

Vol.6 No.2

Haptoglobin polymorphism causes neurodegenerative changes through oxidative stress

Manolov V¹, Hadjidekova S², Vasilev V³, Gramatikova Z⁴, Grozdanova R⁵, Georgiev O⁶, Petrova I⁷, Bogov B⁸, Hadjiev E⁸, Pencheva-Genova V⁶, Tzvetkova G⁸, Angov G⁷, Petrova-Ivanova I⁷, Nikolova M⁵, Karadjova M⁷, Spasova V², Kunchev T⁷, Tzatchev K¹, Traykov L⁷

¹Dept. of Clinical Laboratory and Clinical Immunology, Medical University - Sofia, Bulgaria

²Dept. of Medical Genetics, Medical University – Sofia, Bulgaria

³Clinical Laboratory and Clinical Pharmacology, University "Aleksandrovska" hospital, Sofia, Bulgaria

⁴R.E.D. Laboratories N.V./S.A. - Zellik, Belgium

⁵Dept. of Immunology, NCIPD – Sofia, Bulgaria

⁶Dept. of Propedeutics of Internal Diseases, Medical University – Sofia, Bulgaria

⁷Dept. of Neurology, Medical University – Sofia, Bulgaria

⁸Dept. of Internal Diseases, Medical University – Sofia, Bulgaria

Abstract

Human gene of haptoglobin is presented by two alleles. Haptoglobin types are 1-1, 1-2 and 2-2. Different studies shows role of type 2-2 in cardio-vascular disease occurrence during diabetes. Haptoglobin type 1 is known to suppress hemoglobin based oxygenation of HDL and LDL, acting like antioxidant. We aimed that Bulgarian population is haptoglobin 2-2 type, which causes frequent morbidity by systematic diseases, such as atherosclerosis, diabetes, diabetic nephropathies, gestational diabetes, anemia, etc. 37 volunteers were included, age 33.9 ± 4.1 . IMT, ABI, CBC, iron homeostasis, hsCRP and haptoglobin type were evaluated. Increased serum hepcidin concentrations were established in patients with atherosclerotic a. carotis changes (99.1 \pm 10.8 μ g/L) compared to healthy controls ($20.2 \pm 2.9 \mu g/L$), P<0.001. In haptoglobin type 2-2, was found strong positive correlation between hepcidin levels and changed IMT and ABI (r=0.911, r=0.935, resp.; P<0.05). Three volunteers were with haptoglobin type 2-1; no changes of serum hepcidin concentration and IMT, ABI was found in this phenotype. The main reason for acute coronary thrombosis is atherosclerotic plaque rupture. Extravascular hemoglobin plays role as start mechanism for

inflammation in the plaques. Important contra-active mechanism is played by haptoglobin. Thus, it prevents kidney injury from free hemoglobin. Released iron from destructed erythrocytes forms reactive oxygen radicals through Fenton's reaction. Hepcidin regulates iron homeostasis by its interaction with intracellular iron exporter ferroportin.

Acknowledgements: This project is sponsored by MU-Sofia, as part of Grant <u>J</u>-213/2018.

Keywords-oxidative stress, haptoglobin, iron

Biography

Victor Manolov has completed his PhD at Medical University in Sofia, Bulgaria. He is working as Assist. Prof. at Department of Clinical laboratory and clinical immunology at the same University. His interests are in neurology, pediatrics, gynecology, endocrinology and clinical laboratory. He has published more than 25 papers in reputed journals.

<u>31st International Neuroscience Online Event;</u> Online Event-July 27, 2020.