Identification of genes involved in the effects of hypoxia-inducible factor-2 on articular chondrocytes using bioinformatic analysis and validation in osteoarthritis

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January 30, 2024

Abstract

Although over-expression of hypoxia-inducible factor-2 alpha (HIF-2α) can result in cartilage destruction and osteoarthritis (OA) development, the underlying mechanisms remain poorly understood. Here, we investigated the molecular mechanisms in chondrocytes over-expressing HIF-2α. The GeneCloud of Biotechnology Information platform was used to identify differentially expressed genes (DEGs). Using the GEO GSE104794 dataset of control (empty adenovirus, n = 4) and experimental (recombinant adenovirus expressing HIF-2α, n = 4) groups, we performed DEG, Gene Ontology, pathway, pathway network, and gene signal network analyses. Similarly, DEG analysis was performed for the GEO GSE51588 dataset of control (non-OA, n = 4) and experimental (OA, n = 20) groups. Thereafter, intersection of GSE104794 gene signal network analysis and GSE51588 DEG analysis was performed for the key genes, validated by quantitative reverse transcription-polymerase chain reaction. A total of 542 DEGs were identified, among which, the 10 most significant genes in the gene signal network were Nfkb1, Tlr2, Nt5e, Enpp1, Entpd3, Vegfa, Ptgs2, Socs3, Fos, and Epas1. The key genes in OA were LUM, ENTPD3, SMPD3, FGFR3, GPX3, IRAK3, EREG, HTR2A, TLR2, and CDA. Taken together, we screened key genes that are potentially involved in osteoarthritis, thereby providing a basis for identifying valuable markers for this disease.

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