Abstract:

Hypercholesterolemia, defined as elevated LDL-cholesterol levels in the blood, develops either as a result of genetic predispositions, unhealthy lifestyle, underlying diseases, or as a result of a combination of these factors. LDL-cholesterol is sometimes called "bad" cholesterol because its excess negatively affects the vessel`s innermost layer - endothelium, Endothelium is unique for its vasodilatory, vasoconstrictive, anti-inflammatory, and anti-coagulant function but also for its ability to control vascular permeability. In the case of hypercholesterolemia, cholesterol gradually accumulates in the subendothelial space and reduces levels of the main modulatory molecule of the endothelium - nitric oxide by several mechanisms. In endothelial dysfunction, oxidative stress increases, LDL-cholesterol is oxidized, endothelial cells are activated and produce proinflammatory cytokines and adhesive molecules. Endothelial dysfunction is considered the first stage of atherosclerosis, as monocytes enter the site of inflammation and differentiate into macrophages, which subsequently turn into foam cells by endocytosis of oxidized LDL-cholesterol. In this way, atherosclerotic plaques are formed, which not only narrow the blood vessels, but plaques can erupt, causing subsequent thrombosis, which can result in another very serious cardiovascular disease and even death. This thesis summarizes the main mechanisms by which LDL-cholesterol modulates endothelial dysfunction and subsequent formation of atherosclerotic plaques.