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 Biometeorological influence on cardiovascular mortality

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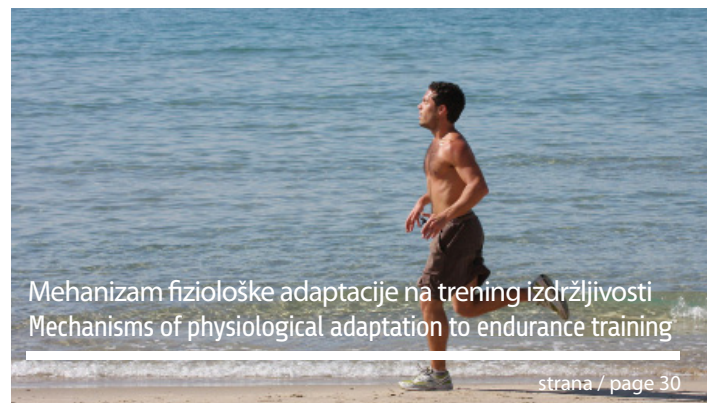
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 regeneraciju nervne
 ćelije

Key Protein Discovered
 That Allows Nerve Cells
 to Repair Themselves

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Klinički značaj blokade renin-angiotenzin-aldosteron sistema u sprečavanju progresije hronične bolesti bubrega

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APSTRAKT

Rad je imao za cilj da utvrdi patogenetske mehanizme uticaja renin-angiotenzin-aldosteron sistema na progresiju hronične bolesti bubrega i da ukaže na klinički značaj blokade renin-angiotenzin-aldosteron sistema. Ovaj sistem ima značajnu ulogu u razvoju glomeruloskleroze i ožiljavanju tubulointersticijuma. Analizirani su publikovani radovi i kliničke studije koje se bave renin-angiotenzin-aldosteron sistemom, progresijom hronične bolesti bubrega i lekovima koji blokiraju aktivnost renin-angiotenzin-aldosteron. Podaci publikovanih studija ukazuju da, pored sistemskog cirkulišućeg RAAS-a, u bubregu je aktivan i lokalni / tkivni RAAS. Renin deluje na angiotenzinogen pri čemu nastaje angiotenzin I; međutim, renin svoje dejstvo ostvaruje i preko receptora za (pro)renin-(P)RR, čije je prisustvo dokazano na mezangijumskim ćelijama piodocitima. Renin se vezuje za receptor i aktivira mezangijumske ćelije dovodeći do aktivacije MPAK (mitogen-activated protein kinase)-ERK („extracellular signal regulated“ kinaze) puta, povećanog stvaranja i oslobađanja proožiljnih medijatora, uključujući transformišući faktor rasta beta 1 (TGFβ1), inhibitor aktivatora plazminogena (PAI-1) i komponente vanćeliskog matriksa (fibronektin i kolagen-1). U podocitima stimulacija (pro)renin receptora dovodi do aktivacije MAPK-ERK puta i povećanog intracelularnog stvaranja angiotenzina II, koji svojim hemodinamskim i drugim dejstvima doprinosi ožiljavanju parenhima bubrega. Blokada renin-angiotenzin-aldosteron sistema smanjuje proteinuriju i usporava progresiju hronične bolesti bubrega.

Ključne reči: renin-angiotenzin sistem; nefroskleroza; inhibitori angiotenzin-kovertirajućeg sistema.

The clinical significance of blockade of renin-angiotensin-aldosterone system in the prevention of chronic kidney disease progression

APSTRAKT

Renin-angiotensin-aldosterone system plays an important role in the development of glomerulosclerosis and tubulointerstitial scarring. The paper was designed to describe the pathogenic mechanisms of influence of the renin-angiotensin-aldosterone in the progression of chronic kidney disease and to show the clinical significance of blocking the renin-angiotensin-aldosterone system. The published research and the clinical studies were analyzed, dealing with the renin-angiotensin-aldosterone, the progression of chronic kidney disease and drugs that block the activity of the renin-angiotensin-aldosterone system. The data of published studies indicate that, in addition to systemic circulating RAAS, there is an active local-tissue RAAS in the kidney too. Renin acts on angiotensinogen, after which angiotensin I appears, however, renin acts on its own receptors called (pro)renin-(P)PR, whose presence was proved on mesangial cells and podocytes. Renin binds to receptor and activates mesangial cells, resulting in activation of MPAK (mitogen-activated protein kinase)-ERK (extracellular signal-regulated transforming and release of pro-scarring mediators, including transforming growth factor beta 1 (TGFβ1), plasminogen activator inhibitor (PAI-1) and components of extracellular matrix (fibronectin and collagen-1). Within podocytes the stimulation of (pro)renin receptors leads to activation of MAPK-ERK pathway and increased intracellular synthesis of angiotensin II, which, through its hemodynamic and other effects, contributes to scarring of kidney parenchyma. Blockade of renin-angiotensin-aldosterone system decreases proteinuria and slows the progression of chronic kidney disease.

Key words: renin-angiotensin system; nephrosclerosis; angiotensin-converting enzyme inhibitors.

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