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RESTORING THE INFLAMMATORY CYTOKINE-INDUCED IMPAIRED CHONDROGENESIS BY DIALLYL DISULFIED IN HUMAN ADIPOSE-DERIVED MESENCHYMAL STEM CELLS: ROLE OF REACTIVE OXYGEN SPECIES AND ANTIOXIDAN ENZYMES.

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SUMMARY

Current strategies based on mesenchymal stem cell (MSC) therapy for restoring injured articular cartilage are not effective enough in osteoarthritis (OA). Due to the enhanced inflammation and oxidative stress in OA microenvironment, differentiation of MSCs into chondrocytes would be impaired. This study aims to explore the effects of diallyl disulfide (DADS) on IL-1β-mediated inflammation and oxidative stress in human adipose derived mesenchymal stem cells (hADSCs) during chondrogenesis. MTT assay was employed to examine the effects of various concentrations of DADS on the viability of hADSCs at different time scales to obtain non-cytotoxic concentration range of DADS. The effects of DADS on IL-1β-induced intracellular ROS generation and lipid peroxidation were evaluated in hADSCs. Western blotting was used to analyze the protein expression levels of IκBα (np), IκBα (p), NF-κB (np) and NF-κB (p). Furthermore, the gene expression levels of antioxidant enzymes in hADSCs and chondrogenic markers at days 7, 14 and 21 of differentiation were measured using qRT-PCR. The results showed that addition of DADS significantly enhanced the mRNA expression levels of antioxidant enzymes as well as reduced ROS elevation, lipid peroxidation, IκBα activation and NF-κB nuclear translocation in hADSCs treated with IL-1β. In addition, DADS could significantly increase the expression levels of IL-1β-induced impaired chondrogenic marker genes in differentiated hADSCs. Treatment with DADS may provide an effective approach to prevent the pro-inflammatory cytokines and oxidative stress as catabolic causes of chondrocyte cell death and enhance the protective anabolic effects by promoting chondrogenesis associated gene expressions in hADSCs exposed to OA condition.