P5: ERYTHROCYTE DEFORMABILITY AND NITRIC OXIDE PRODUCTION IN ANIMAL MODEL OF PRIMARY HYPERTENSION AND THEIR AGE-DEPENDENT CHANGES

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However, stress failed to affect significantly neurogenic contractions of SMA elicited by electrical stimulation of perivascular sympathetic nerves and vasoconstriction induced by exogenous noradrenaline in SMA.

Conclusion: In conclusion, chronic social stress can accelerate the development of hypertension in BHR, which seems to be associated with NO-independent endothelial dysfunction in small resistant arteries. Supported by the grants VEGA No. 2/0190/17 and APVV-16-0263.

Abstracts

P5 ERYTHROCYTE DEFORMABILITY AND NITRIC OXIDE PRODUCTION IN ANIMAL MODEL OF PRIMARY HYPERTENSION AND THEIR AGE-DEPENDENT CHANGES

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Objectives: Reduced deformability of red blood cells (RBC) plays an important role in etiology of various diseases including cardiovascular. The nitric oxide (NO) was identified as one of factors responsible for maintenance of RBC deformability. Reduced bioavailability of NO might be important in the pathogenesis of hypertension. The aim of present study was to determine the effect of hypertension and aging on RBC deformability and NO production of experimental animals.

Methods: Spontaneously hypertensive rats (SHR) and normotensive Wistar-Kyoto (WKY) rats were divided into 6 groups according to age (7, 20 and 52 weeks) and strain: SHR-7, SHR-20, SHR-52, WKY-7, WKY-20, WKY-52. Blood was used for determination of RBC deformability using filtration method and NO production in RBCs using fluorescent NO probe DAF-2 DA.

Results: We found reduced deformability at WKY-52 and SHR-52 as compared to strain-matched 20-week-old animals. Strain-related differences in deformability were observed at 7 and 52 weeks of age, where the SHR-7 had reduced deformability and the SHR-52 had increased deformability as compared to age-matched WKY. We have found that at younger age, deformability and NO production in RBCs was able to increase, while in the older age there was a decrease in both parameters.

Conclusions: Changes in the RBC deformability under hypertensive conditions are unlikely to be related to changes in NO production. On the other hand, age-related changes in deformability of both, WKY and SHR are at least partially associated with changes in NO production. Supported by grants VEGA 1/0032/14 and Slovak Society of Cardiology.

P6 ANGIOTENSIN AT2 RECEPTOR AGONIST, COMPOUND 21, MAINTAINS VASCULAR INTEGRITY AND PREVENTS ABDOMINAL AORTIC ANEURYSM PROGRESSION IN THE RAT

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The effects of the selective angiotensin AT2 receptor agonist, compound 21 (C21), on abdominal aortic aneurysm (AAA) formation were investigated in norepinephrine-stimulated rat aorta.

AAA was induced by perfusion of isolated aortic segments with elastase (Anidjar/Dobrin model). Treatment with C21 (0.03 and 0.3 mg/kg daily) was started after surgery and continued for 14 days. Sham operated animals and vehicle-treated animals after aneurysm induction (AI) served as controls. Aortic diameter and wall properties (distensibility, pulse propagation velocity) were measured infraannularly via ultrasound. Hemodynamic parameters, aortic tissue protein expression and serum cytokines were analysed.

In conclusion, AT2 receptor stimulation with C21 prevented extracellular matrix degradation, maintained vascular integrity of the aorta and prevented AAA progression.

P7 THE URINARY PEPTIDOMIC SIGNATURE OF AORTIC STIFFNESS REVEALS MOLECULAR PATHWAYS AND DRUG TARGETS

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Background: Molecular pathways leading to stiffening of the central arteries are poorly understood. We searched for differentially expressed proteins by urinary peptidomic analysis in patients with arterial stiffness and healthy controls in a case–control study.

Methods: To identify urinary peptides associated with aortic stiffening, we applied capillary electrophoresis coupled to mass spectrometry. We compared 18 cardiovascular disease-free patients with carotid-femoral pulse wave velocity (PWV > 10 m/s standardised to a heart rate of 75/min-1 as measured by the SphygmoCor method) with 18 controls matched for sex, age and mean arterial pressure.

Results: 69 urinary peptides had a different signal amplitude between cases and controls (P < 0.049). Among 33 peptides with known sequence, 26 were members of the extracellular matrix family, including collagen type I α1 and α2, collagen type III α1, collagen type IV α1, collagens IX, XXI and XXVII. Collagen type I was down-regulated, whereas collagen type III was up-regulated. Epidermal growth factor receptor (EGFR), a key regulator of myoblast differentiation, and interactions of laminin with other proteins were down-regulated. Atherosclerosis signalling pathways and intracellular pro-thrombin activation were the top pathways associated with increased PWV. Potential drug targets included collagen type IV α3 and transforming growth factor β3. Angiotensin-converting enzyme inhibitors, which are widely used for vascular protection, were among the possible therapeutic agents.

Conclusions: We suggest that stiffening of large elastic arteries involves changes of the extracellular matrix, as reflected by collagen turnover and regulation of myoblast differentiation. Pathway analysis identified potential drug targets, possibly amenable by angiotensin-converting enzyme inhibition.

P8 PROTEIN CONVERTASE SUBTILISIN/KEVIN TYPE 9 LEVELS AND ARTERIAL FUNCTION

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Purpose/Background/Objectives: Proprotein convertase subtilisin/kinin type 9 (PCSK9) levels are modestly but significantly associated with increased risk of total cardiovascular events. Aortic stiffness and wave reflections are also important predictors of cardiovascular events. The aim...