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Anti VEGFR therapy for osteoarthritis pain

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Osteoarthritis (OA), referred as arthritis, is among the most common chronic conditions among adults. Osteoarthritic symptom, pain, is the key reason to seek medical assistance, yet there is no effective way to relieve OA-induced pain. Despite the major negative impact that severe pain in chronic OA has on quality of life and health care management, we only poorly understand origins of pain in OA, the molecular mechanisms driving the pathology, and the way to effectively cure OA. Many cases eventually require joint replacement with a prosthesis which is costly, and the limited functional life of prostheses (~10 y) can make a second replacement necessary. These factors increase both the overall cost of treatment and the risk for associated morbidity. Significantly, surgical procedures to address the condition typically do not result in a pain-free cure. The central aim of our sutdy is to test that the activation of Flt1 (vascular endothelial growth factor receptor-1) is the major driver of joint pain transmission by plasticity of peripheral (sensory neurons) and central glial activation; Flk1 (vascular endothelial growth factor receptor-2) is primarily responsible for cartilage degeneration during the OA progression, thus, simultaneous inhibition of Flt1 and Flk1 by pazopanib, an FDA-approved small molecule anti-cancer drug, will act as an ideal OA disease-modifying drug (OADMD) with immediate reduction of joint pain and gradually cartilage regeneration. The findings of our proposed research will take the field of OA research a giant step forward: in the short term, by increasing our mechanistic understanding of the causes and progression of OA, and by developing a novel strategy for treating OA and joint pain effectively and safely in our pre-clinical OA animal model; and, in the longer term, by providing a rationale for clinical trials to test pazopanib to treat OA patients.

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