

## Paper highlight: Signaling hope for polycystic kidney disease

September 13 2010

---

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/4153...07331a5bc3d8a1998c3f)

Provided by Journal of Clinical Investigation

Citation: Paper highlight: Signaling hope for polycystic kidney disease (2010, September 13)  
retrieved 15 July 2023 from <https://medicalxpress.com/news/2010-09-paper-highlight-polycystic-kidney-disease.html>

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.