0096

Paraoxonase-1 Gene L55M Polymorphism and Enzyme Activity in Obstructive Sleep Apnea

A. Płóciniczak[1], A. Ludziejewska[2], H. Winiarska[3], S. Michalak[4], G. Kasprzak[2], D. Formanowicz[1], S. Cofta[3], E. Wysocka[2]

[1]Department of Clinical Biochemistry and Laboratory Medicine, Poznan University of Medical Sciences, Rokietnicka 8 60-780 Poznań, Poland; e-mail: alicjaplociniczak@ump.edu.pl

[2]Department of Laboratory Diagnostics, Poznan University of Medical Sciences, Szamarzewskiego 84 St, 60-569 Poznań, Poland

[3]Department of Respiratory Medicine, Allergology and Pulmonary Oncology

Poznan University of Medical Sciences, Szamarzewskiego 84 St, 60-569 Poznań, Poland

[4]Department of Neurochemistry and Neuropathology, Poznan University of Medical Sciences, Przybyszewskiego 49 Str, 60-355 Poznań, Poland

Background: Obstructive sleep apnea (OSA) is in the area of interest in a context of cardiovascular disease (CVD). Antioxidant enzyme paraoxonase-1 (PON1), associated with high-density lipoproteins (HDL) could protect against oxidative stress. The PON1 gene is polymorphic and has been widely investigated in the development of various disorders, especially cardiovascular diseases.

Aim: Determination of PON1 gene L55M polymorphism in OSA-positive and OSA-negative subjects, along with paraoxonase activity of the enzyme.

Methods: Caucasians aged 25-75, with body mass index (BMI) 19-53 kg/m^2 and no acute or severe chronic disorder, were qualified for the study. OSA-suspected subjects underwent full-night polysomnography and apnea/hypopnea index (AHI) was used to diagnose OSA. Finally OSA-negative (n=44) and OSA-positive (n=57) groups were investigated. Risk factors of CVD, including blood pressure, fasting: glucose, lipids, C-reactive protein, homocysteine, were measured. Genomic DNA was extracted from leucocytes and amplified by standard PCR with specific primers, to determine PON1 L55M polymorphism. The presence of the LL, LM and MM genotypes was detected by the method of restriction fragments length polymorphism and was confirmed by automatic sequencing. Serum paraoxonase activity of the enzyme (PON1-act) was measured spectrophotometrically with the use of paraoxon as a substrate, according to the method of Aviram M. et al. (J Clin Invest, 1998).

Results: Different frequency of PON1 L55M polymorphism was established in OSA-negative (LL n=28/64%; LM n=16/36%) and OSA-positive (LL n=26/46%; LM n=20/35%; MM n=11/19%), p<0,05. Increased PON1-act was observed in LL-genotype *versus* LM+MM-genotype individuals in the study population (p<0.05). PON1-act was higher in OSA-negative comparing with OSA-positive subjects (p<0.001) in general and in subgroups presenting LL or LM genotypes, separately. In all study population the negative correlation PON1-act&LDL-cholesterol was observed. OSA-positive group presented the negative relationships PON1-act&fasting glucose. In LM+MM-genotype individuals the positive correlation PON1-act and HDL-cholesterol was observed.

Conclusion: Humans could benefit from LL genotype related with higher activity of plasma paraoxonase-1. OSA pathology might decrease the enzyme activity, despite the presence of LL or LM genotypes.