MedicalXpress

Recipient's immune system governs stem cell regeneration

November 20 2011

A new study in Nature Medicine describes how different types of immune system T-cells alternately discourage and encourage stem cells to regrow bone and tissue, bringing into sharp focus the importance of the transplant recipient's immune system in stem cell regeneration.

The study, conducted at the Center for Craniofacial Molecular Biology at the Ostrow School of Dentistry of USC, examined how mice with genetic bone defects responded to infusions of bone marrow mesenchymal stem cells, or BMMSC.

Under normal conditions, the mice's T-cells produced an inflammatory response and triggered the creation of cellular proteins interferon (INF)-gamma and tumor necrosis factor (TNF)-alpha. These attacked and killed the stem cells, preventing the production of new bone.

"Normally, T-cells protect us from infection," said Professor Songtao Shi, corresponding author for the study, "but they can block healthy regeneration from happening."

However, when the mice were given infusions of regulatory T-cells, or Treg, the levels of the interfering INF-gamma and TNF-alpha decreased, increasing the rate of bone growth and defect repair. Furthermore, administering the anti-inflammatory drug aspirin at the site of the bone defect also increased the rate at which the BMMSCs were able to regrow bone.

Postdoctoral Research Associate and lead author Yi Liu said the findings illustrate the previously unrecognized role of T-cells in tissue regeneration. They also highlight the need for scientists exploring the possibilities of stem cell regeneration to shift their focus to the immune system, she added.

"Based on what we've found, this should be the direction of more research in the future," Liu said.

More information: Yi Liu, Lei Wang, Takashi Kikuiri, Kentaro Akiyama, Chider Chen, Xingtian Xu, Ruili Yang, WanJun Chen, Songlin Wang, and Songtao Shi. (in press) Mesenchymal stem cell - based tissue regeneration is governed by recipient T lymphocytes via IFN-γ and TNF-α. *Nature Medicine* doi: 10.1038/nm.2542

Provided by University of Southern California