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# Selenium Biofortification and the Problem of its Safety

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Additional information is available at the end of the chapter

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## Abstract

Selenium is an essential mineral element to humans and animals because it is an important component of selenoproteins that are important for functioning of the metabolism. Because of poor soil conditions in various regions of the world, the enrichment of edible plants with selenium via the biofortification strategy has been implemented. However, selenium in the context of plant mineral nutrition appears twofold due to its biofortifying character at low concentrations and toxicity at high concentrations. In this sense, understanding of the functional mechanisms in which selenium is involved is important, ranging from its absorption and assimilation in organic compounds to its beneficial or harmful effects, considering its role in food security and human health. Therefore, this chapter addresses the key aspects related to selenium in the soil-plant-man environment and the narrow limit between biofortification and toxicity, as well as the main scientific findings on this mineral element in the biochemical, physiology and plant nutrition contexts.

**Keywords:** selenate, selenite, sulfur, toxicity, metabolism

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## 1. Introduction

The discovery of selenium (Se) by Swedish chemist Jons Jacob Berzelius about 200 years ago initiated the start of several studies with this chemical element in living organisms [1,2]. However, it was [3] who identified Se as an essential nutrient for the growth and development of bacteria, mammals and birds. It is currently considered as an essential micronutrient for humans and animals, as well as for certain lower organisms such as algae, fungi and bacteria [4].

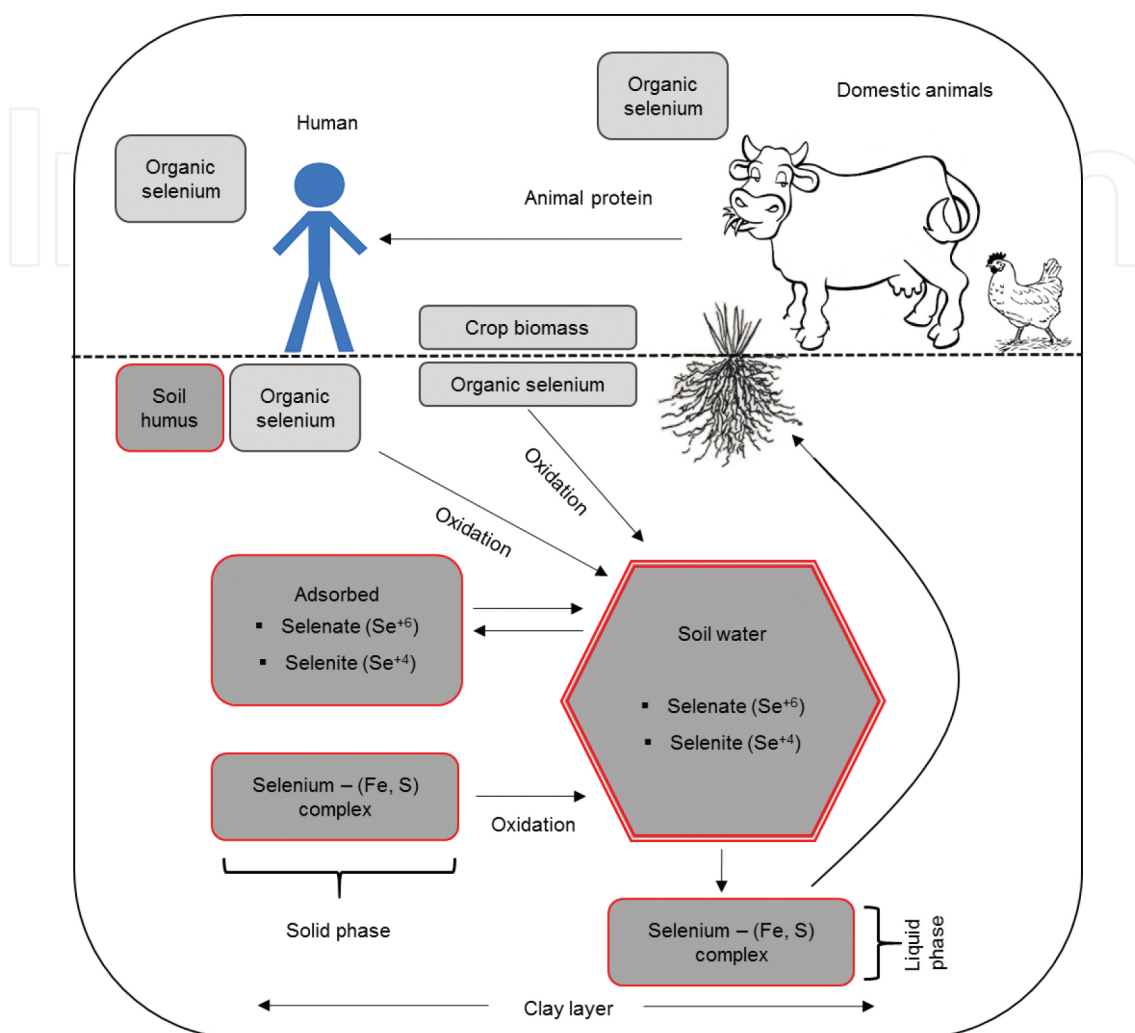
Se is a trace element widespread in various geochemical environments [5], and its average content in the earth's crust is estimated at  $0.05 \text{ mg kg}^{-1}$ . However, concentrations exceeding  $0.5 \text{ mg kg}^{-1}$  can be found. Selenium levels are slightly higher in mafic rocks; however, they rarely exceed  $0.1 \text{ mg kg}^{-1}$ . In sedimentary rocks, Se is associated with the clay fraction, and thus, its abundance is higher in clayey sediments compared to sandstone and limestone. Selenium exists in four valence states, of which the state  $-2$  predominates in organic compounds. The forms selenite ( $\text{Se}^{4+}$ ) and selenate ( $\text{Se}^{6+}$ ) do not form stable compounds in geochemical environments and are preferably adsorbed by minerals, particularly clay minerals, and oxides and hydroxides of Fe and Mn [6]. In the soil-plant system, the plant has the important role of recycling and releasing Se forms from the soil to the food chain. In this sense, the Se concentration in agricultural products and forage depends on the Se content in the soil and its availability [7–9]. However, Se availability in the soil is restricted, and its content is relatively low due to weathering and acidity [6, 7, 9, 10].

Because of the chemical similarity between Se and sulfur (S), the metabolism of Se in higher plants is closely related to S. Therefore, its absorption by the roots of plants is governed by the solubility of adsorbed forms and the transformation of organic Se forms (**Figure 1**). Due to the ability of plants to absorb Se, they can be classified according to Se accumulation in their tissues, including the classes of hyperaccumulators ( $1000\text{--}15,000 \text{ mg Se kg}^{-1}$  dry weight), accumulators ( $>100 \text{ mg Se kg}^{-1}$  dry weight) and non-accumulators ( $<100 \text{ mg Se kg}^{-1}$  dry weight) [11]. Many species of the genera *Astragalus*, *Xylorrhiza* and *Stanleya* are typically Se accumulators because they are capable of growing in selenium-rich soil without showing any negative effect of Se toxicity, accumulating Se contents in the range of  $20\text{--}40 \text{ mg Se g}^{-1} \text{ DM}$  while presenting no toxic effect [12, 13].

Because the range of Se concentrations in the soil varies, for example, from  $8000 \text{ mg kg}^{-1}$  in soils of the Russian city of Tuva to  $0.005 \text{ mg kg}^{-1}$  in soils of Finland and China [15], there is close relationship between Se deficiency in soils and the appearance of disease symptoms related to low Se intake in humans and mammals. Various efforts have thus been made to enrich agricultural feed crops with Se via fertilization or genetic breeding in a strategy called biofortification [16, 17]. According to this strategy, the supply of Se to the plant promotes a positive response in human health, preventing the onset of diseases related to low intake of this nutrient. In this sense, several studies report the positive effect of using Se in biofortification of lettuce [18], tomato [19], broccoli [20, 21], cucumber [22] and carrot crops [23].

In this context, biofortification of edible plants appears to be important from a nutritional point of view because selenium participates in constitution of selenoproteins, for example, the active site of antioxidant enzymes from the group of glutathione peroxidases (GSH-Px), which are reactive oxygen species detoxifying enzymes [24, 25]. There are several isoenzymes of the glutathione peroxidase family (with 4 g atom per mole of protein): cellular glutathione peroxidase (GSH-Px 1); glutathione peroxidase of the intestinal epithelium (GSH-Px 2); plasma glutathione peroxidase (GSH-Px 3); hydroperoxide phospholipid glutathione peroxidase (GSH-Px 4); and glutathione peroxidase present in the sperm (GSH-Px 5). Of these, GSH-Px 1 is the most abundant in mammalian selenoproteins [26]. Additionally, Se is of great importance

to human health because of its presence in chemical constitution of the iodothyronine deiodinase enzyme, which is involved in metabolism of the thyroid hormone [27].



**Figure 1.** Selenium in the soil-plant-atmosphere system. Plants absorb selenate or selenite from the soil solution. Se concentration in the soil solution depends on the solubility of Se forms present and the biological transformation of organic Se forms. Adapted with permission from [14].

Despite the biofortifying character of Se in plants, it also has a toxic effect when provided to plants in high concentrations. This toxicity results from the substitution of S for Se in cysteine and methionine amino acids, reducing the number of disulfide bonds and thus altering the structure and functionality of proteins, causing a negative impact on plant growth. Furthermore, another toxic effect of Se during its assimilation into organic compounds results in depletion of the nonenzymatic antioxidant, glutathione. Therefore, there is an imbalance between detoxification and formation of free radical species, which results in a significant oxidative burst and consequent reduction in plant growth [28].

It should be emphasized that biofortification/toxicity of Se is well reported in literature [18, 21–23, 29]; however, the transition between biofortification and toxicity by Se is narrow and

depends on the concentration and source, as well as the plant genotype. Despite this, literature does not report Se levels in plants based on determination of critical Se concentrations with regard to leaf content and Se concentrations in the culture medium. This shortcoming complicates the adoption of one Se concentration or a narrow range of concentrations that promote plant growth at the expense of biochemical, physiological and nutritional disorders promoted by toxic Se levels.

In this chapter, we sought to address the key aspects inherent to selenium and its functional relationships in the plant environment, as well as the intrinsic importance of food security regarding this nutrient, considering the main current scientific findings in the biochemical, physiological and nutritional fields of plants.

## 2. Selenium metabolism

In previous studies, it was shown that Se was absorbed by passive diffusion [30, 31]; however, it was recently shown that selenate is absorbed by the sulfur carrier, while selenite is absorbed by the phosphate carrier, and both processes are dependent on energy expenditures [32, 33]. Among the inorganic (selenate and selenite) and organic forms of selenium [selenomethionine (SeMet) and selenocysteine], selenomethionine in canola and wheat plants is the form that present the highest absorption rate and rapid translocation to the shoots [34]. Due to the chemical similarity between S and Se, both present the same route of absorption and assimilation, competing for the same carrier membrane [16]. After absorbed through the sulfur carrier (Sultr) and translocated to the shoot, selenate can be assimilated in chloroplasts and reduced to selenite in a reaction catalyzed by the enzyme ATP sulfurylase (APS) and then into selenide [16].

Non-accumulating plants can concentrate Se because APS has limited catalytic activity. On the other hand, accumulating plants overexpress APS resulting in accumulation and tolerance to high Se concentrations. Selenide may be incorporated into the S-amino acid similar to selenocysteine which can be converted to selenomethionine (SeMet) in three enzymatic steps. Incorrect insertion of the amino acid selenomethionine/selenocysteine in proteins can cause the formation of protein aggregates that promote disruption of important cellular functions [35, 28]. The incorporation of Se in proteins may occur when Se is converted to less toxic forms, because some plant species present nonprotein organic compounds containing Se such as methylselenocysteine (MeSeCys),  $\gamma$ -glutamyl-MeSeCys and/or selenocysteine [36]. Se can be volatilized by plants through the dimetilselenide or dimetildiselenide compounds, synthesized from selenomethionine and methylselenocysteine, respectively [16]. Se accumulating plants have the synthesis of methyl-SeCys catalyzed by the enzyme SeCys methyltransferase (SMT), accumulating methyl-SeCys, a nonprotein amino acid. Furthermore, methyl-SeCys may be converted into dimetildiselenide, a volatile compound. Expression of the enzyme SMT in non Se accumulating plants increases accumulation of Se in the form of methyl-SeCys, and its activity is related to tolerance to Se accumulation [16].

In the biochemical field, the metabolic pathways of plants are interconnected by means of some compounds. In the case of Se and nitrogen (N), the metabolism of these inorganic elements is interconnected by means of the O-acetylserine compound. Therefore, alterations to the S metabolism induced by Se interfere with that of N with respect to the metabolism of amino acids and proteins [16], considering that the amino acids methionine, phenylalanine, tyrosine and tryptophan are precursors of glucosinolate, while phenylalanine is a precursor of phenolic compounds. Thus, variations in the synthesis of these amino acids influence the synthesis of nutraceutical compounds such as glucosinolate and phenolic compounds [16].

Several studies report the positive impact of Se on the plant metabolism, particularly due to its abiotic stress mitigating effect. In this sense, Se plays an important role in increasing the activity of antioxidant enzymes to contribute to the detoxification of reactive oxygen species, considering that this mineral element participates in the active site of these enzymes. These enzymes, called glutathione peroxidases, appear quite active in plants subjected to various abiotic stresses such as drought stress [37, 38], salinity [39] and heavy metal toxicity [40], conferring stress tolerance to plants. This effect of Se is evident, because when supplied at concentrations of 10 and 50  $\mu\text{M}$  of selenate beneficial effects were observed in wheat plants grown under appropriate and reduced N availability. In this study, Se promoted a better response of the parameters fluorescence and gas exchange, with a positive impact on the growth of wheat plants [41].

On the other hand, Se in toxic concentrations may compromise energy synthesis by redox reactions (*i.e.*, photosynthesis and respiration) due to substitution of S for Se in the cysteine amino acid residue. Cysteine constitutes an important site for binding and stabilization of Fe-S metal centers, heme groups and ions participating in the flow of electrons in the mitochondria and chloroplasts. In this regard, it is speculated that substitution of the cysteine amino acid residue by selenocysteine in proteins rich in Fe-S metal centers disturbs the flow of electrons in the mitochondria and chloroplasts [28]. This fact implies the reduction of energy synthesis, and consequently reduced plant growth.

Moreover, the reduction of selenite to selenate via the S metabolic pathway demands a great glutathione input, a biochemical component involved in important redox reactions in cellular homeostasis [42]. This fact explains the decrease in root growth of plants induced by selenate when it is assimilated into organic compounds, since there is a depletion in the cellular glutathione content [28]. This was observed in brassica plants of *Stanleya albescent* sensitive to Se toxicity, which when exposed to toxic concentrations of this element had their growth compromised due to oxidative stress caused by the increased leaf accumulation of hydrogen peroxide ( $\text{H}_2\text{O}_2$ ) and superoxide anion ( $\text{O}_2^-$ ) in contrast to the more tolerant genotype *Stanleya pinnata*, which has a high glutathione content [43].

The metabolisms of nitrogen (N) and S are interconnected by means of the compound O-acetylserine [16]. Thus, the supply of Se to plants may interfere with the N metabolism. It was indicated that the supply of Se to barley plants reduced the nitrate assimilatory process because of reduced activity of the nitrate and nitrite reductase enzymes in leaf and root tissues. However, intensity of the reduction was greater when Se was supplied in the form of selenite [44].



It was recently demonstrated that toxic concentrations of Se promote reduction of plant growth in *Arabidopsis thaliana* due to incomplete mobilization of starch reserves overnight, reduced expression of genes encoding the synthesis of endotransglucosylase/endohydrolase enzymes and expansins, as well as nutritional disorders [45]. Therefore, despite the benefits of low Se concentrations, it has a large negative impact on the plant metabolism when provided in high concentrations on plant growth by affecting metabolic processes of energy acquisition, cell expansion, and absorption and assimilation of essential nutrients.

### 3. Selenium biofortification

Selenium is an essential inorganic element for humans and animals, and one of the organic forms of Se, methylselenocysteine, appears to be an effective food source of Se [16]. Se is incorporated into a range of selenoproteins involved in several important metabolic activities such as synthesis of thyroid hormones and antioxidative activity [46, 47]. Selenium is an important inorganic component for the antioxidant metabolism of enzymes, making up part of the active site of enzymes from the group of glutathione peroxidases (GSH-Px) which plays an important role in detoxification of free radicals. The GSH-Px catalyze the reduction of hydroperoxide radicals ( $H_2O_2$  for instance) by the oxidation of glutathione (GSH), a nonenzymatic component of the antioxidative metabolism [48].

It is noted that the accumulation of Se in foods is closely related to the content of this nutrient in the soil. However, consumption of foods poor in Se or low ingestion of foods containing Se is associated with the emergence of numerous diseases such as cancer, type II diabetes, heart disease, pulmonary dysfunction, seizures in children, impaired development and cerebral functions, as well as pregnancy and conception [49–51].

Currently, Se deficiency affects about 1 billion people worldwide due to soils lacking this mineral nutrient in some countries [52]. This edaphic characteristic was registered in countries such as Sweden, Finland, USA and China [16, 53, 54]. Because there is a close relationship between plant mineral nutrition and human health, food fortification with Se, via a strategy known as biofortification, is an effective way to add Se to human food and prevent the emergence of diseases related to deficient Se intake. This strategy proved to be effective due to the fact that Se presents chemical similarity to S, and both have the same carrier membranes and biochemical pathway of assimilation [28, 33].

Because vegetables are considered an important source of bioactive compounds that contain polyunsaturated fatty acids, phytochemicals such as flavonoids and glucosinolates, many of which can inhibit cell proliferation, induce apoptosis and act synergistically when combined in foods [55], some vegetable groups are more suitable for biofortification because they are natural accumulators of Se such as brassicas [16]. This group of plants has significant levels of glucosinolates, a substance of great nutraceutical interest. In this context, recent studies confirm the biofortifying effect of Se in edible plants in the group of brassicas. For example, in a major study [21] showed the positive impact of Se biofortification in broccoli. This study

found that the broccoli extract showed high levels of phenolic compounds, significant antioxidant effect and anti-proliferative activity of tumor cells on the effect of Se in the synthesis of glucosinolates and phenolic compounds, and anticancer activity, see [16, 56].

Although Se biofortification is a strategy undertaken in edible and domesticated plants, it can occur naturally, as in the Brazilian Amazon, where important food sources of Se are the shrub species of the family Lecythidaceae, *Bertholletia excelsa* Humb. and Bonpl and *Lecythis usitata* Miers, which produce nuts presenting high Se concentrations [57, 58]. For these species, biofortification is a natural phenomenon, because the average levels of Se in their nuts range from 0.03 to 512  $\mu\text{g g}^{-1}$  [59, 60]. Thus, consumption of only one nut per day appears to be sufficient to meet the daily Se requirement of an adult, because according to the Scientific Committee on Foods of the European Commission, the human daily intake requirement of Se is around 55  $\mu\text{g dia}^{-1}$  [27]. However, the production of nuts is seasonal and the season of low production affects the nutrition of people near regions of natural occurrence of these two species [61, 62].

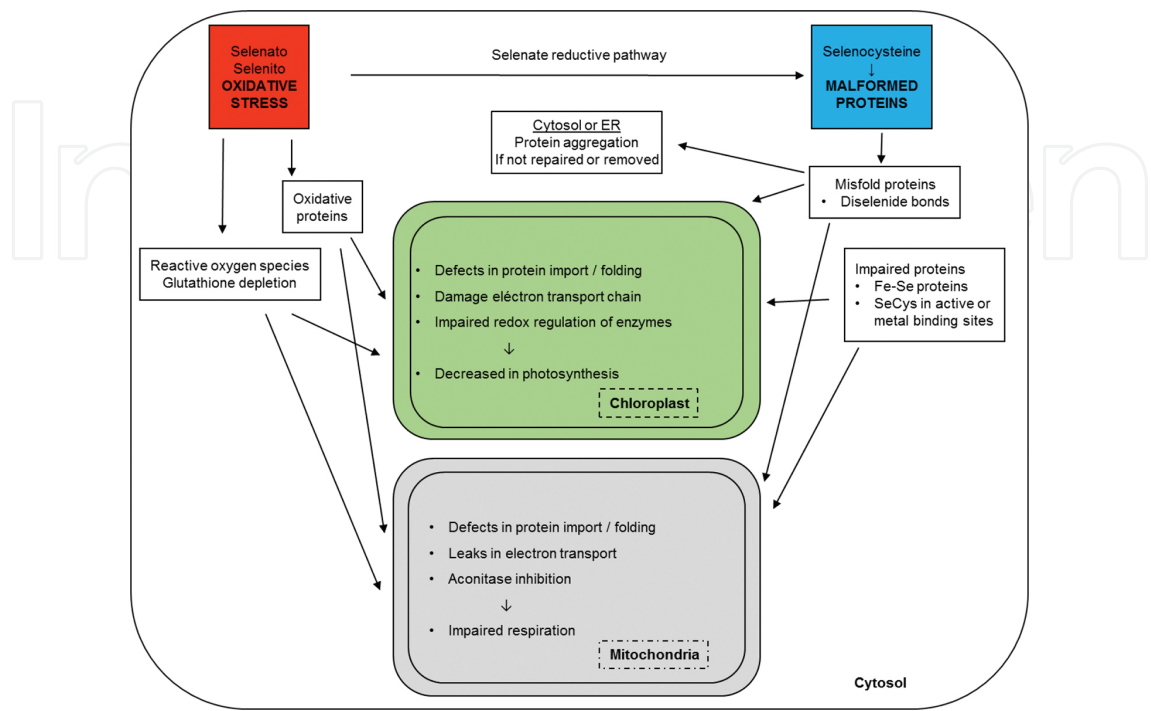
Among domesticated plants of food interest, the biofortification strategy can be carried out by providing Se to the soil, nutrient solution or leaf. However, considering large-scale plantations, the crop cycle and the economic value of the Se sources can make biofortification expensive, with the need for less costly and more practical strategies. One example is pelletizing the seeds of Se accumulator species, like radish, because of its short life cycle. Another strategy is to add Se sources to fertilizers used in agriculture. This strategy was adopted by Whelan and Barrow [63] in cultivation of the grass *Trifolium subterraneum* L in a soil classified as podzol laterite. These authors used a fertilizer containing 1% Se, composed of the sources  $\text{Na}_2\text{SeO}_4$  and  $\text{BaSeO}_4$  in a 1:1 ratio, which is characterized by slow release of Se in the soil. This strategy proved to be interesting due to the fact that there is a synchrony between plant growth and Se liberation, a fact which favors Se absorption and culture biofortification based on soil-plant interaction.

#### 4. Toxicity of selenium

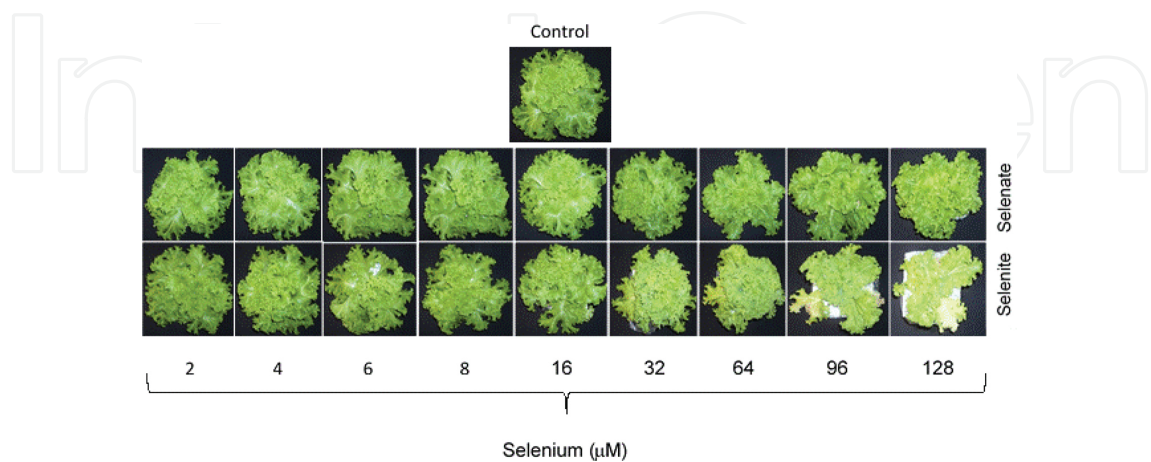
From the point of view of plant nutrition, the effects of Se are twofold, since at low concentrations it can promote biofortification but at high concentrations it triggers toxicity in plants [64]. This duality of effects on plant growth is related to the narrow range between biofortification and toxicity, which in literature is not well established based on determination of critical Se levels. It is understood that the critical toxicity level in the soil or nutrient solution is that which implies 10% reduction in plant growth, since the benefit of fertilization reaches its maximum between 90 and 95% of relative growth of the culture [65]. In plants, the effect of Se toxicity is dependent on the Se concentration and source, as well as plant genotype. However, for the same Se concentration, selenite appears to be more toxic than selenate. This fact is justified by the rapid incorporation of selenite into organic compounds still in the root system [28, 66]. In general, symptoms of Se toxicity in plants are characterized by reduced growth, as observed in lettuce [67], spinach [68], cucumber [64] and pea [29]. Reduced plant growth, as a



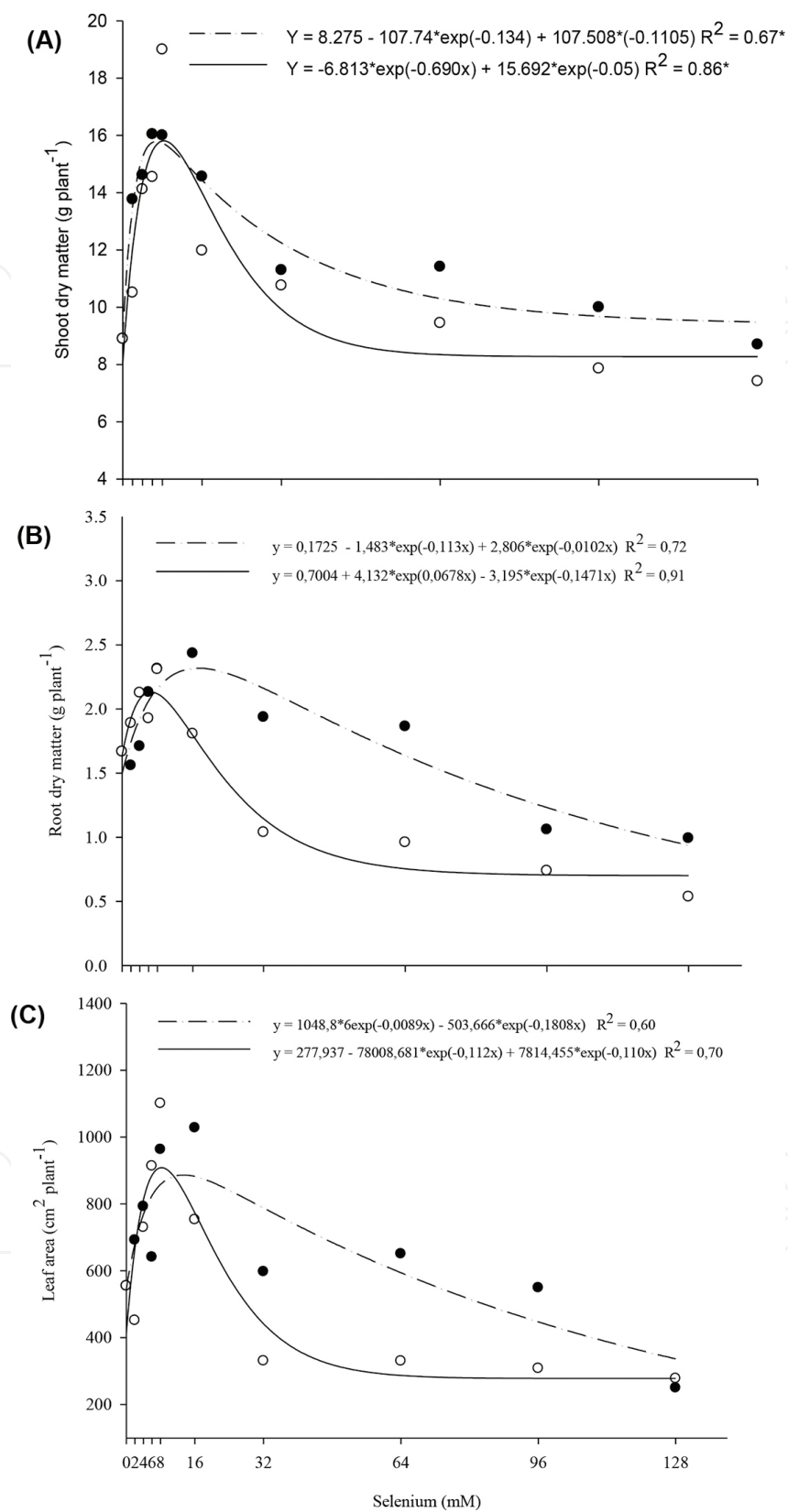
symptom of Se toxicity, is based on two biochemical mechanisms related to Se absorption and assimilation in organic compounds.



**Figure 2.** Model describing the two distinct mechanisms of Se toxicity in plants. Inorganic Se contributes to oxidative stress, while its reduction to selenocysteine may inadvertently substitute the amino acid cysteine and create malformed selenoproteins. The possible targets and ramifications of oxidative stress induced by Se and nonspecific selenoproteins are proposed. Adapted with permission from [28]. These symptoms were observed in the study of toxicity to Se sources and concentrations in the lettuce cultivar Vera, conducted under hydroponic conditions in a study of Ref. [71]. These symptoms were most intense in the source selenite at Se concentrations >8  $\mu\text{M}$  (**Figure 3**). In this study (unpublished data), two response patterns were evident for lettuce plants with respect to Se sources and concentrations in the shoot, root and leaf area growth.



**Figure 3.** Visual aspect of applying increasing concentrations of selenite and sodium selenate to lettuce plants. Sources [71].



**Figure 4.** Leaf area (A), shoot (B) and root dry mass (C) of plants and the lettuce cultivar Vera submitted to selenium sources and concentrations in hydroponic cultivation. Selenite (—○—); Selenate (—●—). Source: [71].

The first mechanism is related to substitution of S for Se in the amino acids cysteine and methionine. This exchange of S for Se implies substitution of the amino acids cysteine and methionine for their analogs selenomethionine and selenocysteine during protein synthesis. This substitution is more deleterious in relation to cysteine, because this amino acid residue is of great importance in the structure and function of proteins and formation of disulfide bonds, enzymatic catalysis, metal bonding sites and redox state regulation. It is also suggested that iron as a metal cofactor complexed to selenocysteine can interrupt the flow of electrons in chloroplasts and mitochondria (**Figure 2**), since these cytoplasmic organelles have electron transport systems with supramolecular organization [28]. Therefore, any replacement of S by Se impedes electron flow due to changes in the protein conformational structure, and hence energy synthesis coordinated through the electron transport chain. The second mechanism is related to the participation of glutathione (GSH), a tripeptide active in cellular redox homeostasis regulation [69] and in the selenite reduction stage [28]. The participation of GSH in this step is crucial, since under Se toxicity conditions there may be a functional imbalance of GSH, since there is increased GSH demand for selenite reduction compared to hydroperoxide reduction in reactions catalyzed by glutathione peroxidase (**Figure 2**).

This imbalance in favor of selenite reduction triggers an oxidative burst that results in reduced plant growth [28]. However, plants considered accumulators or hyperaccumulators have biochemical mechanisms that prevent Se incorporation in proteins, by adding the methyl radical to the intermediate compound selenocysteine which is volatilized [16, 28]. From a symptomatology point of view, Se toxicity is characterized by reduced growth and chlorosis of plants [70].

In the lower concentrations of selenite or selenate, there was greater shoot, root and leaf area growth (**Figure 4A–C**). However, concentrations >14 and 16  $\mu\text{M}$  of Se (selenite and selenate) for the shoots, and 9.7 and 30  $\mu\text{M}$  (selenite and selenate) for the root resulted in growth reduction in these organs and more intense chlorosis in plants grown in the presence of selenite (**Figure 4A–C**). These responses were similar to those observed in lettuce [18] and cucumber [64] grown under sources and increasing Se concentrations.

## 5. Limits between selenium biofortification and toxicity

Plant growth is affected by excess Se, and the sensitivity of plants to toxicity of this mineral element is dependent on its concentration and source. However, in literature, the limits between Se biofortification and toxicity are not evident and based on critical toxicity levels which consider 10% reduction in plant growth [65, 72]. For example, Ríos et al. [18] observed that the concentration of 40  $\mu\text{mol L}^{-1}$  selenate, in contrast to concentrations of selenite, was optimal for lettuce growth and biofortification. However, these authors reported a reduction of plant growth when the selenate concentration was >40  $\mu\text{mol L}^{-1}$ .

It should be noted that the above results reported by the limits between Se biofortification and toxicity are not evident and based on critical toxicity levels which consider 10% reduction in plant growth [65, 72]. For example, Ríos et al. [18] contradict those presented by

Hawrylak-Nowak [67], which places the limit of lettuce biofortification at concentrations 15 and 20  $\mu\text{mol L}^{-1}$  for the sources of selenite and selenate, respectively, by the fact that no negative impact was observed on growth and the content of photosynthetic pigments at these Se concentrations. To investigate the effect of Se in lettuce, Ríos et al. [73] observed that the provision of selenate was not toxic up to the concentration of 80  $\mu\text{mol L}^{-1}$ . On the other hand, selenite concentrations  $>5 \mu\text{mol L}^{-1}$  showed symptoms of toxicity related to reduced growth. These authors attributed growth reduction in plants to the nutritional imbalance of macro- and micronutrients, as well as the oxidative stress caused by toxic levels of selenite used in the study.

In cucumbers, the application of selenate and selenite showed toxic effects with in concentrations  $>80$  and 20  $\mu\text{mol L}^{-1}$ , respectively [64]. In this study, leaf area, leaf and root fresh weight were severely reduced at selenite concentrations  $>20 \mu\text{mol L}^{-1}$ . These authors showed that the concentration of chlorophyllian pigments and fluorescence of chlorophyll were more negatively affected by selenite compared to selenate.

It is known that the human health and plant nutrition are closely linked to soil fertility, and diseases associated with Se deficiency are documented in areas where the content of this element in soil is low, as in China, Denmark, Finland, New Zealand and central and eastern Siberia [74, 75]. However, there is no record of human intoxication by Se due to consumption of plant species of food interest (e.g., corn, rice, beans, wheat, barley, etc.), presumably due to the fact that these plants are not Se accumulators. An interesting fact was recorded on a farm in the state of South Dakota, United States, where the owners recorded low hatchability of chicken eggs or chicks born with deformities and soon died. When investigating the fact, the United States Department of Agriculture identified high levels of Se in wheat grains which were used to feed the chickens [76]. Although literature relates the intake of foods rich in Se to the lower risk of emergence of diseases such as colon, stomach, prostate and lung cancer [51], there are studies that relate Se supplementation to increased risk of death due to prostate cancer [77]. However, the population along the Tapajos River in the Brazilian state of Pará presents high levels of Se in their blood in function of the considerable consumption of nuts from *Bethollethia excelsa* and *Lecythis usitata*, which have high Se levels. In these nuts, the Se concentration is quite variable, within the range from 0.03 to 512  $\text{mg g}^{-1}$  [59], well above the daily intake recommended by the Scientific Committee on Foods of the European Commission, which recommends a daily intake of approximately 55  $\mu\text{g day}^{-1}$  [27]. Nevertheless, in this population, the consumption of nuts rich in Se appears to protect against the emergence of diseases resulting from advancing age such as cataract and motor dysfunction [78]. The recommended daily Se intake (**Table 1**) varies in function of age, gender, pregnancy and lactation. Among the sexes, there is no difference with regard to ingestion demands, except when women are pregnant or lactating, where intake should be increased by 9 and 27%, respectively [79]. However, the maximum tolerable daily Se limit at which there occurs initial toxicity symptoms (**Table 2**) characterized by hair loss, and the appearance of brittle nails is similarly dependent on sex, age, pregnancy and lactation [79].

Age	Male	Female	Pregnancy	Lactation
Birth to 6 months	45 mcg*	45 mcg*		
7–12 months	60 mcg*	60 mcg*		
1–3 years	90 mcg	90 mcg		
4–8 years	150 mcg	150 mcg		
9–13 years	280 mcg	280 mcg		
14–18 years	400 mcg	400 mcg	400 mcg	400 mcg
19* years	400 mcg	400 mcg	400 mcg	400 mcg

Source: [79].  
\*Adequate Intake (AI).

**Table 1.** Recommended dietary allowances for selenium.

Age	Male	Female	Pregnancy	Lactation
Birth to 6 months	15 mcg*	15 mcg*		
7–12 months	20 mcg*	20 mcg*		
1–3 years	20 mcg	20 mcg		
4–8 years	30 mcg	30 mcg		
9–13 years	40 mcg	40 mcg		
14–18 years	55 mcg	55 mcg	60 mcg	70 mcg
19–50 years	55 mcg	55 mcg	60 mcg	70 mcg
51+ years	55 mcg	55 mcg		

Source: [79].  
\*Breast milk, formula, and food should be the only sources of selenium for infants.

**Table 2.** Tolerable upper intake levels for selenium.

## 6. Conclusions and future perspectives

This chapter addressed the main issues referring to the mineral element selenium, its history of discovery, its presence in the soil-plant system and its main functions in mammals and humans, always based on recent scientific findings that can guide students, teachers and researchers in their studies. Also assessed were the mechanisms of absorption, transportation and assimilation of selenium in organic compounds, as well as its biofortifying and toxic effect in plants. Moreover, its essentiality in mammals has been addressed in order to emphasize its importance in food and human health due to its function in the human metabolism. It was therefore sought to present the importance of this nutrient in the plant-human system, and therefore provide information for future studies seeking to clarify other functional aspects of selenium in plants.



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