Flow-mediated dilation, carotid wall thickness and HDL function in subjects with hyperalphalipoproteinemia: the HALA study

F. Zimetti¹, E. Favari¹, F. Bernini¹, E. Satta², S. Boarini², L. Giusto³, E. Pinotti¹, P. Tarugi¹, A. Vanini³ and G.B. Vigna²

¹Dept. of Pharmacy, University of Parma, Parma, Italy; ²Section of Internal Medicine, Gerontology and Clinical Nutrition, Dept. of Medical Sciences, University of Ferrara, Ferrara, Italy; ³Vascular Diagnostic Unit, Medical Dept., Azienda Ospedaliero-Universitaria of Ferrara, Ferrara, Italy; ⁴Dept. of Life Sciences, University of Modena & Reggio Emilia, Modena, Italy

Objective: The HyperAlphaLipoproteinemia and Atherosclerosis (HALA) study is a case-control investigation planned to examine the relationships between very high plasma HDLc, HDL functionality and subclinical atherosclerosis. Results: Subjects with primary hyperalphalipoproteinemia (HAL) were compared with 20 age and sex-matched controls. HDLc levels were 40 mg/dl higher in HAL subjects while LDLc concentration was comparable to control group. Polyacrylamide gel electrophoresis in HAL subjects disclosed larger and more buoyant HDL particles than in controls, while LDL profile was much more similar. Ankle brachial index, carotid-intima-medial thickness and arterial plaques did not differ in cases and controls and the two groups showed comparable flow-mediated dilation at brachial artery examination. HDL cholesterol efflux capacity (CEC) via aqueous diffusion (AD) and SR-BI were higher in HAL subjects (6.41%±0.17 vs 5.16%±0.16; p<0.001 and 4.67%±0.21 vs 2.99%±0.13; p<0.001 respectively), while ABCG1- and ABCA1-CEC were similar between groups; after adjusting efflux values for HDLc levels, AD, ABCA1- and ABCG1-CEC resulted higher in control subjects (0.095% ± 0.021 vs. 0.067% ± 0.009, p<0.001; 0.047% ± 0.021 vs 0.031% ± 0.007, p<0.01; 0.078% ± 0.026 vs 0.050 ± 0.011, p<0.01, respectively) while no differences was detected in SR-BI-CEC between groups. Conclusion: The enhanced HDL functionality observed in control subjects, as indicated by the AD-, ABCA1- and ABCG1-CEC after adjustment for HDLc levels, could explain, at least in part, the lack of differences in subclinical atherosclerosis markers in spite of large differences in HDLc concentration between investigated groups.