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Description

Atherosclerosis is a widespread chronic inflammatory disorder of the arterial wall that often leads to disability and even death. At its final stages, atherosclerosis manifests itself as a lesion of the intimal layer of the arterial wall and accumulation of plaques. Subsequent erosion or rupture of atherosclerotic plaques triggers thrombotic events that can potentially be fatal. Decades of intensive research made it clear that atherosclerosis has complex pathogenesis, the main components of which are lipid accumulation and chronic inflammation in the arterial wall [1]. Atherosclerosis is classically associated with altered lipid metabolism and hypercholesterolemia [2]. An elevated level of circulating modified low-density lipoprotein (LDL) is a known risk factor of cardiovascular diseases [3]. However, the disease pathogenesis appears to be more complex than lipid metabolism changes and involves multiple factors, the most prominent of which is inflammation [4]. The chain of pathological events that leads to atherosclerosis development is believed to be initiated by local endothelial dysfunction, which may be caused by blood flow turbulence near the sites of artery bends or bifurcations [5].

Detailed study of atherosclerotic lesion development is complicated