



Autophagy: Chapter 6. The Role of Autophagy in Atherosclerosis

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Atherosclerosis is a chronic inflammatory disease of large and middle-sized blood vessels, and the leading cause of death among adults in the Western world. Recent evidence suggests that several molecular and cellular mechanisms play an important role in atherosclerosis and plaque progression. One of these mechanisms includes autophagy, a subcellular process for elimination of damaged organelles and protein aggregates via lysosomes. According to in vitro observations, the autophagic machinery is stimulated by several stress-related stimuli inside plaques, such as oxidized lipids, endoplasmic reticulum stress, hypoxia, nutrient deprivation, and inflammation. Although its role in atherosclerosis has not yet been fully established, a growing body of evidence indicates that autophagy has a protective function in atherosclerosis. It stimulates cholesterol efflux and reduces foam cell formation. Moreover, it prevents apoptosis by removing oxidatively damaged hyperpolarized mitochondria before reactive oxygen species production and cytochrome c release. Another important recent finding is that macrophage autophagy plays an essential role in delaying lesion progression by suppressing inflammasome activation. Interestingly, excessive everolimus-induced autophagy leads to selective macrophage death, and is a promising plaque-stabilizing strategy. Overall, autophagy seems to be a major player in atherosclerosis, but further research has to be performed to fully clarify its role in this disease.

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