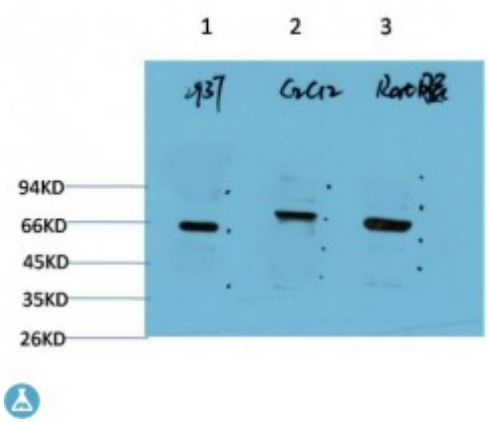
Anti-Beclin-1 antibody



Description	Beclin-1 is a protein encoded by the ECN1 gene which is approximately 51,9 kDa. Beclin-1 is localised to the cytoplasm, Golgi apparatus and mitochondrion membrane. It is involved in the autophagy pathway, deubiquitination, metabolism of proteins, apelin signalling pathway and nanomaterial induced apoptosis. The beclin-1 protein is a component of the phosphatidylinositol-3-kinase (PI3K) complex which plays a role in multiple membrane trafficking pathways. PI3KC3-C1 is involved in initiation of autophagosomes and PI3KC3-C2 in maturation of autophagosomes and endocytosis. It also plays a role in multiple cellular processes, including tumorigenesis, neurodegeneration and apoptosis. Beclin-1 is ubiquitously expressed. Mutations in the ECN1 gene can result in cervical cancer, breast cancer and Wolfram syndrome. STJ97761 was affinity-purified from rabbit. This antibody detects endogenous beclin-1.
Model	STJ97761
Host	Mouse
Reactivity	Human, Mouse, Rat
Applications	IHC, WB
Immunogen	synthetic peptide derived from Beclin-1
Immunogen Region	110-190 aa
Gene ID	8678
Gene Symbol	BECN1
Dilution range	WB 1:1000-2000IHC 1:100-200
Specificity	Beclin-1 Mouse Monoclonal Antibody (5C2) detects endogenous levels of

	BECN1
Tissue Specificity	Ubiquitous.
Purification	The antibody was affinity-purified from mouse ascites by affinity-chromatography using specific immunogen.
Clone ID	5C2
Note	For Research Use Only (RUO).
Protein Name	Beclin-1 Coiled-coil myosin-like BCL2-interacting protein Protein GT197 Beclin-1-C 35 kDa Beclin-1-C 37 kDa
Clonality	Monoclonal
Conjugation	Unconjugated
Isotype	IgG1
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Concentration	1 mg/ml
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:1034OMIM:604378
Alternative Names	Beclin-1 Coiled-coil myosin-like BCL2-interacting protein Protein GT197 Beclin-1-C 35 kDa Beclin-1-C 37 kDa
Function	Plays a central role in autophagy . Acts as core subunit of the PI3K complex that mediates formation of phosphatidylinositol 3-phosphate; different complex forms are believed to play a role in multiple membrane trafficking pathways: PI3KC3-C1 is involved in initiation of autophagosomes and PI3KC3-C2 in maturation of autophagosomes and endocytosis. Involved in regulation of degradative endocytic trafficking and required for the abcission step in cytokinesis, probably in the context of PI3KC3-C2 . Essential for the formation of PI3KC3-C2 but not PI3KC3-C1 PI3K complex forms. Involved in endocytosis . Protects against infection by a neurovirulent strain of Sindbis virus . May play a role in antiviral host defense. Beclin-1-C 35 kDa localized to mitochondria can promote apoptosis; it induces the mitochondrial translocation of BAX and the release of proapoptotic factors.
Sequence and Domain Family	The coiled coil domain can form antiparallel homodimers and mediates dimerization with the coiled coil domains of ATG14 or UVRAG involved in the formation of PI3K complexes.
Cellular Localization	Cytoplasm Golgi apparatus, trans-Golgi network membrane Endosome membrane Endoplasmic reticulum membrane Mitochondrion membrane Endosome Cytoplasmic vesicle, autophagosome. Interaction with ATG14 promotes translocation to autophagosomes. Expressed in dendrites and cell bodies of cerebellar Purkinje cells . Beclin-1-C 35 kDa: Mitochondrion Nucleus Cytoplasm Beclin-1-C 37 kDa: Mitochondrion
Post-translational Modifications	Phosphorylation at Thr-119 by DAPK1 reduces its interaction with BCL2 and BCL2L1 and promotes induction of autophagy . In response to autophagic stimuli, phosphorylated at serine residues by AMPK in an ATG14-dependent manner, and this phosphorylation is critical for maximally efficient autophagy . Polyubiquitinated by NEDD4, both with 'Lys11'- and 'Lys63'-linkages. 'Lys'-11-linked poyubiquitination leads to degradation and is enhanced when

the stabilizing interaction partner VPS34 is depleted. Deubiquitinated by USP10 and USP13, leading to stabilize the PIK3C3/VPS34-containing complexes. Proteolytically processed by caspases including CASP8 and CASP3; the C-terminal fragments lack autophagy-inducing capacity and are proposed to induce apoptosis. Thus the cleavage is proposed to be an determinant to switch from autophagy to apoptosis pathways affecting cellular homeostasis including viral infections and survival of tumor cells.