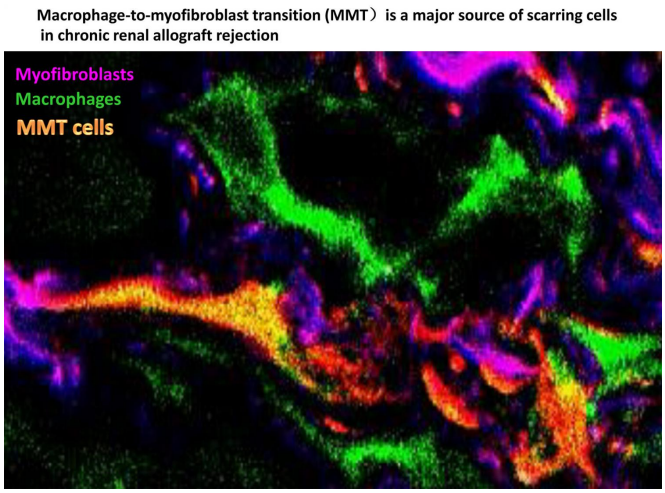
Discovery may help prevent tissue scarring and rejection of transplanted kidneys

16 February 2017



Macrophage-to-myofbiroblast transition is a major cause of scarring in kidney transplant rejection. Credit: Dr. Lan

Researchers have identified a new pathway that likely plays an important role in rejection following kidney transplantation. The findings, which appear in an upcoming issue of the *Journal of the American Society of Nephrology* (*JASN*), point to a promising strategy to help protect the health of recipients and the function of transplanted organs.

Fibrosis, or tissue scarring, is a significant contributor to organ loss after transplantation. Inflammatory immune cells are associated with fibrosis in transplanted kidneys, but how these cells contribute to this damaging response is not clearly understood.

When a team led by Hui Yao Lan MD, PhD (The Chinese University of Hong Kong) and Jiang Hua Chen, MD (Zhejiang University) examined biopsy specimens from patients experiencing kidney rejection, the researchers found that certain immune cells were transforming into connective tissue cells, which produce collagen and other fibers. The extent of this so-called macrophage-to-myofibroblast transition correlated with the severity of fibrosis and with the transplanted kidney's function.

"In this study, we discovered that inflammatory macrophages are an important cell capable of driving the process from acute kidney inflammation to chronic kidney fibrosis during allograft rejection via a new pathway called the macrophage-to-myofibroblast transition," said Dr. Lan.

The macrophage-to-myofibroblast transition was also apparent in mouse transplant models and was mediated through what's known as the TGF-/Smad3 signaling pathway. "These findings suggest that specifically targeting alterative macrophages or the TGF-/Smad3 pathway may help prevent or treat tissue scarring," said Dr. Chen.

More information: "Macrophage-to-Myofibroblast Transition Contributes to Interstitial Fibrosis in Chronic Renal Allograft Injury," DOI: 10.1681/ASN.2016050573

Provided by American Society of Nephrology