10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

AMBRA1 Antibody (C-Terminus)
Rabbit Polyclonal Antibody
Catalog # ALS12703

Specification

AMBRA1 Antibody (C-Terminus) - Product Information

Application	**IF, IHC**
Primary Accession	Q9C0C7
Reactivity	**Human, Mouse, Rat**
Host	**Rabbit**
Clonality	**Polyclonal**
Calculated MW	**143kDa KDa**

AMBRA1 Antibody (C-Terminus) - Additional Information

Gene ID 55626

Other Names
Activating molecule in BECN1-regulated autophagy protein 1, AMBRA1, KIAA1736

Reconstitution & Storage
Short term 4°C, long term aliquot and store at -20°C, avoid freeze thaw cycles. Store undiluted.

Precautions
AMBRA1 Antibody (C-Terminus) is for research use only and not for use in diagnostic or therapeutic procedures.

AMBRA1 Antibody (C-Terminus) - Protein Information

Name AMBRA1 {ECO:0000303|PubMed:17589504, ECO:0000312|HGNC:HGNC:25990}

Function
Substrate-recognition component of a DCX (DDB1-CUL4-X-box) E3 ubiquitin-protein ligase complex involved in cell cycle control and autophagy (PubMed:20921139, PubMed:23524951, PubMed:24587252, PubMed:32333458, PubMed:33854232, PubMed:33854235, PubMed:33854239). The DCX(AMBRA1) complex specifically mediates the polyubiquitination of target proteins such as BECN1, CCND1, CCND2, CCND3, ELOC and ULK1 (PubMed:23524951, PubMed:33854232, PubMed:33854235, PubMed:33854239). Acts as an upstream master regulator of the transition from G1 to S cell phase: AMBRA1 specifically

recognizes and binds phosphorylated cyclin-D (CCND1, CCND2 and CCND3), leading to cyclin-D ubiquitination by the DCX(AMBRA1) complex and subsequent degradation (PubMed:33854232, PubMed:33854235, PubMed:33854239). By controlling the transition from G1 to S phase and cyclin-D degradation, AMBRA1 acts as a tumor suppressor that promotes genomic integrity during DNA replication and counteracts developmental abnormalities and tumor growth (PubMed:33854232, PubMed:33854235, PubMed:33854239). AMBRA1 also regulates the cell cycle by promoting MYC dephosphorylation and degradation independently of the DCX(AMBRA1) complex: acts via interaction with the catalytic subunit of protein phosphatase 2A (PPP2CA), which enhances interaction between PPP2CA and MYC, leading to MYC dephosphorylation and degradation (PubMed:25438055, PubMed:25803737). Acts as a regulator of Cul5-RING (CRL5) E3 ubiquitin- protein ligase complexes by mediating ubiquitination and degradation of Elongin-C (ELOC) component of CRL5 complexes (PubMed:25499913, PubMed:30166453). Acts as a key regulator of autophagy by modulating the BECN1-PIK3C3 complex: controls protein turnover during neuronal development, and regulates normal cell survival and proliferation (PubMed:21358617). In normal conditions, AMBRA1 is tethered to the cytoskeleton via interaction with dyneins DYNLL1 and DYNLL2 (PubMed:20921139). Upon autophagy induction, AMBRA1 is released from the cytoskeletal docking site to induce autophagosome nucleation by mediating ubiquitination of proteins involved in autophagy (PubMed:20921139). The DCX(AMBRA1) complex mediates 'Lys-63'-linked ubiquitination of BECN1, increasing the association between BECN1 and PIK3C3 to promote PIK3C3 activity (By similarity). In collaboration with TRAF6, AMBRA1 mediates 'Lys-63'-linked ubiquitination of ULK1 following autophagy induction, promoting ULK1 stability and kinase activity (PubMed:23524951). Also activates ULK1 via interaction with TRIM32: TRIM32 stimulates ULK1 through unanchored 'Lys-63'-linked polyubiquitin chains (PubMed:31123703). Also acts as an activator of mitophagy via interaction with PRKN and LC3 proteins (MAP1LC3A, MAP1LC3B or MAP1LC3C); possibly by bringing damaged mitochondria onto autophagosomes (PubMed:21753002, PubMed:25215947). Also activates mitophagy by acting as a cofactor for HUWE1; acts by promoting HUWE1- mediated ubiquitination of MFN2 (PubMed:30217973). AMBRA1 is also involved in regulatory T-cells (Treg) differentiation by promoting FOXO3 dephosphorylation independently of the DCX(AMBRA1) complex: acts via interaction with PPP2CA, which enhances interaction between PPP2CA and FOXO3, leading to FOXO3 dephosphorylation and stabilization (PubMed:30513302). May act as a regulator of intracellular trafficking, regulating the localization of active PTK2/FAK and SRC (By similarity). Also involved in transcription regulation by acting as a scaffold for protein complexes at chromatin (By similarity).

Cellular Location

Endoplasmic reticulum. Cytoplasm, cytoskeleton. Cytoplasmic vesicle, autophagosome {ECO:0000250|UniProtKB:A2AH22}. Mitochondrion. Cytoplasm, cytosol {ECO:0000250|UniProtKB:A2AH22}. Nucleus. Cell junction, focal adhesion {ECO:0000250|UniProtKB:A2AH22}. Note=Localizes to the cytoskeleton in absence of autophagy

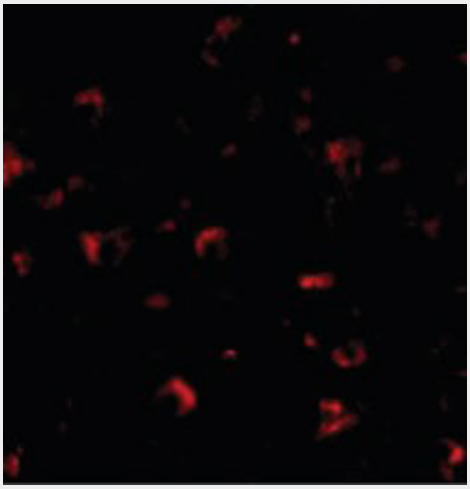
induction (PubMed:20921139). Upon autophagy induction, AMBRA1 relocalizes to the endoplasmic reticulum to enable autophagosome nucleation (PubMed:20921139). Partially localizes at mitochondria in normal conditions (PubMed:21358617). Localizes also to discrete punctae along the ciliary axoneme (By similarity) {ECO:0000250|UniProtKB:A2AH22, ECO:0000269|PubMed:20921139, ECO:0000269|PubMed:21358617}

AMBRA1 Antibody (C-Terminus) - Protocols

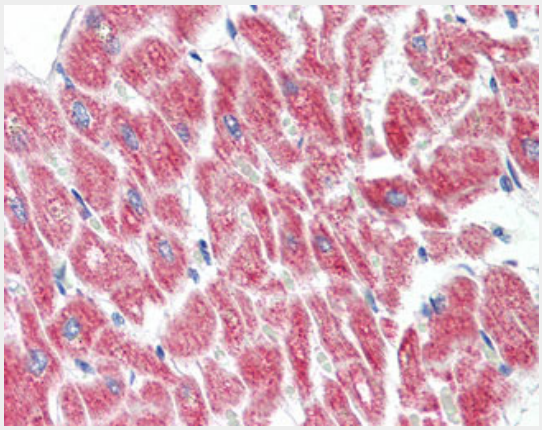
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

AMBRA1 Antibody (C-Terminus) - Images



Immunofluorescence of Ambra1 in Human Brain cells with Ambra1 antibody at 20 ug/ml.



Anti-AMBRA1 antibody IHC of human heart.

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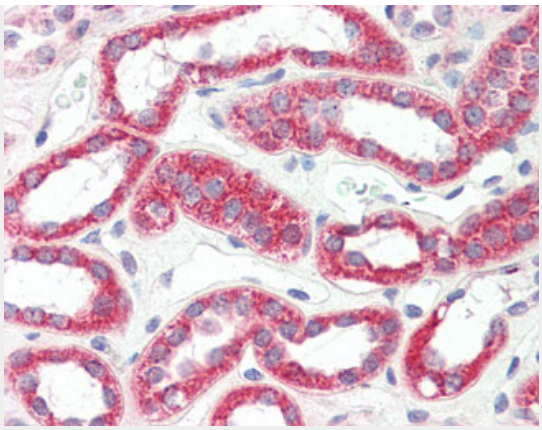

Anti-AMBRA1 antibody IHC of human kidney.

AMBRA1 Antibody (C-Terminus) - Background

Regulates autophagy and development of the nervous system. Involved in autophagy in controlling protein turnover during neuronal development, and in regulating normal cell survival and proliferation (By similarity).

AMBRA1 Antibody (C-Terminus) - References

Maria Fimia G.,et al.Nature 447:1121-1125(2007).
Nagase T.,et al.DNA Res. 7:347-355(2000).
Ota T.,et al.Nat. Genet. 36:40-45(2004).
Bechtel S.,et al.BMC Genomics 8:399-399(2007).
Taylor T.D.,et al.Nature 440:497-500(2006).