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PI3KC3 Antibody (N-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP8014a

Product Information

Application	IHC-P, IF, WB, E
Primary Accession	Q8NEB9
Other Accession	Q6AZN6, O88763, Q5D891, Q6PF93
Reactivity	Human, Rat, Mouse
Predicted	Mouse, Pig, Rat, Xenopus, Bovine, Canine, Rabbit, Chicken
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	101549
Antigen Region	24-53

Additional Information

Gene ID	5289
Other Names	Phosphatidylinositol 3-kinase catalytic subunit type 3, PI3-kinase type 3, PI3K type 3, PtdIns-3-kinase type 3, Phosphatidylinositol 3-kinase p100 subunit, Phosphoinositide-3-kinase class 3, hVps34, PIK3C3, VPS34
Target/Specificity	This PI3KC3 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 24-53 amino acids from the N-terminal region of human PI3KC3.
Dilution	IHC-P~~1:100~500 IF~~1:10~50 WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	PI3KC3 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	PIK3C3 (HGNC:8974)
Synonyms	VPS34 {ECO:0000305}

Function	Catalytic 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 (PubMed:14617358, PubMed:33637724, PubMed:7628435, PubMed:33993848). As part of PI3KC3- C1, promotes endoplasmic reticulum membrane curvature formation prior to vesicle budding (PubMed:32690950). Involved in regulation of degradative endocytic trafficking and required for the abscission step in cytokinesis, probably in the context of PI3KC3-C2 (PubMed:20208530, PubMed:20643123). Involved in the transport of lysosomal enzyme precursors to lysosomes (By similarity). Required for transport from early to late endosomes (By similarity).
Cellular Location	Midbody. Late endosome. Cytoplasmic vesicle, autophagosome. Note=As component of the PI3K complex I localized to pre-autophagosome structures. As component of the PI3K complex II localized predominantly to endosomes (PubMed:14617358). Also localizes to discrete punctae along the ciliary axoneme and to the base of the ciliary axoneme (By similarity) {ECO:0000250|UniProtKB:Q6PF93, ECO:0000305|PubMed:14617358}
Tissue Location	Ubiquitously expressed, with a highest expression in skeletal muscle.

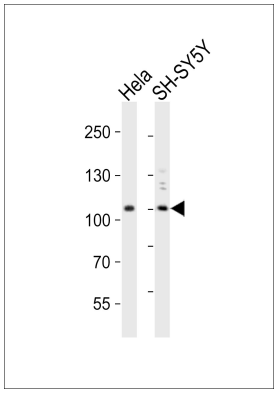
Background

PI3KC3 is a catalytic subunit of the PI3K complex involved in the transport of lysosomal enzyme precursors to lysosomes. This enzyme acts catalytically to convert 1-phosphatidyl-1D-myo-inositol to 1-phosphatidyl-1D-myo-inositol 3-phosphate. Macroautophagy is the major inducible pathway for the general turnover of cytoplasmic constituents in eukaryotic cells, it is also responsible for the degradation of active cytoplasmic enzymes and organelles during nutrient starvation. Macroautophagy involves the formation of double-membrane bound autophagosomes which enclose the cytoplasmic constituent targeted for degradation in a membrane bound structure, which then fuse with the lysosome (or vacuole) releasing a single-membrane bound autophagic bodies which are then degraded within the lysosome (or vacuole). The regulation of the Beclin 1-PI3KC3 complex lipid kinase activity is a critical element in the autophagy signaling pathway.

References

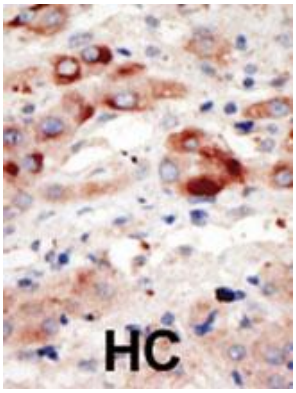
Vergne, I., et al., J. Exp. Med. 198(4):653-659 (2003). Volinia, S., et al., EMBO J. 14(14):3339-3348 (1995).

Images

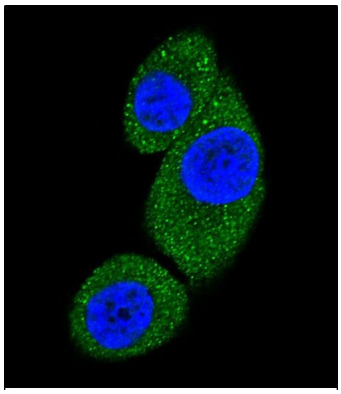


Western blot analysis of lysates from Hela, SH-SY5Y cell line (from left to right), using PI3KC3 Antibody (N-term)(Cat. #AP8014A). AP8014A was diluted at 1:1000 at each lane. A goat anti-rabbit IgG H&L(HRP) at 1:10000 dilution was used as the secondary antibody. Lysates at 20ug per lane.

Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was



peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.



Confocal immunofluorescent analysis of PI3KC3 Antibody (N-term)(Cat#AP8014a) with Hela cell followed by Alexa Fluor 488-conjugated goat anti-rabbit lgG (green).DAPI was used to stain the cell nuclear (blue).

Citations

- A truncating mutation in the autophagy gene UVRAG drives inflammation and tumorigenesis in mice
- HOTAIR/miR-326/FUT6 axis facilitates colorectal cancer progression through regulating fucosylation of CD44 via PI3K/AKT/mTOR pathway.
- Induction of autophagy is essential for monocyte-macrophage differentiation.
- Autophagic and tumour suppressor activity of a novel Beclin1-binding protein UVRAG.
- Is autophagy rather than apoptosis the regression driver in imatinib-treated gastrointestinal stromal tumors?
- The class III kinase Vps34 promotes T lymphocyte survival through regulating IL-7Rα surface expression.
- A non-canonical MEK/ERK signaling pathway regulates autophagy via regulating Beclin 1.
- Deletion of PIK3C3/Vps34 in sensory neurons causes rapid neurodegeneration by disrupting the endosomal but not the autophagic pathway.
- ARD1 contributes to IKKβ-mediated breast cancer tumorigenesis.
- The Class I PI3K inhibitor S14161 induces autophagy in malignant blood cells by modulating the Beclin 1/Vps34 complex.
- Capsaicin Induces Autophagy and Apoptosis in Human Nasopharyngeal Carcinoma Cells by Downregulating the PI3K/AKT/mTOR Pathway.
- Autophagy Mediates Cytotoxicity of Human Colorectal Cancer Cells Treated with Garcinielliptone FC.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.