

Effects of Selenium Mineral on Health

Rabia Melda Karaağaç, Çağla Pınarlı and Fatih Tarlak*

Faculty of Health Sciences, Department of Nutrition and Dietetics, İstanbul Gedik University, İstanbul, Turkey

***Corresponding Author:** Fatih Tarlak, Faculty of Health Sciences, Department of Nutrition and Dietetics, İstanbul, İstanbul Gedik University, Turkey.

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Abstract

Selenium (Se), discovered by Brezilius and Gahn in 1817, is a trace element that is known as a cofactor of many enzymes in our body and has an antioxidant function. It is found in nature and organisms in organic and/or inorganic forms. Se is an important component of selenoproteins that play a role in many biological functions such as antioxidant defense, formation of thyroid hormones, DNA synthesis, fertility, and reproduction. The most important sources of Se are brazil nuts, offal, and fish. Adults should consume 55 micrograms per day. Se toxicity can occur if Se has taken into the body more than necessary. The most common clinical symptoms of Se toxicity are hair loss and split nails. Adults should consume no more than 400 micrograms of Se per day. Se shortage is more common in areas where the soil contains little Se. As a result of deficiency, Keshan and Kashin-Beck diseases occur in people. Se is a mineral that has been linked to chronic illnesses like cancer and cardiovascular disease. There is also a relationship between selenium and gut microbiota. This review aims to discuss the Se mineral from a clinical and metabolic point of view.

Keywords: Se; Mineral; Antioxidant; Selenoprotein; Thyroid; Cardiovascular Disease; Cancer; Enzymes

Introduction

Selenium (Se), which means 'moon goddess' in ancient Greek, is a trace element that is known as a cofactor of many enzymes in our body and has an antioxidant function [1]. Se was discovered by Brezilius and Gahn in 1817 [2]. Brezilius, a Swedish chemist, searched for a toxic substance that caused workers in a chemical factory to become ill and discovered Se found in the mud of sulfuric acid containers [3]. It was understood that it is essential for mammals by Schwartz and Foltz in 1957 [2]. Schwartz was concerned about liver necrosis in research mice fed a *Torula* yeast-based protein diet. When using baker's yeast (*Saccharomyces*) instead of *Torula* yeast, this problem has disappeared. Because *Torula* yeast is poor in Se, while baker's yeast is rich in Se. Thus, Schwarz described the first "Se-sensitive" disease, which soon led to the recognition of Se as an essential trace mineral nutrient.

Se is found in nature and in organisms in organic and/or inorganic forms. Its main organic forms are selenomethionine (Semet)

and selenocysteine (Secys). Inorganic forms are selenite, selenide, selenate, and Se. Se is a metalloid of the same family as oxygen (O) and sulfur (S). It is a semi-metal. It is stable and does not oxidize at normal temperatures. When burned, it produces a blue flame and Se dioxide [4]. Se is similar to sulfur, which is in the same group, in terms of physical and chemical properties [5].

Material and Methods

In this study, "selenium" and "antioxidant," "immune system," "health," "diseases," "reproduction," "microbiota," "cancer," "cardiovascular disease," "thyroid" containing words were reviewed in Pubmed database to evaluate the effects of Se mineral on health. Se values were obtained from TURKOMP (National Food Composition Database) and USDA (U.S. Department of Agriculture) databases. The study is the traditional review.

Functions of Se

Se is a basic component of selenoproteins that play an important role in many biological functions such as antioxidant defense,

formation of thyroid hormones, DNA synthesis, fertility, and reproduction [6].

Antioxidant function

The most important role of Se is its antioxidant effect [7]. The antioxidant enzyme ‘glutathione peroxidase’ requires Se to function properly [8]. Glutathione peroxidases belong to a family of antioxidant enzymes. Their main function in the body is to neutralize hydrogen peroxide and organic hydroperoxides in the intracellular and extracellular parts [9]. Thus, it prevents oxidative stress from damaging the cell and cell membrane. For this task, Se plays a role together with vitamin E [8].

Thyroid function

Iodothyronine deiodinase, a selenoprotein, is responsible for activating thyroid hormones [7]. Iodothyronine deiodinase produces and regulates the active form, T3, from T4 [10].

Immune function

Se is found in excess in the spleen, liver, and lymph nodes. Se has been shown to stimulate cytotoxic T and NK cells as well as antibody formation and the activity of helper T cells. It also plays a role in stimulating phagocytic cell migration and phagocytosis [11,12]. In addition, regard to Se, Se, and some metabolites of selenoproteins such as GPX1 and TR1 has been shown to be involved in immune and inflammatory responses, but the mechanisms by which the beneficial effect is not yet fully understood.

DNA synthesis function

Thioredoxin reductase (selenoprotein) has role in nucleotide reduction in DNA synthesis, antioxidant system regeneration, and maintaining the intracellular redox state, which is necessary for cell viability and proliferation. It regulates gene expression as well as the redox control of transcription factor binding to DNA [13].

Reproductive function

Se plays an important role in fertility, embryonic implantation, placental retention, testosterone and sperm synthesis, and sperm motility [4]. Se is an essential element that plays a role in normal gonadal development, gametogenesis and fertilization. Molecular studies show that gonads actively uptake and store Se, and many of these are incorporated into glutathione peroxidase enzymes [14].

Se sources

According to TURKOMP and USDA data, Se ratios of some foods are shown in Table 1 and Table 2 [15,16].

Food	Average Se (mcg)
Edible offal, veal kidney	155.3
Lentils, green, dry	102.0
Eggs, quail, whole	54.9
Edible offal, veal liver	53.3
Egg, chicken, yellow	53.0
Mussels, Mediterranean, black mussels	48.9
Sardines, canned, in sunflower oil	47.5
Anchovy, canned, in sunflower oil	46.6
Tuna, canned, in sunflower oil	43.9
Sesame seeds, dry	40.8

Table 1: Se Ratios in 100 grams of Some Foods According to TURKOMP.

Food	Micrograms (mcg) per serving	Percent DV (daily value)
Brazil nuts, 1 ounce, 6-8 pieces	544	989
Yellowfin tuna, cooked, dry heat, 3 ounces	92	167
Flounder, cooked, dry heat, 3 ounces	47	85
Sardines, canned in oil, 3 ounces	45	82
Ham, roasted, 3 ounces	42	76
Shrimp, roasted, 3 ounces	40	73
Pasta, enriched, cooked, 1 cup	37	67
Beef steak, roasted, 3 ounces	33	60
Turkey, boneless, roasted, 3 ounces	31	56
Beef liver, fried, 3 ounces	28	51

Table 2: Se Rates of Some Foods According to USDA.

Se requirement

The Recommended Daily Allowance (RDA) of Se is indicated in Table 3 [17].

Age	Male	Female	Pregnancy	Lactation
0 - 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		

Table 3: Recommended Daily Allowance (RDA) of Se.
*Adequate Intake (AI).

Se toxicity

Although Se toxicity is much less common than Se deficiency, it can affect individuals through overdose, accidental or deliberate (suicidal) ingestion of very high doses, or ingestion of high levels with food [18]. For adults, the tolerated upper intake level (UL) is 400 g (5.1 mol) per day [17].

The most common clinical manifestations of chronically high Se intake or selenosis are hair loss and brittle nails. Skin and nervous system lesions, nausea, diarrhea, skin rashes, discolored teeth, weariness, irritability, and nervous system abnormalities are some of the other symptoms [17].

Se deficiency

Se enters the food chain from plants through the soil. Therefore, significant Se deficiency has been detected in volcanic regions of the world due to soils with low Se content [19].

Se deficiency has been recorded in chronic patients who have been receiving total parenteral nutrition (TPN) for an extended period time without Se supplementation. In these patients, muscle weakness, atrophy, and cardiomyopathy (inflammation and damage to the heart muscle) have been seen. Today, TPN solutions are routinely supplemented with Se [20].

Se insufficiency causes Keshan disease, a deadly cardiomyopathy that initially appeared in young women and children in a Se-deficient region of China. The abrupt development of heart failure characterizes the acute stage of the disease. The chronic variant causes mild to severe heart enlargement, as well as variable degrees of heart failure [21]. Kashin-Beck disease, another condition cau-

sed by Se deficiency, is characterized by shortening of the fingers and toes and changes resembling rheumatoid arthritis [22].

Relationship of Se to diseases

Se and cancer

Due to its effects on DNA repair, apoptosis, the endocrine and immunological systems, and other mechanisms, including antioxidant capabilities, Se may have a role in cancer prevention [23].

Se appears to have an inverse connection with the risk of colorectal, prostate, lung, bladder, cutaneous, esophageal, and stomach cancers, according to epidemiological studies [24]. In a Cochrane review of Se and cancer prevention studies, males with the highest Se intake had a 31% lower cancer risk, a 45% lower cancer mortality risk, a 33% lower risk of bladder cancer, and a 22% lower risk of prostate cancer than those with the lowest Se consumption [24].

Se and cardiovascular diseases

Selenoproteins help prevent oxidative modification of lipids, reduce inflammation and prevent platelet aggregation [10].

Epidemiological studies on Se’s effect in cardiovascular disease has yielded mixed results. In certain observational studies, blood Se concentrations were found to be inversely related to the risk of hypertension or coronary heart disease. People with low Se concentrations had a greater risk of coronary heart disease, according to a meta-analysis of 25 observational studies [25]. However, some observational studies have revealed no statistically significant associations between Se levels and the risk of heart disease or cardiac mortality, or that greater Se levels are linked to an increased risk of cardiovascular disease [26,27].

Se and thyroid

The thyroid gland contains a higher concentration of Se than other organs in the body, and Se, like iodine, plays a crucial role in thyroid hormone synthesis and metabolism. A strong inverse connection was identified between serum Se levels and thyroid volume in women in a cross-sectional research conducted in Denmark in 805 people with mild iodine insufficiency [28].

Se-drug interaction

Inorganic chemotherapeutic agent cisplatin is used to treat malignancies of the ovary, bladder, lung, and other organs. Cisplatin may lower Se levels in hair and serum, although it’s unclear if this has a clinically meaningful effect [29].

Se and gut microbiota

The human digestive system contains about 100 trillion microorganisms. Various microorganisms (bacteria, viruses, fungi, and protozoa) live in the digestive tract. This ecosystem is called the microbiota [30]. Bacterial cells have an uneven distribution throughout the gastrointestinal tract, with more than 50 species of bacterial phyla. In almost all individuals, only Bacteroidetes and Firmicutes are preserved [31].

Human microbial colonization occurs at birth. Initially, the microbiota is similar to the mother's vaginal microbiota. Intestinal colonization during birth and breastfeeding is believed to be essential to define the composition of the gut microbiota in adulthood. Determination of microbiota composition is also affected by various external and internal factors related to the host [32].

The microbiome is a structure that can encode more than three million genes. It has various functions, especially the production of some vitamins and bioactive compounds, the synthesis of some amino acids, and the metabolism of indigestible carbohydrates. At the same time, neural, hormonal, and immunological signaling takes place along the gut-brain axis. While the gut microbiota is responsible for the absorption of nutrients, it acts as an epithelial barrier for pathogens [30]. In this sense, problems in the intestinal ecosystem or its two-way interaction with the brain are associated with the risk of many diseases. In this context, strategies have been developed to manipulate the microbiome to prevent and/or reverse unhealthy conditions [33].

Recently, studies have been carried out on anaerobic classes of Deltaproteobacteria and Clostridia, which are organisms rich in selenoproteins. Especially *Syntrophobacter fumaroxidans* comes to the fore at this point [34].

Traces of Se and related key genes have been evaluated in more than 2,300 bacterial and archaeal genomes. A phylogenetic and genomic mosaic pattern has been described among organisms that use Se in different ways. This profile suggests novel genes whose encoded proteins are involved in Se metabolism and homeostasis in prokaryotes [35].

Evolutionary trends in the use of Se and selenoproteins show more than five hundred bacterial genomes. Most of the Se comes from the host. More than half of bacteria do not use Se. This su-

ggests that the ability to use Se is lost over time. Environmental factors and Se utilization have been investigated, showing that anaerobic conditions can significantly support the use of the Se-cofactor property and lead to the evolution of new selenoprotein genes [36]. Thus, since the oxygen level in the colon, where Se absorption is highest, is low and the optimum temperature varies between 25°C and 30°C, the human gut is thought to be a suitable ecosystem for the use of Se by prokaryotes [37].

According to another research, dietary Se affects the composition of the intestinal microflora and colonization of the gastrointestinal tract. This affects the individual's Se status and selenoproteome expression [38].

Conclusion and Recommendations

Se is a very important trace element known as a cofactor of many enzymes in our body and has an antioxidant function. Adults should consume 55 micrograms per day. The most important sources are brazil nuts, offal and fish. Se toxicity and deficiency are both very dangerous for individuals and have alarming clinical consequences. Deficiency is more common in regions with insufficient soils in terms of Se. As a result, people must consume enough Se in their regular diet. There is also a relationship between gut microbiota and Se levels. Adequate Se levels positively affect the microbiota. More studies are needed to clearly define the relationship between Se and gut microbiota.

Conflict of Interest

There is no conflict of interest between the authors.

Bibliography

1. Şimşir IY and Özgen AG. "Tiroid ve selenyum". *Turkish Journal of Emergency Medicine* 14 (2010): 76-79.
2. Orak E., et al. "Selenyum ve kalp hastalıkları ile ilişkisi". *Türk Kardiyoloji Derneği Arşivi* 28 (2000): 230-238.
3. Oldfield JE. "A brief history of Se research: From alkali disease to prostate cancer (from poison to prevention)". *Journal of Animal Science* 11.1 (2001): 1-4.
4. Mehdi Y., et al. "Se in the environment, metabolism and involvement in body functions". *Molecules (Basel, Switzerland)* 18.3 (2013): 3292-3311.
5. Hıncal F and Ataçeri N. "Selenyum'un insan sağlığındaki rolü" *FABAD Farm. Bil. Der* 14 (1989): 23-38.

6. Suttle NF. "Mineral Nutrition of Livestock. 4th ed. MPG Books Group: London, UK (2010): 565.
7. Tóth RJ, Csapó J. "The role of Se in nutrition - A review". *Acta Universitatis Sapientiae, Alimentaria* 11.1 (2018): 128-144.
8. Aksoy M. "Beslenme Biyokimyası (3. Baskı)". *Ankara: Hatiboğlu Yayıncılık* (2011): 546-560.
9. Brigelius-Flohe R and Maiorino M. "Glutathione peroxidases". *Biochimica Biophysica Acta* (2012).
10. Rayman MP. "The importance of Se to human health". *The Lancet* 356.9225 (2000): 233-241.
11. Burk RF. "Se in Biology and Human Health". Springer-Verlag New York Inc.: New York, NY, USA (1994): 221.
12. Finch JM and Turner RJ. "Effects of Se and vitamin e on the immune responses of domestic animals". *Research in Veterinary Science* 60 (1996): 97-106.
13. Allan CB., et al. "Responsiveness of selenoproteins to dietary Se". *Annual Review of Nutrition* 19 (1999): 1-16.
14. Mirone M., et al. "Se and reproductive function: A systematic review". *Journal of Endocrinological Investigation* 36.10 (2013): 28-36.
15. http://www.turkomp.gov.tr/component_result-30
16. U.S. Department of Agriculture. "Agricultural Research Service". *Food Data Central* (2019).
17. Institute of Medicine, Food and Nutrition Board. "Dietary Reference Intakes: Vitamin C, Vitamin E, Se, and Carotenoids". National Academy Press, Washington, DC (2010).
18. Fairweather-Tait., et al. "Se in human health and disease". *Antioxidants and Redox Signaling* 14.7 (2011): 1337-1383.
19. Reilly C. "Se in food and health". London: Blackie Academic and Professional (1996).
20. Cooper A., et al. "Nutritional management of infants and children with specific diseases and other conditions". *Modern Nutrition in Health and Disease*. 11th ed. Baltimore: Lippincott Williams and Wilkins (2012): 988-1005.
21. Sunde RA Se. "Modern Nutrition in Health and Disease". 11th ed. Baltimore: Lippincott Williams and Wilkins (2014): 265-276.
22. Sur Ü., et al. "Selenyum, selenoproteinler ve hashimato tiroiditi FABAD". *Journal of Pharmaceutical Sciences* 45.1 (2020): 45-64.
23. <https://ods.od.nih.gov/factsheets/Se-HealthProfessional/#en10>
24. Dennert G., et al. "Se for preventing cancer". *The Cochrane Database of Systematic Reviews* 5 (2011): CD005195.
25. Flores-Mateo G., et al. "Se and coronary heart disease: a meta-analysis". *The American Journal of Clinical Nutrition* 84.4 (2006): 762-773.
26. Xun P., et al. "Longitudinal association between toenail Se levels and measures of subclinical atherosclerosis: the CARDIA trace element study". *Atherosclerosis* 210.2 (2010): 662-667.
27. Bleys J., et al. "Serum Se and peripheral arterial disease: results from the national health and nutrition examination survey, 2003-2004". *American Journal of Epidemiology* 169.8 (2009): 996-1003.
28. Rasmussen LB., et al. "Se status, thyroid volume, and multiple nodule formation in an area with mild iodine deficiency". *European Journal of Endocrinology* 164.4 (2011): 585-590.
29. Sieja K and Talerczyk M. "Se as an element in the treatment of ovarian cancer in women receiving chemotherapy". *Gynecologic Oncology* 93.2 (2004): 320-327.
30. Bull MJ and Plummer NT. "Part 1: The Human Gut Microbiome in Health and Disease". *Journal of Integrative Medicine (Encinitas)* 13.6 (2014): 17-22.
31. Lozupone CA., et al. "Diversity, stability and resilience of the human gut microbiota". *Nature* 489.7415 (2012): 220-230.
32. Milani C., et al. "The First Microbial Colonizers of the Human Gut: Composition, Activities, and Health Implications of the Infant Gut Microbiota". *Microbiology and Molecular Biology Reviews* 81.4 (2017): e00036-37.
33. Fassarella M., et al. "Gut microbiome stability and resilience: elucidating the response to perturbations in order to modulate gut health". *Gut* 70.3 (2021): 595-605.
34. Zhang Y and Gladyshev VN. "Comparative genomics of trace elements: emerging dynamic view of trace element utilization and function". *Chemical Reviews* 109.10 (2009): 4828-4861.
35. Lin J., et al. "Comparative genomics reveals new candidate genes involved in Se metabolism in prokaryotes". *Genome Biology and Evolution* 7.3 (2015): 664-676.
36. Zhang J., et al. "Dysbiosis of the gut microbiome is associated with thyroid cancer and thyroid nodules and correlated with clinical index of thyroid function". *Endocrine* 34 (2009): 564-574.

37. Koziolok M., *et al.* "Investigation of pH and Temperature Profiles in the GI Tract of Fasted Human Subjects Using the Intellicap (®) System". *Journal of Pharmaceutical Sciences* 104.9 (2015): 2855-2863.
38. Kasaikina MV, *et al.* "Dietary Se affects host selenoproteome expression by influencing the gut microbiota". *The FASEB Journal* 25.7 (2011): 2492-2499.

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