## THE ROLE OF ATP-DEPENDENT POTASSIUM CHANNELS IN REGULATION OF RENAL FUNCTIONS

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Kidneys play a special role in the mechanisms of formation of hypoxic injury. A high need in oxygen leads to the extreme sensitivity of kidneys to hypoxia. The proximal tubules (the S 3 segment and the thick ascending limb of the loop of Henle) usually balance on the edge of hypoxic state, that's why even moderate energy deficit can cause the damage of the cells of tubules and interstitium. The level of violation of the tubular processes that provide water and salt homeostasis largely depends on the state of ion channels. Despite the leading role of ATP-sensitive potassium (KATP) channels in the mechanisms of adaptation to hypoxia their role in the nephrocyte activity continues to refine. In the present research we aimed to assess the functional state of the damaged hypoxic kidney after the experimental activation of potassium ion flow and to clear the role of KATP channels in the regulation of renal functions. The experiments were made on non-linear laboratory white rats 160-170 g of weight after injection of KATP channels activator flocalin (intraventricularly, 5 mg/kg, 7 days) in conditions of acute histohemic hypoxic nephropathy (AHHN). The model of AHHN was made by serial single injection of sodium nitrite (50 mg/kg subcutaneously) and 2,4-dinitrophenol (3 mg/kg intraventricularly). One of the antihypoxic mechanisms of KATP channels activation is the decrease of the intracellular pool of calcium ions so we aimed to compare the effects of flocalin and the blocker of voltage-gated L-type calcium channels diltiazem (intraventricularly, 5 mg/kg). We have shown that both ion channel modulators activated acid regulating kidney function that is responsible for liquidation of metabolic acidosis. The electrolyte balance that had been disturbed by hypoxia was preserved after injection of KATP channels activator due to the decrease of sodium and potassium excretion. The use of calcium channel blocker only prevented the loss of potassium ions in the urine, but with more pronounced kaliuresis than under the influence of flocalin. In contrast to diltiazem flocalin decreased proteinuria at the initial stage of AHHN. It indicated nephroprotective properties of KATP channels activation. It should be noted that particularly flocalin corrected the indexes of proteinuria and tubular transport of the main osmotic ion sodium standardized by glomerular filtration. Therefore, the obtained results point at regulatory activity of KATP channels in AHHN that makes it possible to control the functional state of the damaged nephrocytes by pharmacological activation of this type of channels.

## SPECIFIC CONTRIBUTION OF NEURAL AND HUMORAL MECHANISMS TO THE DEVELOPMENT OF ARTERIAL HYPERTENSION OF DIFFERENT ORIGIN IN RATS

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Aim – to compare the effectiveness of $alfa_1$- and $beta_1$-adrenergic blockade and inhibition of angiotensin-converting enzyme (ACE) used to normalized arterial pressure in rats with hypertension of different etiology: metabolic obesity and alcohol addiction. Chronic hypertension of different genesis was developed in rats during 4 months: under alcoholism (n=10) and metabolic obesity (n=10). Emotional stress was modeled introducing by caffeine. After that hypotensive medicines of different mechanism of action were administered: Concor cor ($beta_1$-adrenergic receptor blocker), Doxazosin (antagonist of $alfa_1$-adrenergic receptor), Prestarium (inhibitor of ACE). These drugs were applied separately of under the stress made by caffeine. Arterial pressure on the tail and the heart rate were registered using sphygmomanometer. In alcohol-depended rats arterial pressure under inhibitor ACE Prestarium was reduced by 17,0±1,5 % after adrenergic receptors blockade – only by 3,6±0,3% (Concor cor) and 9,9±0,9 % (Doxazosin). On the background of adrenaline secretion stimulated by caffeine the reaction described about was similar: Prestarium reduces arterial pressure by 22,9±2,0 %, Concor cor – by 9,9±0,9 % and Doxazosin – by 12,5±1,1 %. In obese rats antagonists of adrenergic receptors reduce arterial pressure

by 20,3±2,2 % (Concor cor) and 12,8±1,0 % (Doxazosin) and ACE inhibitor – only by 8,2±0,8 %. After caffeine induced adrenaline secretion this difference persisted: Concor cor and Doxazosin reduced the pressure by 23,8±2,0 % and 19,8±1,8 % respectively and Prestarium – by 16,0±1,5 %. In control rats the strengths of hypotensive effect was approximately the same for all medicines despite their mechanism of action: both humoral (Prestarium) – 10,5±1,0 % of the pressure redaction and neural (Concor cor and Doxazosin) – 8,8±0,7 % and 11,4±1,0 %. Under the caffeine stress relations were as follows: 6,6±0,6 %, 1,9±0,2 %, 4,7±0,4 %. Concor cor constantly provoked the reduction of the heart rate, Doxazosin and Prestarium – its elevation. In rats drinking alcohol provokes hypertension and tachycardia. Obesity leads to the hypertension. Caffeine causes tachycardia only in the control group and obese rats but doesn't affect the heart rate in alcohol addicted animals. Caffeine results in the arterial pressure elevation only in rats with essential hypertension but in healthy animals with normotony causes the pressure decrease. For the hypertension relief in alcohol treated rats the most effective was the mechanism of ACE inhibition comparatively to $alfa_1$- and $beta_1$-adrenergic receptors blockade. In obese rats the hypertension relief was most potent under the $alfa_1$- and $beta_1$-adrenergic receptors blockade than after ACE inhibition. More specific cause of hypertension in the alcohol addicted organism is the injury of neural adrenergic regulation and in the obese organism – impairment of the humoral regulatory pathways.

## BEHAVIORAL CONSEQUENCES OF ENTEROBIASIS IN RATS

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Representatives of the family Oxyuridae are the most common parasites of laboratory rodents. The aim of the present study was to clarify the behavioral consequences of enterobiasis in laboratory rats. Enterobiasis was induced by infecting of adult Wistar rats with eggs of Syphacia muris and Aspiculuris tetraptera. The level of anxiety and emotionality, sociability and general social interaction, as well as the fear associative memory formation were evaluated in rats with and without pinworm invasion. The significant increase in the anxiety level was observed in rats with enterobiasis. We did not find any changes in other behavioral measures in our studies. The analysis of the leukocyte formula showed a tendency to increase the number of eosinophils in the blood of infected rats, which may indicate an implementation of the immune response due to enterobiasis.

## ДІАГНОСТИЧНЕ ЗНАЧЕННЯ СПІВВІДНОШЕННЯ НЕЙТРОФІЛИ/ЛІМФОЦИТИ У ХВОРИХ З ПІДОЗРОЮ НА ГОСТРИЙ ЗАПАЛЬНИЙ ПРОЦЕС ОРГАНІВ ЧЕРЕВНОЇ ПОРОЖНИНИ

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Одним із важливих завдань діагностичного процесу є встановлення функціонального стану імунної системи за умов запалення. Основними ефекторами неспецифічного імунітету є нейтрофільні гранулоцити, у той час як лімфоцити відповідають за регуляцію імунної відповіді та специфічний імунний захист. Дослідження імунного статусу потребує значних затрат коштів і часу. Тому завданням нашого дослідження було розглянути можливість використання співвідношення нейтрофіли / лімфоцити в діагностиці запальних процесів органів черевної порожнини. Було обстежено 46 хворих на гострий флегмонозний апендицит (ГА), 60 хворих на абдомінальний туберкульоз (АТ) і 36 практично здорових добровольців. Обстеженим проводили забір венозної крові натще до проведення оперативного втручання і визначали загальний вміст та фракційний склад лейкоцитів і розраховували нейтрофіли-лімфоцти співвідношення (НЛС). Загальний вміст лейкоцитів у крові хворих на ГА становив 10,82±0,9 Г/л, що є більше порівняно з показником у здорових осіб (6,5±0,6 Г/л) на 40 % ($P<0,05$). У хворих на АТ абсолютна кількість лейкоцитів становила 6,85±0,6 Г/л, що є менше на 37% порівняно з показником при ГА ($P<0,05$). Аналіз лейкоцитарного профілю периферичної крові показав, що при ГА переважають нейтрофільні гранулоцити, кількість яких становила 8,17±0,7 Г/л проти 4,58±0,42 Г/л у здорових осіб і 4,05± 0,21Г/л у хворих на АТ. У