The (pro)renin receptor and body fluid homeostasis

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Cao T, Feng Y. The (pro)renin receptor and body fluid homeostasis. Am J Physiol Regul Integr Comp Physiol 305: R104–R106, 2013. First published May 15, 2013; doi:10.1152/ajpregu.00209.2013.—The renin-angiotensin system (RAS) has long been established as one of the major mechanisms of hypertension through the increased levels of angiotensin (ANG) II and its resulting effect on the sympathetic nerve activity, arterial vasoconstriction, water reabsorption, and retention, etc. In the central nervous system, RAS activation affects body fluid homeostasis through increases in sympathetic nerve activity, water intake, food intake, and arginine vasopressin secretion. Previous studies, however, have shown that ANG II can be made in the brain, and it could possibly be through a new component called the (pro)renin receptor. This review intends to summarize the central and peripheral effects of the PRR on body fluid homeostasis.

(pro)renin receptor; body fluid homeostasis; renin-angiotensin system
creas, and the kidney (11). Recent studies show that PRR seems to play a role in RAS activation in peripheral organs. Prorenin has been uniquely found to mature into active renin only in the juxtaglomerular cells of the kidney (14), and PRR has been found to colocalize with renin in vascular structures (11), together suggesting a local role of PRR in the kidneys. Previous studies have also shown that renin has the ability to bind to human mesangial cells in vitro and resulted in hypertrophic effects and increased levels of plasminogen activator inhibitor-1; however, renin was not internalized or degraded (4, 10), supporting a functional role of a renin receptor, PRR, in the renal smooth muscle cells. In a study reported by Burckle et al. (2), human PRR was overexpressed in rat smooth muscle cells. The rat models developed higher systolic blood pressure and increased heart rate with aging, but the kidney function and plasma renin levels showed no significant change (2). There was, however, an increase in plasma aldosterone and the aldosterone-to-renin ratio, suggesting that PRR overexpression resulted in increased intra-adrenal RAS activation, therefore, aldosterone synthesis (2). The effects of aldosterone on sodium and water retention in addition to other indirect influences on water-electrolyte balance are well documented (12).

Perspectives and Significance

PRR, through both ANG II-dependent and ANG II-independent pathways, affects body fluid homeostasis (Fig. 1). PRR acts on both the central nervous system and the peripheral organs through the activation of renin or prorenin and the resulting catalysis of the RAS, affects ANG II formation, sympathetic activity, and the AVP synthesis and secretion and, therefore, affects water and electrolyte balance. On the other

![Fig. 1. Central and peripheral effects of (pro)renin receptor (PRR) on body fluid homeostasis. PRR acts on both the central nervous system and the peripheral organs through the activation of renin or prorenin and the resulting catalysis of the renin-angiotensin system, affects ANG II formation, sympathetic activity, and the arginine vasopressin synthesis and secretion, therefore, affects water and electrolyte balance. See text for more information.](http://ajpregu.physiology.org/)

*Review

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hand, the underlying mechanisms of RAS-unrelated PRR action on body fluid homeostasis need further investigation.

REFERENCES


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