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Fund "Nauka" Project № 22028 Resume – Competition-Based Session 2022:
"Investigation of SDF-1α associated activity of endothelial progenitor cells in obesity induced experimental model of endothelial dysfunction"
Project leader: Assoc. prof. Kameliya Zhechkova Bratoeva, MD, PhD

The alarmingly increasing prevalence of obesity over the past two decades results from an interplay between multiple factors such as genetics, dietary mistakes and change in lifestyle. Obesity is associated with multiple comorbidities such as arterial hypertension, insulin resistance, atherosclerosis, ischemic heart disease, alteration in immune response including immune deficiencies, carcinogenesis etc. The latter are associated with high disability and premature mortality rates. Endothelial dysfunction is the major pathogenic factor for all of the above-mentioned complications in obese individuals. It is tightly linked to systemic chronic inflammatory response, oxidative stress, and an imbalance in adipokines from lipidoverloaded adipose tissue. Mechanisms responsible for the development of endothelial dysfunction and endothelial protection in obesity are complex and not fully understood. An in-depth assessment of the early, reversible manifestations of endothelial damage is needed. A previous study on fructose-induced obesity in rat model demonstrates that fructose-rich diet induces obesity, inflammation and oxidative stress, and decreased NOS3 expression in abdominal aorta endothelial cells. These observations also correlated with pathomorphological changes and remodeling of the aorta along with the impaired functional capacity indicative of endothelial dysfunction.

The aim of this study is to investigate the molecular mechanisms for early, reversible manifestation of obesity-associated endothelial damage through the investigation of a complex link between the pathomorphological vascular changes, factors responsible for neovascularization and markers of oxidative stress in fructose-induced obesity model.

Expected results: new knowledge on mechanisms responsible for both endothelial dysfunction and protection of endothelial cells is expected to be obtained. Furthermore, the project team aims to create a panel of clinically relevant markers for early identification cardiovascular disorders and associated pathologies, which in turn will potentially transform into a new therapeutic approach depending on angiogenesis capacity.