

Renal dopamine signaling prevents kidney injury and improves blood pressure in mice

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Dopamine signaling in the kidneys plays a critical role in blood pressure regulation and is responsible for nearly half of the salt and water excretion that occurs in response to increased dietary salt intake. Notably, genetic deletion of any of the 5 dopamine receptors expressed in the kidney causes hypertension in mice. Additionally, genetic polymorphisms in specific dopamine receptor subtypes are associated with susceptibility to kidney injury in humans.

In this issue of *JCI Insight*, Prasad Konkalmatt and colleagues at George Washington University and the University of Maryland Medical School demonstrate that expression of the D2 dopamine receptor (DRD2) protects mice against kidney injury and hypertension.

Konkalmatt and colleagues found that kidney-specific deletion of DRD2 increased expression of inflammatory and profibrotic factors and elevated blood pressure in mice. Moreover, these effects were exacerbated in a mouse model of kidney ischemia/reperfusion injury. Restoration of DRD2 expression decreased the expression of inflammatory and profibrotic factors, normalized [blood pressure](#), and protected mice from [kidney injury](#).

These findings suggest that DRD2-directed therapies may be useful for the treatment of renal injury.

More information: Prasad R. Konkalmatt et al, Renal rescue of dopamine D2 receptor function reverses renal injury and high blood

pressure, *JCI Insight* (2016). [DOI: 10.1172/jci.insight.85888](https://doi.org/10.1172/jci.insight.85888)

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