMet are substrates for the methionyl t-RNA synthetase (Terry et al., 2000).

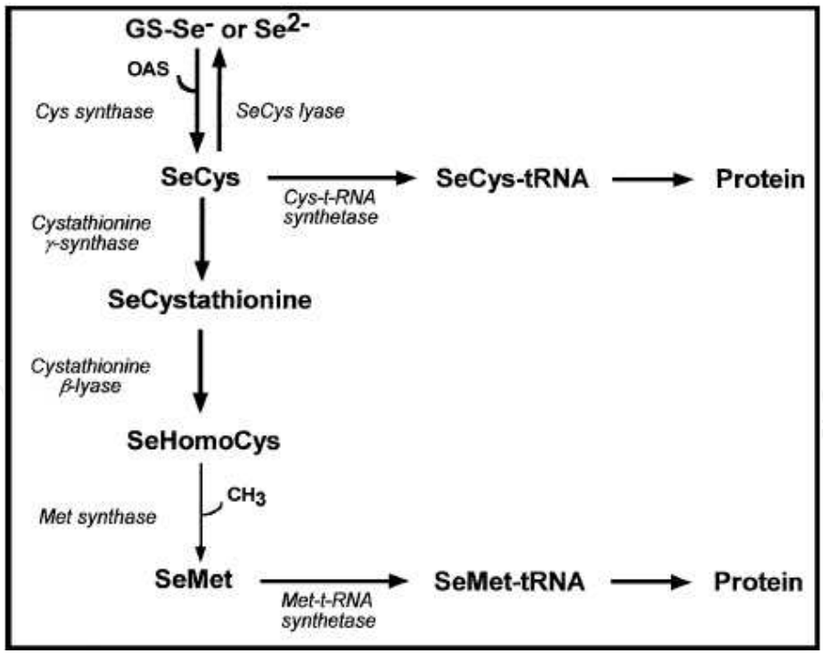


Fig. 3. Schematic pathway of the incorporation of selenide into SeCys, SeMet, and proteins. SeCys lyase is a Se-specific enzyme, whereas all the other enzymes shown recognize both S and Se. The only exception is Met synthase, which is involved in Met synthesis, and is very likely to be involved in SeMet synthesis although there is no evidence supporting this (from Terry et al., 2000).

3. Health benefits of Selenium

Severe selenium deficiency of human and animals have been observed in isolated selenium-poor areas. Although Se deficiency is rare in the US, it does occur in several parts of the world, such as China, where concentrations of Se in the soil are low. Consumption of food containing less than 0.1 mg Se kg^{-1} results in deficiency. Regular consumption of food containing more than 1 mg Se kg^{-1} results in only toxicity, but 1000 mg Se kg^{-1} DW can lead to acute Se poisoning and death for humans and animals **(**Wilber, 1980).

In the 1960s, selenium was proposed to be an essential trace element as a consequence of human and animal studies (Birringer et al., 2002). Since that time, scientists have showed growing interest on Se studies. The toxic effects of excess Se have been known for some time but, in the past decade, it has become more evident that Se has many potential health benefits beyond meeting basic nutritional requirements. In the seventies, Chinese scientists reported that severe selenium deficiency causes diseases in humans: Keshan disease, which is a fatal cardiomyopathy, and Kashin–Beck disease, a disabling chondronecrosis (Birringer et al., 2002). In addition, Se deficiency can lead to heart disease, hypothyroidism and a weakened immune system (Combs, 1980). Concerns about the health hazards from overexposure now tend to become overwhelmed by a bewildering discussion of the benefits. Adequate alimentary selenium supply is claimed to delay the onset of ageing, cardiovascular diseases and cancer, to enable an optimum immune response, to guarantee an appropriate function of the endocrine system, and to be indispensable for male reproduction. For example, in a long-term double-blind studies, supplemental Se was associated with significant reductions in lung, colorectal and prostate cancers (Ip & Ganther, 1992). In 1996, Clark and co-workers reported that supplementation of people with selenized yeast is capable of reducing the overall cancer morbidity by nearly 50%. The possible anti-cancer effect of Se might be summarize according to critical reviews on this area that appear to be widely accepted (Birringer et al., 2002): optimize somehow glutathione peroxidases (GPx) activities; provides optimum selenoprotein expression; the nature of the Se compound has critical importance; although, synthetic selenium compounds do not support selenoprotein synthesis, but also found to be anticarcinogenic. "Need to be proved" advances have led to several mechanisms being proposed for the anticancer activity of Se: antioxidant protection (via selenoproteins); altered carcinogen metabolism; enhanced immune surveillance; regulation of cell proliferation and tumor cell invasion and inhibition of neoangiogenesis (Zeng & Combs, 2008).

4. Molecular approaches to alter Se metabolism in plants

Several different transgenics have been obtained so far. They were showed enhanced Se tolerance, accumulation, and assimilation from inorganic to organic Se, and volatilization. Selenium accumulation was up to nine-fold higher than wild type and volatilization up to three-fold faster, under laboratory conditions. These findings may be useful for cleaning up of excess levels of Se in the environment and also as fortified foods to prevent Se deficiency related diseases. For example, accumulators of MeSeCys would be especially useful for the anticarcinogenic purpose (Unni et al. 2005). In a first step to assess the transgenics' potential for phytoremediation or as Se-fortified food, they were tested for their capacity to accumulate Se from naturally seleniferous soil and from Se-contaminated sediment.

The genetic engineering strategcy for biofortification and phytoremediation are both the same: higher Se levels in harvestable plant parts are purpose. A significant difference between genetic engineering for biofortification and phytoremediation objectives that Se in tissues of renewable plants should not get through toxic concentrations. In biofortification case, some Se compounds have more powerful anti-carcinogenic properties than the others. For example, MeSeCys is the best form of Se to use in biofortified foods, and according to that reason overexpression of SMT may be the best purpose of biofortification. It may also be possible to overexpress some targeted gene(s) in specific plant tissues, such as in the grain, or to overexpress these targeted genes so that anticarcinogenic Se compounds can be readily extracted for production.

4.1 Phytoremediation and biofortification

Selenium accumulator plants can convert inorganic selenate and selenite to SeCys and other organic selenocompounds, including volatile forms. Se hyperaccumulators may have special metabolic pathways for methylation of SeCys and the conversion of methyl-SeCys to volatile DMDSe. Transgenic approaches have been used to further enhance plant Se accumulation, tolerance, and volatilization (Table 3).

Selenate is translocated without chemical modification through the xylem to the leaves after its root absorption via the sulfate transporter (De Souza et al. 1998, Zayed et al. 1998). Afterwards, selenate is metabolized by the enzymes responsible of sulfate assimilation when it enters chloroplasts. ATP sulfurylase catalyzes the key step in the reduction of selenate by activating it to adenosine phosphoselenate (APSe), an activated form of selenate. *In vitro* ATP sulfurylase has been shown to activate selenate, as well as sulfate (Burnell 1981; Dilworth & Bandurski, 1977; Shaw& Anderson, 1972). A gene construct containing the *A. thaliana aps1* gene (Leustek et al. 1997), with its own chloroplast transit sequence, fused to the *Cauliflower* Mosaic Virus 35S promoter cloned into indian mustard plants to overexpress ATP sulfurylase. Molecular studies provided *in vivo* evidence that ATP sulfurylase is responsible for selenate reduction, and that this enzyme is rate limiting for selenate reduction and Se accumulation (Pilon-Smits et al. 1999). X-ray absorption spectroscopy (XAS) analysis of wild-type Indian mustard plants supplied with selenate showed that selenate was accumulated in both roots and shoots, but when selenite was supplied, an organo-Se compound (similar to SeMet) accumulated (De Souza et al. 1998). It is concluded that the reduction of selenate was rate limiting to selenate assimilation. This rate-limiting step was overcome in transgenic plants overexpressing ATP sulfurylase because these plants accumulated a SeMet-like compound when supplied with selenate (Pilon-Smits et al. 1999). In another study, transgenic *A. thaliana* overexpressing both ATP sulfurylase and APR (APS reductase) had a significant enhancement of selenate reduction as a proportion of total Se, whereas SAT (serine acetyl transferase) overexpression resulted in only a slight increase in selenate reduction to organic forms. In general, total Se accumulation in shoots was lower in the transgenic plants overexpressing ATPS, PaAPR (*P. aeruginosa* APR), and SAT. Root growth was adversely affected by selenate treatment in both ATPS and SAT overexpressors and less so in the PaAPR transgenic plants. It is concluded that ATPS and APR are major contributors of selenate reduction in planta. However, Se hyperaccumulation in *Astragalus* is not driven by an overall increase in the capacity of these enzymes, but rather by either an increased Se flux through the S assimilatory pathway, generated by the biosynthesis of the sink metabolites MeCys or MeSeCys (Sors et al., 2005).

The dominant adenosine 5'-phosphosulfate reductase (APR2) in *A. thaliana* converts activated sulfate to sulfite, a key reaction in the sulfate reduction pathway. apr2-1 transgenic plants had decreased selenate tolerance and photosynthetic efficiency. Sulfur metabolism was perturbed in apr2-1 plants grown on selenate, as observed by an increase in total sulfur and sulfate, and a 2-fold decrease in glutathione concentration. Knockout of APR2 also increased the accumulation of total selenium and selenate. However, the accumulation of selenite and selenium incorporation in protein was decreased in apr2-1 mutants. Decreased incorporation of selenium in protein is typically associated with increased selenium tolerance in plants. However, because the apr2-1 mutant exhibited decreased tolerance to selenate, Grant et al. (2011) proposed that selenium toxicity can also be caused by selenate's disruption of glutathione biosynthesis leading to enhanced levels of damaging reactive oxygen species.

As described above, selenium can be assimilated and volatilized via the sulfate assimilation pathway. Cystathionine-γ-synthase (CgS) is the enzyme which catalyzes the synthesis of Se-cystathionine from Se-cysteine, the first step in the conversion of Se-cysteine to volatile dimethylselenide. Overexpression of CgS in *B. juncea*, the first enzyme in the conversion of SeCys to SeMet, resulted in two to threefold higher volatilization rates compared to untransformed control plants (Van Huysen et al., 2003). The CgS transgenics accumulated 40% less Se in their tissues than wild type probably as a result of their enhanced volatilization. Probably due to their lower tissue Se levels the CgS transgenics were also more Se tolerant than wildtype plants. Van Huysen et al. (2003) studied APS and CgS transgenics to evaluate for their capacity to accumulate Se from soil that is naturally rich in Se. In that study, wild-type Indian mustard and the Se hyperaccumulator *S. pinnata* were used for comparison. After growing 10 weeks on Se soil, similar to those of *S. pinnata*, the APS transgenics contained 2,5-fold higher shoot Se levels than wild type Indian mustard. The CgS transgenics contained 40% lower shoot Se levels than wild type. These findings were very significant that they are the first report on the performance of transgenic plants on Se in soil and they showed the potential of genetic engineering for phytoremediation.

Selenocysteine lyase (SL) catalyzes the removal of selenium from L-selenocysteine to yield L-alanine. This enzyme is proposed to have a role in the recycling of the micronutrient selenium from degraded selenoproteins which contain selenocysteine residue. Selenocysteine lyase has a strict substrate specificity for L-selenocysteine and no activity for L-cysteine. However, it is unknown how the enzyme distinguishes between selenocysteine and cysteine. To manipulate plant Se metabolism, another genetic engineering approach is the prevention of the toxic process of its nonspecific incorporation into proteins. A mouse SL was expressed in *A. thaliana* and *B. juncea* (Pilon et al. 2003; Garifullina et al. 2003). Selenocysteine lyase enzyme specifically breaks down SeCys into alanine and elemental Se. The SL transgenics showed reduced Se incorporation into proteins. Se tolerance increased when mouse SL was expressed in the cytosol of *A. thaliana*, but decreased when it was expressed in the chloroplast (Pilon et al. 2003). All the transgenic SL plants showed enhanced Se accumulation, up to twofold compared to wildtype plants. Similar results were obtained when an *A. thaliana* homologue of the mouse SL (called CpNifS) was discovered and overexpressed: the CpNifS transgenics showed less Se incorporation in proteins, twofold enhanced Se accumulation, as well as

enhanced Se tolerance (Van Hoewyk et al., 2005). This enzyme has been cloned from *A. thaliana* and expression of this gene in *B. juncea* originally appeared to reduce selenate toxicity, and Banuelos et al. (2007) attributed this to a reduction in incorporation of Se into proteins. The gene used in this study may be similar to the *AtCpNifS* chloroplast gene used by Van Hoewyk et al. (2005).

In *Arabidopsis* genome, there are three highly conserved homologues of the mammalian 56-kD selenium-binding protein (SBP). A transgenic approach is used to study the function of SBP in this model plant by constitutively overexpressing and down-regulating the endogenous *Atsbp1* gene. It was employed both a conventional antisense method and gene silencing by intron-containing hairpin RNAs. *Atsbp1*-overexpressing and silenced plants were phenotypically normal, under standard growth conditions, when compared with wild type plants. Transgenic plants exhibited different growth responses to exogenously supplied selenite, which correlated with the expression levels of *Atsbp1.* Plants with increased *Atsbp1* transcript levels showed enhanced tolerance to selenite, while plants with reduced levels were more sensitive. Results indicate that *Atsbp1* appears to be involved in processes controlling tolerance of *Arabidopsis* to selenium toxicity (Agalou et al., 2005). A more distant related family of genes that well studied in *A. thaliana,* induce higher levels of binding polypeptides and proteins. It was recently found by Dutilleul et al. (2008) that expression of specific binding proteins for Se also delivered tolerance to cadmium (Cd), most likely also by binding this heavy metal (Dutilleul et al. 2008).

The *Sultr 123* gene family orchestrate sulphate transporters, and by co-operation may also regulate Se transportation. Lydiate et al. (2007) used 'knock-down' technology in *A. thaliana,* determined that *Sultr 123* genes reduced high affinity sulphate transporters transportation of Se and stated that reduced, but had little effect on selenite transportation (Table 3). The *Sultr* gene family are similar to the *SHST* family of sulphate transporter genes.

Se upregulates transcripts that regulate the synthesis and signaling of ethylene and jasmonic acid. *Arabidopsis* mutants which are defective in ethylene or jasmonate response pathways exhibited reduced tolerance to Se, therefore, it suggests an important role for these hormones in Se tolerance. Selenate upregulated a variety of transcripts that were also induced in stress conditions. Selenate seemed to repress plant development, as suggested by the downregulation of genes involved in cell wall synthesis and auxin-regulated proteins. By discovering the Se-responsive genes plants could be created that can better tolerate and accumulate Se, which may enhance the effectiveness of Se phytoremediation or serve as Se-fortified food (Van Hoewyk et al. 2008).

MeSeCys is produced from selenocysteine and S-methylmethionine by SMT enzme. Neuhierl et al. (1999) were cloned successfully the gene encoding SMT from *A. bisulcatus* (AbSMT). This enzyme belongs to a class of methyltransferases involved in metabolism of S-methylmethionine. It shares significant sequence homology with homocysteine S-methyltransferases (HMT). Despite the fact that both SMT and HMT enzymes catalyze methyl transfer using S-methylmethionine as the methyl donor, they exhibit significant Se-containing (for SMT) and S-containing (for HMT) substrate choice as a methyl acceptor *in vitro* (Neuhierl and Bock, 1996; Ranocha et al., 2000). SMT was found to be constitutively

Transgene	Gene orIgIn (plant specIes)	TransgenIc plant specIes	Effects on Se tolerance and accumulatIon	Reference
APS2 isoform of ATP sulphurylase	*A. thaliana*	*N. tabacum*	No significant effects on Se accumulation and Se tolerance	Hatzfeld et al. (1998)
APS1 isoform of ATP sulphurylase	*A. thaliana*	*B. juncea*	Increase in Se accumulation and an increase in Se tolerance	Pilon-Smits et al. (1999)
CgS (crystathionine - γ-synthase)	*A. thaliana*	*B. juncea*	Lower Se levels in shoots and increased Se tolerance	Van Huysen et al. (2003)
SMT (selenocysteine *m*ethyltransferase)	*A. bisulcatus*	*A. thaliana*	Increase in foliar Se levels and increase in tolerance to selenite, but not selenate	Ellis et al. (2004)
SMT(selenocysteine *m*ethyltransferase)	*A. bisulcatus*	*B. juncea*	Increase in total Se levels and increase in tolerance to selenite, but not selenate	LeDuc et al. (2004)
APS isoform of ATP sulphurylase	*A. thaliana*	*B. juncea*	Increase in Se accumulation and an increase in Se tolerance	Van Huysen et al. (2004)
CgS (Crystathionine - γ-synthase)	*A. thaliana*	*B. juncea*	Lower Se levels in shoots and increased Se tolerance	Van Huysen et al. (2004)
APS1 isoform of ATP sulphurylase	*A. thaliana*	*A. thaliana*	Decreased Se accumulation and Se tolerance	Sors et al. (2005)
PaAPR (APS reductase)	*A. thaliana*	*A. thaliana*	Decrease in foliar Se and increase selenate tolerance	Sors et al. (2005)
SATm (Mitochondria serine acetytransferase)	*T. goesingense*	*A. thaliana*	No significant effects on Se accumulation and tolerance	Sors et al. (2005)
Selenium binding polypeptides/proteins (SBP)	*A. thaliana*	*A. thaliana*	Resistance to Se achieved due to overexpression of Se binding proteins	Agalou et al. (2005)
AtCpNifS chloroplast protein like SeCys lyase	*A. thaliana*	*A. thaliana*	Enhanced selenate tolerance by reducing Se incorporation into protein	Van Hoewyk et al. (2005)
ATP sulfurylase *SMT* (selenocysteine *m*ethyltransferase)	*A. thaliana* *Astragalus bisulcatus*	*B. juncea*	Substantial improvement in Se accumulation from selenate (4 to 9 times increase)	Le Duc et al. (2006)

Transgene	Gene orIgIn (plant specIes)	TransgenIc plant specIes	Effects on Se tolerance and accumulatIon	Reference
Selenocysteine lyase (SeCysIyase)	*A. thaliana*	*B. juncea*	Higher selenate tolerance probably by reducing Se incorporation into protein	Banuelos et al. (2007)
SMT (selenocysteine *m*ethyltransferase)	*A. thaliana*	*B. juncea*	Increase in total Se levels and increase in tolerance to selenite, but not selenate	Banuelos et al. (2007)
SULTR 1,2,3 Sulphate proton transporters	*A. thaliana*	*A. thaliana (knock-down gene technology)*	Selenate accumulation reduced by HAST transport, little effect on selenite	Lydiate et al. (2007)
AtCpNifS chloroplast protein like SeCys lyase	*A. thaliana*	*A. thaliana*	Confirm higher selenate tolerance by reducing Se incorporation into protein	Van Hoewyk et al. (2008)
SBP 1,2,3 Se binding protein gene family	*A. thaliana*	*A. thaliana*	Elevated tolerance to heavy metal cadmium (Cd) by Se protein also binding Cd	Dutilleul et al. (2008)
ATPS1 SMT (selenocysteine *m*ethyltransferase)	*A. thaliana* *A. bisulcatus*	*Nicotiana tabacum L. cv. Samsun*	SMT can be utilised to increase the metabolism of Se into MeSe-Cys, the effects of ATPS activity vary depending on the species involved	McKenzie et al. (2009)
Adenosine 5'-phosphosulfate reductase	*A. thaliana*	*A. thaliana (knock-down gene technology)*	decreased selenate tolerance and photosynthetic efficiency	Grant et al. (2011)

Table 3. Molecular genetic studies on selenium tolerance and accumulation, including the origin of the genes (modified from De Filippis, 2010).

expressed in roots and leaves of *A. bisulcatus*, and appear to be not affected by Se induction (Pickering et al., 2003). Heterologous expression of AbSMT in transgenic *A. thaliana* results in the synthesis of MeSeCys and its derivative γ-glutamyl-Se-MeSeCys, these are the compounds not natively produced in *A. thaliana* (Ellis et al., 2004). In transgenic *Brassica juncea* expressing AbSMT accumulated MeSeCys similarly (Le Duc et al., 2004). The SMT transgenics showed increased Se accumulation, in the form of methyl-SeCys, as well as increased Se tolerance. Se volatilization rates also enhanced with the expression of SMT, with more volatile Se synthesized in the form of DMDSe. In SMT expressing trangenics, Se

tolerance, accumulation, and volatilization drawed the attention when the plants were supplied with selenite as opposed to selenate. In this manner, the conversion of selenate to selenite were thought to be a rate-limiting step for the production of SeCys. APS and SMT transgenics were hybridized to create double-transgenic plants that overexpress both APS and SMT (APSxSMT plants) to deal with this rate-limitation. The APS x SMT double transgenics accumulated up to nine times higher Se levels than wild type (LeDuc et al. 2006). The predominant form of the Se compounds in the double transgenics was methyl-SeCys. The APSxSMT double transgenics accumulated up to eightfold more methyl- SeCys than wild type and almost two fold more than the only SMT transgenics. Se tolerance was similar in the single and double transgenics. On the other hand, McKenzie et al (2009) concluded that while the SMT gene from Se hyperaccumulators can probably be utilised universally to increase the metabolism of Se into MeSeCys, the effects of enhancing ATP sulfurylase activity could vary depending on the species involved.

The APS enzyme seems to be rate-limiting for the assimilation of selenate to organic Se compounds, and CgS enzyme is also rate-limiting for DMSe volatilization. Increased APS expression also appears to induce selenate uptake and Se and S accumulation, probably depending on upregulation of sulfate transporter expression. The results from the SL and CpNifS transgenics indicate that SeCys breakdown can decrease nonspecific incorporation of Se into proteins. This situation enhances Se tolerance because elemental Se does not involve with cellular processes. As mentioned above, in plants CpNifS functions in Se tolerance in nature is unknown; it's most serious function is in synthesis of iron–sulfur clusters (Van Hoewyk et al. 2007). The results from the SMT transgenics show that SMT is a key enzyme for Se hyperaccumulation, offering increased Se tolerance and accumulation when expressed in nonaccumulators. Nevertheless, for Se assimilation and detoxification, APS also needs to be overexpressed with SMT. APS x SMT double transgenics link the ability to reduce selenate to selenite and SeCys with the competence to methylate SeCys and thus to detoxify the internal Se. These studies suggest that through genetic manipulation of high biomass, fast-growing plants, Se phytoremediation and biofortification can be improved into a viable option, while producing crops with better nutritional quality.

4.2 Problems and future aspects

The possible transfer of undesirable traits to elite plants and crop cultivars for agriculture is an obvious concern over phytoremediation techniques, especially in using genetically modified plants (Hanson et al. 1997; Terry et al. 2000). The use of phyto-crops for food or animal consumption may be affected by hyperaccumulation and high levels of some elements, for example Se, into plants' part. However technology exists to identify the fate of most of these toxic compounds, and their toxicity as demonstrated by the development of chemo preventitive enriched Se accumulating (fortified) edible crop plants (e.g. potato, radish and other vegetables) in Australia, UK, USA and other parts of the world (Broadley et al. 2004; Lefsrud et al. 2006; Pedrero et al. 2006; Haug et al. 2007; Zhao et al. 2007).

To clean-up Se from constructed wetlands and their waters is the major environmental problem. An affective solution seems to be to use of 'artificially constructed wetlands'. For wetland efficiency for removal of Se the most suitable plant species should be planted and some species like cattail grass (high biomass) and widgeon grass (high amounts hyperaccummulated) removed the most Se in trials (Banuelos 2006; Nyberg 1991). The

world Se resources need to be managed so that this non-renewable vulnerable resource is not squandered. Selenium uptake, mobilisation and assimilation are quite well understood and are similar to sulphur, however there are some steps not well understood, especially enzymatic and non-enzymatic steps about to the reduction of intermediates to selenide.

New genes and proteins will be discovered to improve Se tolerance, accumulation, and volatilization with the arrival of the genomic era. Also, comparative studies of Se hyperaccumulators and related nonaccumulators or of Se-tolerant ecotypes and non-tolerant ecotypes of the same species may reveal new genes that upregulate Se uptake, accumulation, and volatilization. Such new genes may not be involved in the commonly studied sulfur metabolism. For example, a Se-binding protein (SBP) homolog, when overexpressed in *A. thaliana*, increased tolerance to Se as well as cadmium (Agalou et al. 2005). SBP's function is unknown so far, but it has been hypothesized to be similar to glutathione. Moreover, recent genetic and genomic studies (Zhang et al. 2006; Tamaoki et al. 2008; Van Hoewyk et al 2008) have identified new quantitative trait loci (QTL) and genes involved in Se tolerance. The plant hormones jasmonic acid (JA) and ethylene are emerging as important players in plant responses to Se tolerance, possibly via their influence on S and Se assimilation. Further studies may reveal key genes that induce the responses that together provide Se tolerance and accumulation in model plants and hyperaccumulators. These key genes could be the candidates for overexpression, producing the complete Se hyperaccumulation in plants. It is desirable to study the potential ecological implications of growing Se accumulating or volatilizing plants before existing and future transgenics are used at a large scale in the field for phytoremediation or as fortified foods. Additional considerations for the use of transgenics for phytoremediation are the same as those involved with growing transgenics for other purposes and should also be evaluated and weighed against the risks of alternative remediation methods.

Many molecular studies have been reported the overexpression of genes encoding proteins involved in Se uptake, transport and assimilation. In this way further strategies for genetic engineering of Se accumulation, transformation and toxicity will become evident, and the use of transgenic plants for use in a variety of ways could be evaluated. Phytoremediation offers a cost effective and environmentally friendly alternative or complementary technology to conventional bioremediation techniques. However the biological processes of phytoremediation are still largely unknown in many cases, and plant-microbe interactions, mechanisms of degradation and transformation, volatilisation, chelation, binding and detoxification need more detailed investigations. In this point of view there is value in enhancement of traits in plants useful in phytoremediation such as high biomass and growth potential in seleniferous soils, which might otherwise be considered agriculturally non-productive land. Se-hyperaccumulating plants (wether naturally occurring or transgenic plants) have possibilities in that they combine pollutant decontamination with production of a product with beneficial properties to humans and animals.

5. Conclusion

Building on the genomic and biochemical studies described above, follow-up research may reveal key genes that trigger the cascade of responses that provide Se tolerance and accumulation in model plants and hyperaccumulators. Also, genes may be found that encode specific transporters of selenocompounds into and within hyperaccumulators. Such

key genes will be the ultimate candidates for overexpression studies, with the potential of transferring the complete Se hyperaccumulator profile into high-biomass species. Recent research has elucidated many important ecological interactions involving Se in plants. In this chapter, it has been focussed some important areas for future research. Particularly, more research is desirable on the role of soil microbes in plant Se uptake and volatilization, and the movement of Se through the food chain via Se hyperaccumulators or Se-fortified crop plants. The role of Se in below-ground ecological interactions with microbes and other organisms is also a fairly unexplored area. In addition to effects of Se on root–microbe interactions, Se may protect plants from root feeding herbivores, and selenocompounds released from hyperaccumulator roots may be toxic to surrounding vegetation. Similarly, the effects of Se on pollination ecology will be an interesting field of further study. Better knowledge of the processes involved in plant metabolism, the limiting factors involved, the contributions of ecological partners and the effects of Se on ecological partners are all useful for minimizing potential harmful effects of Se while benefiting from the positive effects of plant Se on animal and human health.

The capacity of plants to accumulate and volatilize Se will be very useful for the phytoremediation of Se-contaminated soils and waters (Banuelos and Meek 1990). When plant Se accumulation is well managed, this offers an efficient and cost-effective way to remove Se from the environment. Since plants are an effective source of dietary Se, Se-enriched plant material from phytoremediation or other sources can be considered fortified food. After being grown on Se-contaminated soil or being irrigated with Se-contaminated water, the Se-laden plant material may be used as a feed supplement for livestock, or as a biofuel. If successful, the potential of this strategy may be further enhanced by the use of selected transgenic lines. Of course, any use of Se-accumulating wildtype or transgenic plants will need to be accompanied by careful risk assessment, to avoid escape of transgenes and any adverse ecological effects of the accumulated Se.

6. References

Abrams, M. M., Shennan, C., Zazoski, J. & Burau, R. G. (1990). Selenomethionine uptake by wheat seedlings, *Agron J,* Vol, 82, pp. 1127–1130, ISSN 0002-1962

Agalou, A., Roussis, A.& Spaink, H.P. (2005) The *Arabidopsis* selenium-binding protein confers resistance to toxic levels of selenium. *Functional Plant Biology*, Vol. 31, pp.881–890, ISSN 1445-4408

Andreadou, I., Menge, W. M. P. B., Commandeur, J. N. M., Worthington, E. A. & Vermeulen, N. P. E. (1996). Synthesis of novel Se-substituted selenocysteine derivatives as potential kidney selective prodrugs of biologically active selenol compounds: evaluation of kinetics of b-elimination reactions in rat renal cytosol, *J Med Chem*, Vol, 39, pp, 2040–2046, ISSN 0022-2623

Ari, S., Cakir, O. & Turgut-Kara, N. (2010). Selenium tolerance in *Astragalus chrysochlorus*: identification of a cDNA fragment encoding a putative Selenocysteine methyltransferase, *Acta Physiol Plant*, Vol 32, pp, 1085–1092, ISSN 0137-5881

Baker, A.J.M. & Brooks, R.R. (1989). Terrestrial higher plants which hyperaccumulate metal elements: A review of their distribution, ecology, and phytochemistry, *Biorecovery*, Vol, 1, pp, 81-126, ISSN 269-7572

Banuelos, G., LeDuc, D.L., Pilon-Smits, E.A.H., Tagmount, A.& Terry, N. (2007). Transgenic Indian mustard overexpressing selenoctsteine lyase or selenocysteine methyltransferase exibit enhanced potential for selenium phytoremediation under field conditions. *Environmental Science & Technology,* Vol.41, pp.599–605, ISSN 0013-936X

Banuelos, G. S. & Meek, D. W. (1990). Accumulation of selenium in plants grown on selenium-treated soil, *J Environ Qua1,* Vol, 19, pp, 772-777

Banuelos, G.S. (2006). Phyto-products may be essential for sustainability and implementation of phytoremediation. *Environmental Pollution*, Vol. 144, pp. 19–23, ISSN 0269-7491

Beath, O. A., Draize, J. H., Eppson, H. F., Gilbert, C. S. & McCreary, O. C. (1934). Certain poisonous plants of wyoming activated by selenium and their association with respect to soil types, *J Am Pharm Assoc*, Vol, 23,pp, 94–97, ISSN 1086-5802

Bell, P.F., Parker, D.R. & Page, A.L. (1992). Contrasting selenate sulfate interactions in selenium-accumulating and nonaccumulating plant species. *Soil Sci Soc A. J,* Vol. 56, pp,1818–24, ISSN 0361-5995

Birringer, M., Pilawa, S.& Flohe, I. (2002). Trends in selenium biochemistry. *Natural Product Reports*, Vol.19, pp. 693–718, ISSN 0265-0568

Boyd, R. S. & Martens, S. N. (1993). The raison d'etre for metal hyperaccumulation by plants, In: *The vegetation of ultramafic (serpentine) soils.* A. J. M. Baker, J. Proctor & R. D., pp. 279–289, Reeves, Intercept, Andover, UK

Broadley, M.R., Bowen, H.C., Cotterill, H.L., Hammond, J.P., Meacham, M.C., Mead, A.& White, P.J. (2004) Phylogenetic variation in the shoot mineral concentration of angiosperms. Journal *of Experimental Botany,* Vol. 55, pp. 321–336, ISSN 0022-0957

Brown, T. A. & Shrift, A. (1981). Exclusion of selenium from proteins in selenium-tolerant *Astragalus* species, *Plant Physiol*, Vol, 67, pp. 1951-1953, ISSN: 0032-0889

Brown, T. A. & Shrift, A. (1982). Selenium: toxicity and tolerance in higher plants, *Biol Rev,* Vol, 57, pp, 59–84, ISSN 1464-7931

Burnell, J.N. (1981). Selenium metabolism in *Neptunia amplexicaulis*. *Plant Physiology,* Vol. 67, pp.316–324, ISSN 0032-0889

Cai, X. J., Block, E., Uden, P. C., Zhang, X., Quimby, B. D. & Sullivan, J. J. (1995). *Allium* chemistry: Identification of selenoamino acids in ordinary and selenium-enriched garlic, onion, and broccoli using gas chromatography with atomic emission detection, *J Agric Food Chem*, Vol, 43, pp, 1754–1757, ISSN 0021-8561

Cannon, H. L. (1960). Botanical prospecting for ore deposits, *Science*, Vol, 132, pp, 591–598, ISSN 0036-8075

Cherest, H., Davidian, J. C., Thomas, D., Benes, V., Ansorge, W., Surdin-Kerjan, Y. (1997). Molecular characterization of two high affinity sulfate transporters in *Saccharomyces cerevisiae*. *Genetics,* Vol, 145, pp, 627–635, ISSN 0016-6731

Clark, L.C., Combs, G.F. Jr, Turnbull, B.W., Slate, E.H., Chalker, D.K., Chow, J., Davis, L.S., Glover, R.A., Graham, G.F., Gross, E.G., Krongrad, A., Lesher, J.L. Jr, Park, H.K., Sanders, B.B. Jr, Smith, C.L. & Taylor, J.R. (1996). Effects of selenium supplementation for cancer prevention in patients with carcinoma of the skin a randomized controlled trial – a randomized controlled trial. *Journal of the American Medical Association,* Vol. 276, pp. 1957–1963, ISSN 0098-7484

Combs, G. F. (1980). The search for the nutritional role of selenium: a success story in poultry nutrition, *Feed Management*, Vol, 31, pp, 38-39, ISSN 0014-956X

Combs, G.F. Jr& Gay, W.P. (1998). Chemopreventive agents: selenium. Pharmacology & Therapeutics, Vol.79, pp.179-192, ISSN 0163-7258

Davidian, J.-C., Hatzfield, Y., Cathala, N., Tagmount, A. & Vidmar, J. J. (2000). Sulfate uptake and transport in plants, In: *Sulfur Nutrition and Sulfur Assimilation in Higher Plants: Molecular, Biochemical and Physiological Aspects*, C. Brunold, H. Rennenberg, L. J. De Kok, I. Stuhlen, J.-C. Davidian, pp. 1-19, Bern: Paul Haupt.

Dawson, J. C. & Anderson, J. W. (1988). Incorporation of cysteine and selenocysteine into cystathionine and selenocystathionine by crude extracts of spinach. *Phytochemistry,* Vol, 27, pp, 3453–3460, ISSN 0031-9422

De Fillips, L. F. (2010). Biochemical and molecular aspects in phytoremediation of selenium, In: *Plant Adaptation and Phytoremediation*, M. Ashraf, M. Ozturk, M. S. A. Ahmad, pp. 193-226, Springer, ISBN 9048193699

De Souza, M. P., Pilon-Smits, E. A. H., Lytle, C. M., Hwang, S., Tai, J., Honma, T. S. U., Yeh, L. & Terry, N. (1998). Ratelimiting steps in selenium assimilation and volatilization by Indian mustard. *Plant Physiol,* Vol, 117, pp, 1487–1494, ISSN 0032-0889

Dilworth, G.L.& Bandurski, R.S. (1977). Activation of selenate by adenosine 50- triphosphate sulfurylase from *Saccharmoyces cereviseae*. *Biochemical Journal*, Vol.163, pp.521–529, ISSN 0264-6021

Djanaguiraman, M., Prasad, P. V. V. & Seppanen, M. (2010). Selenium protects *Sorghum* leaves from oxidative damage under high temperature stress by enhancing antioxidant defense system, *Plant Physiol Biohem*, Vol 48, pp, 999-1007, ISSN 0981-9428

Duckart, E. C., Waldron, L. J. & Donner, H. E. (1992). Selenium uptake and volatilization from plants growing in soil, *Soil Sci,* Vol, 153, pp, 94–99, ISSN 0038-075X

Dumont, E., Vanhaecke, F. & Cornelis, R. (2006). Selenium speciation from food source to metabolites: a critical review, *Anal Bioanal Chem,* Vol, 385, pp, 1304–1323, ISSN 1618-2642

Dutilleul, C., Jourdain, A., Bourguignon, J.& Hugouvieux, V. (2008) The *Arabidopsis* putative selenium binding protein family: Expression study and characterisation of SBP1 as a potential new player in cadmium detoxification processes. *Plant Physiology,* Vol. 147, pp.239–251, ISSN 0032-0889

El-Bayoumy, K. & Sinha, R. (2005). Molecular chemoprevention by selenium: a genomic approach, Mutat Res, Vol 591, pp. 224-236, ISSN: 0027-5107

El Mehdawi, A. F., Quinn, C. F. & Pilon-Smits E. A. H. (2011). Effects of selenium hyperaccumulation on plant–plant interactions: evidence for elemental allelopathy?, New Phytologist, Vol, 191, pp, *120–131, ISSN* 1469-8137

Ellis, D.R. & Salt, D. E. (2003). Plants, selenium and human health, *Curr Opin Plant Biol*, Vol, 6, pp. 273–279, ISSN: 1369-5266

Ellis, D.R., Sors, T.G., Brunk, D.G., Albrecht, C., Orser, C., Lahner, B., Wood, K.V., Harris, H.H., Pickering, I.J.& Salt, D.E. (2004). Production of Se-methylselenocysteine in transgenic plants expressing selenocysteine methyltransferase. *BMC Plant Biology, Vol.* 4, pp.1–12, ISSN 1471-2229

Feist, L. J. & Parker, D.R. (2001). Ecotypic variation in selenium accumulation among populations of *Stanleya pinnata*, *New Phytol,* Vol, 149, pp, 61-69, ISSN 1469-8137

Finley, J. W. & Davis, C. D. (2001). Selenium (Se) from high-selenium *broccoli* is utilized differently than selenite, selenate, and selenomethionine, but is more effective in inhibiting colon carcinogenesis, *BioFactors,* Vol, 14, pp, 191-196, ISSN 0951-6433

Foster, S. J., Kraus, R. J. & Ganther, H. E. (1986). The metabolism of selenomethionine, Se-methylselenocysteine, their selenonium derivatives, and trimethylselenonium in the rat, *Arch Biochem Biophys,* Vol, 251, pp, 77–86, ISSN 0003-9861

Galeas, M. L., Zhang, L. H., Freeman, J. L., Wegner & Pilon-Smits, E. A. H. (2007). Seasonal fluctuations of selenium and sulfur accumulation in selenium hyperaccumulators and related nonaccumulator, *New Phytol*, Vol, 173, pp,517-525, ISSN 1469-8137

Ganther, H. E. & Lawrence, J. R. (1997). Chemical transformations of selenium in living organisms. Improved forms of selenium for cancer prevention. *Tetrahedron*, Vol, 53, pp, 12299–112310, ISSN 0040-4020

Garifullina, G.F., Owen, J.D., Lindblom, S-D., Tufan, H., Pilon, M. & Pilon-Smits, E.A.H. (2003). Expression of a mouse selenocysteine lyase in *Brassica juncea* chloroplasts affects selenium tolerance and accumulation. *Physiologia Plantarum*, Vol.118, pp.538–544, ISSN 0031-9317

Germ, M., Stibilj, V., Osvald, J. & Kreft, I. (2007a). Effect of selenium foliar application on chicory (*Cichorium intybus* L.), *J Agricult Food Chem*, Vol, 55,pp, 795-798, ISSN 0021-8561

Germ, M., Stibilj, V. & Kreft, I. (2007b). Metabolic Importance of Selenium for Plants, *The European Journal of Plant Science and Biotechnology*, Vol, 1, pp, 91-97, ISSN 1752-3842

Gladyshev, V. N. & Kryukov, G. V. (1999). Evolution of selenocysteinecontaining proteins: significance of identification and functional characterization of selenoproteins, *Biofactors*, Vol 14, pp, 87-92, ISSN 0951-6433

Goldhaber, S.B. (2003). Trace element risk assessment: essentiality vs. toxicity. *Regul Toxicol Pharmacol,* Vol. 38, pp. 232–242, ISSN: 0273-2300

Goulet, A. C., Watts, G., Lord, J. L., Nelson, M. A. (2007). Profiling of Selenomethionine Responsive Genes in Colon Cancer by Microarray Analysis, *Canc Biol Ther*, Vol, 6, pp, 1-10 , ISSN 1538-4047

Grant, K., Carey, N.M., Mendoza, M., Schulze J., Pilon, M., Pilon-Smits, E. A. H. & van Hoewyk D. (2011). Adenosine 5′-phosphosulfate reductase (APR2) mutation in *Arabidopsis* implicates glutathione deficiency in selenate toxicity. *Biochemical Journal*, Vol. 438, pp. 325–335, ISSN 0264-6021

Guo, X. & Wu, L. (1998). Distribution of free seleno-amino acids in plant tissue of *Melilotus indica* L. grown in selenium laden soils, *Ecotoxicol Environ Safety,* Vol, 39, pp, 207-214, ISSN 0147-6513

Hanson, A.D., Trossat, C., Nolte, K.D. & Gage, D.A. (1997). 3-dimethylsulphonioproprionate biosynthesis in higher plants. In: *Sulphur nutrition and assimilation in higher plants: Regulatory agricultural and environmental aspects*, Cram, W.J., De Kok, L.J., Stulen, I., Brunold, C., Rennenberg, H., pp.147-154, Backhuys Publishers, USA

Hasanuzzaman, M. & Fujita, M. (2011). Selenium pretreatment upregulates the antioxidant defense and methylglyoxal detoxification system and confers enhanced tolerance to drought stress in rapeseed seedlings. Biol Trace Elem Res, DOI: 10.1007/s12011-011-8998-9, ISSN 1559-0720

Hatzfeld, Y., Cathala, N., Grignon, C.& Davidian, J.C. (1998). Effect of ATP sulphurylase overexpression in bright yellow 2 tobacco cells. *Plant Physiology,* Vol. 116, pp.1307–1313, ISSN 0032-0889

Haug, A., Graham, R.D., Christophersen, O.A.& Lyons, G.H. (2007). How to use the world's scarce selenium resources efficiently to increase the selenium concentration in food. *Microbial Ecology in Health and Disease*, Vol. 19, pp.209–228, ISSN 0891-060X

Ip, C. & Ganther, H. E. (1992). Relationship between the chemical form of selenium and anticarcinogenic activity, In: *Cancer Chemoprevention*, I. Wattenberg, M. Lipkin, C. W. Boon, G. J. Kellott & R. Boca, pp. 479-488, CRC Press

Ip, C. (1998). Lessons from basic research in selenium and cancer prevention, *J Nutr,* Vol, 28, pp, 1845–1854, ISSN 0022-3166

Ip, C., Birringer, M., Block, E., Kotrebai, M., Tyson, J., Uden, P. C. & Lisk, D. (2000). Chemical speciation influences comparative activity of selenium-enriched garlic and yeast in mammary cancer prevention, *J Agric Food Chem,* Vol, 48, pp, 2062–2070, ISSN 0021-8561

Jhee, E. M., Dandridge, K. L., Christy, Jr., A. M. & Pollard, A. J. (1999). Selective herbivory on low-zinc phenotypes of the hyperaccumulator *Thlaspi caerulescens* (*Brassicaceae*), *Chemoecology*, Vol, 9, pp, 93 – 95, ISSN 0937-7409

Kim, T., Jung, U., Cho, D. Y. & Chung, A. S. (2001). Se-methylselenocysteine induces apoptosis through caspase activation in HL-60 cells, *Carcinogenesis,* Vol, 22, pp, 559–565, ISSN 0143-3334

LeDuc, D.L., AbdelSamie, M., Montes-Bayo´n, M., Wu, C.P., Reisinger, S.J. & Terry, N. (2006). Overexpressing both ATP sulfurylase and selenocysteine methyltransferase enhances selenium phytoremediation traits in Indian mustard. *Environmental Pollution*, Vol. 144, pp. 70–76, ISSN 0269-7491

LeDuc, D.L., Tarun, A.S., Montes-Bayon, M., Meija, J., Malit, M.F., Wu, C.P., Abdel-Samie, M., Chiang, C.Y., Tagmount, A., De Souza, M., Neuhierl, B., Bock, A., Caruso, J.& Terry, N. (2004). Overexpression of selenocysteine methyltransferase in *Arabidopsis* and Indian mustard increases selenium tolerance and accumulation. *Plant Physiology,* Vol. 135, pp.377–383, ISSN 0032-0889

Leustek, T., Smith, M., Murillo, M., Singh, D.P., Smith, A.G., Woodcock, S.C., Awan, S.J. & Warren, M.J. (1997). Siroheme biosynthesis in higher plants. Analysis of an *S*-adenosyl-L-methionine-dependent uroporphyrinogen III methyltransferase from *Arabidopsis thaliana*. *J Biol Chem,* Vol. 272, pp, 2744-2752, ISSN 0021-9258

Lefsrud, M.G., Kopsell, D.A., Kopsell, D.E. & Randle, W.M. (2006). Kale carotenoids are unaffected, whereas biomass production, elemental concentration, and selenium accumulation respond to, changes in selenium fertility. *Journal of Agricultural and Food Chemistry,* Vol.54, pp.1764–1771, ISSN 0021-8561

Lindblow-Kull, C., Kull, F. J. & Shrift, A.(1985). Single transporter for sulfate, selenate, and selenite in *Escherichia coli* K12, *J Bacteriol,* Vol, 163, pp. 1267–1269, ISSN 0021-9193

Low, S. C. & Berry, M. J. (1996). Knowing when not to stop: selenocysteine incorporation in eukaryotes, *Trends Biochem Sci*, Vol, 21, pp, 203-208, ISSN 0968-0004

Lu, J. & Jiang, C. (2001). Antiangiogenic activity of selenium in cancer chemoprevention: metabolite-specific effects, *Nutr Cancer,* Vol, 40, pp, 64–73, ISSN 0163-5581

Lu, J., Pei, H., Ip, C., Lisk, D. J., Ganther, H. & Thompson, H. J. (1996). Effect of an aqueous extract of selenium-enriched garlic on in vitro markers and *in vivo* efficacy in cancer prevention, *Carcinogenesis,* Vol, 17, pp, 1903–1907, ISSN 0143-3334

Lydiate, D., Higgins, E., Robinson, S., Korbas, M., Yang, S.I.& Pickering, I. (2007). Selenium acquisition by *Arabidopsis* plants. *Canadian Light Source*, Vol. 27, pp.106–107

Lyi, S.M., Heller, L.I., Rutzke, M., Welch, R.M., Kochian, L.V. & Li, L. (2005). Molecular and biochemical characterization of the selenocysteine Se-methyltransferase gene and Se-methylselenocysteine synthesis in broccoli. *Plant Physiol,* Vol. 138, pp,409–420, ISSN 0032-0889

McCluskey, T. J., Scarf, A. R. & Anderson, J. W. (1986). Enzyme-catalyzed elimination of selenocystathionine and selenocystine and their sulfur isologues by plant extracts. *Phytochemistry,* Vol, 25, pp, 2063– 2068, ISSN 0031-9422

McKenzie, M.J., Hunter, D.A., Pathirana, R., Watson, L.M., Joyce, N.I., Matich, A.J., Rowan, D.D.& Brummell, D.A. (2009). Accumulation of an organic anticancer selenium compound in a transgenic Solanaceous species shows wider applicability of the selenocysteine methyltransferase transgene from selenium hyperaccumulators. *Transgenic Research*, Vol. 18, (3), pp.407–424, ISSN 0962-8819

Medina, D., Thompson, H., Ganther, H. & Ip, C. (2001). Se-Methylselenocysteine: a new compound for chemoprevention of breast cancer. *Nutrition and Cancer*, Vol, 40, pp, 12 – 17, ISSN 0163-5581

Neuhierl, B.& Bock, A. (1996). On the mechanism of selenium tolerance in selenium accumulating plants. Purification and characterization of a specific selenocysteine methyltransferase from cultured cells of *Astragalus bisulcatus*. *European Journal of Biochemistry*, Vol. 239, pp. 235–238, ISSN 0014-2956

Neuhierl, B., Thanbichler, M., Lottspeich, F.& Boeck, A. (1999). A family of S-methylmethionine dependent thiol/selenol methyltransferases: Role in selenium tolerance and evolutionary relation. *Journal of Biological Chemistry*, Vol. 274, pp.5407–5414, ISSN 0021-9258

Ng, B. H. & Anderson, J. W. (1978). Synthesis of selenocysteine by cysteine synthases from selenium accumulator and non-accumulator plants, *Phytochemistry,* Vol, 17, pp, 2069–2074, ISSN 0031-9422

Nyberg, S. (1991). Multiple use of plants: Studies on selenium incorporation in some agricultural species for the production of organic selenium compounds. *Plant Foods for Human Nutrition*, Vol. 41, pp.69–88, ISSN 0921-9668

Pedrero, Z., Yolanda, M. & Carmen, C. (2006). Selenium species bioaccessibility in enriched raddish (*Raphanus sativa*). A potential dietary source of selenium. *Journal of Agricultural and Food Chemistry* Vol.54, pp.2412–2417, ISSN 0021-8561

Pickering, I.J., Wright, C., Bubner, B., Ellis, D., Persans, M.W., Yu, E.Y., George, G.N., Prince, R.C.& Salt, D.E. (2003). Chemical form and distribution of selenium and sulfur in the selenium hyperaccumulator *Astragalus bisulcatus*. *Plant Physiology,* Vol. 131, pp.1-8, ISSN 0032-0889

Pilon, M., Owen, J.D., Garifullina, G.F., Kurihara, T., Mihara, H., Esaki, N. & Pilon-Smits, E.A.H. (2003). Enhanced selenium tolerance and accumulation in transgenic Arabidopsis thaliana expressing a mouse selenocysteine lyase. *Plant Physiology,* Vol. 131, pp. 1250–1257, ISSN 0032-0889

Pilon-Smits, E. H. A. & Quinn, C. F. (2010). Selenium Metabolism in Plants. In: *Cell Biology of Metals and Nutrients, Plant Cell Monographs 17*, R. Hell & R. R. Mendel, Springer-Verlag, Berlin Heidelberg

Pilon-Smits, E.A.H., Hwang, S., Lytle, C.M., Zhu, Y., Tai, J.C,, Bravo, R.C., Chen, Y., Leustek, T. & Terry N. (1999). Overexpression of ATP sulfurylase in Indian mustard leads to increased selenate uptake, reduction, and tolerance. *Plant Physiology,* Vol.119, pp.123–132, ISSN 0032-0889

Ranocha, P., Bourgis, F., Ziemak, M.J., Rhodes, D., Gage, D.A. & Hanson, A.D. (2000). Characterization and functional expression of cDNAs encoding methionine-sensitive and –insensitive homocysteine S- methyltransferases from Arabidopsis. *Journal of Biological Chemistry*, Vol. 275, 15962-15968, ISSN 0021-9258

Reeves, R.D. & Baker, A.J.M. (2000). Metal- Accumulating Plants, In: *Phytoremediation of toxic metals: using plants to clean-up the environment*, I. Raskın & , B.D. Ensley, pp. 193-230, New York, John Wiley and Sons

Shaw, W.H.& Anderson, J.W. (1972). Purification, properties and substrate specificity of adenosine triphosphate sulphurylase from spinach leaf tissue. *Biochemical Journal*, Vol. 127, pp.237–247, ISSN 0264-6021

Shrift,A. & Ulrich, J. M. (1976). Transport of selenate and selenite into *Astragalus* roots, *Plant Physiol,* Vol, 44, pp. 893–896, ISSN 0032-0889

Sinha, R., Kiley, S. C., Lu, J. X., Thompson, H. J., Moraes, R., Jaken, S. & Medina, D. (1999). Effects of methylselenocysteine on PKC activity, cdk2 phosphorylation and gadd gene expression in synchronized mouse mammary epithelial tumor cells, *Cancer Lett* , Vol, 146, pp, 135–145, ISSN 0304-3835

Sors, T.G., Ellis, D.R. & D.E. Salt (2005). Selenium uptake, translocation, assimilation and metabolic fate in plants. *Photosynthesis Research*, Vol. 86, pp.373–389, ISSN 0166-8595

Sors, T.G., Martin, C.P., Salt, D.E. (2009). Characterization of selenocysteine methyltransferases from *Astragalus* species with contrasting selenium accumulation capacity. *Plant J* 59(1):110–122 ISSN 0960-7412

Tamaoki, M., Freeman, J.L. & Pilon-Smits, E.A.H. (2008). Cooperative ethylene and jasmonic acid signaling regulates selenite resistance in Arabidopsis thaliana. *Plant Physiol,* Vol.146, pp,1219–1230, ISSN 0032- 0889

Terry, N., Zayed, A. M., Souza, M. P. & Tarun, A. S. (2000). Selenium in higher plants, *Annu Rev Plant Physiol Plant Mol Biol*, Vol, 51, pp, 401-432, ISSN 1040-2519

Tsang, M. L. S. & Schiff, J. A. (1978). Studies of sulfate utilization by algae. 18. Identification of glutathione as a physiological carrier in assimilatory sulfate reduction by *Chlorella*, *Plant Sci Lett,* Vol, 11, pp, 177–183, ISSN 0304-4211

Unni, E., Koul, D., Alfred Yung, W-K. & Sinha R (2005). Semethylselenocysteine inhibits phosphatidylinositol 3-kinase activity of mouse mammary epithelial tumor cells in vitro. *Breast Cancer Research*, Vol.7, pp.699-707, ISSN 1465-5411

Van Hoewyk, D., Abdel-Ghany, S.E., Cohu, C., Herbert, S., Kugrens, P., Pilon, M.& Pilon-Smits, E.A.H. (2007). The Arabidopsis cysteine desulfurase CpNifS is essential for maturation of iron-sulfur cluster proteins, photosynthesis, and chloroplast development. *Proceedings of the National Academy of Sciences of the United States of America*, Vol. 104, pp.5686–5691, ISSN 0027-8424

Van Hoewyk, D., Garifullina, G.F., Ackley, A.R., Abdel-Ghany, S.E., Marcus, M.A., Fakra, S., Ishiyama, K. Inoue, E., Pilon, M.& Takahashi, H. (2005). Overexpression of

AtCpNifS enhances selenium tolerance and accumulation in *Arabidopsis*. *Plant Physiology,* Vol. 139, pp.1518–1528, ISSN 0032-0889

Van Hoewyk, D., Takahashi, H., Hess, A., Tamaoki, M.& Pilon-Smits, E.A.H. (2008). Transcriptome and biochemical analyses give insights into selenium-stress responses and selenium tolerance mechanisms in *Arabidopsis*. *Physiologia Plantarum*, Vol. 132, pp.236–253, ISSN 0031-9317

Van Huysen, T., Abdel-Ghany, S., Hale, K.L., LeDuc, D., Terry, N. & Pilon-Smits, E.A.H. (2003). Overexpression of cystathionine-gamma-synthase enhances selenium volatilisation in *Brassica juncea*. *Planta,* Vol. 218, pp.71–78, ISSN 0032-0935

Van Huysen T, Terry N, Pilon-Smits EAH (2004). Exploring the Selenium phytoremediationpotential of transgenic Brassica juncea overexpressing ATP sulfurylase or cystathionine g-synthase. *Int J Phytoremed* 6:111–118

Wang, Z., Jiang, C. & Lu, J. (2002). Induction of caspase-mediated apoptosis and cell-cycle G1 arrest by selenium metabolite methylselenol, *Mol Carcinog*, Vol, 34, pp, 113–120, ISSN 1098-2744

White, P. J., Bowen, H. C., Marshall, B. & Broadley, M. R. (2007). Extraordinarily high leaf selenium to sulfur ratios define 'Se-accumulator' plants, *Ann Bot*, Vol, 100, pp, 111–118, ISSN 0305-7364

Wilber, C. G. (1980). Toxicology of selenium: a review. *Clin Toxicol,* Vol, 17, pp, 171–230, ISSN 1556-3650

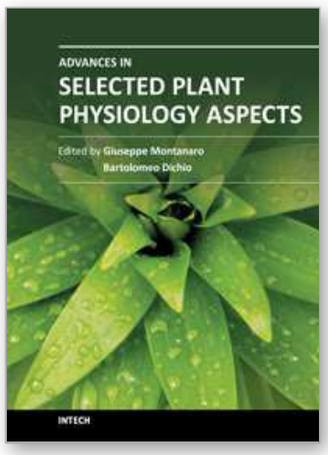
Zayed, A., Lytle, C.M. & Terry N. (1998). Accumulation and volatilization of different chemical species of selenium by plants, *Planta,* Vol, 206, pp. 284–292, ISSN 0032-0935

Zeng, H. & Combs, G. F. (2008). Selenium as an anticancer nutrient: roles in cell proliferation and tumor cell invasion, *Journal of Nutritional Biochemistry,* Vol 19, pp,1-7, ISSN 0955-2863

Zhao, F., McGrath, S., Gray, C. & Lopez-Bellido, J. (2007). Selenium concentrations in UK wheat and biofortification strategies. *Comparative Biochemistry and Physiology - Part A: Molecular & Integrative Physiology*, Vol.146, pp.S246, ISSN 1095-6433

Zhang, L.H., Byrne, P.F.& Pilon-Smits, E.A.H. (2006). Mapping quantitative trait loci associated with selenate Tolerance in *Arabidopsis thaliana*, *New Phytol,* Vol. 170, pp, 33–42, ISSN 1469-8137

Zhu, L., Jiang, C.J., Deng, W.W., Gao, X., Wang, R.J. and Wan, X.C. (2008). Cloning and expression of selenocysteine methyltransferase cDNA from *Camellia sinensis*. *Acta Physiol Plant*, Vol.30, pp, 167–174, ISSN 0137-5881



Advances in Selected Plant Physiology Aspects

Edited by Dr. Giuseppe Montanaro

ISBN 978-953-51-0557-2
Hard cover, 388 pages
Publisher InTech
Published online 25, April, 2012
Published in print edition April, 2012

The book provides general principles and new insights of some plant physiology aspects covering abiotic stress, plant water relations, mineral nutrition and reproduction. Plant response to reduced water availability and other abiotic stress (e.g. metals) have been analysed through changes in water absorption and transport mechanisms, as well as by molecular and genetic approach. A relatively new aspects of fruit nutrition are presented in order to provide the basis for the improvement of some fruit quality traits. The involvement of hormones, nutritional and proteomic plant profiles together with some structure/function of sexual components have also been addressed. Written by leading scientists from around the world it may serve as source of methods, theories, ideas and tools for students, researchers and experts in that areas of plant physiology.

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Özgür Çakır, Neslihan Turgut-Kara and Şule Arı (2012). Selenium Metabolism in Plants: Molecular Approaches, Advances in Selected Plant Physiology Aspects, Dr. Giuseppe Montanaro (Ed.), ISBN: 978-953-51-0557-2, InTech, Available from: http://www.intechopen.com/books/advances-in-selected-plant-physiology-aspects/selenium-metabolism-in-plants-molecular-approaches

INTECH
open science | open minds

InTech Europe
University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China
Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

IntechOpen