Manganese

Introduction

Chemically, manganese is closely related to iron. It is a catalytic cofactor for arginase, pyruvate carboxylase and mitochondrial superoxide dismutase (SOD), but also functions as a specific or unspecific activator for a large number of other enzymes, some of which participate in the synthesis of proteins, mucopolysaccharides and cholesterol.

Dietary sources and intake

Unrefined cereals, nuts and leafy vegetables have high manganese content. Tea is a substantial contributor to manganese intake, containing about 2.7 mg/L. Accordingly, manganese intake varies from very low, <2 mg/day, to high, >8 mg/day, in vegetarian diets.

The average content of manganese in the Swedish diet analysed in supermarket baskets and duplicate portions collected in the late 1980s was 3.6-3.7 mg/day (Becker et al 1997, Jorhem et al 1998). A Danish study where 100 men collected duplicate portions of their regular diets for 48 hours showed a manganese intake of 3.9 mg/day (Bro et al 1990). The manganese intake of Finnish children 3-18 years of age was in the range of 3-7 mg/day calculated from food consumption data and food contents (Bro et al 1990). These data indicate that manganese intake is adequate in these countries. Multivitamin-mineral and mineral supplements for adults may provide 2-5 mg manganese/dose.

Physiology and metabolism

The total body content of manganese is estimated to be 10-20 mg. The concentration is relatively high in bone and in organs rich in mitochondria, such as liver, pancreas and kidney, while muscle and plasma have low concentrations. Absorption from the diet is low, approximately 5%, and excretion is primarily through the bile. Animal studies have shown that iron, calcium and phytic acid reduce the absorption of manganese (Hurley et al 1987). A negative effect of calcium has been shown in humans, while the effect of iron and phytic acid does not seem to be pronounced (Davidsson et al 1991).

High intakes of manganese inhibit iron absorption (Rossander-Hulten 1991), and a higher absorption of manganese has been reported in iron deficiency (Mena et al 1969, Meltzer et al 2010).

Manganese deficiency in experimental animals results in reduced growth, skeletal abnormalities and defects in lipid and carbohydrate metabolism (Hurley et al 1987). In humans, only a limited number of possible manganese deficiency symptoms have been described in experimental studies with a manganese-deficient diet (Friedman et al
Dermal changes and hypocholesterolaemia are possible signs of manganese deficiency, as well as diffuse bone demineralization and poor growth in children. Very little information is available concerning the relationship between manganese intake and health endpoints or disease prevention (Brown et al. 2012).

**Requirement and recommended intake**

Our knowledge of manganese metabolism and the consequences of low intakes are insufficient for determining requirements and recommended daily intakes for humans. Balance studies have suggested that an intake of 0.74 mg/day should be sufficient to replace daily losses of manganese (Freeland et al. 1988). Intakes over 1 mg/day generally result in a positive manganese balance (Brown et al. 2012).

The EU Scientific Committee for Food (1993) considered a ‘safe and adequate intake’ to be 1-10 mg/person/day.

The US Food and Nutrition Board (2001) found data to be insufficient for setting an Estimated Average Requirement (EAR) for manganese, but used median intakes reported from the US Total Diet Study 1982-9 as a basis for setting adequate intakes (Pennington and Young 1991). The AI for adult men and women is set at 2.3 and 1.8 mg/day, respectively. In 1993, the EU Scientific Committee for Food (1993) suggested 1-10 mg/day to be an acceptable intake of manganese.

The Nordic Recommendations of 2004 did not include recommendations for manganese intake. As very few relevant human studies have been conducted since then, requirements are also difficult to determine this time, and accordingly, recommendations are not given for any age group.

Data are also too limited to determine requirements for manganese during pregnancy and lactation, and manganese deficiency of pregnant or lactating women has not been observed in humans. Manganese excretion from breast milk is estimated to be below 1% of the total manganese excretion. There is no clear correlation between dietary intake and breast milk manganese concentration (Brown et al. 2012). In a systematic review of studies, including studies published from January 1990 to October 2011, 15 studies reporting breast milk manganese concentration were retrieved (Brown et al. 2012), with levels ranging from 0.8-30 µg/L. Median (SD) manganese concentration of 31 Swedish milk samples was found to be 3.23 (0.27) µg/L (Parr et al. 1991).

**Upper intake levels and toxicity**

Manganese is regarded as one of the least toxic trace elements. Manganese toxicity, which manifests as psychological and neurological changes, has been observed in workers in manganese mines (Mena et al 1969). The neurological symptoms are reminiscent of those seen in Parkinson’s disease. Inhalation of manganese dust may be the explanation, while toxicity caused by a high dietary intake is unknown. The EU Scientific Committee for Food (2006) found that data for setting a Tolerable Upper Intake Level of manganese were too uncertain. The UK Foods Standards Agency (2003) has also found data to be insufficient to establish a Safe Upper Level for manganese.
References


