

PaLMS (PATHOLOGY NORTH) TRACE ELEMENTS FACT SHEET

SELENIUM

Introduction

Atomic mass 79. A widely, but unevenly distributed essential micronutrient that can also be toxic in excess. Selenium has multiple functions as an anti-oxidant, in thyroid hormone metabolism, reproduction and immune function. There is enormous geographic variation in selenium intakes worldwide. These variations reflect differences in the selenium content of the soil, which translates to differences of selenium content throughout the food chain. Limited Australian data suggests an average intake of around 90µg/day^{i, ii, iii}. The NHMRC RDI is 70µg/day for men and 60µg/day for women. In contrast, intakes in New Zealand may be as low as 28µg/day.

Overt deficiency, generally manifesting as cardiomyopathy, is rare. Due to its role as an anti-oxidant, there has been great interest in the potential deleterious effects of sub-optimal selenium status. Indeed, epidemiological evidence has associated low selenium status in a range of health effects including viral infections, cardiovascular disease, inflammatory conditions and immune function. Additionally, there is initial evidence that increased selenium status following supplementation is associated with decreased incidence of a range of cancers. These areas provide exciting areas for ongoing research.

Selenium is known to have a key role in the oxidation-reduction reaction. Therefore, there has been an explosion of interest in the potential health implications of selenium status. However, a dearth of quality prospective trials mean the link with many diseases are still controversial and, in many cases, speculative. There is prospective epidemiological evidence linking low selenium status with total cancer mortality and increased prostatic cancer incidence.

Exposure

Selenium is used in photoelectric cells, glass and ceramics, cosmetics, pigments and sheep dips. Occupational hazards result from exposure to dust or fume. Diets that include selenium at a rate of 1 µg per kilogram per day appear optimal. The selenium content of foodstuffs varies markedly in proportion to the selenium content of the soils in which the animal feed or cereals and vegetables have grown. Seafood provides a stable and relatively abundant source of selenium.

Absorption

Selenium absorption after inhalation of aerosol is dependent upon particle size. If the particles are respirable most selenium will be deposited in the deep lung where soluble oxides are absorbed rapidly. Non-respired selenium is cleared to the gut. Inorganic selenium compounds are not absorbed well from the gut. Organo-selenium compounds found in cereals (mainly selenomethionine) are well absorbed.

Distribution

Once absorbed, organo-selenium compounds are rapidly transferred to plasma and red blood cells. Selenate, in comparison, is more slowly transferred from the gut and is excluded from the red blood cells. Circulating selenium is primarily partitioned to brain, kidney and liver, but body stores are not large relative to metabolic demand. Selenium is incorporated into a range of enzymes that function as antioxidants (eg. glutathione peroxidase) and in thyroid hormone metabolism.

Excretion

Body balance of selenium is maintained through regulation of excretion of trimethyl selenonium to urine. Given normal intakes, about half the absorbed selenium load will be excreted to urine. In deficiency states and during pregnancy with a normal diet, very little selenium is excreted to urine. With increasing selenium body burden, selenium is exhaled as the volatile dimethyl selenide.

Pathology

Toxicity from selenium in Australia is rare in the absence of supplementation well above recommended. Acute intoxication may result in continuous vomiting, garlicky breath, mucosal irritation, abdominal pain, hypersalivation, haemolysis, necrosis of the liver, cerebral and pulmonary oedema, coma and death.

Selenosis from chronic exposure to selenium compounds in food and water has been associated with hair and nail brittleness and loss, dental caries, dry hair, misshapen nails, skin lesions, polyneuritis, garlic-like breath odour, fatigue, irritability, nausea and vomiting.

Occupational exposure to selenium containing fume or dust may result in eye redness, sneezing, coughing, respiratory difficulty and abdominal distress. Selenium exposure has been associated with lethal liver damage. Selenium hydride gas and selenium oxychloride liquid are both potentially lethal.

Selenium deficiency is caused by decreased dietary intake. The deficiency results in muscle abnormalities manifesting as cardiomyopathy (potentially fatal), muscle pain (commonly in the thighs) and reduced plasma glutathione peroxidase activity. Both these symptoms have been associated with long term parenteral nutrition without supplementation.

Monitoring

Plasma selenium levels respond rapidly to either stress (decreasing) or selenium uptake (increasing). Red cell selenium content changes more slowly in response to variations in selenium uptake and is not affected by stress. Whole blood selenium gives a good, inclusive measure of medium-term selenium status. Urinary selenium content varies markedly in normals and is subject to feeding and clearance effects that confound its use in either exposure or deficiency. Glutathione peroxidase activity in plasma may be used as a marker of selenium deficiency if selenium determinations are not available. Determinations in hair and nails are confounded by environmental contamination and so are not recommended.

Treatment

Selenium deficiency is treated by diet supplementation with selenomethionine or sodium selenite.

Analysis

Selenium determination may be made with graphite furnace or hydride generation atomic absorption spectrometry. **PaLMS** Trace Element Service employs inductively coupled plasma-mass spectrometry for improved turn around time and quality of results.

ⁱ Fardy JJ, McOrist GD, Farrar YJ. The determination of selenium status in the Australian diet using neutron activation analysis. *Journal of Radioanalytical and Nuclear Chemistry* 1989;133:397-408.

ⁱⁱ Reilly C. Selenium in Australian health and disease. *Proceedings of the Nutrition Society of Australia* 1992;17:109-114.

ⁱⁱⁱ Baghurst K, Worsley A, Crawford D. the Victorian Nutrition Survey Part 2.70 (GENERIC) Ref. Type: Report. CSIRO Division of Human Nutrition, Adelaide, 1987.

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