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Germ Rice Extract Improves Lipid Metabolism in High Fat Feeding C57bl/6 Mice

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Obesity stems from an imbalance between calorie intake and expenditure, leading to the accumulation of excess fat in the body and is associated with the development of various metabolic abnormalities, such as insulin resistance and dyslipidemia. The incidence of obesity is dramatically increasing and by 2025, 18% of the world's men and 21% of the world's women are expected to be obese. Although many medications are available for the management of hyperlipidemia and hyperglycemia, they fail to completely control glucose and lipid homeostasis. Furthermore, they often cause side effects and are expensive. Therefore, there is a need for natural products that can inhibit obesity, are safe for human consumption, and are available at an accessible price.

Here, we evaluated the effects of a germ rice extract on lipid metabolism with the aim to find anti obesity effects in mice. The germ rice (Koiazusa) produced in Ishikawa, Japan, in 2016, contained 6.5 times more γ-aminobutyric acid than normal rice (Koshihikari). We used water extract of germ rice that was roasted for 20 s at 220 °C to prepare pre-gelatinized starch. Although germ rice extract (GRE) did not affect water and food consumption and high fat diet (HFD)-induced gain in body weight in C57BL/6 mice, it reduced glucose, AST, ALT, total cholesterol and the arteriosclerotic index in serum samples. Moreover, oral administration of GRE inhibited lipid accumulation in the liver of HFD-fed mice, thus reducing the liver damage induced by HFD-feeding. We found a significant difference in PPAR-α gene expression levels in the liver between HFD-fed and GRE- and HFD-fed mice. No differences in the expression levels of FAS, PPAR-γ and SREBP1c, which are associated with lipid production, were found. PPAR-α is a ligand-activated transcription factor that activates fatty acid oxidation and lipoprotein metabolism and improves plasma lipid profiles. Collectively, our data indicate that oral administration of GRE inhibits fatty liver through the activation of β-oxidation by up-regulating of PPAR-α mRNA.