Curcumin, a Potential Inhibitor of Up-regulation of TNF-alpha and IL-6 Induced by Palmitate in 3T3-L1 Adipocytes through NF-kappaB and JNK Pathway

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Objective To investigate the attenuating effect of curcumin, an anti-inflammatory compound derived from dietary spice turmeric (Curcuma longa), on the pro-inflammatory insulin-resistant state in 3T3-L1 adipocytes. Methods Glucose uptake rate was determined with the \(^{[3]H}\) 2-deoxyglucose uptake method. Expressions of tumor necrosis factor-\(\alpha\) (TNF-\(\alpha\)) and interleukin-6 (IL-6) were measured by quantitative RT-PCR analysis and ELISA. Nuclear transcription factor kappaB p65 (NF-kB p65) and mitogen-activated protein kinase (MAPKs) were detected by Western blot assay. Results The basal glucose uptake was not altered, and curcumin increased the insulin-stimulated glucose uptake in 3T3-L1 cells. Curcumin suppressed the transcription and secretion of TNF-\(\alpha\) and IL-6 induced by palmitate in a concentration-dependent manner. Palmitate induced nuclear translocation of NF-kB. The activities of Jun NH2-terminal kinase (JNK), extracellular signal–regulated kinase 1/2 (ERK1/2) and p38MAPK were decreased in the presence of curcumin. Moreover, pretreatment with SP600125 (inhibitor of JNK) instead of PD98059 or SB203580 (inhibitor of ERK1/2 or p38MAPK, respectively) decreased the up-regulation of TNF-alpha and IL-6 induced by palmitate. Conclusion Curcumin reverses palmitate-induced insulin resistance state in 3T3-L1 adipocytes through the NF-kB and JNK pathway.

Key words: Curcumin; Insulin resistance; Inflammation; Adipocyte; Free fatty acids

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