

Kidney fibrosis in older transplants links to failure

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Clinically, kidney fibrosis can be used to assess stage, progression, and prognosis for both kidney transplants and kidney disease. There is debate as to whether kidney fibrosis is a maladaptive, injury-triggered process that inherently progresses to kidney failure or an adaptive wound-healing process that stabilizes the injury site.

A new study in the inaugural issue of *JCI Insight* supports the hypothesis that fibrosis in kidney transplants is driven by continuous injury and not the result of an unstoppable cascade of events. Philip Halloran and colleagues at the University of Alberta in Edmonton, Canada performed transcriptome analysis on [kidney biopsies](#) from 681 transplant recipients. There was no evidence of fibrosis in kidneys at the time of transplant.

However, biopsies taken a few months after transplant exhibited mild fibrosis that correlated with the expression of genes associated with [acute kidney injury](#), and this early fibrosis did not associate with progression to failure. Conversely, fibrosis in biopsies taken more than a year after transplant exhibited a different gene signature, infiltration of immune cells, and elevated levels of fibrillar collagen, likely reflecting ongoing injury. Moreover, the presence of fibrosis in older transplants was associated with an increased risk of kidney failure.

This study reveals that detection of fibrosis in transplanted kidneys is indicative of injury and that progression to failure is the result of continuous injury.

More information: Jeffery M. Venner et al. Relationships among injury, fibrosis, and time in human kidney transplants, *JCI Insight* (2016). [DOI: 10.1172/jci.insight.85323](https://doi.org/10.1172/jci.insight.85323)

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