USANA Technical Bulletin

Manganese

Technical Background

- While manganese is abundant and widely distributed in nature, it is required only in trace amounts in the body. It does, however, play a number of essential roles in cellular function and human metabolism.
- At the biochemical level, manganese can function both as a constituent of metallo-enzymes and as an enzyme activator. The enzymes involved in urea production, carbohydrate synthesis, and preventing lipid peroxidation all contain manganese. A large number of enzymes with broad functions affecting multiple processes central to cellular metabolism, growth, replication, and differentiation, require manganese for their activation.[1]
- Manganese's effects on skeletal development have been extensively studied. It is now known that manganese-activated enzymes are involved in the synthesis of proteoglycan molecules, which add structural integrity to bone and joint cartilage.[1] Manganese may play a further role in bone development and remodeling,[2] and there are reports that women with osteoporosis tend to have low levels of blood manganese.[3]
- Studies indicate that manganese deficiency may lead to bone and joint abnormalities, impaired pancreatic function, ataxia, reduced growth, impaired reproductive performance, and abnormal carbohydrate and lipid metabolism.[1] Supplementation with manganese (combined with other nutrients) may improve joint health[4] and strengthen bones.[5]
- Acute (i.e. short-term) manganese deficiencies are rare in humans, in part because manganese is required in trace amounts, and in part because other divalent cations (particularly magnesium) can substitute for manganese in many functions involving enzyme activation.
- Manganese toxicity is an infrequent but serious human health hazard, resulting in severe and often irreversible disorders of the central nervous system.[6] The majority of reported cases involve exposure to high levels of airborne manganese (dust). There are few isolated cases of manganese toxicity resulting from excessive dietary exposure.[1]

Sources and Recommended Intake

- Whole grains, cereals, and nuts are the richest dietary sources of manganese. Tea and coffee are also relatively rich in manganese, but some believe that the element is not well-absorbed from these sources.[1]
- The estimated safe and adequate daily dietary intake for manganese is 0.3-1.0 mg for infants, 1.0-3.0 mg for children, and 2.0-5.0 mg for adults. Some believe that the lower end of the range for adults (2.0 mg) is too low, and should be raised to 3.5 mg/day.[7]

- Manganese's safe supplemental range extends to 10 mg/day. Some feel that an occasional intake of 20 mg per day may be safe.[8] Canada permits a maximum daily dosage of 30 mg/day.

Abstracts

Rico H, Gomez-Raso N, Revilla M, Hernandez ER, Seco C, Paez E, Crespo E. Effects on bone loss of manganese alone or with copper supplement in ovariectomized rats. A morphometric and densitomeric study. Eur J Obstet Gynecol Reprod Biol. 2000 May;90(1):97-101. OBJECTIVE: The aim of this study was to examine the effect of manganese (Mn) alone and with the addition of copper (Cu) in the inhibition of osteopenia induced by ovariectomy (OVX) in rats. STUDY CONDITIONS: Four lots of 100-day-old female Wistar rats were divided into experimental groups of 15 each. One group received a diet supplemented with 40 mg/kg of Mn per kilogram of feed (OVX+Mn). The second group received the same diet as the first, but with an additional 15 mg/kg of copper (OVX+Mn+Cu). The third group of 15 OVX and the fourth group of 15 Sham-OVX received no supplements. At the conclusion of the 30-day experiment, the rats were slaughtered and their femurs and fifth lumbar vertebrae were dissected. Femoral and vertebral length were measured with caliper and bones were weighed on a precision balance. The bone mineral content (BMC) and bone density (BMD) of the femur (F-BMC, mg and F-BMD, mg/cm(2)) and the fifth lumbar vertebra (V-BMC, mg and V-BMD, mg/cm(2)) were measured separately with dual energy X-ray absorptiometry. RESULTS: The F-BMD, mg/cm(2) was lower in the OVX than in the Sham-OVX group ($P<0.0001$) and in the other two groups receiving mineral supplements ($P<0.005$ in both). F-BMC, mg was significantly lower in the OVX group than in the other three ($P<0.0001$ in all cases). Calculations for V-BMC, mg and V-BMD, mg/cm(2) are similar to findings in the femur. CONCLUSIONS: These data show that a Mn supplement is an effective inhibitor of loss of bone mass after OVX, both on the axial and the peripheral levels, although this effect is not enhanced with the addition of Cu.

References

[1] Ken CL, Zidenberg-Cher S. Manganese. In Ziegler EE, Filer LJ (eds). Present Knowledge in Nutrition. Washington (DC): ILSI Press. p 334-41.
[2] Strause L et al. The effect of deficiencies of manganese and copper on osteoinduction and on resorption of bone particles in rats. Calcif Tissue Int 1987; 41: 145-50.
[3] Strause L et al. Spinal bone loss in post menopausal women supplemented with calcium and trace minerals. J Nutr 1994; 124: 1060-4.
[4] Leffler CT, Philippi AF, Leffler SG, Mosure JC, Kim PD. Glucosamine, chondroitin, and manganese ascorbate for degenerative joint disease of the knee or low back: a randomized, double-blind, placebo-controlled pilot study. Mil Med. 1999 Feb;164(2):85-91.
[5] Rico H, Gomez-Raso N, Revilla M, Hernandez ER, Seco C, Paez E, Crespo E. Effects on bone loss of manganese alone or with copper supplement in ovariectomized rats. A morphometric and densitomeric study. Eur J Obstet Gynecol Reprod Biol. 2000 May;90(1):97-101.
[6] Dobson AW, Erikson KM, Aschner M. Manganese neurotoxicity. 2004. Ann NY Acad Sci. 1012:115-128.
[7] National Research Council. Recommended Dietary Allowances. Washington (DC): National Academy Press. 1989. 284 pp.
[8] Cohen RD, Braunstein NS. 1996. Vitasearch. Newmarket (NH):Great Bay Nutrition Resources. 1996. 49 pp.