Hypomagnesemia, obesity and inflammatory cytokines

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In a preceding letter to the editor, I reported that various generations with chronic hypomagnesemia developed hypertension and dyslipidemia but not obesity, insulin resistance and diabetes mellitus type 2 [1]. These results indicate that chronic hypomagnesemia does not induce insulin resistance but hypertension. This conclusion was questioned [2].

Due to an inborn defect of intestinal magnesium (Mg) absorption or renal Mg excretion, chronic hypomagnesemic patients developed cardiomyopathy, convulsions, tetany or neuropsychiatric symptoms but not insulin resistance and diabetes [3, 4].

Chronic hypomagnesemic patients with various forms of the magnesium deficiency syndrome or tetanic syndrome expressed many symptoms but did not develop insulin resistance and diabetes [4-9].

The major risk factor for insulin resistance and type 2 diabetes is obesity. The formation of various adipokines and inflammatory cytokines, among others the formation of IL-1, IL-6 and TNF-α, is increased in obesity [10, 11]. Some of these substances induce the development of insulin resistance [10] and hypertension [11] and contribute to the development of the obesity-induced metabolic syndrome [1, 11].

Increased formation of inflammatory cytokines was also found in Mg-deficient animals. Increased concentrations of IL-1, IL-6 and TNF-α were measured in plasma of hamsters and rats when total plasma Mg concentration was reduced by more than 75% [15]. However, data on the increase in proinflammatory cytokines in blood from Mg-deficient rats are conflicting [14].

An interaction of inflammatory cytokines and Mg in humans may be possible in obese subjects. In obese subjects, TNF-α and serum Mg exhibited an inverse correlation [19]. Possibly the obesity-induced formation of inflammatory cytokines is enhanced by Mg deficiency. An experiment with Zucker fat rats and different Mg statuses could help to clarify the relationship between obesity, Mg and inflammatory cytokines.

References

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