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Antiobesity effect of *Lactobacillus reuteri* 263 associated with energy metabolism remodeling of white adipose tissue in high-energy-diet-fed rats☆,☆☆

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Abstract

Obesity is a serious and costly issue to the medical welfare worldwide. Probiotics have been suggested as one of the candidates to resolve the obesity-associated problems, but how they combat obesity is not fully understood. Herein, we investigated the effects of *Lactobacillus reuteri* 263 (*L. reuteri* 263) on antiobesity using four groups of Sprague–Dawley rats (n=10/group), namely, C (normal diet with vehicle treatment), HE [high-energy diet (HED) with vehicle treatment], 1X (HED with 2.1×10^9 CFU/kg/day of *L. reuteri* 263) and 5X (HED with 1.05×10^{10} CFU/kg/day of *L. reuteri* 263), for 8 weeks. *L. reuteri* 263 improved the phenomenon of obesity, serum levels of proinflammatory factors and antioxidant enzymes. More importantly, *L. reuteri* 263 increased oxygen consumption in white adipose tissue (WAT). The mRNA expressions of thermogenesis genes *uncoupling protein-1*, *uncoupling protein-3*, *carnitine palmitoyltransferase-1 and cell death-inducing DFFA-like effector-a* were up-regulated in WAT of the 5X group. Moreover, *L. reuteri* 263 might induce browning of WAT due to the higher mRNA levels of browning-related genes *peroxisome proliferator-activated receptor-γ*, *PR domain containing-16*, *Pparγ coactivator-1α*, *bone morphogenetic protein-7* and *fibroblast growth factor-21* in the 1X and 5X groups compared to the HE group. Finally, *L. reuteri* 263 altered the expressions of genes involved in glucose and lipid metabolisms in WAT, including increasing the levels of *glucose transporter type 4* and *carbohydrate-responsive element-binding protein* and decreasing the expression of *Acetyl-CoA carboxylase-1*. The results suggest that *L. reuteri* 263 may treat obesity through energy metabolism remodeling of WAT in the high-energy-diet-induced obese rats.

Keywords: Antiobesity; Browning of white adipose tissue; Energy metabolism remodeling; Lactobacillus reuteri 263; Probiotics

1. Introduction

Obesity is defined as abnormal accumulation of fat in the body. It is a medical condition that is linked to, and may increase the likelihood of, multiple other health disorders. According to World Health Organization, the prevalence of obesity increases more than twofold from 1980 to 2014 [1]. Since obesity-linked diseases, such as diabetes, hypertension, atherosclerosis and cancer [2,3], place an enormous personal and economic burden on those affected, it is critical to find ways to prevent and treat obesity.

Because obesity is caused by an imbalance of energy intake and expenditure, the best way to treat obesity is to cause a decrease in the former and an increase in the latter. Previous studies have shown that decreasing energy intake also leads to decreased energy expenditure [4]. Thus, current obesity treatments have focused on increasing energy expenditure. WAT, which was previously believed to be an energy storage tissue, has been found to be also involved in energy expenditure. WAT can up-regulate an *uncoupling protein* (*Ucp*) 1 and thus remodel its energy metabolism through a process called browning [5]. Browning transforms an original WAT to a beige adipose tissue, which has a higher respiratory rate, thermogenesis rate and energy consumption rate, similar to BAT [6,7]. After browning, the WAT displays a metabolism that expended glucose and lipid energy instead of storing it [8]. Thus, browning of WAT can be one of the ways to increase energy expenditure and prevent obesity.

Browning of WAT is regulated by several genes, including *peroxisome proliferator-activated receptor γ* (*Pparγ*), *PR domain containing 16* (*Prdm16*), *Pparγ coactivator-1α* (*Pgc1α*), *bone morphogenetic protein 7* (*Bmp7*) and *fibroblast growth factor 21* (*Fgf21*). Pparγ is the master transcription factor for fat differentiation and survival of adipocytes [9]. Pparγ stabilizes and recruits Prdm16 to activate *Pgc1α*, which results in browning of WAT [10,11]. Bmp7 and Fgf21 have been reported to induce *Pgc1α* and *Ucp1*, which increase energy expenditure [10,12,13]. Browning also induces mitochondrial biogenesis genes such as *carnitine palmitoyltransferase 1* (*Cpt1*) and *cell death-*

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