SIGNIFICANCE STATEMENT

Pendrin, an integral membrane protein expressed in β-intercalated cells. of the collecting duct, regulates acid-base balance by mediating bicarbonate secretion. There is growing evidence that it is also implicated in regulation of blood pressure and salt balance in part via the renin-aldosterone system (RAAS). Angiotensin II and aldosterone are both known to upregulate pendrin expression. The authors find that in adrenalectomized mice, angiotensin II alone no longer increases renal pendrin abundance unless administered together with aldosterone replacement. Furthermore, in pendrin deficient mice, blood pressure falls on a low salt diet. The results indicate a critical role of the MR-pendrin pathway in BP maintenance during RAAS activation by dietary salt restriction.