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arginine: NO system, cyclooxygenase metabolites of arachidonic acid, as well as, their interactions in the control of CA of the isolated rat heart.. In our study rat hearts autoregulate CF between 50 and 90 cm H2O of CPP. Basal release (at 60 cm H2O) of NO (as nitrite), cAMP, cGMP and HX+X (i.e. adenosine) amounted to 2.85+/-0.25 nmol/min/g wt, 29.45+/-2.22 pmol/min/g wt, 0.43+/-0.08 pmol/min/g wt and 37.50+/-2.89 nmol/min/g wt respectively. Release of NO, cAMP and cGMP were strictly parallel with CPP-CF curve, while release of adenosine (i.e. HX + X) was an inverse function of perfusion pressure. Inhibition of NOS (L-NAME, 30 μM) significantly widened autoregulatory range (40-100 cm H2O), with significant reduction in CF and NO- and cGMP release, while release of cAMP was completely reversed in the presence of L-NAME. However, inhibition of cyclooxygenase (indomethacin, 3 μM) didn't influence autoregulatory range, with similar changes of NO- and cAMP-release and completely inversed values of released adenosine. When L-NAME and indomethacin where added together, they exhibit interactions between these two enzymatic systems. Namely, when L-NAME was added first, indomethacin didn't influence hemodynamic effects of NOS-inhibitor. On the other hand, when COX-inhibitor was added first, L-NAME widened autoregulatory range in small manner as after control autoregulatory experiments (40-90 cm H2O). All hemodynamic changes were followed with similar changes in NO-release, what suggest that exist interaction between L-arginine: NO system and COX-metabolites in the regulation of coronary autoregulation.

A18.

LIPIDNI I ANTIOKSIDACIONI STATUS U ANIMALNOM MODELU EKSPERIMENTALNE ATEROSKLEROZE

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Postoji veliki broj podataka o pokušajima uspostavljanja pouzdanog animalnog modela ateroskleroze, ali još nije poznat dovoljno dobar analog humanoj aterosklerozi. Kao prilog ovim istraživanjima, pokušali smo da procenimo doprinos reaktivnih vrsta kiseonika (ROS) u ranoj aterogenezi i preventivnu ulogu endogenog antioksidacionog zaštitnog sistema (AOS). Odrasli mužjaci Wistar albino pacova, telesne mase oko 400 g, korišćeni su za sprovođenje specijalne aterogene dijete koja se sastojala od smese masti (buter, holesterol) i drugih supstanci (holna kiselina, holin hlorid, tiouracil). Kontrolne i eksperimentalne životinje su bile smeštene u plastične kaveze i imale su dostupne peletiranu hranu i vodu po volji. Eksperimentalne životinje su dobijale svakoga dana u toku 8 nedelja po 5 g aterogene smeše, sondom, intragastrično. Na kraju dijete sve životinje su žrtvovane i sakupljena je krv za određivanje (1) **lipidnog statusa**: koncentracija triglicerida (TG), ukupnog holesterola (CH). LDL holesterola i HDL holesterola u plazmi, odnosa koncentracija LDL i HDL holesterola (LDL/HDL) i (2) **antioksidacionog statusa** u krvi: aktivnost katalaze u eritrocitima (Er CAT), aktivnost glutation-S-transferaze (GST) u plazmi, koncentracija glutationa (GSH) u plazmi i koncentracija vitamina E (Vit. E) u plazmi. Uzimani su isečci abdominalne aorte za histološku verifikaciju ateroskleroze. Razlike između vrednosti kontrolnih i eksperimentalnih životinja testirane su statistički.

Analiza hitoloških praparata aorte pokazala je normalnu građu arterijskog zida u kontrolnih životinja, dok su u eksperimentalnih životinja nađene inicijalne promene u intimi (zadebljanje i ćelijska akumulacija). Vrednosti svih praćenih varijabli lipidnog i antioksidacionog statusa u kontrolnih životinja bile su normalne. U eksperimentalnih životinja postojalo je statistički značajno povećanje koncentracije TG ($p < 0,05$), koncentracije CH ($p < 0,01$), koncentracije LDL ($p < 0,01$) i odnosa LDL/HDL ($p < 0,01$) u poređenju sa kontrolnim životinjama. Koncentracija HDL bila je nepromenjena ($p > 0,05$). Aktivnost Er CAT i koncentracija GSH u plazmi bile su povećane u eksperimentalnih životinja u odnosu na kontrolne, ali ne značajno ($p > 0,05$). Aktivnost GST u plazmi nije bila promenjena ($p > 0,05$). Samo koncentracija Vit. E u plazmi eksperimentalnih životinja bila je statistički značajno veća u poređenju sa kontrolnim životinjama ($p < 0,01$). Možemo zaključiti da antioksidacioni status u krvi nije bio u korelaciji sa lipidnim statusom u toku rane aterogeneze u odraslih pacova.

A19.

THE NEUROBIOLOGY OF SOMATOSTATIN

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The purpose of this study is to describe the recent findings on the neurobiology of somatostatin, with special emphasis on its multiple neuronal and neuroendocrine functions. Since its discovery in 1968, as the neuroendocrine hormone responsible for inhibiting growth hormone (GH) secretion, our understanding of the function of somatostatin (SRIH), both in the CNS and endocrine system has grown enormously.

Somatostatinergic neurons occur in high densities throughout the CNS, particularlly in hypothalamus – median eminence, cerebral cortex, hippocampus, striatum, and different areas of the limbic system. According to their locations, somatostatinrgic neurons