Autosomal dominant polycystic kidney disease (ADPKD) represents a life-threatening monogenic condition stemming from mutations in PKD1 and PKD2, the genes encoding polycystin 1 (PC1) and polycystin 2 (PC2). PC1 and PC2 are localized to the cilia of renal epithelial cells, and their function is understood to involve an inhibitory activity that suppresses the cilia-dependent cyst activation (CDCA) signal. The deficiency of PCs leads to the activation of CDCA, thereby stimulating cyst growth. Recent studies have demonstrated that re-expression of PCs in established cysts can reverse PKD, suggesting a mode of action akin to a 'counterbalance in cruise control' to maintain lumen diameter within a designated range. This review explores recent findings pointing to novel avenues for future research on PCs with therapeutic potential for treating ADPKD.