

## Paper highlight: Signaling hope for polycystic kidney disease

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Autosomal Dominant Polycystic Kidney Disease (ADPKD) is a common genetic disease that results in chronic kidney failure.

Although the genes responsible for ADPKD have been identified (PKD1, PKD2), relatively little is known about how mutations in these [genes](#) promote cyst growth molecularly.

In this paper, scientists at Children's Hospital in Boston, lead by Jordon Kreidberg, investigated the signaling pathways that go awry in the disease using mouse [kidney](#) epithelial cells in which Pkd1 was genetically deleted.

They found that the protein c-Met was hyperactive in Pkd1-deficient cells, resulting in increased mTOR signaling, a pathway that had previously been linked to cyst formation. The increase in c-Met activity was related to sequestration of the protein c-Cbl in a cellular compartment known as the golgi, which increased c-Met protein stability.

In support of a critical role for c-Met activity in disease progression, pharmacological inhibition of c-Met decreased mTOR activity and blocked cyst formation in a [mouse model](#) of ADPKD, leading the authors to suggest that c-Met is a potential [therapeutic target](#) in patients with ADPKD.

**More information:** Failure to ubiquitinate c-Met leads to

hyperactivation of mTOR signaling in a mouse model of autosomal dominant polycystic kidney disease: [www.jci.org/articles/view/4153 ... 07331a5bc3d8a1998c3f](http://www.jci.org/articles/view/415307331a5bc3d8a1998c3f)

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