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REVIEW

Biogeochemistry of selenium and its impact on food chain quality and human health

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Abstract

In areas where soils are low in *bioavailable* selenium (Se), potential Se deficiencies cause health risks for humans. Though higher plants have been considered not to require this element, the experience with low-Se soils in Finland has provided evidence that the supplementation of commercial fertilizers with sodium selenate affects positively not only the nutritive value of the whole food chain from soil to plants, animals and humans but also the quantity of plant yields. The level of Se addition has been optimal, and no abnormally high concentrations in plants or in foods of animal origin have been observed. Se levels in serum and human milk indicate that the average daily intake has been within limits considered to be safe and adequate. In fact, plants act as effective buffers, because their growth is reduced at high Se levels. They also tend to synthesize volatile compounds in order to reduce excess Se. On the other hand, when added at low concentrations, Se exerts a beneficial effect on plant growth via several mechanisms. As in humans and animals, Se strengthens the capacity of plants to counteract oxidative stress caused by oxygen radicals produced by internal metabolic or external factors. At proper levels it also delays some of the effects of senescence and may improve the utilization of short-wavelength light by plants. High additions are toxic and may trigger pro-oxidative reactions. Thus, the present supplementation of fertilizers with Se can be considered a very effective and readily controlled way to increase the average daily Se intake nationwide.

Keywords: Selenium intake; Plant nutrition; Food chain; Antioxidant; Human health

Contents

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Introduction

Selenium (Se) is a complex trace element possibly of greatest concern worldwide. It is an essential nutrient for animals and humans, but it is toxic at high concentrations. Because either insufficient or excess Se intake can have dramatic consequences; this element is often described as a ''two-edged sword''. Se deficiency is generally caused by low concentrations in forage and food, whereas toxicity problems usually result from build-up in body tissues and biomagnification in the food chain. The Se concentration in feed should meet minimum requirements of livestock, in the order of 0.05–0.1 mg/kg dry matter [1]. Toxic effects can be expected at chronic intakes in feed that exceed about 1 mg/kg.

A worldwide atlas compiled by Oldfield [2] shows that the geographical distribution of Se in its ultimate source, namely in rocks and soils, is very uneven, ranging from almost zero up to 1250 mg/kg in some seleniferous soils in Ireland. In most soils the concentration is between 0.01 and 2 mg/kg. Large areas in the world can be characterized as Se-deficient or as Se-toxic. Experience with Se in animal production has led to the recognition that on a worldwide scale, vast land areas do not supply enough of this element for optimum animal nutrition. Although other problems related to seleniferous soils are also well known, Se-deficient areas seem to be far more common than Se-toxic areas. They are typically derived from igneous rocks, whereas high-Se soils largely come from sedimentary deposits [3]. Because Se can cause serious problems at both ends of its supply spectrum, it is important to recognize risk areas. Even though the toxic levels in some areas originate from industrial operations including the combustion of fossil fuels and sulphide ore mining, more often they occur naturally.

Selenium toxicity

The symptoms of Se toxicity were probably described for the first time long before the discovery of the element. While travelling along the Silk Road to remote parts of China in the middle of 13th century, Marco Polo recorded the presence in the province of Shanxi of certain poisonous plants that had serious effects on the beasts that ate them: the hoofs of the animals dropped off [2]. It is likely that these plants were Se accumulators, whose existence as such was not recognized until the end of the 1930s. At about the same time, the occurrence of ''alkali disease'' was reported in the western United States (Rosenfeld and Beath 1964, [4]). The symptoms of this disorder were necrotic and sloughed hoofs, hair loss, poor growth and reproduction, and in extreme cases it caused the death of horses grazing on ranches with saline seeps. Researchers identified ''alkali disease'' as chronic Se toxicosis (selenosis). In seleniferous areas, it still occurs in ruminants and monogastrics. Against this background it is easy to understand that until the 1950s, Se had been considered merely as an environmental toxicant. The findings in the 1980s that Se excess caused death of aquatic birds, malformation of bird embryos and poisoning of fish in Kesterson Wildlife Refuge and Reservoir in California gave rise to further environmental concern regarding this element.

Selenium deficiency

Schwarz and Foltz [5] changed forever the bias that Se is merely a nuisance. In a series of laboratory experiments they made the stunning finding that Se is critical in preventing liver necrosis in rats. Subsequent studies with domestic food-producing animals showed that Se deficiency was the underlying cause of several metabolic diseases previously considered untreatable, such as ''white muscle disease'' and ''ill-thrift'' in calves, liver damage in pigs [6] and exudative diathesis in chicks. Data published in 1963 [4] showed that ''white muscle disease'', a myopathy affecting both cardiac and skeletal muscles in young animals, was diagnosed in about 30 of the 50 states of the USA.

Furthermore, evidence was also presented that Se supplementation was able to enhance the growth rate of young animals [7] and reproduction [8]. In fact, in low-Se areas worldwide, livestock producers have adopted methods of ensuring that their animals obtain adequate amounts of Se. In Finland, for instance, because inadequate Se intake was shown to cause nutritional disorders in pigs [9] and thus to reduce the profitability of their production, all commercial animal feeds have been supplemented with selenite since 1969.

Diseases caused by Se deficiency can be fatal, but in any case they result in sickly, unproductive animals of low nutritive quality to humans. Low Se intake in agricultural products has negative effects on human health. The most serious consequences have been reported in low-Se areas in China and Eastern Siberia, where deficiency has been found to cause two endemic diseases. Keshan disease is a multifocal myocarditis that occurs primarily in children (2–10 years old) and, to some extent, among women of child-bearing age. Kashin-Beck disease, an ankylosis (''big-joint disease'') is a human rheumatoid condition that results in enlarged joints, shortened fingers, toes, and, in extreme cases, dwarfism.

In China, researchers found that both endemic diseases are mainly distributed in a wide belt running geographically from the northeast to the southwest part of the country [10]. The belt is mainly characterized by dark brown, brown earth and black soils, very low in water-soluble (bioavailable) Se. The problems in human and animal health (''white muscle disease'' in lambs, ''mulberry heart disease'' in pigs) in the belt area are related to the low flux of Se in the soil–plant–human system [10]. Recent studies on human Se-responsive diseases in China [11,12] have revealed a negative correlation between soil organic matter and water-soluble Se. However, this relationship cannot be taken to indicate a direct adsorption of Se onto organic matter. A more probable mechanism is that organic matter acts as a source of electrons reducing Se into the selenite (Se^{4+}) form which has been shown to form a strong inner-sphere complex with iron oxides [13]. The selenate anion (Se^{6+}), on the other hand, is adsorbed weakly as an outer-sphere complex.

Recently, Beck et al. [14,15] have shown that Se-deprivation of mice facilitated the mutation of Coxsackie virus B3 to a highly virulent phenotype that produced cardiomyopathy. This suggests that Keshan disease likely has a dual etiology that involves both Se deficiency and an infection of enterovirus. Similarly, a too low Se intake alone seems not to be sufficient to cause Kashin-Beck disease. It is possible that this disorder results from combined Se and iodine deficiency [16].

Western countries have also shown an increasing interest in the Se status of humans, since its health-protective and therapeutic effects are now better understood. For instance, low dietary ingestion of Se has been assumed to contribute to an increased risk of cardiovascular disease and cancer [17,18] and to promote infectious viral diseases related to heart disease and AIDS [19]. In fact, at supranutritional levels of dietary intake, many Se compounds have been shown to be effective in reducing the risk of cancer in animals and humans [20].

Biochemical functions of selenium

Rotruck et al. [21] created a scientific basis for understanding the function of Se in biological systems. They identified Se as an essential component of the antioxidative enzyme glutathione peroxidase (GSH-Px), which is a scavenger of hydrogen peroxide and lipid and phospholipid hydroperoxides in human cells. This provided additional impetus to carry out research on the effects of Se and produced more information about other selenoenzymes that regulate, e.g. hormone balance, electron transfer in the biosynthesis of nucleotides and redox status of vitamin C in human and animal cells [22–24]. It has also been shown that through its antioxidative function Se can ameliorate UV-B radiation damage in humans and experimental animals [25–27].

The prevailing view is that higher plants do not require Se. This has placed the scientific community in a dilemma, for plants play a key role in cycling Se from soil to animals and humans. Are plants only conveyers in this system, or do they derive some direct benefit from Se for themselves? Recent studies with ryegrass, lettuce, potato and soybean [28–31] give indications that at proper addition level Se is able to enhance the growth rate of plants. Furthermore, when considering this question we have to recall two other facts. Firstly, some plants have developed a remarkable ability to take up Se from soil [32]. This means that they have metabolic systems to treat and accumulate this element. Secondly, it has been shown repeatedly that Se is more bioavailable to animals and humans in organic forms than in inorganic forms [33]. Plants have an important role in synthesizing organic Se, including amino acids, and providing it to animals and humans. In wheat, soybean, and Se-enriched yeast, Se is found predominantly as selenomethionine [34 and references therein]. It can be hypothesized that protein-bound Se may have similar roles in animal and plant cells.

Selenium cycle in soil

While the principal source of Se for most individuals is the daily diet, intake with food and drinking water is ultimately dependent on geochemical factors. The Se cycle begins and ends with soil, and the chemical forms (dissolved in soil solution, adsorbed on the oxide surfaces, fixed in the mineral lattice) and concentrations of Se in soil determine its *bioavailability* and thus the need for dietary supplementation. In theory, Se exhibits a broad range of oxidation states: +6 in selenates, +4 in selenites, 0 in elemental Se, and −2 in inorganic and organic selenides. It also forms catenated species, such as volatile diselenides (RSeSeR). Selenate, which is

weakly adsorbed on oxide surfaces and thus the most mobile Se form, can be expected to occur under high oxidative conditions [35]. At low redox potential it can be reduced to selenite, which has a much higher adsorption affinity. It is strongly retained by ligand exchange on oxide surfaces, especially at low pH, which reduces its bioavailability. Volatile Se is lost to the atmosphere from plants or through microbial activity, but Se also returns to the soil from the atmosphere with precipitation.

Selenium in the food chain

The Se problem in Finland and its solution can be used as an example to describe various aspects of food chain quality. Finnish soils are not exceptionally low in Se, but they are young and weakly weathered, acid and high in adsorptive oxides. All these soil characteristics contribute to the low bioavailability of this element. Thus, in the mid-1970s, the extensive "Mineral Element Study" [36] revealed that Finnish cereal crops, beef, milk and dairy products were distinctly poorer in Se than the comparable agricultural products in other European countries, North America and Australia, and that the average daily Se intake in Finland was clearly below the recommended safe and adequate intake level (0.05 mg/day) defined by the National Research Council [37] in the USA.

Even though severe human diseases directly related to Se deficiency could not be detected, the quality of domestic food products and the health risks due to the potential Se deficiency raised questions. The Finnish authorities became concerned about human well-being: epidemiological studies gave indications that low Se intake correlated positively with increased risk of cardiovascular disease, coronary heart disease and cancer [17,38,39]. These findings made Se an issue of prime public interest, and all sorts of Se supplements became very popular. It became urgently necessary to restore consumer confidence in Finnish agricultural products and to control the exaggerated interest in Se medication.

The Working Group of the Ministry of Agriculture and Forestry made a proposal for supplementation of all multinutrient fertilizers with Se. This was considered the safest and cheapest way to solve the Se problem. The main goal of the operation was to increase the Se concentration of Finnish cereal grains to 0.1 mg/kg dry matter and the average Se intake to the safe and adequate range of 0.05–0.20 mg/day [37].

Impact of selenium fertilization on food quality

In 1985, Se-containing fertilizers came into general use. In the manufacturing process, sodium selenate is added to the fertilizer slurry in order to obtain a uniform Se concentration in the granules. Since the commencement of Se fertilization, its impact has been regularly monitored by analyzing Se in agricultural soils and plants, water, all types of feeds, plant and animal foods, and human sera, the results of these studies appearing in numerous publications [e.g. 40–42]. The Se level in fertilizers has been adjusted on the basis of these findings. In 1991, the initial level of 16 mg/kg used for cereal crop fertilizers was reduced to 6 mg/kg. Since this measure had an adverse effect on the crop quality, in 1998 the Se concentration was raised to the present level of 10 mg/kg.

Fertilization induced drastic changes in the Se concentration in agricultural products. For instance, in spring cereals the increase was generally 20–30 fold during the first years of supplementation (Fig. 1). The present level is about 13 times higher than in the mid-1970s. In winter cereals, the Se levels increased first 2–5 fold to 0.07 mg/kg dry weight in 1990, the present level being about 10–12 times higher than that in the 1970s.

Throughout the monitoring program, milk has been the most sensitive indicator, and it was the first foodstuff to reveal the changes in food quality induced by Se fertilization (Fig. 2). The temporary reduction in Se supplementation in 1991 was also quickly and clearly reflected in lower Se concentrations of dairy products. When compared with the Se levels in milk, cheese and eggs in the mid-1970s, the increase was about 10-, 6- and 3-fold, respectively [41]. In meat and meat products, the Se concentration increased 13-fold during 1985–1991. The increase in pork was lower, about 4-fold, because the generally used Se supplementation had previously improved the Se intake by pigs. The effects of Se fertilization on Se bovine and porcine liver have been only moderate.

The Se supplementation of fertilizers has substantially affected the average Se intake. On the basis of Finnish food balance sheets it can be estimated that a plateau of 0.11–0.12 mg/day (at an energy level of 10 MJ) was

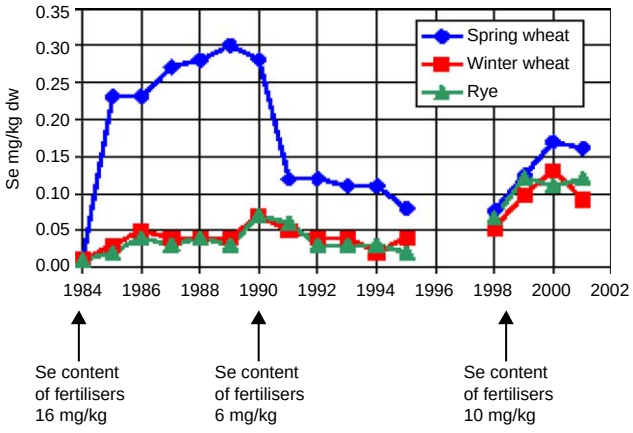

Fig. 1. Trends in Se concentrations in wheat and rye grains during 1984–2001 (data compiled by M. Eurola, see [40]).

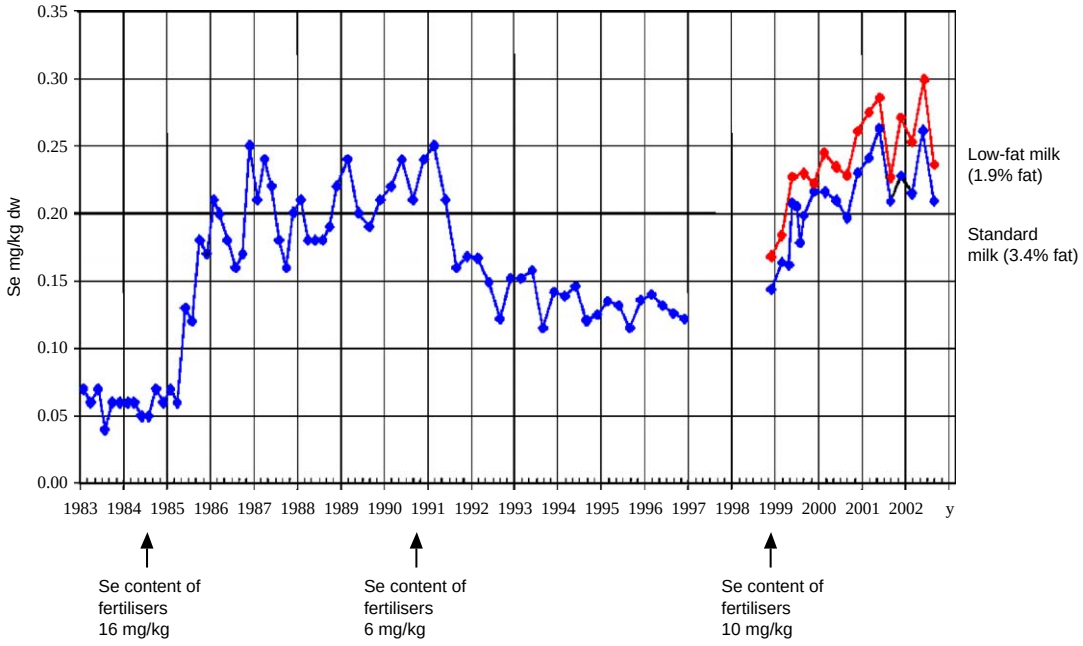

Fig. 2. Se concentrations in milk in Finland during 1984–2001 (data collected by M. Eurola, see [40]).

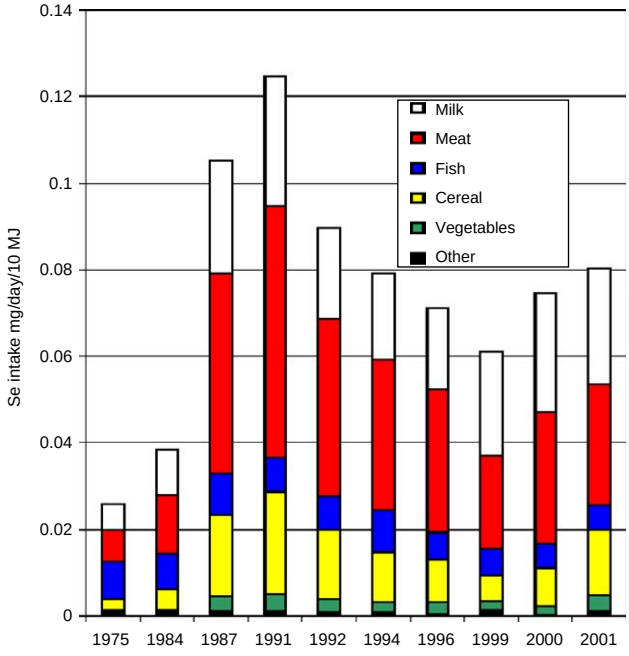

Fig. 3. Trends in Se intake in Finland during 1975–2001 (data compiled by P. Ekholm, see [40]).

reached in 1987 (Fig. 3). The reduction in the supplementation level in 1991 lowered the daily human Se intake in the subsequent years. However, the increase in the Se concentration in fertilizers in 1998 resulted in a new rising trend in the intake. These responses clearly show that agricultural products are a rather sensitive reflection of the status of available Se in soil.

Impact of fertilizer selenium on humans

In Finland, Se in human serum has been monitored in healthy adults since the 1970s. Before Se supplementation of fertilizers, the low dietary intake was reflected in serum Se levels (0.63–0.76 μmol/l) that were among the lowest reported in the world [43]. Interestingly, the situation improved in the years when high-Se wheat was imported. The present level, varying between 1.2 and 1.4 μmol/l, is slightly higher than in most other European countries, but lower than that generally found in Canada and in the USA [43]. This indicates a good response to the supplemented fertilizers.

To date, studies of Se in plants have focused on the concentration assumed to be a quality index for animal fodder and human food. However, an ultimate quality index would be the bioavailability of Se needed to regulate biochemical functions. A review by Rayman [44] clearly shows that Se is more bioavailable in organic than in inorganic form. It also depicts how the metabolic pathway of Se depends on its species. Cereals and forage crops convert Se mainly into selenomethionine and incorporate it into proteins in competition with methionine. In this form Se is well retained in the body. In contrast, selenocysteine cannot be directly incorporated into proteins but it is better utilized for the selenocysteine enzymes. Furthermore, Se-methyl-Se-cysteine produced in Se-enriched garlic and broccoli is largely converted in animals and humans directly to methyl selenol, a potent anti-carcinogen [45].

Alfthan et al. [46] reported an interesting study showing the biochemical impact of Se fertilization on

humans. They measured the activity of the antioxidative selenoenzyme GSH-Px in platelets of healthy men supplemented with 200 μg of inorganic Se (sodium selenate) or organic Se (Se-enriched yeast) or with a placebo. Their study was carried out before and after the introduction of Se fertilizers. The results revealed that before the use of Se-containing fertilizers, sodium selenate increased the enzyme activity by 104%, while yeast-Se increased it by 75%. The corresponding figures during Se fertilization diminished to 41% and 6%, respectively. This result provides evidence that the fertilizer Se had positively affected the antioxidative capacity of the platelets, even though it had not completely saturated the GSH-Px activity.

In China, Se supplementation has been widely used to control Keshan and Kashin-Beck diseases [47,48], even though the latter disease probably is a combined result of deficiencies of two trace elements, Se and iodine [16]. In chemoprevention of cancer, the Se dosages are far beyond normal dietary intake [49]. When supplied with fertilizers, the Se intake remains far below the supranutritional level generally used in the intervention studies. Eurola et al. [40] have stated that the use of Se-containing fertilizers in Finland is a nationwide experiment affecting all individuals, not a placebo-controlled clinical trial. Therefore the impact of Se fertilization on the occurrence of human diseases is difficult to judge.

Nevertheless, age-adjusted mortality from coronary heart disease has declined continuously in Finland since the end of the 1960s. This positive trend has been attributed to favourable changes in the composition of the diet and to the decrease in classical risk factors such as serum cholesterol, smoking, etc. However, it should be noted that since 1969 commercial animal feeds have been supplemented with Se in order to gain control of deficiency symptoms in domestic animals. This suggests that the supplementation of feed as such might have had some prophylactic effect on humans who ingested animal products. As humans are at the top of the food chain, we can hypothesize that the subsequent introduction of Se fertilizers may have further contributed to the continuously decreasing trend, especially in mortality of men (Fig. 4). Humans benefit from fertilizer Se in two forms: as incorporated in plant products and as plant-derived Se bioaccumulated in animals.

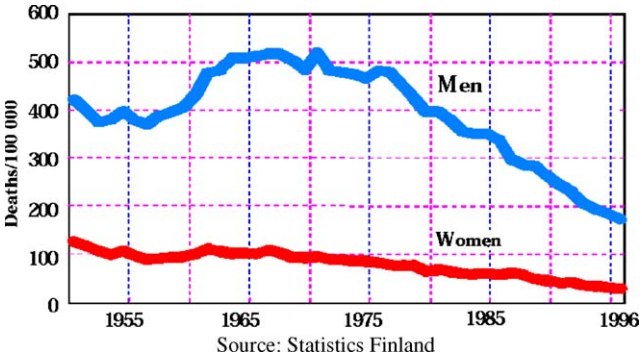

Fig. 4. Age-standardized mortality from coronary heart disease in Finland during 1955–1996, deaths per 100 000 of the population aged 35–64 (figure prepared by G. Alfthan, see [40]).

The role of selenium in plants

Despite the present positive trend in daily Se intake in Finland, the advantages of fertilization compared to a direct supplementation of food and feed with Se have been questioned. Some sceptics ask why the plants are forced to take up an element they do not require. On the other hand, the prevailing view that this element has no role in plants can also be questioned. Maybe Singh et al. [50] in 1980 were the first to publish a study indicating a growth-promoting effect of Se in non-accumulator plants. They cultivated raya (*Brassica juncea* Coss.) in sand culture and in sandy soil and found that at a low addition level Se increased the dry matter yield, particularly when sulphur was not added. For this reason, we conducted a series of greenhouse experiments to study the biological role of Se in plants. The initial working hypothesis was that as in human and animal cells, at proper levels Se is able to defend plants against oxidative stress caused by internal factors such as oxygen radicals produced in photosynthesis or respiration, or by external factors such as short-wavelength light. The experiments have been carried out with plant species used for food and feed.

Antioxidative and pro-oxidative effects

In a study of the possible antioxidative function of Se [28], ryegrass was cultivated at Se addition levels ranging from low to very high. Two subsequent harvests revealed that the effect of Se on ryegrass is dependent on dosage and plant age (Table 1). The very low Se addition had an indifferent effect on the shoot yields, but when the Se addition was increased, the ryegrass yield increased by 13% in the second harvest. The growth-promoting response agreed with the enhanced antioxidative capacity manifested in decreasing lipid peroxidation (TBARS). This response coincided with a marked increase in GSH-Px activity and a peak concentration of tocopherols (vitamin E), scavengers of lipid peroxide radicals and singlet oxygen. The results show that Se treatment may not only increase the yield of forage plants but also improve their nutritive quality

Table 1. Fresh weight (FW) of the ryegrass shoot yields, GSH-Px activity, TBARS and α-tocopherol concentration of the crops at various Se addition levels

	Se added (mg/kg)	FW (g/pot)	TBARS[a]	GSH-Px[b]	α-tocopherol (μg/g FW)
1st harvest	0	85.1[a]	26.2[c]	UD	16.5[b]
	0.1	90.4[a]	23.2[d]	4.17[c]	17.1[b]
	1.0	68.7[b]	22.1[d]	7.55[b]	18.4[b]
	10.0	1.3[c]	170[b]	56.5[a]	36.7[a]
	30.0	0.1[d]	236[a]	ND	ND
2nd harvest	0	92.9[b]	35.2[b]	2.57[d]	49.4[c]
	0.1	91.5[b]	32.9[b]	3.26[c]	47.4[c]
	1.0	105.0[a]	26.1[c]	3.96[b]	62.3[b]
	10.0	0.2[c]	304.5[a]	13.90[a]	58.0[b]
	30.0	0.05[d]	ND	ND	124[a]

Each column and crop was tested separately for statistical significance. Values followed by the same letter are not statistically different at $p \leqslant 0.05$ (Duncan test).

ND, no data; UD, undetectable.

[a]nmol malondialdehyde (MDA)/g FW.

[b]nmol reduced glutathione (GSH)/mg protein/min.

Table 2. Dry weight (DW) of the lettuce shoot yields, Se taken up by the crops[a], TBARS concentration and GSH-Px activity of the lettuce crops at various Se addition levels

Se added (mg/kg)	Shoot yield (g DW/pot)		Se taken up (μg/pot)		TBARS[b]		GSH-Px[c]	
	YS	SS	YS	SS	YS	SS	YS	SS
0	20.7[a]	46.9[b]	1.66[c]	6.24[c]	128[a]	268[a]	5.58[c]	5.94[b]
0.1	20.3[a]	53.5[a]	98.0[b]	103.0[b]	74[c]	159[c]	6.95[a]	6.86[a]
1.0	6.9[b]	27.9[c]	1858[a]	1150[a]	110[b]	199[b]	6.55[b]	6.78[a]

Each column and crop was tested separately for statistical significance. Values followed by the same letter are not statistically different at $p \leqslant 0.05$ (Duncan test).

[a]YS, young seedlings; SS, senescent seedlings.

[b]nmol malondialdehyde (MDA)/g FW.

[c]nmol reduced glutathione (GSH)/mg protein/min.

in many ways. On the other hand, a severely toxic effect of Se on yield was observed in both harvests, where the highest additions appeared to have a pro-oxidant effect as seen from TBARS concentration (Table 1).

Senescence - delaying function

Senescence, an integral part of plant development, may coincide with the production of free oxygen radicals and be regulated by a variety of environmental and autonomous factors. Free radical reactions diminish the value of vegetables on the market and cause post-harvest losses. We hypothesized that through its antioxidative function Se is able to delay plant senescence and to promote plant growth. We cultivated lettuce in two pot sets at various Se addition levels and harvested it at a stage fit for sale (young seedlings, 49 days old), or at a senescent stage (98 days old) [29]. Table 2 shows that at the low addition level, Se effectively counteracted senescence-induced lipid peroxidation and enhanced the growth of senescent lettuce by 14%. The fertilizer Se enhanced GSH-Px activity in the young seedlings, but no further increase was found in senescent plants presumably because of the decrease in the Se concentration. It is likely that Se was lost as volatile compounds from the shoots, because the amount of Se in the senescent seedlings was lower than in the young ones (Table 2). Volatilization of Se from lettuce may have taken place as dimethylselenide [51].

The concentration of tocopherols (vitamin E) is an important quality factor of vegetables used in the human diet. Total tocopherols diminished during senescence, but the added Se counteracted their decrease and thus the impairment of the nutritive value of lettuce (Fig. 5). The role of Se in maintaining tocopherol concentration at later stages in plant development was confirmed in a later study [52].

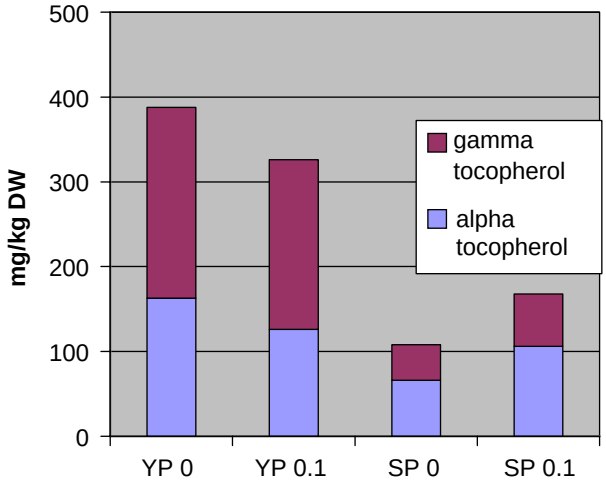

Fig. 5. Tocopherol concentrations in young (YP) and senescent (SP) lettuce seedlings cultivated without added Se or at an Se addition level of 1.0 mg/kg of soil.

Defending role against external stress factors

Plants may respond via numerous mechanisms to stress induced by increased UV-B radiation. As some of the detrimental effects of UV-B radiation in plants are due to oxidative stress, we tested the hypothesis that through its antioxidative function Se is able to increase the ability of plants to tolerate UV-B [53]. Ryegrass and lettuce were cultivated without Se or with Se (added at 0.1 and 1.0 mg/kg) under normal greenhouse light conditions or under short UV-B irradiation episodes of 1–2 min. Without Se, UV-B diminished the lettuce yield markedly (by 23%), but ryegrass showed a higher tolerance. A low Se dosage had no effect on yields under normal light conditions, but in combination with high-energy light it enhanced plant growth, i.e. UV-B light triggered the growth-promoting effect. Interestingly, UV-B alleviated the toxicity of the higher Se dosage. In conformity with previous experiments, Se reduced lipid peroxidation irrespective of light conditions.

Growth-promoting effect

All these studies have shown that proper levels of Se are able to enhance plant growth. We are now studying the physiological factors behind this effect. The results obtained in two experiments hitherto [30,52] have revealed that Se enhances the accumulation of starch and sugars in plants.

Conclusions

In areas where soils are low in *bioavailable* Se, potential Se deficiencies cause health risks for humans. The supplementation of commercial fertilizers with sodium selenate has proven to be an effective and safe way to increase the Se intake of the whole population in Finland. The Se concentrations of all foods studied have remained at consistently safe levels since the inception of this program. Se levels in serum and human milk also indicate that the average daily intake has been within limits considered safe and adequate. In fertilizers, the level of Se addition has been optimal, and no abnormally high concentrations in plants or in foods of animal origin have been observed. In fact, plants act as effective buffers, because their growth is reduced at high Se levels. They also tend to synthesize volatile compounds in order to reduce excess Se. On the other hand, the systematic pot experiments we have carried out have provided evidence that Se, when added at low concentrations, exerts a beneficial effect on plant growth via several mechanisms. As in humans and animals, Se strengthens the capacity of plants to counteract the oxidative stress caused by oxygen radicals produced by internal metabolic or external factors. At proper levels it also delays some of the effects of senescence. There are also indications that Se may improve the utilization of short-wavelength light by plants. High additions of Se are toxic. Pro-oxidative reactions may be one of the toxicity mechanisms of Se excess. The present supplementation of fertilizers with Se is a good example of how the use of fertilizers can affect positively not only the quantity of the yield but also the nutritive value of the whole food chain from soil to plants, animals and humans.

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