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# BRAIN-0181

# Non-Registered Abstracts
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## IMMUNOLOGICAL CHARACTERISTICS AND ANTIGENS OF HLA-SYSTEM IN NEURORHEUMATISM IN UZBEK POPULATION

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**Abstract**
**Introduction:** Rheumatic fever(RF) and rheumatic heart disease are still remaining an important public healthproblem. The purpose of the study was determination of immunological parametersand analysis of antigens HLA-class system for RF in a case of neurorheumatism inUzbek population.
**Materialsand Methods:** We examined 44 patients Uzbek nationality with RF - 26 (59%) women and18 (41%) men at 15–59 years (mean age - 31,9 ± 2,0 years). The control group composed245 healthy individuals of the same nationality. Immunoanalysis performed inperipheral blood of patients.
**Resultsand discussion:** RF patients showed a significant decrease in the peripheral blood of T-cellpool and immunoregulatory subpopulation (1.4 times), B cells (1.7 times) of thenumber of phagocytic neutrophils, increase of zero lymphocytes and the level ofCircular Immune Complex (3 times). It should be noted that the decrease in T-suppressor was more pronounced than that of T-helper cells, which resulted inincrease in the ratio Th/Ts (3,30 ± 0,15 in the control group and 4,10 ± 0,20- patients).
Occurrence of antigens of HLA-systemwas observed only at loci A and B. Thus, a locus A in 2 times more commonantigen was HLA-A10 (RR = 2,70), in a locus - the greatest risk of developing RFwas for antigen B7 (RR = 2,55), B8 (RR = 2,12), B27 (RR = 5,29), B40 (RR = 2,61).
**Conclusion:** Neurorheumatismaccompanied by disturbances in the immune system. Genetic markers ofpredisposition to RF in Uzbek people were HLA-A10, B7, B8, B27, B40.

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## INFLUENCE OF ANGIOLIN ON NEUROAPOPTOSIS, CAUSED BY AN IMBALANCE OF REDOX POTENTIAL

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**Abstract**
It is shown that an imbalance of redox potential caused by displacement of thiol-disulfide balance and the accumulation of oxidized forms of thiol-containing molecules under conditions of cerebral pathology can cause neuronal apoptosis. Our studies have shown high neuroprotective activity of Angiolin ((S)-2,6 diaminohexane acid 3-methyl-1,2,4-triazolyl-5-thioacetate) on various models of cerebral pathology and in experiments in vitro. Experiments on cerebral cortex neurons of 14-days-old Wistar rats showed that introduction of oxidized glutathione in 1–5 mM concentration into suspension of neurons results in the activation of caspase-3 and expression of p53. After processing of neurons with ethidium bromide the increase of the number of *apoptotically changed cells was noted*. The experimental data on dynamics of neuronal apoptosis allow us indirectly connect the action of oxidized glutathione with cascade of reactions of Ras-signaling pathway, as Ras-mediated apoptosis may develop from the level of the protein p53. Pre-processing of neurons with Angiolin ($10^{-5}$ M) resulted in a significant reduction of the number of *apoptotically changed cells*. At the same time in the samples with cerebrocurine significant decrease of p53 expression was found. Pronounced antiapoptotic effect of Angiolin takes place indirectly through reduction of p53 protein, which is involved in Ras-mediated apoptosis and causes increase of oxidative stress. Processing of Ras-protein and several other members of this family of proteins of apoptosis intracellular regulation depends on the status of glutathione pool in the cell. Our experiments suggest that Angiolin can increase the ratio reduced/oxidized glutathione, and thus affect the Ras-signaling cascade.