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Commentary

Comprehensive study on renin–angiotensin system

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ABOUT THE STUDY

The hormone system known as the Renin Angiotensin System (RAS) or Renin Angiotensin Aldosterone System (RAAS) controls systemic vascular resistance, fluid and electrolyte balance, and blood pressure. Juxtaglomerular cells in the kidneys transform the precursor prorenin, which is already present in the blood, into renin and secrete it directly into the circulation when renal blood flow is reduced. Angiotensinogen, generated by the liver, is then transformed into a decapeptide known as angiotensin I by plasma renin. The Angiotensin Converting Enzyme (ACE) is located on the surface of vascular endothelial cells, primarily those in the lungs, and converts angiotensin I into angiotensin II. Angiotensin II only lasts for one to two minutes. Angiotensinases, which are found in red blood cells and vascular beds in many tissues, then quickly degrade it into a heptapeptide known as angiotensin III.

Angiotensin III, which has 100% adrenocortical stimulating action and 40% vasopressor activity of angiotensin II, raises blood pressure and causes the adrenal cortex to secrete more aldosterone. Additionally, angiotensin IV has vasopressor and adrenocortical functions. A powerful vasoconstrictive peptide, angiotensin II raises blood pressure by constricting blood arteries. The hormone aldosterone is stimulated by angiotensin II to be released from the adrenal cortex. Aldosterone increases the amount of sodium that is reabsorbable by the renal tubules, which leads to the reabsorption of water into the blood and the excretion of potassium. The body's extracellular fluid volume rises as a result, raising blood pressure as well. The RAS will function inappropriately if the blood pressure is too high. A number of different types of medications, such as ACE inhibitors, renin inhibitors, and angiotensin II receptor blockers, interfere with various processes in this system to lower blood pressure. These medications are a key component in managing diabetes-related complications such as high blood pressure, heart failure, renal failure, and others.

Local renin–angiotensin system: Numerous tissues, such as the kidneys, glands, the heart, the vasculature, and the nervous system, have locally expressed renin-angiotensin systems. These systems have a variety of functions, including local cardiovascular regulation, either in conjunction with or independently from the systemic renin-angiotensin system, as well as non-cardiovascular ones. While prorenin, the precursor to renin, is highly expressed in tissues and accounts for more than half of the circulating prorenin, its physiological function beyond acting as a precursor to renin is still unknown. Renin is primarily taken up from the circulation outside of the kidneys but may also be secreted locally in some tissues. Angiotensinogen is taken up from the circulation outside of the liver or expressed locally in some tissues; it combines with renin to generate angiotensin I, which can then be converted into angiotensin II by locally expressed angiotensin-converting enzyme, chymase, or other enzymes. Either an intracellular or an interstitial procedure can take place. It is probably involved in the paracrine control of aldosterone secretion in the adrenal glands; remodelling or vascular tone in the heart and vasculature; and possible local blood pressure regulation in the brain, where it is essentially independent of the circulatory RAS.

Angiotensin can also be used for sympathetic neurotransmission by the central and peripheral nervous systems. The digestive system, the skin, and the reproductive system are other sites of expression. Drugs that target the systemic system may have positive or negative effects on how those local systems express themselves.

Foetal renin–angiotensin system: Because angiotensin II has little to no impact on aldosterone levels, the renin-angiotensin system in the foetus is primarily a sodium-losing mechanism. Because of the restricted pulmonary blood flow, the renin levels in the foetus are high while angiotensin II levels are noticeably lower. This prevents ACE, which is mostly present in the pulmonary circulation, from acting to its full potential.

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