

NEWS UPDATES



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Magnesium and Bone Health

Magnesium supplementation has indeed shown promise in improving bone density, particularly in postmenopausal women who are at higher risk for osteoporosis and bone fractures. Studies have suggested that magnesium may play a crucial role in bone health by influencing bone metabolism and mineralization.

The effectiveness of magnesium supplementation in increasing bone density in postmenopausal women has been demonstrated in various research studies. One notable study involved a group of osteoporotic women who were given magnesium supplements for a period of two years. The results of this study indicated that magnesium supplementation not only helped prevent fractures but also led to a significant increase in bone density among the participants.

It's important to note that while calcium is commonly associated with bone health, magnesium appears to play a complementary role, and in some cases, it may even be more effective than calcium alone in restoring bone mineralization. This suggests that a balanced intake of both calcium and magnesium may be beneficial for maintaining optimal bone health, especially in populations at risk for osteoporosis. *Vikkanski, L. Magnesium may Slow Bone Loss. Med. Trib. Jul. 22, 1993. Sojka, JE, et al: Magnesium Supplementation and Osteoporosis. Nutr. Rev. 53, 1995.*

Magnesium plays a crucial role in regulating the transport and utilization of calcium in the body. It is involved in various physiological processes that are essential for maintaining bone health, including the activation of vitamin D, which in turn helps in the absorption of calcium from the intestines.

When there is an imbalance between calcium and magnesium levels, it can disrupt the normal transport of calcium into the bones, potentially leading to decreased bone density and increased risk of fractures. Therefore, correcting this imbalance is essential for ensuring optimal calcium metabolism and bone health.

Magnesium helps regulate calcium levels by influencing the activity of certain enzymes involved in calcium transport and metabolism. Additionally, magnesium is a cofactor for the production of calcitonin, a hormone that helps regulate calcium levels in the blood by promoting calcium deposition into the bones. *Sojka, JE, et al: Magnesium Supplementation and Osteoporosis. Nutr. Rev. 53, 1995.*

Overall, correcting any disturbances in the balance between calcium, magnesium and other minerals is crucial for supporting bone health and preventing bone-related disorders. It highlights the importance of considering the interrelationship between minerals in the management and prevention of conditions such as osteoporosis. However, it's essential to consult with a healthcare professional before starting any supplementation regimen, as many factors can contribute to bone loss. HTMA testing would provide a specific and targeted nutritional approach in assessing individual's needs.

Copper and Neurological Disease

Mineral imbalances, particularly deficiencies in copper relative to zinc and iron, have been implicated in various neurological disorders. Copper is an essential trace mineral that plays a crucial role in the central nervous system, including neurotransmitter synthesis, myelination, and antioxidant defense mechanisms. One of the key antioxidant enzymes that require copper for activation is superoxide dismutase (SOD). SOD is responsible for neutralizing harmful free radicals in the body, particularly the superoxide radical, which can cause oxidative damage to cells and tissues, including those in the brain. Reduced activity of SOD due to copper deficiency can lead to increased oxidative stress, which has been associated with neurological disturbances and neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis (ALS).

Mutations in genes encoding copper- and zinc-containing proteins, such as the copper transporter ATP7A and the copper/zinc-superoxide dismutase (SOD1) gene, have been linked to various neurological disorders. These mutations can disrupt copper homeostasis and impair the function of copper-dependent enzymes, leading to neurological dysfunction.

Furthermore, copper deficiency relative to zinc and iron imbalance can affect the balance of metal ions in the brain, leading to neurotoxicity and neuronal damage. Copper is involved in maintaining the balance of other metal ions, such as zinc and iron, and alterations in this balance can contribute to oxidative stress and neurodegeneration.

Overall, maintaining proper copper homeostasis is essential for neurological health, and imbalances in copper levels relative to other minerals can contribute to the pathogenesis of neurological disorders. It underscores the importance of ensuring adequate intake of copper and other essential minerals through a balanced diet and proper supplementation, when necessary, particularly in individuals at risk for neurological diseases. Mutations in the Copper- and Zinc- Containing Superoxide Dismutase Gene are Associated with "Lou Gehrig's Disease." Nutr. Rev. 51,8, 1993.

Past Heavy Metal Exposures

Heavy metals can persist in the body for extended periods, and their effects may not manifest immediately after exposure. The concept of half-life is crucial in understanding the long-term effects of heavy metal exposure.

Heavy metals such as lead, mercury, cadmium, and arsenic have long half-lives in the body, which means they can remain present for years after initial exposure. This persistence can lead to ongoing health effects, even if exposure occurred in the past.

For instance, lead exposure, especially in childhood, can result in lead accumulation in bones, where it can persist for decades. Similarly, mercury can accumulate in tissues such as the brain and kidneys, leading to chronic toxicity over time.

Therefore, even if a clinician is unable to identify a current source of heavy metal exposure, it's essential to consider past exposure history. Additionally, long-term monitoring and management may be necessary to address any potential health effects associated with previous exposure to heavy metals.