

A combination of Mesenchymal stem cells and Low Intensity Ultrasound for Knee meniscus regeneration: A preliminary study

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Abstract

Background

Meniscus defects critically alter knee function and lead to degenerative changes. Regenerative medicine applications including stem cell transplantation have showed a promising efficacy in finding alternatives to overcome traditional treatment limitations. However, stem cell therapy remains limited due to the substantially reduced viability and inhibitory microenvironment. Since tissue growth and repair are under the control of biochemical and mechanical signals, several approaches have recently been investigated (e.g., low intensity pulsed ultrasound [LIPUS]) to promote the regeneration process. This study employed LIPUS to improve growth and osteogenic differentiation of mesenchymal stem cells derived from human embryonic stem cells to improve the regeneration of meniscus tissue.

Methodology:

The MSCs were transplanted into the epicenter of the injured meniscus in rabbits, which were randomized into two main groups: a treatment group (n=32 New Zealand rabbits) including 4 subgroups of 8 rabbits in each subgroup (LIPUS treatment, MSC treatment, LIPUS with MSC and control), and a second group (n=9) to track .

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Biography: Mohammad Nasb's research program is focused on understanding the epigenetic neural gene control mechanisms that govern regulation of higher order brain function via chromatin packaging control in neurons. Her research group focuses on understanding the role(s) of specific HATs in cognition and neurodegenerative disorders such as Alzheimer's disease (AD). Her research group generated a robust Drosophila model system that enables them to modulate Tip60 HAT levels in neural circuits of choice under AD

neurodegenerative conditions, in vivo. Its use led to their exciting discovery that Tip60 is critical for cognitive processes and protects multiple cognitive neural circuits impaired in the brain during early AD progression. Her group is currently deciphering the mechanisms underlying Tip60 HAT action in neuroprotective gene control using fly and mouse AD models and determining how these Tip60 epigenetic processes go awry in the brains of human AD patients.