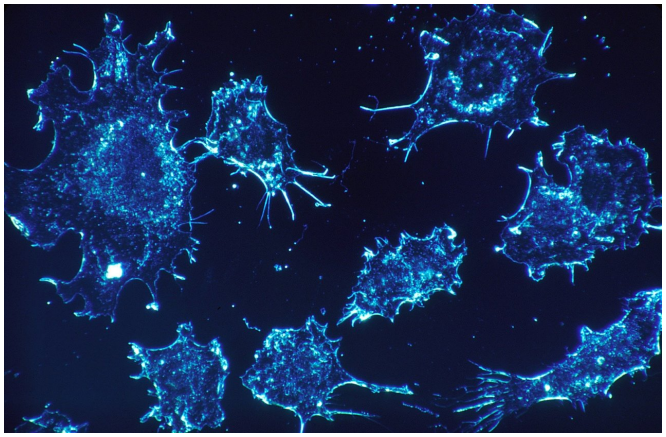
# New window on fibrosis

9 August 2019, by Bill Snyder



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DDR1 is a receptor tyrosine kinase (RTK)—a cell surface receptor—that regulates multiple functions including the maintenance of the normal structure of tissues, but which also contributes to pathological conditions including cancer, inflammation and fibrosis.

In the kidney, upregulation of DDR1 expression contributes to inflammation and progression of fibrotic responses that can damage the kidney. How it does this has been unclear.

Now, in a report in the *Journal of the American Society of Nephrology*, Ambra Pozzi, Ph.D., and colleagues demonstrate that, like other RTKs, activated DDR1 can translocate to the nucleus of the cell, where it acts as a co-transcription factor.

In the nucleus DDR1 regulates the transcription of pro-fibrotic molecules including collagen IV. A structural protein, collagen IV is a component of the extracellular matrix. Together with collagen I, collagen IV is upregulated in fibrosis.

This previously unrecognized nuclear role of DDR1 opens unanticipated therapeutic options for the treatment of fibrotic diseases, the researchers concluded.

**More information:** Manuel Chiusa et al. The Extracellular Matrix Receptor Discoidin Domain Receptor 1 Regulates Collagen Transcription by Translocating to the Nucleus, *Journal of the American Society of Nephrology* (2019). DOI: 10.1681/ASN.2018111160

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