RAT AORTIC SMOOTH MUSCLE CELLS IN CULTURES ON COLLAGEN I MODIFIED BY MAST CELLS. ***L. Bačáková*** [1, 2], *R. Vytášek* [2, 3], *E. Filová* [1], *J. Herget* [2, 3], [1]Institute of Physiology, Academy of Sciences of the Czech Republic, Prague, [2]Center for Experimental Cardiovascular Research, Prague, [3]Department of Physiology, Second Medical School, Charles University, Prague

Degradation of extracellular matrix by mast cells is an important pathogenetic factor of several vascular diseases, including systemic and pulmonary hypertension or atherosclerosis [1, 2]. In this study, collagen I on polystyrene Petri dishes was pretreated for 72 hours with mast cells (line RBL-2H3, 400 000 cells/dish) in atmosphere containing air and 5% CO_2 (sample "N"), under the same normoxic conditions with stimulation by a ionophore A23187 for last 24 hours (sample "A") or under hypoxia (10% of O_2, sample "H"). Dishes coated with unmodified collagen (sample "I") as well as uncoated dishes (sample "G") also were prepared. After removal of mast cells by EDTA, the dishes were seeded with rat aortic smooth muscle cells (RASMC, 100 000 cells/dish, DMEM with 10% of fetal bovine serum). Six hours after seeding, the cells on collagen samples I, N and A usually adhered at significantly higher numbers than those on uncoated dishes (by 79-153%), whereas on H samples, the cell number was similar than that on G. Twenty-four hours after seeding, the cells on A samples adhered through the smallest cell spreading area (1520 ± 160 μm^2 *vs.* 2450 ± 280 μm^2 and 2720 ± 230 μm^2 on I and N, respectively, $p \leq 0.05$). The cells on A and H also contained a significantly lower concentration of focal adhesion proteins talin and vinculin (by 18-67% compared to I, measured by ELISA per mg of protein). During the first 2 days of cultivation, the highest mitotic index was found on A samples (2.8 ± 1.2% *vs.* 0.5 ± 0.3% to 1.2 ± 0.5% on the remaining samples), and shortest doubling times on A and H (11.0 ± 0.1 h and 13.2 ± 0.7 h, respectively, *vs.* 12.0 ± 0.6 h to 17.9 ± 0.2 h on the remaining samples). These results indicate that mast cells activated by hypoxia or ionophore A23187 attenuate the adhesion of RASMC and stimulate their proliferation, probably by degradation of collagen growth support by proteolytic enzymes. *Supported by the grant No.304/02/1348 of GA CR.*

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CONTROL OF VASCULAR SMOOTH MUSCLE CELL (VSMC) PROLIFERATION BY CVT-313, A CDK2 INHIBITOR. ***E. Filová***[1], *L. Bačáková*[1,2], *V. Lisá*[1], *M. Lapčíková*[3], *D. Kubies*[3], *F. Rypáček*[3], [1]Institute of Physiology, Academy of Sciences of the Czech Republic, Prague, [2]Center of Experimental Cardiovascular Research, Prague, [3]Institute of Macromolecular Chemistry, Academy of Sciences of the Czech Republic, Prague

Cardiovascular diseases belong to leading causes of death in the contemporary world. Implantation of artificial vascular prostheses represents a promising approach used in therapy of heavy damage of vessels. For this purpose, prostheses resistant to immune rejection and obstruction with excessively proliferating VSMC are requested. The VSMC proliferation could be regulated by a controlled release of cell cycle inhibitors from the prosthesis material. Therefore, we studied changes in number of VSMC cultured in presence of an inhibitor of cyclin-dependent kinase 2 (CDK2), CVT-313, which is known to stop the cell cycle progression through G1/S and G2/M phases [1]. The VSMC were isolated by explantation method from adult pigs, adult and newborn Wistar rats or adult spontaneously hypertensive rats (SHR), and exposed to CVT-313 concentrations from 3.91 to 1000 ng/ml in Dulbecco`s modified Eagle`s medium with 10% of fetal bovine serum. The maximum decrease in the number of VSMC was obtained at the concentration of 1000 ng/ml in newborn rats (by 90.9 and 57% compared to the number of untreated cells, 6 days and 2 days of incubation, respectively, $p<0.001$). On day 2 of exposure, the same concentration decreased the number of VSMC from adult rats only by 41% ($p<0.01$), and no inhibition was found in both VSMC from pigs and SHR. In VSMC from newborn rats, the half maximal inhibition of VSMC proliferation (IC-50) was obtained at 900 and 120 ng/ml of CVT-313 on day 2 and 6 of exposure, respectively. These results indicate that CVT-313 could be a promising drug for the control of VSMC growth on vascular prostheses, but its effects markedly depend on animal species, strain, age and time of exposure. *This work was supported by Grant Agency AS CR (No. A4050202).*

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CHRONIC HYPOXIA ALTERS NITRIC OXIDE IN EXHALED BREATH: NEWBORN RATS DIFFER FROM ADULTS. ***A. Baňasová,*** *J. Bíbová, D. Miková, J. Herget, V. Hampl*, Department of Physiology, Charles University, Second Medical School, Prague and Center for Experimental Cardiovascular Research, Prague

While studying the role of endogenous NO in the mechanism of chronic hypoxic pulmonary hypertension, we have found recently that chronic hypoxia elevates no concentration in exhaled air of adult rats (1). As there are indications that the role of NO in the pulmonary circulation is profoundly different in neonates than in adults (2), we hypothesized that the effect of chronic hypoxia on exhaled NO is different in neonatal than in adult rats. Rats were placed into a hypoxic chamber (10 % O_2) 24 hr after birth and compared to controls kept in room air. once every 1-2 days, the rats were removed from the chamber for ~ 1hr for NO measurement. Exhaled NO was measured as 15 min NO accumulation in a closed, initially no-free flask (2.1 l) using a cld 77 am chemiluminescence no analyzer (EcoPhysics, Dürnten, Switzerland). During the first postnatal week, the exhaled NO was near the detection limit of the analyzer (~1 ppb) in both the normoxic and hypoxic pups. From day 8, exhaled NO was significantly lower in the hypoxic (0.46 ± 0.13 ppb/) than in the normoxic (2.51 ± 0.45 ppb) pups. However, when the rats were 14 days old, exhaled NO in the hypoxic group (6.03 ± 0.67 ppb) exceeded that in the normoxic controls (2.27 ± 0.32 ppb) and remained elevated for the rest of the exposure, resembling the situation in adult rats (1). The exhaled NO remained low in normoxic controls. When the hypoxic exposure was terminated on day 27, exhaled NO fell rapidly to low level similar to that in controls. We conclude that chronic hypoxia reduces exhaled NO concentration in neonatal rats. By contrast, exhaled NO is elevated by chronic hypoxia in rats older than ~2 weeks. This developmental difference is likely to have implications in pulmonary hypertension. *Supported by MSMT CR 111300002.*

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SLOWLY REVERSIBLE NORMALIZATION OF BLOOD PRESSURE BY 2-WEEK hypoxia in ren-2 transgenic rat MODEL OF HYPERTENSION. ***J. Bíbová,*** *A. Baňasová, J. Herget, V. Hampl*, Department Of Physiology, Charles University, Second Medical School, Prague and Center for Experimental Cardiovascular Research, Prague

Chronic hypoxia reduces systemic hypertension in several experimental models. However, what happens with systemic arterial blood pressure after termination of the hypoxic exposure is not characterized. Therefore, we aimed to answer two questions: 1) Does chronic hypoxia reduce blood pressure also in a novel, genetically well characterized model of hypertension, the transgenic rat harboring mouse Ren-2 renin gene; and 2) how does blood pressure change during recovery from chronic hypoxia? As Ren-2 transgenic homozygotes have high mortality, we used heterozygous (+/-) males. Systolic arterial pressure (SAP) was measured in conscious rats by tail plethysmography every few days. Each measurement was repeated 5 times. Starting at 66 days of age, rats were exposed to hypoxia (10% O_2) for 2 weeks. Just before the exposure to hypoxia, SAP was elevated in +/- rats (210 ± 3 mmHg) compared to wild-type controls (-/-; 145 ± 1 mmHg). SAP did not change appreciably during hypoxia in the -/- rats. In the +/- group, SAP started to drop in hypoxia, so that by day 3 it was significantly lower (195 ± 4 mmHg) than before the exposure. By day 14 of hypoxia, it did not differ from the normoxic -/- group (166 ± 3 vs. 161 ± 2 mmHg). Measurements in a separate set of rats anesthetized with thiopental confirmed that mean carotid artery pressure was lower in +/- rats at the end of 2-wk hypoxia (111 ± 7 mmHg) than in +/- rats kept in room air (151 ± 3mmHg) and did not differ from that in -/- rats (111 ± 5 mmHg). There were no significant differences among the groups in cardiac output estimated from ascending aorta blood flow measured in open-chest, ventilated animals. After cessation of hypoxia, SAP in the +/- rats rose first quickly (184 ± 2 mmHg on day 3 post hypoxia) and very slowly thereafter. Six weeks after hypoxia, SAP in the +/- rats (200 ± 4 mmHg) still did not quite reach the pre-hypoxic level. Thus, hypoxia as brief as two weeks completely normalizes systemic hypertension in the Ren-2 transgenic rat model, and this beneficial effect partly persists for a period several times longer than the exposure itself. *Supported by MSMT CR 111300002*

INDUCIBLE NITRIC OXIDE SYNTHASE PARTICICATES IN THE MECHANISM OF HYPOXIC PULMONARY HYPERTENSION.
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Accumulating evidence suggests that a key event in the development of chronic hypoxic pulmonary hypertension is vascular wall injury during the first few days of hypoxia and that nitric oxide (NO) may participate in this process (1). In many tissues, NO produced by endothelial isoform of NO synthase (eNOS) acts as a vasodilator, whereas NO produced by the inducible isoform (iNOS) can cause tissue injury. We hypothesized that NO produced in the pulmonary circulation during the first few days of hypoxia by iNOS contributes to the vascular wall injury and thus promotes the development of pulmonary hypertension, while the eNOS activity throughout the hypoxic exposure may reduce vascular tone and thus limit pulmonary hypertension. To test this hypothesis, we used NG-nitro-L-arginine methyl ester (L-NAME), a non-selective NOS inhibitor, and L-N6-(1-iminoethyl)lysine (L-NIL), a selective iNOS blocker. Rats were treated with L-NAME (500 mg/l) or L-NIL (8 mg/l) in drinking water either during the first or the last week of a 3-wk hypoxic exposure. Mean pulmonary artery pressure (PAP) was measured after 1 or 3 weeks of hypoxia in spontaneously breathing rats under thiopental anesthesia (40 mg/kg bw i.p). In rats treated with L-NIL during the first week of hypoxia, PAP was reduced at the end of both 1-wk and 3-wk hypoxic exposure compared to untreated hypoxic controls. L-NIL treatment only during the last week of a 3-wk hypoxia had no significant effect, suggesting that iNOS activity during the first week promoted pulmonary hypertension. In support of this interpretation, rats treated with L-NAME during the first week of hypoxia had reduced PAP at the end of 1-wk hypoxia. However, their PAP was increased at the end of a 3-wk exposure, perhaps due to the known poor reversibility of the effect of L-NAME. PAP was also elevated in rats treated with L-NAME only during the last week of a 3-wk hypoxic exposure, implying that endogenous NO reduces vascular tone in pulmonary hypertension. We conclude that iNOS activity in the first week of hypoxia contributes to the mechanism of pulmonary hypertension. *Supported by MSMT CR 111300002.*
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NO PRODUCTION IN AN EXPERIMENTAL MODEL OF HYPERLIPOPROTEINEMIA - APOLIPOPROTEIN E-DEFICIENT MICE.
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Nitric oxide (NO) is a very important vasorelaxation factor. We tested the effect of diets and unselective and selective inhibition of inducible NO synthase by N^{ω}-nitro-L-arginine methyl ester (L-NAME) and L-N^{6}-(1 iminoethyl)lysine (L-NIL), respectively, on the plasma NO concentrations of apolipoprotein E-deficient (apo E KO) mice. Homozygous (-/-) and heterozygous (+/-) apo E KO mice and wild-type mice (+/+) were used. Compared with +/- and +/+ mice, -/- mice have higher cholesterol concentrations (~ 2.3 mmol/L vs. ~11 mmol/L) and develop atherosclerotic changes. Whilst on cholesterol diet, cholesterolemia increased dramatically by ~100% in -/- mice, and only by ~30% in +/- and +/+ mice. When on chow diet, the NO concentrations in +/+ and +/- mice were comparable (1363 ppb and 2065 ppb, respectively) but were lower compared with -/- mice (4079 ppb). On cholesterol diet, NO concentrations rose in all groups; to 3917 ppb in +/+ mice, to 4985 ppb in +/- mice, and to 8868 ppb in -/- mice. Compared with chow diet, the NO concentrations on cholesterol diet with L-NAME decreased in -/- and +/- mice (606 ppb and 715 ppb), but remained unaltered in +/+ mice (1488 ppb). In -/- mice on cholesterol diet, the inhibition of NO production by L-NIL leads to a decrease in NO concentrations to levels (2492 ppb) below those seen on chow diet. We suggest that increasing plasma NO concentrations in -/- mice as an effect of higher NO production via inducible and endothelial NO synthase makes part of the protective mechanism against higher plasma cholesterol concentration and atherosclerotic vascular changes.

INTESTINAL FUSOBACTERIAL INFECTION DOES NOT STIMULATE ATHEROGENESIS IN POLYGENIC HYPERCHOLESTEROLEMIC RATS.
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The role of infectious pathogens in human atherogenesis is a controversial topic. It has been proved that "pathogen burden"(the total number of seropositivities against different pathogenes in one person) is a risk factor for atherosclerosis progression. Individual immune response might be related to the increased risk, but chronic infections, causing persistent mild inflammation, e.g. periodontitis, might play also an important role. In this experiment, the influence of intestinal fusobacterial infection on atherosclerosis initiation was studied in hypercholesterolemic rats. Fusobacteria were chosen because in humans it is a common infectious agent in periodontitis and poor dental hygiene. Prague hypercholesterolemic female rats (PHHC) were used. In spite of substantial increase in cholesterol concentration, no atherosclerosis was documented in this strain after long-term feeding of high cholesterol diet in earlier studies. After weaning, the experimental animals were inoculated by fusobacterial infusion into their gastrointestinal tract, and the inoculation was repeated after 4 weeks. The effect of the inoculation was controlled by microbial analysis of the stools, where massive fusobacterial infection in experimental animals was proved. The control (the same strain without infection) and experimental animals were fed 2% cholesterol chow diet. Total blood cholesterol concentration was controlled at the third month and in the end of the study. After 6 moths the animals were sacrificed, blood collected and their hearts and aortas were removed for further histological analysis. The cholesterol concentrations were 20.73 ± 5.43 mmol/l in the experimental group (n=7) and 27.81 ± 8.05 mmol/l in the control group (n=6), p=0,16. In this strain, more than 70% of cholesterol is carried in VLDL and LDL particles, thus resembling an atherogenic lipoprotein profile of man, on the contrary from other rat strains. After sacrifice, no macroscopic changes on the abdominal or thoracic aorta were found in neither of the groups. No differences in the vascular wall composition between the study and control groups were found in histological analysis. Thus intestinal fusobacterial infection did not cause acceleration of atherogenesis in PHHC rats.

EXPOSURE TO CHRONIC HYPOXIA DOES NOT PROTECT AGAINST ARTERIAL HYPOXEMIA INDUCED BY ACUTE HYPOXIC CHALLENGE.
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Hypoxic pulmonary vasoconstriction is an important regulatory mechanism, which redistributes lung blood flow from hypoventilated lung regions towards better-ventilated lung areas and thereby improves arterial blood oxygenation. In isolated rat lungs challenged by acute ventilation hypoxia we found an inversely proportional relation between the increase in perfusion pressure and decrease of P_{O2} in the lung outflow *(1)*. Exposure to chronic hypoxia results in pulmonary hypertension. In present experiment we tested whether pulmonary hypertension induced by chronic hypoxia improves lung oxygen transport in acute hypoxic challenge. One group of adult male rats (n=4) was exposed to chronic normobaric hypoxia for 16 days (F_{iO2}=0.01), the control group of rats (n=9) stayed in room air. We used isolated lungs perfused with salt solution with albumin (4g/100ml), meclofenamate ($17x10^{-6}$ M) and L-NAME ($5x10^{-5}$ M) by constant flow (4 ml/min/100 g b.w.) The lungs were ventilated with normoxic (21% O_2 + 5% CO_2) or hypoxic (10, 3, 0% O_2 + 5% CO_2) gas mixtures. Lung perfusion pressure and P_{O2} in the lung outflow were continually monitored. Lung outflow P_{O2} during ventilation with hypoxic gas mixtures (3% O_2, 0% O_2) was lower in the group of animals exposed to chronic hypoxia than in control group ($p<0,001$) and chronic exposition to hypoxia inhibited hypoxia-induced vasoconstriction. We didn't find any correlation between perfusion pressure and pO_2 measured in the lung outflow (R=0,021). We conclude that exposition of chronic hypoxia decreases the acute hypoxic pulmonary vasoconstriction and doesn't prevent arterial hypoxemia.
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CARDIAC TOLERANCE TO ISCHEMIA IN NEONATAL SHR AND WKY. ***Z. Chvojková****, I. Ošťádalová, J. Kuneš, J. Zicha, B. Ošťádal*, Institute of Physiology, Academy of Sciences of the Czech Republic, Prague

Cardiac tolerance to ischemia in healthy Wistar rats decreased significantly during early postnatal development. Ischemic preconditioning (IP) failed to improve cardiac tolerance on day 1 and its protective effect developed only at the end of the first postnatal week [1]. The aim of the present study was to analyze cardiac tolerance to oxygen deprivation and possible effect of IP in neonatal SHR. SHR and their controls WKY were used at the ages of 1 and 10 days of postnatal life. Hearts were isolated and perfused in the Langendorff mode with Krebs-Henseleit solution at constant pressure, temperature and stimulation rate. Perfusion pressure was adjusted to mean arterial pressure for given developmental period [2]. Recovery of the contractile function after global ischemia (40 min) was measured by an isometric force transducer and analyzed using an on-line computer. IP (on day 10) was induced by three 3-min periods of global ischemia, each separated by a 5-min period of reperfusion. Relative heart weight was significantly higher in SHR as compared with WKY in both investigated periods. However, developed force / heart weight ratio was in SHR markedly lower. Cardiac tolerance to ischemia was significantly higher in SHR in 1-day-old animals; on the other hand, there was no difference between SHR and WKY on day 10. IP failed to increase cardiac tolerance in 10-day-old SHR and WKY. It can be concluded that i) cardiac tolerance to ischemia in 1-day-old SHR is significantly higher than in WKY, ii) cardiac tolerance to ischemia in both strains decreases after birth, and iii) IP was without any effect in both strains on postnatal day 10.

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ANTIOXIDANT N-ACETYLCYSTEINE (NAC) INHIBITS HYPOXIC PULMONARY HYPERTENSION (HPH) MORE EFFECTIVELY IN EARLY PHASES OF EXPOSURE. ***V. Lachmanová****, O. Hniličková, J. Herget*, Department of Physiology, Charles University, 2nd Medical School, Prague and Center for Experimental Cardiovascular Research, Prague

Exposure to chronic hypoxia results in HPH. Hypoxic injury to pulmonary vascular wall, which causes HPH, is most pronounced in the first week of exposure (2), than HPH stabilizes and reaches the steady state after 3 – 4 weeks of hypoxia (1). Free radical damage of lung tissue, the important pathogenetic mechanism of HPH, is most significant in the first week of exposure (3). We hypothesize that antioxidant applied before and at beginning of hypoxia should be more effective than antioxidant treatment of developed pulmonary hypertension. We studied adult male rats exposed for 3 – 4 weeks to isobaric hypoxia (F_{iO2} = 0.1) and treated with an antioxidant NAC (20g/l of drinking water). NAC was given preventively (7 days before and first 7 days of sojourn in hypoxia, n=9) or therapeutically (last two weeks of hypoxic exposure, n=6). Experimental groups were compared with normoxic controls (n=9) and not-treated hypoxic rats (3 weeks hypoxia, n=9). In thiopental anesthesia (40mg/kg i.p.) we measured pulmonary arterial mean blood pressure (PAP), cardiac output (CO), hematocrit and heart weight. All animals kept in hypoxia had significantly higher pressure in pulmonary artery than normoxic animals. PAP was significantly smaller in hypoxic animals treated preventively with NAC than after therapeutic application (preventive 27.1 ± 0.9 torr, therapeutic NAC 30.5 ± 1.0 torr, P = 0.02, not-treated with NAC 32.6 ± 1.2). CO was significantly lower and hematocrit significantly higher in all hypoxic groups. Therapeutic NAC treatment did not influenced hypoxia-induced right ventricle weight. We conclude that oxygen radicals are involved in the pathogenesis of hypoxic pulmonary hypertension. They induce the injury to pulmonary vasculature in early phases of exposure to hypoxia that leads to structural remodelling of peripheral pulmonary arteries and HPH. *The study was supported by grant GACR 304/02/1348.*

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POLYMORPHISMS IN ABCA1 TRANSPORTER AND PLASMA LIPIDS. ***J. Štefková****, R. Poledne, J.A. Hubáček,* Institute for Clinical and Experimental Medicine, Atherosclerosis Research Department, Prague and Center for Experimental Cardiovascular Research, Prague

ABCA1 transporters are members of large group of transmembrane proteins. They transport cellular cholesterol and phospholipids (mostly phosphatidylcholine) to cell surface-bound apolipoproteins. These proteins represent the first and rate-controlling step in the reverse cholesterol transport pathway. Mutations in ABCA1 gene cause autosomal recessive disorder, Tangier disease. Patients have severe deficiency or absence of high-density lipoprotein in plasma and accumulation of cholesterol esters in macrophages. Recent studies show that some SNPs in ABCA1 gene can affect plasma lipids too. Using nested PCR and than restriction analyses, we have measured two polymorphisms, one in promoter region (-191G/C) and one in 5´ untranslated region (C69T), and their effect to plasma lipids in 300 unrelated men, 300 women and 300 patients with myocardial infarction. We have found no associations between these two polymorphisms and plasma lipid levels in any group. We have found that computation of 69T homozygotes in women is statistical lower than in men. We have found no differences between patients with myocardial infarction and population. In conclusion, these two polymorphisms have no association to plasma lipid levels in Czech population and there are no differences in frequencies of genotypes between patients with myocardial infarction and Czech population.

LONG-TERM EFFECT OF LIFESTYLE CHANGES ON LIPOPROTEIN CONCENTRATIONS. ***P. Suchanek****, A. Dvorakova, D. Bobkova, P. Stavek, R. Poledne.* Center for Experimental Cardiovascular Research, Institute for Clinical and Experimental Medicine, Prague, Czech Republic.

Obesity is a growing problem in many countries, and its role in cardiovascular disease has recently drawn increasing attention. The aim of our study was to determine the effect of long-term, self-monitored physical activity after 6 months on the serum lipid profile and body composition in obese women. The group included 21 women (50.1±8.4 years; 85.7±10.1 kg, BMI range 33.2±4.1 kg/m^2) with abdominal obesity. Women volunteering to participate in the study underwent 9-week intervention comprising controlled physical activity (6 units/week) and a continuous individualized dietary regimen (3-day food consumption recalls). The women were examined before, after the 9-week intensive lifestyle intervention, and then after six months. Lifestyle intervention resulted in a significant decrease in body weight and LDL-cholesterol, whereas the concentrations of the protective HDL particle increased. At the end of the six-month period after intervention, one part of volunteers (A, 60%) continued to adhere to the recommendation of self-monitored physical activity, whereas the other part (B) returned to their original sedentary lifestyle. Group A demonstrated further improvement (a 13% decrease in LDL-cholesterol and a 14% increase in HDL-cholesterol). Group B had completely opposite results (a 22% increase in LDL and a 4% decrease in HDL). It is evident that although self-monitored continuous increased physical activity was less intensive than during the controlled lifestyle intervention, lipoprotein parameters continue to improve.

INFLUENCE OF THE IMMATURITY OF ERYTHROCYTES ON THE ION TRANSPORT IN DAHL RATS WITH SALT HYPERTENSION. ***M. Vokurková****, Z. Dobešová, J. Kuneš, J. Zicha,* CECR and Institute of Physiology AS CR, Prague, Czech Republic

It is generally accepted that hypertension is often accompanied by a certain degree of anemia resulting in the enhanced entry of immature erythrocytes to the circulation. Immature erythrocytes exhibit lower mean cellular hemoglobin content (MCHC) compared to mature erythrocytes. Moreover, an increased K^+ content and an augmented activity of the Na^+-K^+ pump, a reduced activity of the Na^+-K^+ cotransport and slightly lowered Na^+-Li^+ exchange were observed in the immature human erythrocytes. In this study performed under the conditions of high salt intake, salt-sensitive Dahl rats had significantly higher blood pressure (151±5 *vs* 107±4 mm Hg, $p<0.001$), elevated erythrocyte Na^+ content (4.116±0.185 *vs* 3.400±0.056 mmol/l ery, $p<0.001$), increased Na^+ (7.697±1.200 *vs* 3.407±0.114 mmol/l ery /h, $p<0.001$) and Rb^+ leak (0.537±0.025 *vs* 0.408±0.014 mmol/l ery /h, $p<0.001$) together with the enhanced ouabain-sensitive Na^+ net extrusion (7.981±1.208 *vs* 3.483±0.104 mmol/l ery /h, $p<0.001$) and ouabain-sensitive Rb^+ uptake mediated by the Na^+-K^+ pump (8.600±1.252 *vs* 4.255±0.130 mmol/l ery /h, $p<0.001$) and bumetanide-sensitive Na^+ net transport mediated by Na^+-K^+ cotransport system (1.014±0.224 *vs* 0.194±0.078 mmol/l ery /h, $p<0.001$) compared to salt-resistant animals which remained normotensive. Furthermore, mean hemoglobin content in erythrocytes of Dahl rats with salt hypertension was significantly lower than in erythrocytes of their normotensive controls (4.53±0.15 *vs* 4.91±0.05 mmol/l ery, $p<0.01$). Influence of the immaturity of erythrocytes on the ion transport in erythrocytes of Dahl rats with salt hypertension was studied by correlation analysis. MCHC was used as a marker of the maturity of the erythrocytes. MCHC correlated negatively with activity of the Na^+-K^+ pump (ouabain-sensitive Na^+ net extrusion: $r = -0.935$, $p<0.001$ or ouabain-sensitive Rb^+ uptake: $r = -0.947$, $p<0.001$) and Na^+ leak ($r = -0.884$, $p<0.01$). In contrast, there was a positive correlation between MCHC and activity of the Na^+-K^+ cotransport (bumetanide-sensitive Na^+ net extrusion $r = 0.735$, $p<0.05$ or bumetanide-sensitive Rb^+ uptake $r = 0.725$, $p<0.05$). We can conclude that a considerable part of the ion transport abnormalities usually associated with the pathogenesis of salt hypertension is caused by enhanced amount of immature erythrocytes in the circulation. *This work was partially supported by the research grants 305/00/1638 (GA CR, Prague), NB6682-3/2001 (Ministry of Health CR) and by the research project AVOZ 5011922.*