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alpha-Mangostin Improves Glucose Uptake and Inhibits Adipocytes Differentiation in 3T3-L1 Cells via PPAR gamma, GLUT4, and Leptin Expressions

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Abstract

Obesity has been often associated with the occurrence of cardiovascular diseases, type 2 diabetes, and cancer. The development of obesity is also accompanied by significant differentiation of preadipocytes into adipocytes. In this study, we investigated the activity of alpha-mangostin, a major xanthone component isolated from the stem bark of *G. malaccensis*, on glucose uptake and adipocyte differentiation of 3T3-L1 cells focusing on PPAR gamma, GLUT4, and leptin expressions. alpha-Mangostin was found to inhibit cytoplasmic lipid accumulation and adipogenic differentiation. Cells treated with 50 μ M of alpha-mangostin reduced intracellular fat accumulation dose-dependently up to 44.4% relative to MDI-treated cells. Analyses of 2-deoxy-D-[H-3] glucose uptake activity showed that alpha-mangostin significantly improved the glucose uptake ($P < 0.05$) with highest activity found at 25 μ M. In addition, alpha-mangostin increased the amount of free fatty acids (FFA) released. The highest glycerol release level was observed at 50 μ M of alpha-mangostin. qRT-PCR analysis showed reduced lipid accumulation via inhibition of PPAR gamma gene expression. Induction of glucose uptake and free fatty acid release by alpha-mangostin were accompanied by increasing mRNA expression of GLUT4 and leptin. These evidences propose that alpha-mangostin might be possible candidate for the effective management of obesity in future.

Keywords

KeyWords Plus: [LIPID DROPLET ACCUMULATION](#); [GARCINIA-MANGOSTANA](#); [SIGNALING PATHWAY](#); [DOWN-REGULATION](#); [ADIPOSE-TISSUE](#); [TUMOR-GROWTH](#); [OBESITY](#); [ADIPOGENESIS](#); [XANTHONES](#); [MECHANISM](#)

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