## Investigation the molecule mechanism of adipokines in human chondrocytes

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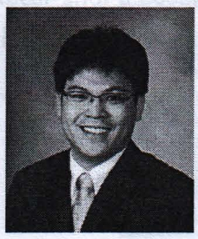

**Abstract:**

Articular adipose tissue is a ubiquitous component of human joints, and adiponectin is a protein hormone secreted predominantly by differentiated adipocytes and involved in energy homeostasis. The adiponectin is significantly higher in synovial fluid of patients with osteoarthritis and rheumatoid arthritis. Matrix metalloproteinases (MMP)-3 may contribute to the breakdown of articular cartilage during arthritis. We investigated the signaling pathway involved in MMP-3 caused by adiponectin in human chondrocytes. Adiponectin increased the secretion of MMP-3 in cultured human chondrocytes, as shown by qPCR, western blot and ELISA analysis. Adiponectin-mediated MMP-3 expression was attenuated by AdipoR1 but not AdipoR2 siRNA. Pretreatment with 5'-AMP-activated protein kinase (AMPK) inhibitor (araA and compound C), p38 inhibitor (SB203580) and NF- B inhibitor (PDTC and TPCK) also inhibited the potentiating action of adiponectin. Activations of p38, AMPK and NF- B pathways after adiponectin treatment were demonstrated. Taken together, our results provide evidence that adiponectin acts through AdipoR1 to activate p38 and AMPK, resulting in the activations of NF- B on the MMP-3 promoter and contribute cartilage destruction during arthritis.