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Positive and negative aspects of cooperative action of angiotensin II and vasopressin in the kidney

Pozytywne i negatywne skutki współdziałania angiotensyny II i wazopresyny w nerkach

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Słowa kluczowe

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Summary

In many instances renin-angiotensin system (RAS) and vasopressin (AVP) systems are activated simultaneously and some components of the RAS and AVP cooperate in the regulation of renal functions via effects exerted either indirectly through the sympathetic nervous system or through active compounds released to the systemic circulation. Growing evidence shows that there is also local cooperation between angiotensin II (Ang II) and AVP in the kidney. Angiotensin II cooperates with AVP in the regulation of water-electrolyte balance and blood pressure in several ways: 1) through a cross-talk between AT1 receptor and V2 receptor signaling pathways which facilitates reabsorption of sodium and water in the thick ascending loop of Henle (TALH) and the collecting duct, 2) through an interaction between AT1 and V1a receptors pathways in the smooth muscles cells of renal glomeruli and vessels, which plays a role in the regulation of glomerular filtration rate and renal medullary blood flow, and 3) through a joint stimulation of release of aldosterone from the adrenal cortex by Ang II and AVP acting on AT1 and V1a receptors, respectively. The aim of this review is to highlight the mechanisms and the targets of these interactions, and to show their significance for blood pressure regulation under physiological and pathological conditions.

Streszczenie

W wielu przypadkach dochodzi do jednoczesnej aktywacji układu renina-angiotensyna (RAS) i układu wazopresyny (AVP), a niektóre związki układów RAS i AVP współpracują w regulacji czynności nerek poprzez działania wywierane bezpośrednio w nerkach lub też za pośrednictwem współczulnego układu nerwowego oraz innych czynników uwalnianych do układu krążenia. Wzrastająca liczba informacji wskazuje, że w samych nerkach również dochodzi do lokalnego współdziałania między angiotensyną II (Ang II) i AVP. W nerkach Ang II współpracuje z AVP w regulacji równowagi wodno-elektrolitowej i ciśnienia tętniczego w różnorodny sposób: 1) poprzez wzajemne oddziaływania między szlakami sygnalizacyjnymi receptorów AT1 i V2 w komórkach grubej części ramienia wstępującego pętli Henlego (TALH) oraz w cewce zbiorczej, dzięki którym jest ułatwiona resorpcja sodu i wody, 2) poprzez liczne interakcje pomiędzy szlakami sygnalizacyjnymi receptorów AT1 i V1a w komórkach mięśni gładkich kłębuszków nerkowych i naczyń rdzenia nerki, które odgrywają istotną rolę w regulacji filtracji kłębuszkowej i przepływu krwi przez rdzeń nerki oraz 3) poprzez wspólne działanie stymulujące Ang II i AVP na uwalnianie aldosteronu z kory nadnerczy w wyniku aktywacji odpowiednio receptorów AT1 i V1a. Celem obecnej pracy przeglądowej jest zwrócenie uwagi na najważniejsze mechanizmy i cele tych interakcji oraz na ich znaczenie w regulacji ciśnienia tętniczego w warunkach fizjologicznych i patologicznych.

Conflict of interest
Konflikt interesów

None
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INTRODUCTION

There is general acknowledgment that the kidney plays a pivotal role in the regulation of body fluid volume and blood pressure, and that angiotensin II (Ang II) and vasopressin (AVP) participate in these regulations through multiple direct and indirect effects exerted on renal blood flow and water electrolyte transport. Excessive activation of RAS and AVP under pathological conditions is involved in the generation of a common set of features typical for a progressive renal damage.

REGULATION OF RAS AND AVP SYSTEMS

All components of the renin-angiotensin system, namely prorenin, renin, angiotensinogen, angiotensin converting enzymes (ACE and ACE2) and other enzymes necessary for formation of a specific angiotensin as well as angiotensin receptors (AT1R, AT2R and Mas) are present in the kidney. The receptors can be stimulated by angiotensin peptides supplied into the kidney from the systemic circulation or by those generated locally in the kidney. Specialized juxtaglomerular (JG) granular cells, which derive from smooth muscle cells, and which are situated in the wall of the afferent arteriole of the kidney, are the most abundant source of prorenin and renin in the body. The JG cells are located in close vicinity to specialized granular epithelial cells of the macula densa of the distal tubule. Both groups of juxtaglomerular and macula densa cells are situated in the angle between the afferent and efferent arteriole. Renin is also synthesized in the principal cells of the collecting ducts (CD) (1-5).

The renal RAS can be activated by multiple hemodynamic, endocrine and chemical factors. Even small decreases of intraglomerular hydrostatic pressure, caused by relaxation of afferent sympathetic arteriole, as well as small decreases of NaCl delivery to the macula densa are able to elicit significant increase of renin release. In addition, sympathetic innervation of the kidney exerts tonic excitatory effect on secretion of renin by JG cells. Nitric oxide (NO) and prostaglandin E2 which are released by the macula densa cells as a result of reduced sodium delivery to the proximal tubule, and which act in the mechanism of the tubuloglomerular feedback, cause increase of cGMP and cAMP in JG cells and subsequent release of renin. Systemic hypovolemia or hypotension are also potent stimuli of intrarenal renin release. They can act directly by decreasing intraglomerular pressure, or indirectly by unloading systemic baroreceptors and subsequent activation of the sympathetic nervous system (1, 4-7). Hypoxia, which can act either directly on the renin-secreting cells or indirectly by stimulating peripheral chemoreceptors, and subsequently activating the sympathetic nervous system, is another powerful stimulus for renin release (8, 9). There is also evidence for stimulation of renin release by vasopressin. Although earlier studies suggested that systemic AVP administration inhibits release of renin, the later investigations demonstrated that this was an indirect effect, related to AVP-induced increase of blood pressure (10). It has been also shown that local stimulations of V2 receptors (V2R) by AVP and of AT1 receptors (AT1R) by Ang II elevate synthesis of renin in CD cells (11, 12).

Vasopressin secreting neurons are located mainly in the supraoptic and paraventricular nuclei of the hypothalamus and are releasing AVP to the systemic circulation and into specific regions of the brain in response to hyperosmolality, Ang II, unloading of baroreceptors, and stimulation of chemoreceptors and/or afferent renal nerves (13-15). Angiotensin receptors located on vasopressinergic neurons can be stimulated either by angiotensin peptides produced locally in the brain or by those reaching the brain from the systemic circulation via the circumventricular organs (16, 17).

ANGIOTENSIN AND VASOPRESSIN RECEPTORS. PATHWAYS OF INTRACELLULAR STIMULATION

In the kidney, AT1R and AT2R proteins or genes were identified in walls of vessels and tubules. In particular, they were found in the arcuate arteries, afferent arterioles and outer medullary descending vasa recta, and in the proximal tubule, thick ascending limb (TALH) of the loop of Henle, and the collecting duct (3, 18, 19). Prolonged exposure of cultured proximal tubule cells to Ang II results in concentration-dependent increases of AT1R density (20). The Mas receptors for Ang-(1-7) are located mainly in proximal tubules and presumably their stimulation acts oppositely to activation of Ang II (21). The studies in which cultured renal cells were exposed to Ang II, ACE inhibitors or AT1R antagonists, and experiments with intrarenal infusions of these compounds provided evidence that Ang II increases contractility of smooth muscle cells and induces vasoconstriction. It has been shown that Ang II constricts afferent and efferent arterioles, reduces the cortical and papillary blood flow, enhances sodium absorption in the proximal tubule and inhibits tubuloglomerular feedback. Prolonged exposure to Ang II may cause renal injury (5, 22, 23).

Similarly as Ang II, vasopressin exerts a variety of biological effects by means of specific G-protein-coupled receptors, designated V1a, V1b and V2. Vasopressin V1aR are necessary for vasoconstriction, cellular proliferation, platelet aggregation and metabolic effects of AVP, whereas V2R participate in the regulation of body fluid homeostasis (24, 25). Receptors of V1aR subtype were found in the interlobular arteries, efferent arterioles, vasa recta, glomerular mesangium, juxtaglomerular apparatus, macula densa, tubular ascending loop of the loop of Henle, and the collecting duct principal and alpha intercalated cells (25, 26). Activation of V1aR initiates cellular responses regulated by phospholipase C/diacylglycerol - IP3-STAT-protein kinase C pathway (27-29).

There is evidence for an interaction between Ang II and AVP in the regulation of smooth muscle tone. Experiments on preglomerular arterioles have shown that both Ang II and AVP produce significant increases in Ca^{2+} intracellular concentration, which depend on stimulation of AT1 and V1 receptors and engages activation of dihydropyridine-sensitive voltage-gated calcium channels (30). Strong evidence argues for positive interaction of AVP and Ang II with regard to calcium mobilization, which means that a combination of subthreshold doses of these peptides elicits significant cytosolic Ca^{2+} mobilization in vascular smooth muscle cells, though separate administration of the same doses is not effective. In addition, application of both peptides simultaneously increases intracellular Ca^{2+} in a more than additive manner (31).

Stimulation of vasopressin V1a and angiotensin II AT1 receptors induces contraction of mesangial cells and vasoconstriction of glomerular afferent and efferent arterioles (32-34). In acute experiments on rats Ang II was found to reduce cortical blood flow whereas vasopressin was effective both in the cortical and papillary regions of the kidney (35). The effect of vasopressin on regional kidney perfusion was mediated by V1aR stimulation (36-38). Experiments of Silva et al., on TALH suspensions from Wistar rats (39) and *in vivo* study of Calzavacca et al. (40) provided evidence that Ang II and AVP have significant, though different impact on oxygen consumption in the kidney. Thus, Ang II was found to decrease renal blood flow, renal oxygen delivery and oxygen consumption and this was associated with a decrease of medullary (but not cortical) pO_2 whereas AVP elicited comparable decreases in RBF and renal oxygen delivery but did not have significant influence on oxygen consumption or pO_2.

There is also significant interaction between AT1 and V2 receptors in the kidney. The strongest expression of V2R was found in the TALH, macula densa, connecting tubule, and cortical and medullary collecting ducts, whereas it was less visible in the cortical TALH and distal convoluted tubule cells (41-43). It is now well established that stimulation of V2 receptors initiates cellular responses activated by adenylyl cyclase – cAMP-dependent protein kinase A and cascade of reactions which cause the translocation of phosphorylated aquaporine-2 (AQP-2) to the cellular membrane (44, 45). Vasopressin V2R are engaged in AVP-induced stimulation of sodium reabsorption in the thick ascending limb of Henle's loop and in increased $Na^+,K^+,2Cl^-$ cotransporter protein expression in this portion of the renal tubule. Stimulation of V2R also causes reabsorption of water and regulates urea permeability in the collecting duct (44, 46, 47). In CD cells a specific V2 agonist dDAVP increases mRNA expression of β an γ subunits of the epithelial sodium channel (ENaC) (48).

Several studies based on elimination of various genes of the RAS and/or vasopressin system indicate that genes of both these systems play important role in the regulation of blood pressure and handling of water and electrolytes by the kidney. It has been shown that the mice deprived of one of the genes of the RAS manifest hypotension, and polyuria with urine concentration defects which are associated with abnormalities in the renal inner medulla structure, including hypoplastic papilla (2, 49, 50). In addition, a growing evidence draws attention to several points of intracellular interactions between vasopressin and angiotensin receptors in the kidney. Earlier studies suggested indirectly that there may be an interplay between stimulation of vasopressin V1aR and expression of Ang II receptors. It has been shown that pharmacological blockade of AVP receptors significantly increases density of glomerular Ang II receptors (51) whereas Ang II was found to increase expression of V2R mRNA in the inner collecting duct (52, 53).

In 2000 the study of Oliverio et al. (54) revealed that the mice lacking AT1aR for Ang II show significant abnormalities in water handling, which are manifested as a defect of urine concentration. As the mice responded with normal increases of plasma AVP concentration to water deprivation, and were able to concentrate the urine (though less efficiently) after administration of V2R agonist DDAVP, the authors suggested that the abnormal urine concentration results from interruption of AT1aR functions in the nephron (54). The authors did not take into account the possibility that AT1aR ablation could result in inadequate stimulation of V2R. Later studies draw attention to significant interaction between Ang II and AVP locally in the kidney and proved that activation of AT1R is necessary for efficient regulation of water, sodium and urea reabsorption executed by vasopressin by means of V2R (55). Namely, it has been shown that blockade of AT1R in rats treated with a selective V2 agonist (DDAVP) results in decreased urine concentration and reduced AQP2 and AQP1 expression. There was also a decrease in expression of NHE3, NCC, and Na-K-ATPase (55).

Subsequent studies of Aoyagi et al. (27, 56) and Birumachi et al. (57) on V1aR (-/-) mice provided evidence that V1aR can participate in the regulation of blood pressure and body fluid balance through multiple effects mediated by RAS and V2R. Thus, this mice excrete greater volume of urine, and show lower GFR, UNaV, and V2R and AQP2 expressions. In addition, they show reduced cAMP generation, and decreased expression of neuronal nitric synthase (nNOS) and cyclooxygenase-2 (COX-2) in macula densa cells, i.e. in the place where these enzymes play important role in the regulation of release of renin. The mice manifested also lower concentrations of plasma renin, angiotensin and aldosterone and reduced expression of renin in granule cells. They also showed reduced AVP-stimulated release of aldosterone (27, 57).

Studying functional consequences of AT1aR knockout, Li et al. (58), were able to demonstrate that in comparison to wild-type mice the AT1R(-/-) mice are hypotensive, excrete more urine with reduced osmolality and have three times lower plasma AVP. The AT1R knockout mice exhibited also reduced amounts of AQP2, adenylyl cyclases III, V and VI and phosphorylated MAP kinases and ERK 1/2 proteins in the inner medulla. Thus, this study provided evidence that stimulation of AT1aR is necessary for fully efficient urine concentrating action of vasopressin. The importance of cooperative action of Ang II and AVP in the regulation of AQP2 trafficking in renal collecting duct principal cells has been confirmed in 2011 by Li et al. (59), who showed that both V2 receptors agonist DDAVP and Ang II can increase expression of AQP2 and trafficking of AQP2 protein to the apical membrane of the principal cell. Furthermore, they found that Ang II, like DDAVP, increases accumulation of AQP2 mRNA in a time-dependent manner and that its effect is mediated by AT1R because It can be eliminated by losartan. Altogether,

the experiments of Li et al. (59) provided evidence that the urine concentrating effect of Ang II engages both AT1 and V2 receptors and that the combined stimulation of these receptors is mediated by cAMP and PKA, and by PKC and calmodulin signaling pathways.

The above studies indicate that in the kidney Ang II cooperates with AVP in the regulation of water electrolyte balance and blood pressure in several ways: 1) through a cross-talk between AT1 and V2 receptor signaling pathways in the TALH and collecting ducts, 2) through an interaction between AT1 and V1a receptors in the smooth muscles of renal glomeruli and vessels, and 3) through a combined stimulatory effect of V1aR and ATR activation on aldosterone release from the adrenal cortex (fig. 1).

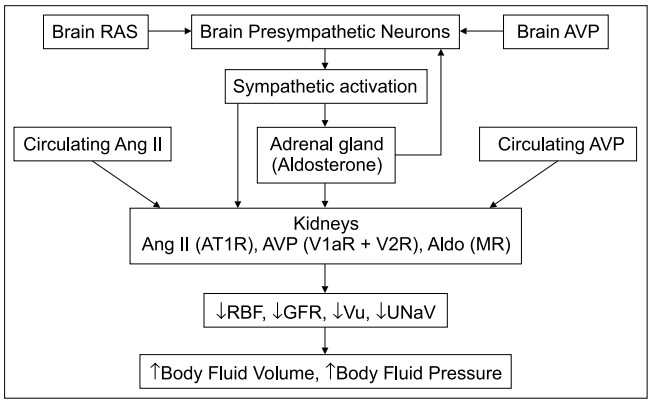

Fig. 1. Effects of joined stimulation of renal angiotensin II (Ang II) AT1 (AT1R) receptors, vasopressin (AVP) V1a (V1aR) and V2 (V2R) receptors, and aldosterone (Aldo) receptors (MR) on body fluid volume and pressure. The figure also shows effects of stimulation of the sympathetic innervation of the kidney by the brain presympathetic neurons which are activated by components of the brain renin-angiotensin system (RAS) and vasopressinergic neurons, and by aldosterone
GFR – glomerular filtration rate; RBF – renal blood flow; Vu – urine volume; UNaV – sodium excretion

SIGNIFICANCE OF INTRARENAL INTERACTION OF ANG II AND AVP UNDER PATHOLOGICAL CONDITIONS

In many pathological conditions, including various types of hypertension, heart failure, nephropathy and diabetes mellitus, there is a parallel elevation of concentration of renin and angiotensin II and AVP or its surrogate biomarker – copeptin in the blood (60-62). Więcek and Kokot were the first who provided evidence for elevated concentration of AVP and Ang II in the same cohort of patients with renovascular hypertension (62). Experimental studies provided evidence that AVP together with Ang II participates in the progression of chronic kidney disease by increasing intraglomerular capillary pressure. It has been also shown that suppression of AVP release or its absence due to genetic defect, is associated with reduced blood pressure and renal hypertrophy. Specifically, reduced glomerular hyperfiltration, glomerulosclerosis and tubulointerstitial fibrosis was found in the rats subjected to ablation of 5/6 of the kidneys mass and in Brattleboro rats with diabetes mellitus, when they were compared to their respective controls (63-66).

Studying effects of prolonged treatment of nephropathy induced by an ablation of 5/6 kidney mass Perico et al. (67) were able to show positive effects of prolonged (39 days) therapy with ACE inhibitor enalapril, AT1R antagonist losartan or dual V1a and V2 vasopressin receptor antagonist (RWJ-676070). Each of these treatments significantly reduced elevation of systolic blood pressure, and diminished proteinuria, glomerulosclerosis, tubular damage and interstitial accumulation of ED-positive cells monocytes/macrophages. Importantly, combination of RWJ 676070 with RAS inhibitors further substantially ameliorated renoprotective effect of RWJ 676070 or enalapril applied alone (68 vs 38% and 56% reduction in proteinuria, respectively). It is worth of noting that the authors introduced the treatments with RAS and AVP inhibitors 21 days after ablation of the kidney mas, when the animals manifested symptoms of overt nephropathy. Thus, the study of Perico et al. (67) suggested that the combined blockade of RAS and AVP systems can be therapeutically beneficial even in an advanced kidney nephropathy. Earlier, similar positive effects of blockade of V1aR with OPC-21268 antagonist and V2R with OPC-31260 antagonist on proteinuria, and glomerular and tubulointerstitial damage were found in adriamycin + DOCA-hypertension nephropathy and in streptozotocin-induced diabetic nephropathy (63, 68-70). In agreement with these findings, administration of V2R agonist DDAVP to five/sixth nephrectomized Brattleboro rats significantly aggravated the symptoms of nephropathy (66).

CONCLUSIONS

In many physiological and pathological circumstances renin-angiotensin system and vasopressin system are activated simultaneously. There is a solid background to believe that angiotensin II and vasopressin closely cooperate in the regulation of transport of water and electrolyte in the kidney, and in the regulation of glomerular filtration and blood flow through the renal medulla. They also jointly stimulate secretion of aldosterone. Under some pathological situations in which RAS and vasopressin are stimulated concurrently there may be a reinforcing interplay between them which may result in worsening of the kidney function.

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