JASN® Journal of the American Society of Nephrology

2006 JASN IMPACT FACTOR 7.371

Published ahead of print on November 21, 2007
J Am Soc Nephrol 19: 102-108, 2008

doi: 10.1681/ASN.2007060688

Basic Research

Vasopressin Directly Regulates Cyst Growth in Polycystic Kidney Disease

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Received for publication June 20, 2007. Accepted for publication August 2, 2007.

The polycystic kidney diseases (PKD) are a group of genetic disorders causing renal failure and death from infancy to adulthood. Arginine vasopressin (AVP) V2 receptor antagonists inhibit cystogenesis in animal models of cystic kidney diseases, presumably by downregulating cAMP signaling, cell proliferation, and chloride-driven fluid secretion. For confirmation that the protective effect of these drugs is due to antagonism of AVP, PCK (*Pkhd1*$^{-/-}$) and Brattleboro (*AVP*$^{-/-}$) rats were crossed to generate rats with PKD and varying amounts of AVP. At 10 and 20 weeks of age, PCK *AVP*$^{-/-}$ rats had lower renal cAMP and almost complete inhibition of cystogenesis compared with PCK *AVP*$^{+/+}$ and PCK *AVP*$^{+/-}$ rats. The V2 receptor agonist 1-deamino-8-d-arginine vasopressin increased renal cAMP and recovered the full cystic phenotype of PCK *AVP*$^{-/-}$ rats and aggravated the cystic disease of PCK *AVP*$^{+/+}$ rats but did not induce cystic changes in wild-type rats. These observations indicate that

AVP is a powerful modulator of cystogenesis and provide further support for clinical trials of V2 receptor antagonists in PKD.

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