Stres i kardiovaskularni sustav

Stress and the cardiovascular system

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Postoji više pretpostavki kojim mehanizmima stres utječe na kardiovaskularni sustav (KV) u cijelosti: kemijskim stresorima, fizikalnim i/ili biološkim. Svi oni djeluju putem cirkulirajućih hormona, staničnih receptora, stresom aktiviranih kinaza i proteina te enzima koji održavaju oksidativno-redukcijsku ravnotežu u KV sustavu. Krajnji rezultat je sinteza molekula koje su biološki odgovor: utječu na vaskularni tonus, arterijski tlak, frekvenciju srca, koagulabilnost i adheziju trombocita. Postoje razlike između akutnog odgovora na stres i kroničnog stresa: visoke koncentracije „stresnih hormona“ dovode do promjene u ekspresiji gena i promjena u funkciji ciljnih stanica. Na unutarstaničnoj razini glukokortikoidi, mineralokortikoidi, estrogeni i androgeni djeluju vezujući se na steroidne receptore i posljedično reguliraju fosforilaciju stresom aktiviranih kinaza. To dovodi do promjena u transkripciji gena, promjena u metabolizmu glukoze i dr. proteina te do promjena u energetske homeostazi KV sustava. Kaskada enzima kinaza tijekom fosforilacije je dio molekularnog odgovora na signale stresa, koji djeluju putem membranskih receptora (adrenoreceptora, receptora za hormon rasta) i posljedično putem cAMP, cGMP ili inozitol-trifosfata. Stresom aktivirane fosfokinaze (SAPK) nisu uključene samo u upalne procese, već i u formiranje aterosklerotskih promjena i postishemičnih ozljeda srca.

There are several hypothesized mechanisms by which stress might trigger changes in cardiovascular (CV) system: chemical stressors, physical and/or biological 'stressors'. All of them are acting through endogenous pathways with complex interaction between the circulating hormones, cellular receptors, stress-activated phosphoprotein kinases, stress-induced proteins, and enzymes maintaining oxidoreductive balance in the CVS. Final result is that stressor's message translates into biological responses: increase in blood pressure, heart rate, vascular tone, and platelet aggregability. There are differences between acute and chronic stress: high glucocorticoid (GCs) and mineralocorticoid (MCs) concentrations in chronic stress lead to substantial changes in expression of genes regulating reception, transmission, processing and response to other cellular signals. At the intracellular level, the steroids, GCs, MCs, estrogens, and androgens exert their action through binding to the respective steroid receptor (SRs) and subsequently regulating functions of SRs are controlled through phosphorylation by the stress-activated kinases. Such modifications lead to sequential recruiting of different components of the transcription machinery to the promoter regions of the genes under steroid regulation. It is known that GR primarily regulates genes of the intermediate metabolism of glucose thus maintaining the energy homeostasis of the CVS. Protein phosphoprotein kinase cascade is a part of molecular mechanism, which enables adaptive CVS response to the stress signals. It is triggered in response to activation of receptor systems from the cell membrane, such as adrenoreceptors, growth factor receptors and others, acting via cAMP, cGMP or inositol-triphosphate mechanism. Stress activated phosphokinases (SAPK) are not only included in regulation of the inflammatory processes, but also in progression of the atherosclerotic plaques and postischemic cardiac injuries.

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