Study Shows how Tafa2 Protein Induces Stem Cell Migration to Heal Bone Fractures

Skeletal (mesenchymal) stem cells (MSCs) are being used in an increasing number of clinical trials for their therapeutic benefits in tissue regeneration and fracture healing. However, their poor homing capacity to the injured site presents a major challenge to realizing their full capabilities. A new study recently published in STEM CELLS shows a possible way to overcome that.

DURHAM, N.C. (PRWEB) December 17, 2018 -- Skeletal (mesenchymal) stem cells (MSCs) are being used in an increasing number of clinical trials for their therapeutic benefits in tissue regeneration and fracture healing. However, their poor homing capacity to the injured site presents a major challenge to realizing their full capabilities. A new study recently published in STEM CELLS shows a possible way to overcome that.

The study was conducted by a team led by Moustapha Kassem, M.D., Ph.D., of the Novo Nordisk Foundation Center for Stem Cell Biology (DanStem), Department of Cellular & Molecular Medicine at the University of Copenhagen and Department of Endocrinology, University Hospital of Odense, Denmark. It focused on understanding the mechanisms regulating recruitment of human MSCs (hMSCs) to the injury site — a prerequisite for their successful use in cell replacement therapy.

“In order to identify secreted factors with the capacity for enhancing recruitment of MSCs to the skeleton, we examined a number of factors known to regulate migration and motility of adult stem cells in various tissues,” Dr. Kassem said. “We identified Tafa2 — a recently discovered neurokine involved in cell migration in the central nervous system — in a screening experiment using chemo-attraction of MSCs as an end point.”

The team was able to demonstrate that Tafa2 stimulates hMSC migration through activation of Rac1-p38 signaling. “We were also able to show that Tafa2 has a chemo-attractant activity for hMSC and is overexpressed at the site of the bone fracture,” Dr. Kassem said.

Upregulation of Tafa2 levels was detected during the inflammatory phase of fracture healing in the mice, as well as in patients with hip fractures. The team found that Tafa2 did not affect the differentiation capacity of the cells, but that it enhanced their proliferation and cell recruitment to sites where bone formation is needed.

“As a result, we think that our findings demonstrate that Tafa2 enhances hMSC migration and recruitment to sites of bone fracture and, thus, is relevant for regenerative medicine applications,” Dr. Kassem said.

Dr. Nolta, Editor-in-Chief of Stem Cells, commented, “From this important report, Tafa2 is identified as one of the key signals needed to recruit MSCs to the site of injury to begin fracture healing. With hip fracture such a major health problem, especially in older patients, this is an important advancement toward improving and hastening healing.”


About the Journal: STEM CELLS, a peer reviewed journal published monthly, provides a forum for prompt
publication of original investigative papers and concise reviews. The journal covers all aspects of stem cells: embryonic stem cells/induced pluripotent stem cells; tissue-specific stem cells; cancer stem cells; the stem cell niche; stem cell epigenetics, genomics and proteomics; and translational and clinical research. STEM CELLS is co-published by AlphaMed Press and Wiley.

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