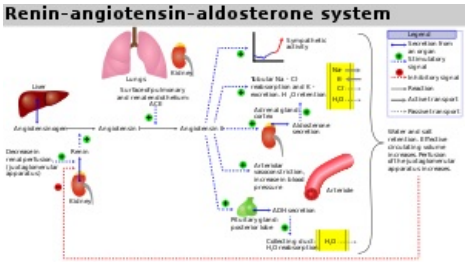
The renin-angiotensin-aldosterone system

The **Renin-angiotensin-aldosterone system** (RAAS, renin-angiotensin-aldosterone axis) is one of the main **neurohumoral** regulators of **physiological homeostasis**. The primary stimulus for its activation is the outflow of **renin** from juxtaglomerular cells, located in the media of afferent renal arterioles. This can happen based on:

- decreased **blood flow** through afferent arteriole (eg hypotension of various etiologies, renal artery stenosis);
- reduction of **NaCl** supply to the macula densa region of the distal tubule;
- activation of **β_1-adrenoreceptors** in the region of juxtaglomerular cells.

Renin secretion is also **stimulated'** by prostaglandins (PGI2, PGE2). On the other hand, renin secretion is **inhibited** by β-blockers.



The renin–angiotensin system (RAS) or the renin–angiotensin–aldosterone system (RAAS)

Mechanisms of transformations

The main function of renin is the cleavage of the decapeptide **angiotensin I** (AGI) from **angiotensinogen**. Histidine and leucine in positions 9 and 10 are subsequently cleaved from angiotensin I by angiotensin-converting enzyme (ACE). This results in the octapeptide angiotensin II (AGII). *Conversion diagram and function of AGII see picture*.

1. AGII causes significant **vasoconstriction** predominantly in the arteriolar blood stream, with a lesser intensity it also affects the veins. Vasoconstriction is most evident in the skin and kidneys. At least, on the contrary, it affects the vessels of the brain and muscles.
2. In the kidneys, AGII leads to **vas efferens constriction** and by a not entirely clear mechanism, it *increases sodium reabsorption in the proximal tubule*.
3. In the adrenal cortex, it **stimulates the release of aldosterone**, which subsequently potentiates the reabsorption of sodium and the excretion of potassium in the distal tubule and collecting duct.
4. In the adrenal medulla, AGII **facilitates the release of catecholamines**.
5. Centrally **increases sympathetic tone** (stimulates the release of catecholamines from nerve endings).
6. In the neurohypophysis, AGII **stimulates the secretion of vasopressin'** (ADH) with subsequent water retention. Most of these effects are mediated by the binding of AGII to the specific AT1-receptor.
7. With a long-term increase in the concentration of AGII, its strong **pro-oncogenic effect'** is manifested - the growth of vascular smooth muscle cells and striated heart muscle is stimulated, the synthesis of collagen increases and the production increases superoxide radicals.

Links

Related Articles

- Kidneys
- Diuretics
- Kidney blood flow and its autoregulation
- Angiotensin converting enzyme inhibitors
- Angiotensin II receptor blockers

Used literature

- MARTÍNKOVÁ, Dahlia – MICHUDA, Stanislav – CERMANOVÁ, Jolana. *Selected chapters in clinical pharmacology for undergraduate studies : Cardiovascular system* [online]. ©2000. [cit. 2010-06-28]. <https://www.lfhk.cuni.cz/farmakol/predn/bak/kapitoly/prednasky/kardio-bak.ppt/>.