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The ethanolic extracts of *Brassica rapa* L. prevents high-fat diet induced obesity via beta3-adrenergic regulation of catabolic activity of white adipocytes

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Objectives

Obesity is the most common nutritional disorder in the developed world and it is considered to be a risk factor associated with the genesis or development of various diseases, including coronary heart disease, hypertension, type 2 diabetes mellitus, and cancer. Many attempts have been made to correct the metabolic disparity of the obesity condition, producing a number of reagents. However, administration of these drugs is known to often cause undesirable side effects. Therefore, there has been high demand for therapeutically potent, and yet safe, anti-obesity reagents such as botanical drugs. In the current study, we demonstrated the anti-obesity effects of the ethanolic extracts of *Brassica rapa* L. (EBR) and investigated the underlying mechanism at the molecular level.

Materials and Methods

○ Material

The roots of *Brassica rapa* L. (100 g) obtained from Ganghwa County Agricultural Technology Service Center, Incheon, Korea, was cut and extracted with 95% ethanol with mechanical agitation for 24 h. The extract solutions were filtered and concentrated under reduced pressure to obtain the ethanolic extracts (10. 6 g) for use in further tests.

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○ Methods

It is evaluated the effects of EBR on adipocyte differentiation and lipolysis using 3T3L1 adipocyte. Furthermore it is examined if EBR could prevent the body weight gain induced by feeding a high-fat diet to female ICR mice for 8 weeks. We assessed lipid distribution of plasma and adipokine profile of plasma and adipose tissue. To investigate the physiological mechanism of EBR on obesity, the gene

expressions were analyzed from each organ by real-time RT-PCR and western blot technology.

Results

EBR has been found to inhibit the lipid accumulation in 3T3-L1 adipocytes increasing lipolysis, if added at an early phase of differentiation. RT analysis revealed that the expression of beta3 adrenergic receptor (β3-AR) and its downstream genes including HSL, ATGL, and UCP-2, were highly induced by EBR treatment while that of other adipogenic genes were not affected. EBR treatment also restored normal expression of adipokine including leptin, adiponectin, and IL-6. In the animal experiment, the increases in body weight and epididymal fat accumulation were highly suppressed by 6-week oral administration of EBR at 50 mg/kg/day, which is greater than that of 10 mg/kg/day of Xenical, a lipid absorption inhibitor (XEN), while the overall amount of food intake was not affected. Results from blood analysis showed that the plasma lipid profile was not changed by EBR administration, and that the level of leptin was reduced by 59%, while that of adiponectin was increased by 14%, compared to high fat diet-fed control (HFD). Surprisingly, the gene expression pattern in the epidydymal adipose tissue of EBR-treated mice was highly similar with that in 3T3L1 cells. Our data suggest that EBR may have great potential as a new anti-obesity agent in that beta3 adrenergic regulation of lipolysis and diet-induced thermogenesis is highly induced by this reagent.