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Renin angiotensin system

The renin-angiotensin system (RAS) or the renin-angiotensin-aldosterone system (RAAS) is a hormone system that regulates blood pressure and fluid balance.

When renal blood flow is reduced, juxtaglomerular cells in the kidneys convert the precursor – prorenin, already present in the blood into renin and secrete it directly into the circulation. Plasma renin then carries out the conversion of angiotensinogen, released by the liver, to angiotensin I.

Angiotensin I is subsequently converted to angiotensin II by the angiotensin-converting enzyme (ACE) found in the lungs. Angiotensin II is a potent vasoconstrictive peptide that causes blood vessels to narrow, resulting in increased blood pressure.[3] Angiotensin II also stimulates the secretion of the hormone aldosterone from the adrenal cortex. Aldosterone causes the renal tubules to increase the reabsorption of sodium and water into the blood, while at the same time causing the excretion of potassium (to maintain electrolyte balance). This increases the volume of extracellular fluid in the body, which also increases blood pressure.

If the RAS is abnormally active, blood pressure will be too high. There are many drugs that interrupt different steps in this system to lower blood pressure. These drugs are one of the primary ways to control high blood pressure, heart failure, kidney failure, and harmful effects of diabetes.

The renin-angiotensin system (RAS), the primary regulator of cardiovascular circulation, exhibits local action and works as a paracrine system. In the context of this local regulation, the expression of RAS peptides and receptors has been detected in different kinds of tumors, including gliomas. The dysregulation of RAS components plays a significant role in the proliferation, angiogenesis, and invasion of these tumors, and therefore in their outcomes. The study and potential application of RAS peptides and receptors as biomarkers in gliomas could bring advantages against the limitations of current tumoral markers and should be considered in the future. The targeting of RAS components by RAS blockers has shown potential of being protective against cancer and improving immunotherapy. In gliomas, RAS blockers have shown a broad spectrum for beneficial effects and are being considered for use in treatment protocols. This review aims to summarize the background behind how RAS plays a role in gliomagenesis and explore the evidence that could lead to their use as biomarkers and treatment adjuvants ¹⁾.

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