The Mechanism of the Reducing Effects of Korean Red Ginseng on Triacylglycerol Content in 3T3-L1 Adipocytes Cultured under Enriched Lipid Conditions

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Obesity, the commonest nutritional disorder in modern societies, is associated with serious morbidities including a high incidence of type II diabetes, cardiovascular disease, osteoarthritis, and the increased risk of many cancers. Changes in body weight are resulted from the difference between energy intake and expenditure that can be regulated by several factors.

Ginsenosides are the components of saponin extracted from ginseng and they are known to possess various physiological and pharmacological activities. One of these activities is related to decreased lipid levels in plasma.

In this study, the effects of the Korean red-ginseng extract and ginsenosides on fat metabolism were examined in 3T3-L1 adipocytes cultured in high fatty acid conditions. The amounts of triacylglycerol (TAG) in the 3T3-L1 adipocytes cultured in high fatty acid conditions were greatly increased compared to a control condition without fatty acid. This TAG increment in high fatty acid conditions was relieved back to the control level by the individual treatments of Korean red-ginseng extracts or ginsenosides, especially Rh2 and compound K, suggesting that ginsenosides have a lowering effect of TAG level in adipocytes. The effects of ginsenosides on 3T3-L1 preadipocyte differentiation were also examined. And to obtain a detailed understanding of molecular level on the mechanism of action of the ginsenosides, we have performed mRNA differential display analysis on differentiating 3T3-L1 cells and identified genes exhibiting altered expression after 4 days and 9 days ginsenosides treatment. Our data showed that ginsenosides increased the expression of the genes participating in the activation of energy expenditure such as leptin and MAP kinase. These results indicated that ginsenosides decreased the TAG level in 3T3-L1 adipocytes and up-regulated the transcription of leptin which is known for lowering TAG content in adipocytes through transcriptional activation of the crucial genes involved in peroximal and mitochondrial β-oxidation. To elucidate the mechanism of the effects of ginsenosides on lowering TAG content in 3T3-L1 adipocytes, we examined whether ginsenosides modulate the expressions

of other adipokines besides leptin and transcription factors related to control of energy expenditure process because adipokines regulate adipocyte mass and increased energy expenditure may consume much TAG in adipocytes. In these studies, ginsenosides increased the expressions of mRNAs for leptin, adipsin, and TGFβ and their corresponding proteins in 3T3-L1 adipocytes, resulting in decreased TAG content. In addition, ginsenosides also increased intracellular cAMP level that signal transducer on differentiation and lipolysis and up-regulated the expression of PPARδ, which is known to increase the transcription of target genes of energy expenditure.

In conclusion, ginsenosides could control TAG content in differentiated adipocytes by up-regulation of adipokines including leptin and transcription factors related to fat burn, and these processes might be mediated by cAMP-related signaling. Taken together, it can be suggested that ginsenosides may be developed as a new contributable drug for treatment of obesity.

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