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Editorial

Tyrosine Kinases Role in Chondrocyte Hypertrophy

Emma Walker*

Editorial office, Journal of Bone Research, Spain

EDITORIAL

Osteoarthritis (OA) is a major health issue that damages joints and causes severe impairment around the world. Pain and low-grade inflammation are common symptoms. OA is a common degenerative joint illness that affects people all around the world. Slow-progressive deterioration of the articular cartilage, dysregulation of subchondral bone remodelling, and synovial inflammation are all symptoms of OA, which eventually lead to loss of joint function and persistent pain. Despite the fact that the key risk factors for OA have been recognised, no effective disease-modifying medication is currently available. However, the specific cause of the disease is unknown, and treatment choices are limited. Articular chondrocytes in OA go through a phenotypic change, becoming hypertrophic, which causes cartilage degradation and worsens the disease. As a result, a medication that inhibits hypertrophy might be a promising disease-modifying treatment. The use of tyrosine kinase inhibitors for therapeutic purposes has primarily been centred on oncology, but the Food and Drug Administration. The overexpression of proteolytic enzymes, which cause the destruction of extracellular matrix (ECM) components such as type II Collagen and Aggrecan, is one of the key hallmarks of hypertrophic chondrocytes. Matrix Metallopeptidase (MMP) 13 and a desintegrin, as well as Metallopeptidase with Thrombospondin Motif, are the major enzymes involved (ADAMTS).

Tyrosine kinases (TKs) may play a key role in chondrocyte hypertrophy regulation. These enzymes play an important role in cellular differentiation and are key regulators in a number of pathways. There is, however, no study that gives a comprehensive summary of the TKs linked to chondrocyte hypertrophy and the mechanisms that lead to this phenotypic change. Chondrocyte hypertrophy, which occurs during bone growth, has been linked to a number of mechanisms. The negative feedback loop between Indian hedgehog (IHH) and parathyroid hormone related protein (PTHrP) is known to maintain chondrocyte homeostasis. PTHrP inhibits hypertrophy while IHH promotes it. TGF-b has also been demonstrated to suppress hypertrophy by regulating the amounts of key chondrocyte phenotype-determining transcriptional regulators including SOX9 and RUNX2 via Smad2/3 signalling.

TGF-b, on the other hand, may induce hypertrophy by activating Smad1/5/9 phosphorylation. Wingless-type (Wnt) signalling is another mechanism that contributes to chondrocyte hypertrophy. Wnt causes b-catenin to be stabilised in the cytoplasm, allowing it to be translocated to the nucleus and eventually allowing RUNX2 transcription. Similarly, pro-inflammatory cytokines like interleukin (IL)-1b and tumour necrosis factor (TNF)-a have been demonstrated to upregulate hypertrophic markers and suppress SOX9 expression through stabilising nuclear factor kappa-lightchain-enhancer of activated B cells (NF-kb) and hypoxia inducible factor 2a (Hif-2a). Other pathways, such as mitogen-activated protein kinase (MAPK) signalling, phosphoinositide 3-kinase (PI3K)-protein kinase B (AKT), and janus kinase (JAK) 2- (signal transducer and activator of transcription factor (STAT) signalling, may, depending on the ligand-receptor by which the transduction is initiated, produce opposite responses.

Correspondence to: Emma Walker, Editorial office, Journal of Bone Research, Spain, E-mail: orthopedicsurgery@journalsoa.org

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