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Selenium: Essential Trace Element or Toxic Threat? Navigating Its Role in Modern Nutrition

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Abstract

Selenium is a trace element and a necessary micronutrient for human health. The use of organic selenium as a trace element supplement in microorganisms and plants is becoming more and more popular. The objective of this study is to review the biological functions, molecular mechanisms, nutritional significance, metabolism, supplementation methods, and research needs of selenium in both health and disease across species. In the light of modern knowledge of molecular mechanisms that underlie their pathogenesis, Se-responsive diseases in man and animals, and tracing the discovery history towards selenium being established as an essential nutrient. The role of selenium in dietary sources, nutritional utilization, metabolic processes, and dietary needs of selenium in various species are all critically discussed in this paper. In addition, the function and regulation of Selenium in humans, food animals, and rodents are discussed. In addition to the discoveries of Se metabolites, approaches to enhance Se levels, and the cycling of Se in food systems and ecosystems, the review on the metabolic effects of high intakes of dietary Se in various species would be included. Lastly, the basic science and real-world uses of dietary selenium in food, nutrition, and health across species are supported by the research needs that have been identified.

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Introduction

Selenium is a vital trace element that plays a significant role in the health and metabolism of nearly all living organisms [1]. Its unique chemical properties and biological significance make selenium essential across a wide range of animal species, highlighting its importance in both nutrition and disease prevention [2]. Chemically, selenium (Se^{34}_{79}) is a metalloid that is similar to sulfur (S). The amount of selenium in the soil is influenced by factors such as rainfall, type, texture, and organic matter content of the soil [3]. Numerous hormones and enzymes contain biologically active microelement selenium. Selenium, a potent antioxidant, benefits the animal cardiovascular system by preventing myocardiosis and lowering the chance of cardiovascular disease development [4]. Selenium (Se), initially considered a harmful substance present in soils and feed for grazing animals, has now been acknowledged as a vital nutrient for numerous species, including humans. In addition to its recognized nutritional benefits, selenium is now known to have vital metabolic functions essential for preserving health and physiological activities. Due to its antioxidative properties, selenium plays a significant role in the metabolism of certain plants and cyanobacteria. Despite being detrimental to plants at high concentrations, it has positive effects at low concentrations. It postpones senescence, encourages the growth of ageing seedlings and boosts plant resistance to oxidative stress brought on by UV light. It has recently been demonstrated that Se can control the water status of plants during drought [5]. This review discusses the diet sources of selenium, its physiological mechanisms of fit digestion, metabolism, absorption, its deficiency and consequences of excess intake (Fig. 1). Schwarz et al. were among the first to suggest that selenium is an essential nutrient by showing its ability to prevent exudative diathesis in chicks and liver necrosis in rats [6, 7]. Later studies showed that a deficiency of both vitamin E and selenium led to the development of skeletal myopathies, white muscle disease in pigs, hepatic, cardiac, and smooth muscle pathologies in turkeys, lambs, and calves, placental retention in cows and toms' infertility [8]**.** Vitamin E is known to have an antioxidant function, so Se is also considered an antioxidant in the prevailing paradigm for nearly 20 years, because these lesions were avoided by supplementing those diets with either vitamin E or selenium.

How do plants get selenium?

Selenium concentration in food depends on the content of selenium in the soil where crops are grown. Selenium exists mainly in two inorganic forms in soil: selenate (Se^{6+}) and selenite (Se^{4+}). Selenate predominates in soils with a high redox potential and basic pH, while selenite is more common in slightly acidic to neutral soils [9]. These species are absorbed from the roots by plants and then converted into organic compounds such as selenomethionine (SeMet) and selenocysteine (Sec) [10]. Regions of China, New Zealand, and Eastern Europe are dominated by soil selenium deficiencies, where crops and forages are typically low in selenium. Areas where soils are replete with selenium, such as specific parts of the United States, India, and Venezuela, result in foods rich in selenium. The uptake mechanisms involve selenate absorption primarily through sulfate transporters, while selenite uptake may involve phosphate and silicon transporters. After absorption, selenite is often metabolized and retained in roots, whereas selenate is more readily translocated to shoots, where it is incorporated into organic forms [11].

Sources of Selenium

Selenium contents in wheat grain vary from deficient soils with less than 0.005 mg/kg to above 5 mg/kg in abundant selenium areas [12]. Most of the plants take up selenium in the form of SeMet [13], although variation exists with the species and the type of soil. Other cruciferous, including garlic and broccoli, may store much larger quantities of selenium if the selenium content in the soil is large, or even in cases where fertilizers are enriched through fertilizers. Such Se-bearing crops are fed to both animals and humans as additives against Se deficiency conditions in those locations where it is deficiently present in sufficient availability (Fig. 2). Other forms are SeMet and Sec, which occur in animal products, except for muscle meat that normally contains 0.3 to 0.5 mg/kg selenium (liver, kidney, and other organ meats) that are rich sources [14]. In addition, selenium can be obtained from eggs, milk, and seafood. Foods and diets rich in selenium should be provided in selenium-deficient regions, such as sodium selenite, selenate, and seleno-yeast (Fig. 3). Population-specific deficiencies targeted by interventions are effective.

Fig. 1 Selenium metabolism, critical intake stages, and biological cascade understanding and future directions.

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Absorption, Metabolism, and Utilization of Selenium

Selenium from the diet is highly absorbed by the gastrointestinal tract. These are broken down and are absorbed by amino acids like SeMet and Sec via the active transport system [15]. The inorganic forms, like selenite and selenate, are acquired through simple diffusion or carrier-mediated transport [16]. Species and forms of chemicals express the rates of selenium absorption. The bioavailability of Se is between 98% for SeMet and as low as 84% selenite [13]. For instance, organic selenium is readily available to animals, but the inorganic forms are not. Selenium metabolism in ruminants is controlled by the action of microbes in the rumen that either reduce the inorganic form of selenium into less bioavailable forms or absorb it into microbial proteins. Thus, the bioavailability of forage-based diets is lower than that of concentrate-based diets in ruminants. The main absorption site of selenium, especially the duodenum and jejunum of the small intestine, where the species of selenium must be carried by the hepatic portal vein to the liver to be further metabolized [17]. It gets metabolically activated upon ingestion into the liver. The Selenite gets reduced into a selenide, which is an intermedi-ary metabolite. This selenide can be utilized in three ways. There can be a reduction of the selenide into Sec through the action of SPS2 and SecS, where the latter is a catalyst to the co-translational process of inserting Sec into selenoproteins [18].

Excretion and Utilization of Excess Selenium

As the excess selenium in the body causes selenium toxicity, that's why its removal is necessary. The kidney's glomerular filtration rate, which is measured by creatinine clearance, determines how much selenium is excreted. Since muscle makes up almost half of the body's selenium in West German adults, the direct correlation between selenium excretion and creatinine excretion suggests that muscle affects selenium excretion in urine [19]. In an experiment, regardless of the Se-form that was given, excess dietary Se was excreted as Se-sugar, and the rate of total Se excretion was dose-dependent. methylselenocysteine (Se-DRS) and dietary selenomethionine (soybean meal) had distinct excretion dynamics from the mineral Se (Se-salt). The Se source had a significant impact on the amount of Se species in the urine [20]. The exhalation of volatile methylated selenium metabolites causes

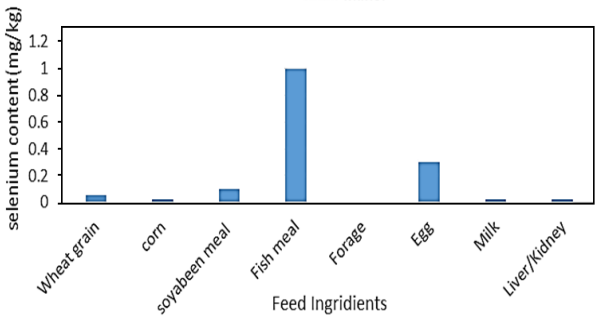

Fig. 2 Selenium content in common feed ingredients
Figure illustrates the selenium content (mg/kg) in various common feed ingredients. The data clearly show that fish meal contains the highest concentration of selenium among the feeds analyzed, reaching approximately 1.0 mg/kg. Eggs also present a relatively high selenium content compared to plant-based ingredients, followed by soybean meal and forage. In contrast, wheat grain, corn, milk, and liver/kidney exhibit much lower selenium concentrations, typically below 0.1 mg/kg.

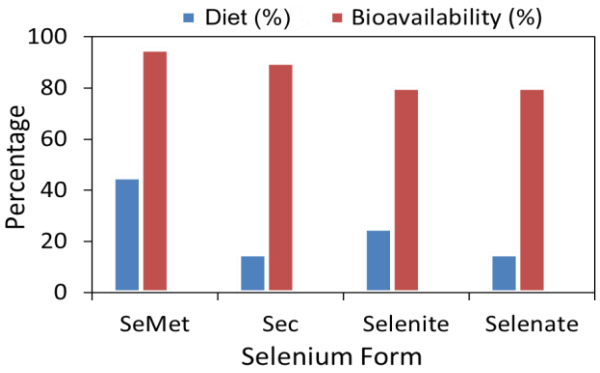

Fig. 3 Distribution and bioavailability of selenium forms in animal diets.
This bar chart illustrates both the dietary proportion in diet and the bioavailability of four major selenium forms commonly found in animal diets: Selenomethionine (SeMet), Selenocysteine (Sec), Selenite, and Selenate. Organic selenium forms (SeMet and Sec), primarily derived from plant and animal proteins, constitute a larger share of dietary selenium (SeMet: 45%, Sec: 15%) and exhibit the highest bioavailability (SeMet: 95–98%, Sec: 90–95%). Inorganic forms (Selenite and Selenate), typically present in mineral supplements, represent a smaller fraction of dietary selenium (Selenite: 25%, Selenate: 15%) and have comparatively lower bioavailability (both 80–84%).

animals that receive such acute doses of selenium compounds to develop a garlicky breath odour. Trimethylselenonium ion, a urinary metabolite, and dimethyl selenide, the main volatile metabolite, have comparatively low orders of toxicity. Animals acutely poisoned with selenium experience tetanic spasms, vomiting, dyspnea, and respiratory failure in addition to garlicky breath odour. Excess selenium is methylated to metabolites such as trimethylselenonium, excreted through urine [13] or as dimethylselenide through breath [21]. SeMet may be incorporated non-specifically into body proteins; it acts here as a reservoir source of selenium. Selenoproteins are the proteins that are associated with protection against oxidative stress, regulation of redox state, thyroid hormone biosynthesis, and immunity. It catalyzes peroxides to their breakdown; therefore, there is less damage to cells. It balances the redox status during thioredoxin reduction. It influences thyroid hormone metabolism by facilitating deiodination.

Selenium Requirement of Different Species

The requirement for selenium intake daily depends on age, sex, and physiological condition. The recommended dietary allowances (RDA) of the element in adults are in a range between 55 µg/day, while pregnant women require 60 µg/day, and lactating women require 70 µg/day [13]. It depends upon optimum selenoprotein activity, and this avoids deficiency disorders. The amount required by cattle in terms of dietary intake varied between 0.1 to 0.3 mg/kg of DMI depending upon growth stages and physiological needs [13]. Selenium supplementation has been thought to be of highest priority in selenium-deficient regions to avoid diseases like white muscle disease. Sheep and goats require the same amount of 0.1 to 0.2 mg/kg DMI as cattle (Table 1). Selenium-enriched feeds and mineral supplements are commonly used to meet their requirements. The needs of pigs in terms of selenium amount to about 0.15 mg/ kg of feed [22]. Supplemental selenium and organic or inorganic selenium increase growth performance and immune functions. In poultry feed, the selenium level must be within the range of 0.15 to 0.3 mg/kg for both chicken and turkey breeds [23]. An adequate level of selenium ensures good growth, good performance and better reproduction and antioxidant defense

Table 1 Recommended selenium intake by different species.

Species	Intake (mg/kg feed DMI)	References
Cattle	0.1–0.3	[13]
Sheep/Goat	0.1–0.2	[13]
Pigs	0.15	[22]
Poultry	0.15–0.3	[23]
Cats/Dogs	0.3–0.35	[24]
Fish/Shrimp	0.2–0.5	[25]

mechanisms in birds. The required amount of selenium is about 0.3 to 0.35 mg/kg in the cat and dog diet [24]. Commercial foods for pets contain selenium fortifications to meet such requirements. Fish and shrimp require from 0.2 to 0.5 mg/kg of feed selenium [25]. Aquatic species require selenium for growth, reproduction, and resistance to oxidative stress. Selenium requirements in wildlife vary with species and dietary habits.

Selenium Deficiency and Diseases Associated with Its Deficiency

Selenium, a major trace mineral, is deficient and causes major problems and diseases in many species. In an experiment, it was found that the chick body and testicle weights were substantially reduced because of Se deficiency, and their body organ coefficients also decreased, and their testis tissue was damaged. Se deficient group had significantly lower levels of the sex hormones testosterone, estradiol, as well as the activities of the serum antioxidant enzymes T-AOC, GSH-ST, and GSH-PX. Additionally, the Se-deficient group had a significantly higher level of autophagy. According to the data, chicks that are dietary deficient in selenium show lower antioxidant enzyme activity, poor testicular growth, impaired synthesis of sex hormones, and elevated mRNA and protein expression of factors linked to autophagy [26]. If sheep, rabbits, horses, and deer are raised in regions where selenium is deficient, then they may show deficiency symptoms. It would require some good practices to make selenium available for them, such as selenium-fortified salt licks. Selenium deficiency, by interrupting the synthesis of selenoproteins, breaks the antioxidant defense. It makes the organism vulnerable to oxidative stress. The diseases that result from selenium deficiency are these. Keshan disease, a cardiomyopathy found in selenium-deficient areas, and worsens with viral infections [27]. Kashin-Beck disease, a degenerative osteoarthropathy brought about by inadequate intake of selenium [28]. White Muscle disease affects livestock, causing degeneration of skeletal and cardiac muscles [29]. In immune function impairment, the effect of selenium deficiency reduces the activity of immune cells and thus increases susceptibility to infection. High-risk groups are those with dietary intakes deficient in selenium; these include parts of China [30] and New Zealand. Those individuals who have low levels or are suffering from any kind of disorder also show an increased risk of suffering from deficiency.

Diseases Associated with High Intake of Selenium

As selenium is an important trace mineral, a variety of illnesses, ranging from acute respiratory distress syndrome, myocardial infarction, and renal failure (Se overloading) to chronic inflammatory diseases like rheumatoid arthritis, inflammatory bowel disease, and atherosclerosis (Se unloading), are linked to an imbalance in the bioavailability of selenium [32]. Excessive intakes of selenium accumulate the methylated metabolites of selenium and bring about oxidative damage and interfere with cellular metabolism [13]. The acute manifestations of Selenium toxicity are nausea, vomiting, diarrhea, and severe abdominal pain. Chronic Selenosis leads to nail brittleness of nails, garlic odor, breathing, and neural disturbances. In diabetogenic effects, high selenium status has been related to an increased risk of type 2 diabetes [33]. Tolerance to high selenium intake varies with species. Livestock that consume selenium-rich forage in seleniferous regions are associated with hoof deformities, reduced fertility, and weight loss. Chronic intakes above the tolerable upper intake level of 400µg/day will likely pose risks [34].

Conclusions

Selenium is used as a nutrient and as a poison. Thus, the crucial aspect is to maintain its balance in the diet in order to avoid the deleterious effects. The enormous role of selenium to health, from antioxidants to thyroid function, illustrates its significance in nutrition, but deficiency and excess represent serious health risks, showing the necessity

Table 2 Selenium deficiency and toxicity symptoms in animals.

Species	Deficiency symptoms	Toxicity symptoms	Reference
Cattle	White muscle disease, infertility	Hoof deformities, weight loss	[29]
Poultry	Exudative diathesis, reduced growth	Decreased egg production	[28]
Humans	Keshan disease, Kashin-Beck disease	Selenosis: hair/nail loss, garlic breath	[27, 28)
Sheep	Poor growth, muscular dystrophy	Emaciation, lameness	[31]
Pigs	White muscle disease, reproductive failure	Alopecia, hoof separation	[27]

for exact recommendations in dietary intake based on regional and individual needs. Future research should focus on closing gaps in understanding selenium molecular mechanisms, improving bioavailability where needed in deficient regions, and the development of innovative forms of bio-fortification approaches for addressing global nutritional challenges. Such efforts will enhance the capacity to benefit from selenium in an appropriate manner while ameliorating its risks by further contributing to better health across species.

Conflict of interest

The authors declare no conflict of interest.

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