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RHO-KINASE ACTIVATION CONTRIBUTES TO THE FORMATION OF HIGH BLOOD PRESSURE IN RAT MODEL

Our aim was to characterize the spontaneously hypertensive stroke prone (SHRSP), and the normotensive wistar kyoto (WKY) rat strains, to find the possible difference in their blood pressure and cardiac system.

In our experiments, we detected the diameter changes of cannulated muscle arterioles, and the contractile force of the femoral artery. We also measured the body and the heart weight, and also the blood pressure. The body weight of the SHRSP rats (257±2g) was smaller than the weight of the WKY rats (361±10g, $p<0,01$). The heart/body ratio (0,0047), the systolic (210±12 Hgmm) and diastolic (161± 8 Hgmm) blood pressure of the SHRSP was significantly higher than we found in WKY rats (0,0027, 147±9Hgmm, 115±7 Hgmm). The myogenic tone of the muscle arterioles was similar in the two groups. After pretreatment with Rho-kinase inhibitor fasudil (10 uM) the myogenic tone decreased more in the SHRSP group (82,5±6,2%) than in the WKY group (34,4± 9,8 %). Pretreatment with other substances, such as acetylcholine, norepinephrine, serotonin, and U46619 we did not find any difference, not even after adding fasudil to the system.

In the case of the femoral artery, treatment with 10-60 mM KCL resulted in higher contractile force in SHRSP (10,33±1,7mN) than in WKY (6,53±1,3mN). Pretreatment with fasudil resulted in the normalisation of the higher contractile force in the SHRSP group (decreased to 6,3 ±1,8mN), but had no effect on the contractile force of the WKY group (6,3±1,6mN). In case of using serotonin we detected similar reaction. (contraction for SHRSP: 13,7±1,6 mN, which decreased to 9,3±1,5mN in the presence of fasudil, contraction for WKY: 8,7±2mN, which was 8,9±1,8mN after using fasudil.)

According to our results, the Rho-kinase system probably plays an important role in the pathogenesis of high blood pressure, and better understanding of the pathogenesis may lead to new and hopefully more effective drug development.