Elevated triglyceride level (hypertriglyceridemia - HTG) affects about 25% of people and is closely related to the development of obesity, considered to be a civilization disease of the 21st century. HTG contributes to the development of atherosclerosis, which leads to coronary heart disease, myocardial infarction or stroke, some of the most common causes of mortality worldwide. For this reason, research on the development and treatment of HTG is very important. HTG is a result of disturbed metabolism of very low density lipoprotein (VLDL) responsible for transporting triglycerides (TG) in the plasma. In healthy individuals, the TG in VLDL undergo lipolysis, i.e. they are broken down by lipoprotein lipase (LPL). In people with HTG, TG lipolysis is decreased resulting in the accumulation of VLDL and partially metabolised VLDL particles (so called remnants) in the plasma. In our previous study, we found that the efficiency of VLDL lipolysis can be increased by the presence of high density lipoprotein (HDL), which has an antiatherogenic protective effect in the body. HDL are a very heterogenic group of lipoproteins, with two major subfractions: large HDL-2 and small HDL-3. It is also known that HTG causes a decrease in HDL concentration, especially in HDL-2. However, it is not known whether quantitative and qualitative changes in HDL in patients with HTG have an impact on their effect on VLDL lipolysis.

For this reason, experiments will be carried out to clarify whether the beneficial effect of HDL on VLDL lipolysis is due to the influence of HDL-2 and whether this effect is altered by adverse changes in HDL resulting from HTG.

Fasting morning blood samples will be collected from patients with HTG and healthy controls. VLDL and HDL, and then HDL-2 and HDL-3, will be isolated from the blood. Then, VLDL isolated from patients with HTG will be incubated with LPL, in the presence of HDL, HDL-2, or HDL-3 acquired from patients with HTG or from healthy controls. After the reaction, concentrations of lipolyzed TG in VLDL will be determined for each reaction mixture. Additionally, other VLDL components (cholesterol, phospholipids, proteins) released from the VLDL during lipolysis will also be measured. This will allow for the assessment of the impact of HDL and HDL subfractions obtained from patients with HTG and from healthy controls on the efficiency of the VLDL lipolysis efficiency.

The results obtained from the presented experiments will allow a better understanding of the mechanism of HDL impact on the VLDL transformation and on the HTG development. It is very likely that in the near future these findings could become a basis for the development of new drugs for the treatment of HTG, which would consequently reduce the risk of atherosclerosis and reduce the mortality due to diseases caused by atherosclerosis.