JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue
JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR)
An International Scholarly Open Access, Peer-reviewed, Refereed Journal

"Study of Role of Renin with special reference to Physiology –A Literature Review."

[1.] Dr. Raghavendra Nadargi
Professor,
Department of Kriya sharir,
Shivajirao Pawar Ayurved Medical College and Research Centre, pachegao, Tal., Shrirampur, Dist., Ahmednagar, Maharashtra, India..

[2] Dr.Tambe Geeta Shridhar
Associate professor,
Department of Kriya sharir,
Shivajirao pawar ayurved medical College and research centre, pachegao,Tal. Shrirampur , District Ahmednagar, Maharashtra, India.

Abstract:

Renin (etymology and pronunciation), also known as an angiotensinogenase, is an aspartic protease protein and enzyme secreted by the kidneys that participates in the body's renin–angiotensin–aldosterone system (RAAS)—also known as the renin–angiotensin–aldosterone axis—that increases the volume of extracellular fluid (blood plasma, lymph and interstitial fluid) and causes arterial vasoconstriction. Thus, it increases the body's mean arterial blood pressure.

Renin is not commonly referred to as a hormone as well as enzymatic activity with which it hydrolyzes angiotensinogen to angiotensin .

Renin is a central hormone in the control of blood pressure and various other physiological functions. So this article is focusing on Renin function with special reference to physiology.

Key words: Renin, Physiology.

Introduction:

Renin, enzyme secreted by the kidney (and also, possibly, by the placenta) that is part of a physiological system that regulates blood pressure. In the blood, renin acts on a protein known as angiotensinogen, resulting in the release of angiotensin I. Angiotensin I is cleaved by angiotensin-converting enzyme, splitting off two amino acids from the 10-amino-acid chain of angiotensin I, to form angiotensin II. The resultant angiotensin II octapeptide (previously called hypertensin, or angiotonin) acts via receptors to constrict arterioles, causing a rise in both systolic and diastolic blood pressure. Angiotensin II is one of the most active vasoconstrictors known; on a weight basis it is significantly more potent than norepinephrine. It also increases the secretion of cortisol and aldosterone by a direct action on the adrenal cortex. Renin was discovered in 1898 by Robert Tigerstedt and Per Bergman, researchers at the Karolinska Institute in Stockholm.[1]

Renin is a central hormone in the control of blood pressure and various other physiological functions. In spite of the very early discovery of renin over 100 years ago, we have only recently gained a deeper understanding of the origin of renin-producing cells and of the mechanisms responsible for renin synthesis and secretion. The main source of renin is the juxtaglomerular cells (JGCs), which release renin from storage granules. Besides the renin-angiotensin system (RAS) in the JGCs, there exist local RASs in various tissues. [2]

Objectives:

To study of Role of Renin with special reference to Physiology.

Methods:

Literature Survey Methods:

Renin**,** also known as angiotensinogenase, is an aspartyl protease and belongs to the protein family peptidase A1. Aspartyl proteases are endopeptidases that typically use two aspartate residues in the active site to specifically cleave peptide substrates using an acid-base hydrolysis mechanism. Mature renin circulates in the blood stream and contains 340 amino acid residues and has a mass of approximately 37 kDa. The function of renin is to cleave angiotensinogen to produce angiotensin I.[3]

1. Physical inactivity:[4]

Renin is secreted by the kidneys. The kidneys act both directly and indirectly to regulate arterial blood pressure and provide the major long term mechanism of blood pressure and control. The direct mechanism changes blood volume independently of hormones. When blood pressure and blood volume increase the kidneys can not filter all of the liquids and thus liquids are lost in the urine to decrease blood pressure and blood volume.

The indirect mechanism, or the renin-angiotensin system (RAS), controls blood volume and blood pressure through renin and two forms of angiotensin. Renin is involved in the rate limiting first step of a cascade that eventually produces angiotensin II. The specialized granular cells of the juxtaglomerular apparatus secrete renin when stimulated by the macula densa when blood pressure or blood volume decreases. Renin circulating in the blood stream cleaves a small 10 residue portion of plasma protein angiotensinogen that is secreted by the liver. Cleavage of angiotensinogen produces the inactive precursor angiotensin I that is converted to angiotensin II by angiotensin-converting enzyme primarily in the lungs. Angiotensin II increases blood pressure in three ways.

- Angiotensin II constricts blood vessels by influencing smooth muscle tissue. The heart pumps faster to overcome the constricted arteries and blood pressure rises.
- Angiotensin II stimulates the adrenal cortex to secrete aldosterone that causes renal adsorption of sodium. When sodium moves into the blood stream water follows to increase the blood volume.
- Angiotensin II causes the posterior pituitary gland to release vasopressin, also known as antidiuretic hormone, to induce water reabsorption.

Secretion & function: [5]

The enzyme renin is secreted by pericytes in the vicinity of the afferent arterioles and similar microvessels of the kidney from specialized cells of the juxtaglomerular apparatus—the juxtaglomerular cells, in response to three stimuli:

1. A decrease in arterial blood pressure (that could be related to a decrease in blood volume) as detected by baroreceptors (pressure-sensitive cells). This is the most direct causal link between blood pressure and renin secretion (the other two methods operate via longer pathways).
2. A decrease in sodium load delivered to the distal tubule. This load is measured by the macula densa of the juxtaglomerular apparatus.
3. Sympathetic nervous system activity, which also controls blood pressure, acting through the β_1 adrenergic receptors.

Other functions:

Additionally, angiotensin II triggers the sensation of thirst.

The release of renin into the blood stream ultimately raises blood pressure. The kidneys restore and maintain blood pressure homeostasis by regulating blood volume through the action of renin. Although blood volume varies with age, body size, and sex, renal mechanisms usually maintain it to 5 liters.[6]

Discussion:

. Renin secretion is stimulated by a decrease in arterial blood pressure, a decrease in sodium chloride levels in kidney nephrons, or sympathetic nervous system activity.

Renin can be referred to as both a hormone and an enzyme because it displays properties of both. Renin is secreted by cells in the kidneys, circulates in the bloodstream, binds to the renin receptor, and ultimately helps to regulate the body's blood pressure.

The renin angiotensin system (RAS) is a peptide hormone system that is composed of various enzymes, as well as inactive and active peptides that play pivotal roles in the regulation of fluid homeostasis, blood pressure, and cardiovascular function.[7]

Renin secretion and renin synthesis are coordinated, but they are regulated on different time scales. Whereas renin secretion from the kidneys can change within seconds, changes in renin synthesis require several hours. This time gap creates no problem, however, since the kidneys contain enormous stores of renin, sufficient to maintain a normal renin secretion rate over days, even if *de novo* synthesis of renin ceased.

Renin secretion and synthesis are essentially triggered by sympathetic nerve activity with noradrenaline as the main stimulatory transmitter acting via β1 receptors on JGE cells. Renin secretion and synthesis are further controlled by several negative feedback loops.[8]

Conclusions:

1. Renin hormone enzyme initiates the enzymatic cascade generating the angiotensin peptides that regulate blood pressure, cell growth, apoptosis and electrolyte balance, to mention only some of the foremost-recognized functions. Renin is rate limiting in the production of angiotensin II (Ang II), a hormone that ultimately integrates cardiovascular and renal function in the control of blood pressure as well as salt and volume homeostasis.
2. Renin can be used for the treatment of hypertension.
3. In current medical practice, the renin–angiotensin–aldosterone system's overactivity (and resultant hypertension) is more commonly reduced using either ACE inhibitors (such as ramipril and perindopril) or angiotensin II receptor blockers (ARBs, such as losartan, irbesartan or candesartan) rather than a direct oral renin inhibitor.

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