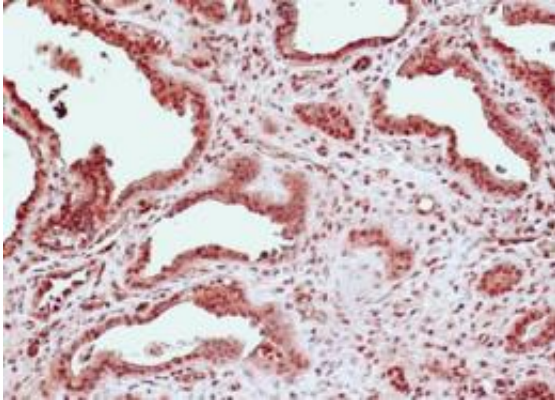


Aurora A may contribute to kidney disease

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Red staining indicates the presence of activated Aurora A kinase in the epithelial cells lining cysts in the kidneys of patients with polycystic kidney disease (PKD). A new study in *The Journal of Cell Biology* suggests that Aurora A may contribute to PKD, a common genetic disease, by inactivating a key calcium channel in kidney cells. Credit: Plotnikova, O.V., and E.A. Golemis. 2011. *J. Cell Biol.* doi:10.1083/jcb.201012061.

The Aurora A kinase may contribute to polycystic kidney disease (PKD) by inactivating a key calcium channel in kidney cells, according to a study in the June 13 issue of *The Journal of Cell Biology*.

Aurora A is an oncogene best known as a regulator of mitotic progression. But the kinase has important functions during interphase as well, when it can promote cilia disassembly and can be activated by elevated calcium levels. Because both [calcium signaling](#) and cilia are defective in PKD, researchers from the Fox Chase Cancer Center in

Philadelphia wondered whether Aurora A might contribute to the pathology of this common genetic disease.

The researchers found that Aurora A was up-regulated and activated in epithelial cells lining the cysts in PKD patient kidneys. In addition, Aurora A bound to and phosphorylated a calcium channel called polycystin-2, whose gene, PKD2, is often mutated in autosomal dominant forms of PKD.

Polycystin-2 mediates the release of calcium from storage in the endoplasmic reticulum and [calcium influx](#) into cilia. Inhibition or knockdown of Aurora A boosted intracellular calcium levels, but this effect was less pronounced in [kidney cells](#) lacking polycystin-2, indicating that Aurora A normally lowers calcium levels by inactivating polycystin-2. Only small doses of inhibitor were required to increase calcium levels, suggesting that Aurora A may be a viable [therapeutic target](#) for boosting polycystin-2 activity in certain PKD patients. Senior author Erica Golemis now wants to investigate how Aurora A becomes up-regulated in PKD and whether inhibitors of the kinase can slow cyst formation in mouse models of the disease.

More information: Plotnikova, O.V., and E.A. Golemis. 2011. J. Cell Biol. [doi:10.1083/jcb.201012061](https://doi.org/10.1083/jcb.201012061)

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