+HSFLGLQ TXDQWL¿FDWLRQ LQ 1HXURGHJHQHUDWLYH GLVHDVHV

Manolov V, Hadjidekova S, Petrova J, Vasilev V, Petrova M, Kuntchev T, Jelev Y, Tzatchev K, Jeliazkov P and Traykov L 0 H G L F D O 8 Q L Y H U V L W \ 6 R À D % X O J D U L D

Aim: Neurodegenerative diseases are conditions in which the nervous system progressively and irreversibly deteriorates. Neurodegenerative diseases are of en late manifestation of disorders typified by Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD) and amyotrophic lateral sclerosis (ALS). AD depends on age, is chronic disease, a leading cause for dementia. Brain atrophy is main sign in AD cases. PD is another neurodegenerative disease, and also usually involves elder people. HD is characterized by abnormal involuntary writhing movements called chorea. We aimed to find a connection between iron homeostasis regulator hepcidin and neurodegenerative diseases patients.

Materials and Methods: 17 patients with Huntington's disease, 23 with Alzheimer's and 19 with Parkinson's disease were included; 31 females (52.5%). ey had clinical and neurological examination, EMG. ey were evaluated for routine biochemical parameters, and additional serum hepcidin and glutathione peroxidase (GPX) were quanti ed. Hepcidin and GPX were evaluated by ELISA methods. e results obtained from HD, AD and PD patients were compared to age and gender matched healthy controls. Statistical analysis of established results was performed using Pearson's correlation and Student's paired t-test.

Results: We found statistically signicant elevated serum hepcidin levels in HD patients compared to healthy controls (51.6 μ g/L \pm 10.2 μ g/L; 20.4 μ g/L \pm 4.9 μ g/L; P<0.001). In AD and PD cases we found also increased serum hepcidin (61.4 μ g/L \pm 12.3 μ g/L; and 54.9 μ g/L \pm 5.7 μ g/L), compared to controls (P<0.001). GPX activity was de (s)-8 (e)-4.9 (d s)-8 (er)dd-5 (d t-t)6 1Fd5.9 (a)8tients compared to