Neha D. Shah, MPH, RD, CNSC, CHES, Series Editor
Elizabeth Wall, MS, RDN-AP, CNSC, Series Editor

Selenium: A Micronutrient with a Macro-Impact


Sebastian Dobrow


Rangesh Modi

Selenium, a naturally occurring trace element, serves many bodily functions through its role in a family of proteins called selenoproteins. The average daily intake in the United States is about 116 mcg/day, well above the recommended dietary reference intake of 55 mcg/day for adults. Thus, deficiency is rare among Americans. Deficiency can occur in areas of low environmental selenium or in disease states that impair intestinal absorption, such as short bowel syndrome or inflammatory bowel disease, and affects the cardiovascular, respiratory, immune/hematologic, gastrointestinal, reproductive, central nervous, and neuromusculoskeletal systems. This review will briefly summarize the general physiologic roles of selenium, emphasizing deficiency due to various gastrointestinal disorders, monitoring, and replenishment. A summary of recent investigations into selenium levels of those on parenteral nutrition is also provided.

INTRODUCTION

Selenium is a non-metallic trace element, named for Selene, the Greek goddess of the moon.[1] It is found naturally in foods and is widely available as a dietary supplement. In the United States (US), the most selenium-deficient areas are the Northwest, the Great Lakes region, and the Northeast and Atlantic coastal areas, particularly Florida.[2] Selenium is an essential component in a family of proteins called selenoproteins. There are 25 known human selenoproteins, and each helps carry out diverse functions, namely redox reactions. A few of the most well-described selenoproteins include glutathione peroxidases, thyroid hormone deiodinases, and thioredoxin reductases.[3,4]

Selenoproteins function largely as antioxidants and have multiple physiologic roles. These roles include the protection of cells and DNA; supporting thyroid function, modulating inflammation, and regulating the nervous and immune systems. Selenoproteins also promote muscle (heart and skeletal), bone, and cartilage health, support male reproductive function and testosterone synthesis,

Sebastian Dobrow, MD, MPH Rangesh Modi, MD
The University of Chicago Medicine, Chicago, IL

assist in regulating lipid metabolism, and may even play a role in cancer prevention.[4-7]

Selenoproteins Vital to Human Health

Glutathione peroxidases are a family of eight (thus far discovered) selenoproteins, abbreviated GPx1, GPx2, and so on. Each serves slightly different biological roles; however, they function primarily as antioxidants – reducing hydrogen peroxide and lipid peroxides to protect cells from oxidative stress and apoptosis.[8] Genetic differences in genes coding these proteins have been associated with cardiovascular disorders, including coronary and peripheral vascular disease, hypertension, aneurysm, and stroke, as well as Keshan and Kashin-Beck disease, which are described below. Malignancies such as colorectal cancer, thyroid, and breast cancer have also been associated with variations in glutathione peroxidase genetics.[9]

Iodothyronine deiodinases (Dio) are selenoproteins responsible for proper thyroid function. Dio proteins are responsible for converting thyroid hormone from its inactive T4 to its active T3 form and between inactive forms (T4 to reverse T3; and T3 and reverse T3 to T2).[9] T2 is the only form of thyroid hormone available as an over-the-counter supplement to stimulate metabolism.[10] T2 supplementation is likely to cause disruption of normal thyroid hormone regulation before desired metabolic effects and is not recommended.[10]

Thioredoxin reductases (TrxR) are another family of redox selenoproteins with antioxidant functions that are vital to DNA synthesis. TrxR enzymes are crucial to proper cardiac cell function and appear to play a role in disease states such as familial amyotrophic lateral sclerosis (ALS, or Lou Gehrig's Disease) and gastrointestinal cancers, among others.[9]

Other selenoproteins, including Selenoproteins P, S, N, and 15kDa selenoprotein, also have human health implications moderating the risk and progression of various cancers, fertility, and cardio- and neurovascular disease.[9]

Selenium Homeostasis - Uptake, Metabolism, and Excretion

Uptake and Metabolism

Selenium, found largely in soil, is incorporated into humans through the intake of both dietary plants and animals. Plants, especially wheat and grains, are the predominant source of selenium for humans; however, red meat, poultry, and fish represent other dietary sources.[1] Selenium uptake occurs mainly in the duodenum, proximal jejunum, and cecum.[1,4,11] Absorption occurs via various mechanisms involving both organic and inorganic forms of selenium. The organic forms, bound to amino acids such as selenomethionine (predominantly) and selenocysteine (less commonly), are more readily absorbed than inorganic forms and likely occur via active transport similar to their corresponding amino acids.[12] Several inorganic forms, predominantly selenite and selenate, are less readily bioavailable when consumed.[3,8] Selenate absorption occurs in an energy-dependent fashion, whereas selenite occurs by passive diffusion.[12] Several elements impair selenium absorption through various mechanisms, including iron (specifically in the ferric state), calcium, mercury (which may reduce selenium uptake from fish sources), sulfur, lead, and arsenic.[4]

Once absorbed, the liver takes up selenium via the portal system, where much of it is incorporated into the 10 selenocysteine residues present on the hepatically synthesized selenium transporter, selenoprotein P (SELENOP). SELENOP is secreted into plasma for delivery to the tissues.[12] Bound selenium is then taken up by receptors on target tissues for local use. It has been shown that the brain and testes uptake SELENOP via the apolipoprotein E receptor-2 (ApoER2), a low-density lipoprotein (LDL) receptor. ApoER2 is present in several other tissue types and thus may be involved in SELENOP uptake. Lipoprotein receptor megalin appears responsible for renal reuptake of SELENOP from glomerular filtrate, preventing excess selenium loss in urine.[12]

Selenium circulates in plasma bound to other liver-synthesized proteins in lesser quantities, such as albumin, alpha and beta globulins, and cholesterols, namely LDL.[4,13] Selenium storage occurs mainly in the form of selenomethionine and takes place largely in the liver and muscle (about 30% each), kidney (15%), and plasma (10%).[4]

Excretion

Selenium excretion occurs primarily through urine (with decreased excretion in low glomerular filtration states) and feces; however, losses can

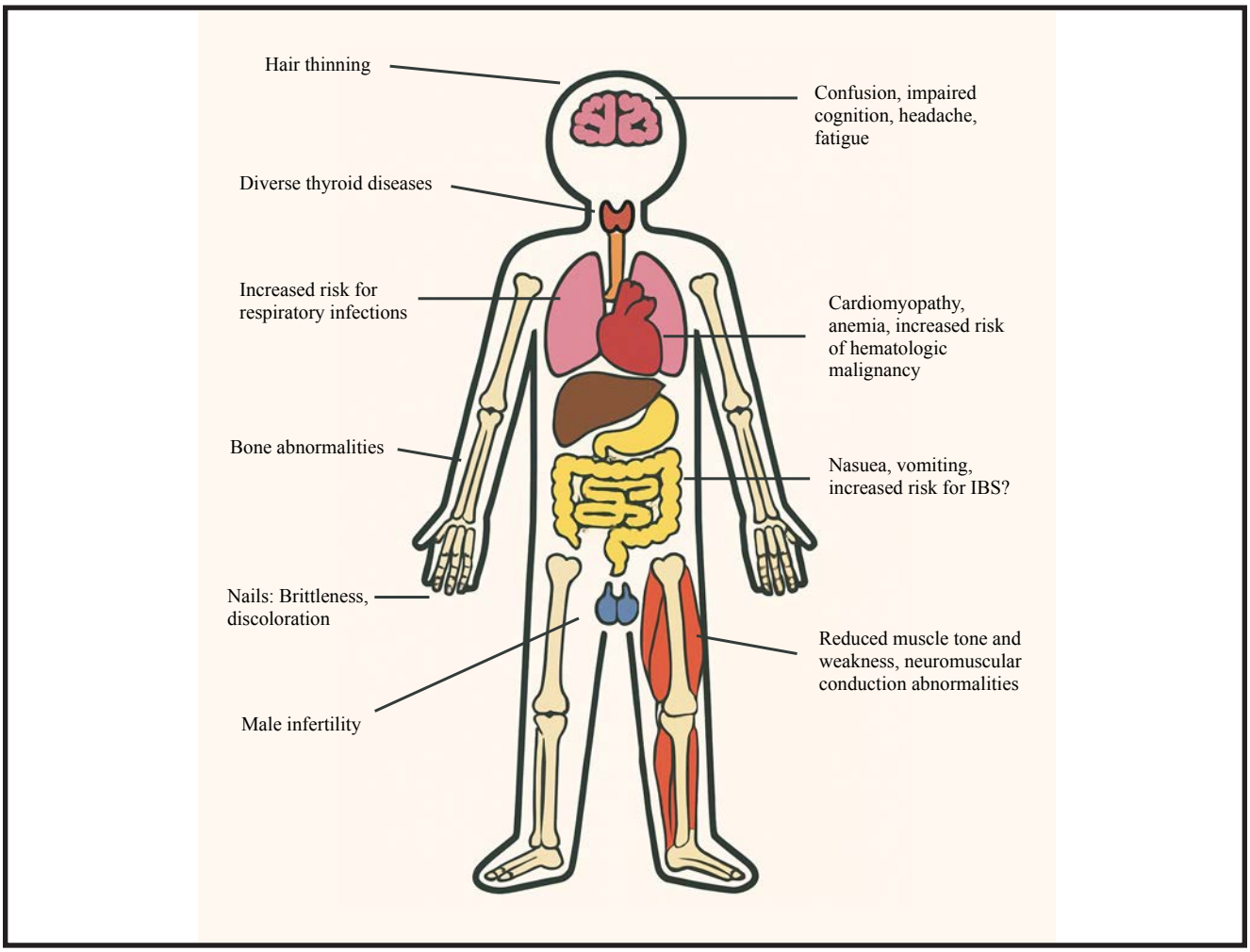

Figure 1. Effects of Selenium Deficiency by Body Part or System

occur through expired air in states of excess consumption.[1,4] Breast milk contains relatively low amounts; however, levels rise with increased selenium intake.[4]

Selenium Deficiency

Signs and Symptoms

According to data from the United States National Health and Nutrition Examination Survey (NHANES) 2017-2022, the average daily dietary selenium intake is 116 mcg/day, with men taking in slightly more than women.[14] Assuming adequate absorption, the average American is in a safe range.

Recent analysis of the United Kingdom Biobank data revealed a significant correlation between low dietary selenium levels and increased irritable bowel syndrome incidence.[15] Mouse model studies have shown that a low selenium diet causes disturbances in the gut microbiota, characterized by an increase in *Faecalibaculum* and *Helicobacter* and a decrease in *Bifidobacterium* and *Akkermansia*.[15] Lower selenium levels have also been linked to increased risk for gastric and esophageal cancers.[16]

As the biological roles of selenoproteins vary, so do the features of selenium deficiency. These are illustrated in Figure 1 and include:[4,7,17,18]

- Fatigue
- Nausea, vomiting
- Headache, confusion
- Decreased muscle tone and neuromuscular conduction issues
- Hair thinning
- Nail changes (especially leukonychia)

- Anemia
- Male infertility
- Necrotizing cardiomyopathy
- Increased susceptibility to infections including, bacterial infections, progression from HIV to AIDS, COVID-19, and many others
- Increased cancer susceptibility

Decreased Intake

As selenium content in foods depends largely on soil levels, individuals living in regions with low soil selenium, especially those with volcanic soil, such as regions of Europe, Russia, and China, are at risk for selenium deficiency.[4] In the US, the most selenium-deficient areas are the Northwest, Northeast, Atlantic coastal area, Florida, and the Great Lakes. Obesity and poverty may be risk factors for low selenium levels, possibly owing to poor quality of nutritional intake.[1,5] Chronic insufficient dietary intake can result in major endemic syndromes:

- Keshan disease - cardiomyopathy typically found in the Keshan region of rural China and Tibet, where the soil is volcanic and selenium deficient. Selenium supplementation has proven effective in protecting residents against the disease.[1]
- Kashin-Beck disease (KBD) - osteochondropathy characterized by short stature, joint deformities, and osteoarthritic changes endemic to parts of eastern Russia (Siberia), China, Tibet, and North Korea. Selenium deficiency is considered one of the major risk factors for KBD; however, fungal toxins, genetic susceptibilities, and many other factors are thought to play roles. A Chinese interventional study found that KBD rates and other bone cartilage measures improved with selenium supplementation; however, it did not prevent the disease.[7]

Impaired Absorption and Selenium Wasting

Patients with altered intestinal anatomy, including short-bowel syndrome and post-bariatric surgery, are at particular risk for selenium deficiency. Restrictive procedures such as a sleeve gastrectomy and gastric banding may reduce intake to the point of causing deficiency, while combination restrictive-malabsorptive procedures such as Roux-en-Y gastric bypass and duodenal switch result in an even higher risk of deficiency due to reduced absorptive capacity.[11]

Other malabsorptive states, including inflammatory bowel disease (especially Crohn's disease and celiac disease), may lead to selenium deficiency, with some studies suggesting potential use of selenium levels as a biomarker for disease activity.[19-21] Those on renal replacement therapy are also at risk for low selenium levels due to excess removal during dialysis.[22] Importantly, chyle contains large amounts of selenium; chylous loss due to various conditions can lead to deficiency.[23]

Table 1 summarizes studies looking at selenium deficiency in patients with chronic intestinal failure (CIF) on home parenteral nutrition (HPN).[19–22] Although deficiency is rare overall in healthy adults eating a balanced diet, patients who receive the majority of their nutrition from parenteral nutrition (PN) are at the highest risk for developing selenium deficiency, irrespective of etiology. This is true despite standard trace element supplementation.

Excess Selenium

Despite its essential role in human health, chronically high selenium levels can also be toxic. Selenosis is a syndrome that can result from brittle or loss of hair and nails, GI issues, rashes, garlic-smelling breath, and nervous system dysfunction. Selenosis has been reported in China in cases of those consuming >850 mcg/day. This has influenced the recommendations for the upper tolerated range of 400 mcg/day set by the US Institute of Medicine and 300 mcg/day by the World Health Organization.[1] Patients with chronic kidney disease are particularly susceptible to selenium toxicity.

Measuring Selenium Levels

Plasma selenium levels are often measured and represent short-term status, while red blood cell levels can be used to determine longer-term status. In inflammatory states with an elevated erythrocyte sedimentation rate or C-reactive protein (CRP), interpretating plasma selenium levels becomes difficult due to the down-regulation of carrier and storage proteins. According to the European Society

for Clinical Nutrition and Metabolism (ESPEN), depending on the severity of the inflammatory response, a "correction" of the value is required: CRP concentrations of 10-40, 41-80, and greater than 80 mg/L would be expected to produce falls in plasma selenium of approximately 15-25%, 35%, and 50% respectively. Normal plasma selenium levels are 110-165 mcg/L in the US, though reference ranges vary between laboratories.

Urinary selenium concentration can also be

Table 1. Studies of Selenium Deficiency in Patients with CIF on HPN[24-27]

Study	Population	Study Details	Key findings
Culkin et al.[24]	• 93 adult patients on HPN for CIF • Mean age (range): 54 years (21-81 years)	• Retrospective cohort study (25 months long) • Etiology for HPN: IBD (30%), mesenteric ischemia (25%), motility disorder (24%), surgical complication (6%), others (14%) • Patients on stable micronutrient dosing for at least 6 months • Excluded CRP >15 mg/L or changes in micronutrients within 6 months of study	• 13% patients had selenium deficiency despite standard trace elements • Highest risk in patients with surgical complications
Uzzan et al.[25]	• 73 patients on HPN for CIF • Mean age (range): 49 years (18-86 years)	• Prospective cohort study • Etiology: SBS (63%), dysmotility (23%), others (14%) • Included patients needing HPN at least 8 times/month, stable formula for at least 1 month and serum trace element dosage available • Excluded if no available trace element dosage or unstable HPN formula • Median follow-up was 19.6 months	• 21.9% had selenium deficiency despite standard trace minerals • No difference in deficiency risk based on anatomy type • Low serum selenium associated with higher infection risk (HR 2.65, 95% CI [1.01-6.97])
Rannem et al.[26]	• 165 patients (CD, ulcerative colitis, or other GI illnesses) • 27 (16%) HPN patients • Mean age (range): 43 years (15-75 years)	• Case control study • Cases included CD (52%), UC (24%), ischemia or obstruction (9%), others (15%) • Controls were 50 healthy subjects	• 85% of PN patients had reduced selenium vs 20% on EN • 26% of CD patients had reduced selenium • Strongest predictors were stool mass, vitamin B12 absorption, and small-bowel resection length
Fleming et al.[27]	• 12 patients with IF pre-PN • 26 on HPN (mean 29 months) • Age group not mentioned	• Case control study • Cases included CD (37%), non-specific SBS (18%), ischemia (10%), others (35%) • Controls were 30 CD patients not on HPN and 27 healthy subjects	• 92% of pre-PN patients had low selenium levels (42 ng/mL) vs. controls (88 ng/mL) and Crohn's controls (76 ng/ml) • 85% of patients on HPN for 2–109 months had low selenium (mean 38.4 ng/mL)

SBS - short bowel syndrome CD - Crohn's disease UC- ulcerative colitis EN- enteral nutrition CD-Crohn's disease

Table 2. Selenium Content of Selected Foods[30]

Food	Micrograms per serving	Percent DV (%)
Brazil nuts 1 ounce (6-8 nuts)	544	989
Tuna, yellowfin, cooked 3 ounces	92	167
Shrimp, cooked 3 ounces	42	76
Pork chop, bone-in, broiled 3 ounces	37	67
Beef steak, bottom round, roasted 3 ounces	37	67
Spaghetti, cooked 1 cup	33	60
Beef liver, pan fried 3 ounces	28	51
Cottage cheese, 1% milkfat 1 cup	20	36
Egg hard boiled 1 large	15	27
Oatmeal, cooked with 1 cup water	13	24
Baked beans 1 cup	13	24
Mushrooms, portabella, grilled ½ cup	13	24
Brown rice, cooked 1 cup	12	22

measured, and usually correlates with daily intake.[28] Concentration in toenails can be used to measure longer-term storage, as can hair; however, selenium content in many shampoos often limits accuracy.[1,28] Activity levels of certain selenoproteins such as glutathione peroxidase and SELENOP may also be used; however, the availability of such assays may be limited. In general, ascertaining functional selenium status may be difficult given the numerous forms and functions selenium takes on.[28]

Repletion and Supplementation

Oral and Enteral Support

The recommended daily intake of selenium for adult men and women in the US (over 4 years) is 55 micrograms (mcg).[29] Pregnant women should increase their intake to about 60 mcg/day and to 70 mcg/day if lactating or breastfeeding.[29] Table 2 summarizes some foods rich in selenium and percent daily values (DV).[30]

For people living in geographic regions with selenium deficient soil and water, daily supplementation of 60-100 mcg of selenium should be sufficient to prevent deficiency. Generally, over-the-counter (OTC) selenium supplements are selenium-amino acid compounds (e.g., L-Selenomethionine or L-Selenocysteine). The supplements are available in tablet or capsule form and can be swallowed whole, crushed, or opened and sprinkled over food or liquids.

Patients with selenium deficiency from malabsorption or low dietary intake can try oral (or via enteral tube) repletion. Most OTC selenium supplements provide 200 mcg/capsule or tablet, though multi-mineral supplements range from 50-200 mcg selenium/capsule or tablet. The recommended safe, upper limit for selenium is 300-400 mcg/day for adults.[1]

Parenteral Dosing

In the US, selenium is available in the IV form as selenious acid injection in 6 mcg/mL or 60 mcg/mL. The neonatal/pediatric trace element mixture used for body weight under 10 kg is Multrys™, and each vial contains 6 mcg of selenium. For children with weights above 10 kg and adults, Tralement™ is used, which has 60 mcg of selenium in a 1 mL vial.[31]

Recommended selenium dosing in PN per the American Society for Parenteral and Enteral Nutrition (ASPEN) is:[32]

- 2 mcg/kg for babies under 10 kg
- 2 mcg/kg for children with weight 10-40 kg with a maximum dose of 100 mcg
- 40-60 mcg for adolescents with weight >40 kg
- 60-100 mcg for adults

No consensus exists for selenium repletion in states of deficiency for patients requiring enteral nutrition (EN) formulas or PN.

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According to ESPEN:[22]

- The typical adult EN regimen containing 1500 kcal should include 50-150 mcg of selenium/day. (GRADE B*)
- The typical PN regimen should include 60-100 mcg of selenium for adults. Doses of 100 mcg selenium/day, whether IV or oral (depending on absorption capacity), are usually sufficient to replenish selenium levels within 2 weeks. (GRADE B*)
- Post PN initiation, selenium must be measured every 3 to 6 months along with CRP levels. (GRADE B*)
- Patients with selenium levels <60 mcg/L and CRP<20 mg/L should also get additional selenium supplementation promptly; the route depends on intestinal absorption capacity, and IV is a more reliable route for rapid repletion. (No GRADE^)
- Patients with selenium levels of <32 mcg/L require prompt supplementation with 100 mcg selenium/day. (No GRADE^)
- Patients with selenium levels <30 mcg/L can be treated with high dose IV selenium 400 mcg/day for 7-10 days and then recheck levels. (No GRADE^)
- No clear guidelines exist on rechecking selenium levels when being treated for deficiency.

*GRADE B was provided if the recommendation came from a body of evidence including well-conducted cohort or case-control studies directly applicable to the target population and demonstrating overall consistency of results or extrapolated evidence from high-quality or well-conducted studies

^No GRADE was considered as a good practice point and was provided if the guideline group found that there is an important practical point that they wish to emphasize but for which there is not, nor is there likely to be, any research evidence but only evidence from clinical experience.

Selenium in Acute Illness

Higher needs may exist in some illnesses; however, existing evidence is weak:[33,34]

- Burn patients with selenium losses, benefit from large IV supplies of around 375 mcg/day, with more rapid healing and fewer infections.
- Patients with major trauma and cardiac surgery may similarly benefit from a supplement of 275 mcg/day.

Patients with continuous renal replacement therapy (CRRT) or intermittent hemodialysis require higher doses (60-100 mcg) in PN given losses during dialysis.[35,36] Given impaired excretion, lower doses of 40 mcg are generally used in patients with renal failure, not on CRRT.

Retrospective studies with adequate numbers of patients on home PN have shown that standard amounts of selenium in routine home PN formulas may not be sufficient for all patients to replenish selenium levels or prevent deficiencies even after several months of PN. In addition, no clear guidelines exist to point to the correct dose of selenium in patients with IF receiving PN. Most authors recommend 100 mcg/day to as high as 400 mcg/day in deficiency. However, the duration of treatment for deficiency and ideal monitoring guidelines do not exist. Lastly, it is difficult to determine if low plasma selenium levels correlate with reduced whole-body stores or at what levels a person may develop clinical signs and symptoms of selenium deficiency. Each individual is unique, and genetic polymorphisms play a key role in selenium metabolism. Hence, more research and randomized trials are required to better understand selenium supplementation, especially in patients with IF receiving long-term PN.

CONCLUSION

Most healthy adults in the US consume enough dietary selenium to prevent clinically relevant deficiency. Selenium is absorbed in the duodenum, jejunum, and partly in the cecum; hence altered surgical anatomy, IF, and inflammatory bowel disease are associated with an increased risk of deficiency, even when receiving PN support. While some signs and symptoms of deficiency are difficult to measure (increased risk of infection

or cancer, for example), dilated cardiomyopathy is well-described in certain individuals with selenium deficiency.

For patients receiving EN or PN, an average of 60-100 mcg/day of selenium oral supplements or parenteral trace elements is usually sufficient to avoid deficiency. However, despite standard trace element dosing in home PN, patients remain at risk for selenium deficiency. Plasma selenium levels along with CRP must be monitored every 3-6 months for routine screening purposes, or sooner if the patient is being actively treated for selenium deficiency. The rising selenium cost may influence infusion, and insurance companies may reduce or omit it from PN formulations. However, given the deficiency risks, home PN providers must advocate for inclusion. ■

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