

internists, etc. were provided. PASI and DLQI-Dermatology Life and Quality Index were calculated

The study was conducted on two groups of patients. Different cardio metabolic violations were determined in 213 patients, respectively 81,6%. Considering high comorbidity of psoriasis and cardio metabolic disorders, we proposed combine therapy of psoriatic patients with using metabolic and cardioprotective drugs: infusions of Pentoxiphylline 0,5 mg in Ringer's lactated solution 200 ml every other day № 5, alternating with 1,5% solution of Meglumine sodium succinate infusions 400 ml on alternate days № 5, intravenous injections of essential phospholipids 5 ml in 5 ml autoblood once a day № 10 and Magnesium sulfas intravenous injections 5 ml 25% solution in 5 ml physiological saline once a day № 10.

Using of metabolic therapy of psoriasis combined with cardio metabolic disorders will make possible to avoid medication for cardio metabolic comorbidity correction, or eliminate the use of already assigned symptomatic therapy. This is especially true for the use of antihypertensive drugs, because they are known risk factor for exacerbation of psoriasis and formation of the so-called "drug induced psoriasis".

## **EFFECT OF WATER AND SALINE OVERLOAD ON THE CONCENTRATION OF PYRUVIC ACID IN THE BLOOD OF STREPTOZOTOCIN DIABETIC RATS**

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Type 1 Diabetes Mellitus (T1DM) is a widespread chronic disease among children and adolescents. Salt plays an important part in the control of blood pressure in obesity and diabetes mellitus. High-sodium intake may increase blood pressure and diabetes is a salt-sensitive condition.

The aim of the present study was to evaluate the effect of water and saline overload on concentration of pyruvic acid in the blood of streptozotocin (STZ) diabetic rats.

Material and methods. Male Wistar rats weighing 180 +/- 50 g were made diabetic by injection with a single intraperitoneally (i.p.) dose of STZ (65 mg/kg b. w.). After the induction of diabetes, animals were maintained for 5 days with free access to standard rat chow and tap water. After 5 and 12 days was carried out to determine the level of glucose in vivo. Blood was taken from the tail vein evaluate the basal glycemia level with the use of One

Touch Ultra (Life Scan, USA). Water stress was carried out by introducing the animals water at the rate of 5 % of body weight. Saline loading diabetic rats was performed by introducing a 0,1 % NaCl at a rate of 5 % of the body weight of rats. Blood samples were collected at day 12 post STZ injection (from diabetic group serum glucose level significantly elevated  $< \text{or} = 300 \text{ mg \%}$ ,  $p < \text{or} = 0.05$ ). The animals were divided into subgroups: 1) intact rats (the control group); 2) STZ- diabetic rats with overt (basal glycemia  $>150 \text{ mg \%}$ ) diabetes; 3) animals with overt diabetes undergoing water stress; 4) animals with overt diabetes undergoing saline stress. Determinations of pyruvic acid concentration in the blood was made by standard methods.

Our results showed increase in concentration of pyruvate in groups of diabetic rats and diabetic rats with water overload by an average of 195 % respectively compared to the same indexes of control rats. According to the results obtained in the blood of rats with STZ diabetes, which had saline stress, concentration of pyruvate increased by 250 % compared to the same indexes of control rats. These changes are more pronounced in the group of diabetic rats undergoing saline load.

Increase of pyruvate in plasma is likely to be due to the growth of free radical lipid oxidation of biomembranes and its release from the cells into blood plasma.

Conclusion. Salt load slows the process of pyruvate use in diabetic rats.

## **T-2 TOXIN INDUCES DEGENERATIVE ARTICULAR CHANGES IN RATS**

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Osteoarthritis (OA) is a degenerative joint disease that is characterized by joint pain and a progressive loss of articular cartilage. Kaschin-Beck disease is a form of endemic OA in China (and Transbaykal area of Russia as well) which etiology is unclear, but epidemiological data indicate a possible link to trichothecenes mycotoxin exposure. In vitro, T-2 toxin, a trichothecenes mycotoxin, has been demonstrated to inhibit aggrecan synthesis and promote aggrecanase and proinflammatory cytokine production in cultured chondrocytes. To assess the effects of T-2 toxin on articular cartilage in vivo, Wistar rats were fed a diet containing T-2 toxin (100 ng/kg chow) for six and ten months. Following six months of T-2 toxin exposure, histopathological changes in femorotibial cartilage were characterized by chondrocyte degener-

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