

PI3KC3 Antibody (S164) Blocking Peptide
Synthetic peptide
Catalog # BP1851g**Specification**

PI3KC3 Antibody (S164) Blocking Peptide - Product InformationPrimary Accession [Q8NEB9](#)**PI3KC3 Antibody (S164) Blocking Peptide - 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

The synthetic peptide sequence used to generate the antibody [AP1851g](/product/products/AP1851g) was selected from the S164 region of human PI3KC3. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

PI3KC3 Antibody (S164) Blocking Peptide - 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](http://www.uniprot.org/citations/14617358), PubMed: [7628435](http://www.uniprot.org/citations/7628435), PubMed: [33637724](http://www.uniprot.org/citations/33637724)). As part of PI3KC3-C1, promotes endoplasmic reticulum membrane curvature formation prior to vesicle budding (PubMed: [33637724](#)).

href="http://www.uniprot.org/citations/32690950" target="_blank">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). Localizes also 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.

PI3KC3 Antibody (S164) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

PI3KC3 Antibody (S164) Blocking Peptide - Images

PI3KC3 Antibody (S164) Blocking Peptide - 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.

PI3KC3 Antibody (S164) Blocking Peptide - References

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