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Oral administration of *Lactobacillus* reuteri GMNL-263 improves insulin resistance and ameliorates hepatic steatosis in high fructose-fed rats

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Abstract

Background: Type 2 diabetes mellitus (DM), characterized by peripheral insulin resistance, is the most common form of diabetes. Probiotics are live micro-organisms that, when administered in adequate amounts, confer delaying effect on DM development. In this study, the effects *Lactobacillus reuteri* GMNL-263 (Lr263), a new probiotic strain developed by our laboratory, on insulin resistance and the development of hepatic steatosis in high-fructose fed rats were explored. Furthermore, the relevant regulatory pathways involved were also investigated.

Method: Male Sprague–Dawley rats were fed a high-fructose diet with or without Lr263 administration for 14 weeks. The composition of fecal microbiota, oral glucose tolerance, glycated haemoglobin, insulin, leptin, C-peptide, and incretins were measured. The markers of liver injury, serum and hepatic lipids profile, activity of hepatic antioxidant enzyme, and proinflammatory cytokines in adipose tissue were investigated. Additionally, the expression of hepatic lipogenic genes and insulin signaling related genes in adipose tissue were also studied. Liver sections were examined for hepatic steatosis using hematoxylin-eosin staining.

Results: The levels of serum glucose, insulin, leptin, C-peptide, glycated hemoglobin, GLP-1, liver injury markers, lipid profile in serum and liver were significantly increased in high-fructose-fed rats. However, after Lr263 administration, the elevation of these parameters was significantly suppressed. Feeding of Lr263 reversed the decreased number of *bifidobacterium* species and *lactobacillus* species and increased number of *clostridium* species induced by high fructose treatment. The decreased activities of hepatic antioxidant enzymes in HFD rats were dramatically reversed by Lr263 treatment. Concentrations of IL-6 and TNF-α in adipose tissue which were elevated in high fructose treatment were markedly decreased after Lr263 feeding. Decreased levels of PPAR-γ and GLUT4 mRNA after high fructose treatment were significantly enhanced by Lr263 administration. Lr263 consumption normalized the increased lipogenic gene (Srebp-1c, FAS, and Elvol6) expressions stimulated by high fructose. Administration of Lr263 significantly ameliorated hepatic steatosis observed in high fructose treated rats.

Conclusion: Our study provided evidences clarifying the effectiveness of Lr263 on reducing insulin resistance as well as hepatic steatosis formation in high-fructose-fed rats and suggested that Lr263 may be a promising therapeutic agent in treating type 2 diabetes.

Keywords: *Lactobacillus reuteri* GMNL-263, High-fructose-diet, Type 2 diabetes, Insulin resistance, Hepatic steatosis

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