No. 1147

Basic Study for Metabolisms of Bittern and Biometal in the Heart

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Summary

Breakdown of biometal's homeostasis are closely contributed pathogenesis of several diseases, such as lifestyle-related disease and cardiac disease. The magnesium (Mg) is essential element for all living organisms as a cofactor for ATP, DNA, RNA, and metabolic enzymes. Recently, the relationship of Mg and cardiac disease has attracted attention. Animals and clinical studies have shown that Mg-deficiency has consequence of cardiac disease. However, molecular mechanisms of Mg on the development of cardiac disease are not well understood. To figure out molecular mechanisms of Mg, we investigated the histological change and the alterations of gene expression of Mg transporter family in the cardiac muscle of Mg-deficient mice.

Mg-deficient mice were fed by Mg-deficient diets (AIN93G MgO free) and distillated water. Control mice were fed by normal diets (AIN93G) and distillated water. Refeeding mice were fed by Mg-deficient diets for 25 days, and then displaced to normal diets for 7 days. Mg concentration or other biomarkers of serum were measured. To analyze the status of cardiac muscle in each group, we performed the histological staining and transmission electron microscopic study. Finally, we analyzed the gene expressions of several Mg transporters in the cardiac muscle of each group by quantitative RT-PCR (qRT-PCR).

Mg-deficient mice showed a significant reduction in serum Mg concentration compared with control mice. The levels of aspartate amino transferase and creatine kinase that are biomarker of myocardial infarction were higher than those of control mice. The structure of cardiac muscle in Mg-deficient mice showed the breakdown of cardiac myofibrillar structure and swelling of the mitochondria with the histological staining and transmission electron microscopic analysis. From qRT-PCR analysis, Trpm7 mRNA was significantly increased than that of control mice. On the other hand, the mRNA expressions of Slc41a3, MagT1, ACDP2 and Mrs2 of Mg-deficient cardiac muscle were decreased. Our results suggested that ultrastructural alterations in Mg-deficient mice occurred in connection with varying expressions of Mg transporters. In future, we plan to proceed in a more diversified research, but not limited to the involvement of Mg transporter in the onset of heart disease.