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CARDIOMETABOLIC EFFECTS OF RENIN-ANGIOTENSIN SYSTEM

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Renin-angiotensin system (RAS) is the combination of enzymes, peptydes and receptors playing a great role in regulation of important homeostatic function of organism. In modern consideration have been distinguished two axis RAS – classical and non-classical. The classical RAS axis includes next components: angiotensinogen, renin, angiotensin-converting enzyme (ACE), angiotensin II, receptors type 1 (AT_1R), receptors type 2 (AT_2R). Dysregulation of classical RAS is accompanied by formation of cardiovascular pathology. The main player RAS is angiotensin II which via the AT_1R increases sympathetic nervous system tension, exerts renal sodium reabsorption and potassium excretion, promotes inflammation, oxidant stress. Angiotensin II is critical to elevation of blood pressure levels depending from contraction of vascular smooth muscle cells. Arterial hypertension is the dominant effect of angiotensin II/ AT_1R. Stimulation of AT_1R causes pro-fibrotic cascade in heart and vessels resulted in myocardial hyperthrophy and vessels wall remodeling. Components of classical RAS are responsible for glucose metabolism with negative effect provoking resistance to insulin, incident type 2 diabetes mellitus [2, 3]. Beyond the border of classical RAS the concept of alternative RAS have been formed with such components angiotensin-converting enzyme 2 (ACE 2), angiotensin -(1–7), angiotensin -(1–9), Mas-receptor [5]. ACE 2 is biological active peptide and serves as key modulator of the RAS in physiological condition and pathological state regulating of angiotensin II activity due to its property converts one to other molecules angiotensin-(1–7), angiotensin-(1–9) that diminished the effects of angiotensin II.

Multiple pathways of non-classical RAS related beneficial effects associated with control of blood pressure, regulating metabolic process, anti-inflammation, anti-oxidant properties providing the defense some organs. [6]. Cardio-protective features of ACE 2, angiotensin -(1–7) reflect in inhibition of fibrosis, suppressing myocytes grows, pathological hypertrophy, myocardial remodeling in patients due to the adverse influences of angiotensin II both in formation of hypertensive heart and in outcomes of myocardial infarction. [4]. According to results of experimental research anti-fibrotic properties of alternative components RAS has been provided the preventive role under development of heart failure [7]. Alternative branch of RAS corresponds with metabolic disorders correction. Data about facilitative actions of angiotensin-(1-7)/Mas receptor on glucose metabolism increasing skeletal muscle glucose uptake and improving insulin sensitivity in experimental animals have been shown [1].

Hence should be established future research in patients directed to identification the potential relationship of components RAS activation and depression for comprehensive understanding the pathogenesis of widespread internal diseases and implementation the advanced knowledge in clinical practice.

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