Role of G Protein Receptor Kinase 3 (GRK3) on Pathogenesis of Osteoarthritis

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Osteoarthritis (OA) is a musculoskeletal disease often caused by overuse or injury but recent literature has suspected G protein coupled receptor (GPCR) signaling of cell surface receptors as playing a role in the complex cellular process involved in disease pathogenesis. In our study of OA, we aimed to understand the regulation of GPCRs by investigating the role G protein coupled receptor kinase 3 (GRK3) plays in the development of the disease. GRKs are intracellular kinases that turn off (desensitize) GPCR signaling. Based on the histopathology analysis of this study, there was no statistically significant difference in the age-induced OA control mice compared to the mice deficient in GRK3 (Grk3-/-). However, we did observe a clinical difference in that Grk3-/- mice had half the amount of cartilage degradation. These results suggest that the initial pilot study may be underpowered and that more mice would be needed to determine statistically significant results. Further, in vitro studies would be necessary to understand the mechanistic role GRK3 plays in the regulation of specific GPCRs in OA.

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