Commentary

DOI: http://dx.doi.org/10.53043/2347-3894.acam11009

ACAM

Magnesium – The Metabolic Blockbuster

Uwe Gröber

Academy of Micronutrient Medicine, Essen 45130, Germany

Asian Journal of Complementary and Alternative Medicine. Volume 11 Issue 02

Published on: 05/07/2023

***Author for Correspondence:** Uwe Gröber, Academy of Micronutrient Medicine, Essen 45130, Germany.

Cite this article as: Uwe Gröber. *Magnesium – The Metabolic Blockbuster.* Asian Journal of Complementary and Alternative Medicine, Vol 11(2), 54-54:2023.

Magnesium is primarily found within the cell where it acts as a counter ion for the energy-rich ATP and nuclear acids. Magnesium is a cofactor in more than 600 pacemaker enzyme systems, encompassing approximately 80% of all known metabolic functions, that regulate elementary biochemical reactions in the body, including protein synthesis, muscle and nerve transmission, neuromuscular conduction, blood glucose control, and blood pressure regulation. Some magnesium-dependent enzymes are Na+/K+-ATPase, hexokinase, creatine kinase, protein kinase, and cyclases. Magnesium is also necessary for structural function of proteins, nucleic acids, or mitochondria. It is required for DNA and RNA synthesis, reproduction, and for both aerobic and anaerobic energy production—oxidative phosphorylation and glycolysis - either indirectly as a part of magnesium-ATP complex, or directly as an enzyme activator [1-3].

Magnesium also plays a key role in the active transport of calcium and potassium ions across cell membranes, a process that is important to nerve impulse conduction, muscle contraction, vasomotor tone, and normal heart rhythm. Hypomagnesemia is frequently linked with hypokalemia owing to disturbances in renal K+ secretion in the connecting tubule and collecting duct. Magnesium is a natural calcium antagonist—the block of N-methyl-d-aspartate (NMDA) receptor channels by external magnesium is believed to be of great physiological importance. Moreover, it contributes to the structural development of bone and is required for the adenosine triphosphate-dependent synthesis of the most important intracellular antioxidant glutathione. Magnesium absorption and excretion is influenced by different hormones. It has been shown that 1,25-dihydroxyvitamin D [1,25(OH)2D] can stimulate intestinal magnesium absorption. On the other hand, magnesium is a cofactor that is required for the binding of vitamin D to its transport protein, vitamin D binding protein (VDBP). Moreover, conversion of vitamin D by hepatic 25-hydroxlation and renal 1α-hydroxylation into the active, hormonal form 1,25(OH)2D is magnesium-dependent. Magnesium deficiency, which leads to reduced 1,25(OH)2D and impaired parathyroid hormone response, has been implicated in ”magnesium-dependent vitamin-D-resistant rickets”. Magnesium supplementation substantially reversed the resistance to vitamin D treatment.

Of special importance is parathyroid hormone (PTH). Absorption of both magnesium and calcium appears to be inter-related, with concomitant deficiencies of both ions well described. A common link is that of PTH, secretion of which is enhanced in hypocalcemia. Hypomagnesemia impairs hypercalcemic-induced PTH release, which is corrected within in minutes after infusion of magnesium. The rapidity of correction of PTH concentrations suggests that the mechanism of action of magnesium is enhanced release of PTH. PTH release enhances magnesium reabsorption in the kidney, absorption in the gut and release from the bone [1, 3].

REFERENCES

1. Gröber U, Magnesium and Drugs. Int J Mol Sci, 2019; 20(9). pii: E2094. doi: 10.3390/ijms20092094
2. Rosanoff A, West C, Elin RJ, et al., Recommendation on an updated standardization of serum magnesium reference ranges. Eur J Nutr, 2022; 61(7): 3697-3706.
3. Kisters K, Kisters L, Gröber U, et al., Increased serum vitamin D concentration under oral magnesium therapy in elderly hypertensives. Magnes Res, 2020; 33(4):131-132.