Heat-Killed *Lactobacillus reuteri* GMNL-263 Inhibits Systemic Lupus Erythematosus–Induced Cardiomyopathy in NZB/W F1 Mice

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Abstract

It has been increasingly recognized that accelerated atherosclerosis is a major cause of morbidity and mortality in patients with systemic lupus erythematosus, a multisystem autoimmune disease. In this study, we investigated the anti-apoptotic effects of heat-killed *Lactobacillus reuteri* GMNL-263 on the cardiac tissue of NZB/W F1 mice. The myocardial architecture of the mice heart was observed and evaluated using different staining techniques such as hematoxylin and eosin, TUNEL assay, Masson’s trichrome, and fluorescent immunohistochemistry. Additionally, the probiotics-related pathway proteins were analyzed via western blot analysis. Our results showed prevention of enlarged interstitial spaces and abnormal myocardial structures in the hearts of NZB/W F1 mice with *L. reuteri* GMNL-263 feeding. Significant reduction in TUNEL-positive cells, Fas death receptor–related components, and apoptosis was also detected in the cardiac tissues of the NZB/W F1 mice after *L. reuteri* GMNL-263 feeding compared with the control group. These findings are the first to reveal the protective effects of *L. reuteri* GMNL-263 against cardiac abnormalities in NZB/W F1 mice and suggest the potential clinical applications of *L. reuteri* GMNL-263 in the treatment of SLE-related cardiovascular diseases.

Keywords Systemic lupus erythematosus · Atherosclerosis · Cardiomyopathy · Probiotic bacteria · *Lactobacillus reuteri* GMNL-263

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