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# Role of calcium in polycystic kidney disease: From signaling to pathology

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**Abstract**

Autosomal dominant polycystic kidney disease (ADPKD) is the most common inherited monogenic kidney disease. Characterized by the development and growth of cysts that cause progressive kidney enlargement, it ultimately leads to end-stage renal disease (ESRD). Approximately 85% of ADPKD cases are caused by mutations in the *PKD1* gene, while mutations in the *PKD2* gene account for the remaining 15% of cases. The *PKD1* gene encodes for polycystin-1 (PC1), a large multi-functional membrane receptor protein able to regulate ion channel complexes, whereas polycystin-2 (PC2), encoded by the *PKD2* gene, is an integral membrane protein that functions as a calcium-permeable cation channel, located mainly in the endoplasmic reticulum. In the primary cilia of the epithelial cells, PC1 interacts with PC2 to form a polycystin complex that acts as a mechanosensor, regulating signaling pathways involved in the differentiation of kidney tubular epithelial cells. Despite progress in understanding the function of these proteins, the molecular mechanisms associated with the pathogenesis of ADPKD remain unclear. In this review we discuss how an imbalance between functional PC1 and PC2 proteins may disrupt calcium channel activities in the cilium, plasma membrane and endoplasmic reticulum, thereby

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