Menisci are fibrocartilaginous structures in knee joints integral to stabilization during locomotion. They are made of mostly water, Type I and II collagens, and contain chondrocytes and fibroblast-like cells. There is no optimal treatment option due to diversity in tear types and ages of patients. There is incentive in finding better treatment methods as such injuries increase probability of joint degeneration, worsening degeneration if there is pre-existing osteoarthritis.

Treatment methods involve anabolic growth factors, which bind to receptors to initiate a sequence resulting in cell proliferation, differentiation, or death. Fibroblast growth factor two (FGF-2) can stimulate mitosis in chondrocytes and mesenchymal stem cells (MSCs) to form connective tissue. FGF-2 is known to causing breakdown of articular chondrocytes and human extracellular matrix, unlike in mammalian model organisms. However, paired with transforming growth factor beta 1 (TGFB1), FGF-2 has shown significant chondrocyte regeneration. Zhou et al. investigated both FGF-2 and TGFB1 effects on MSC proliferation, concluding FGF-2 promoted MSCs, but that it also inhibited anabolic cartilage growth. There is potential in adjusting FGF-2 and TGFB1 ratios to enhance regenerative capabilities.

Past studies commonly involved expensive in vivo experiments with menisci of mammalian organisms, at the expense of the organism’s lives. Hunziker et al. instead used a less costly in vitro model to study the combination of FGF-2 and TGFB1, while also producing faster results. There is potential with investigating the combination of FGF-2 and TGFB1 on in vitro models used by Hunziker et al. to develop better treatment methods for meniscal degeneration.