

Autophagy mediates tumor necrosis factor- α -induced phenotype switching in vascular smooth muscle A7r5 cell line

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This is an open access article, free of all copyright, and may be freely reproduced, distributed, transmitted, modified, built upon, or otherwise used by anyone for any lawful purpose. The work is made available under the Creative Commons CC0 public domain dedication. Vascular smooth muscle cells (VSMC) dedifferentiation from a contractile to a synthetic phenotype contributes to atherosclerosis. Atherosclerotic tissue has a chronic inflammatory component with high levels of tumor necrosis factor- α (TNF- α). VSMC of atheromatous plaques have increased autophagy, a mechanism responsible for protein and intracellular organelle degradation. The aim of this study was to evaluate whether TNF- α induces phenotype switching of VSMCs and whether this effect depends on autophagy. Rat aortic Vascular smooth A7r5 cell line was used as a model to examine the phenotype switching and autophagy. These cells were stimulated with TNF- α 100 ng/mL. Autophagy was determined by measuring LC3-II and p62 protei