Magnesium deficiency results in an increased formation of osteoclasts.

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OBJECTIVE: Magnesium (Mg(2+)) deficiency is a frequently occurring disorder that leads to loss of bone mass, abnormal bone growth and skeletal weakness. It is not clear whether Mg(2+) deficiency affects the formation and/or activity of osteoclasts. We evaluated the effect of Mg(2+) restriction on these parameters.

METHODS: Bone marrow cells from long bone and jaw of mice were seeded on plastic and on bone in medium containing different concentrations of Mg(2+) (0.8 mM which is 100% of the normal value, 0.4, 0.08 and 0 mM). The effect of Mg(2+) deficiency was evaluated on osteoclast precursors for their viability after 3 days and proliferation rate after 3 and 6 days, as was mRNA expression of osteoclastogenesis-related genes and Mg(2+)-related genes. After 6 days of incubation, the number of tartrate resistant acid phosphatase-positive (TRACP(+)) multinucleated cells was determined, and the TRACP activity of the medium was measured. Osteoclastic activity was assessed at 8 days by resorption pit analysis.

RESULTS: Mg(2+) deficiency resulted in increased numbers of osteoclast-like cells, a phenomenon found for both types of marrow. Mg(2+) deficiency had no effect on cell viability and proliferation. Increased osteoclastogenesis due to Mg(2+) deficiency was reflected in higher expression of osteoclast-related genes. However, resorption per osteoclast and TRACP activity were lower in the absence of Mg(2+).

CONCLUSION: In conclusion, Mg(2+) deficiency augmented osteoclastogenesis but appeared to inhibit the activity of these cells. Together, our in vitro data suggest that altered osteoclast numbers and activity may contribute to the skeletal phenotype as seen in Mg(2+) deficient patients.

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