Tulane researcher shows enhanced therapeutic stem cell migration improves neurodegenerative disease

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Jean-Pyo Lee, PhD, assistant professor in the Department of Physiology at Tulane School of Medicine, in collaboration with colleagues at Sanford Burnham Prebys Medical Discovery Institute and the University of California San Diego, has shown for the first time that injection of a synthetic drug that attracts stem cell migration can improve neurological outcome in a mouse model of neurodegenerative disease. (Photo provided)

Stem cell therapy, especially neural stem cells, offers great promise in treating brain injury. Neural stem cells exhibit a broad repertoire of potentially multiple therapeutic actions including functional neural replacement and acute and chronic anti-inflammatory action via the delivery of therapeutic gene products synthesized inherently by the stem cells in the disease environment.

For optimal stem cell therapy, neural stem cells should migrate quickly and extensively to the site of injury and neurodegeneration.

Jean-Pyo Lee, PhD, assistant professor in the Department of Physiology at Tulane School of Medicine, in collaboration with colleagues at Sanford Burnham Prebys Medical Discovery Institute and the University of California San Diego, has shown for the first time that injection of a synthetic agonist (drug), that attracts stem cell migration can improve neurological outcome in a mouse model of neurodegenerative disease. The research was recently published by the Proceedings of the National Academy of Sciences of the United States of America and can be viewed here: https://www.pnas.org/content/early/2020/11/19/1911444117.

The study addresses the important nature of tropism of stem cell migration using neural stem cells, Lee said. Chemokines and chemokine receptors found in neural stem cells can mediate this activity.

In this study, human neural stem cells derived from induced pluripotent stem cells were used. Transplantation of human induced pluripotent stem cell-derived neural stem cells in a mouse model of a prototypical neurodegenerative disease improves neurological function and increases the life span of neurodegenerative mice.

The study also found that a synthetic chemokine analog attracts these neural stem cells and increases the beneficial impact of these stem cells on neurological disorders, Lee said. When this analog is co-administered with transplanted neural stem cells, the agonist (drug) enhanced stem cell migration, dissemination and integration into the diseased brains. Considering the prevalence of neurological diseases and current limitations of stem cell therapy, the findings will contribute to advancing the stem cell field and will be of great interest to further neurodegenerative disease research.