Angiotensin II-regulated autophagy is required for vascular smooth muscle cell hypertrophy

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phosphorylation, as well as the number of autophagic vesicles, suggesting that this peptide induces autophagy by activating phagophore initiation and elongation. These findings were confirmed by the assessment of autophagic flux by co-administering Ang II together with chloroquine (30 μM). Pharmacological antagonism of the angiotensin type 1 receptor (AT1R) with losartan and RhoA/Rho Kinase inhibition prevented Ang II-induced autophagy. Moreover, Ang II-induced A7r5 hypertrophy, evaluated by α-SMA expression and cell size, was prevented upon autophagy inhibition. Taking together, our results suggest that the induction of autophagy by an AT1R/RhoA/Rho Kinase-dependent mechanism contributes to Ang II-induced hypertrophy in VSMC.