

MTM1 Antibody (N-term) Blocking Peptide
Synthetic peptide
Catalog # BP6809a**Specification**

MTM1 Antibody (N-term) Blocking Peptide - Product InformationPrimary Accession [Q13496](#)**MTM1 Antibody (N-term) Blocking Peptide - Additional Information**

Gene ID 4534

Other NamesMyotubularin, Phosphatidylinositol-3, 5-bisphosphate 3-phosphatase,
Phosphatidylinositol-3-phosphate phosphatase, MTM1, CG2**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP6809a](/product/products/AP6809a) was selected from the N-term region of human MTM1. 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.

MTM1 Antibody (N-term) Blocking Peptide - Protein InformationName MTM1 ([HGNC:7448](#))

Synonyms CG2

Function

Lipid phosphatase which dephosphorylates phosphatidylinositol 3-monophosphate (PI3P) and phosphatidylinositol 3,5-bisphosphate (PI(3,5)P2) (PubMed: [11001925](http://www.uniprot.org/citations/11001925), PubMed: [10900271](http://www.uniprot.org/citations/10900271), PubMed: [12646134](http://www.uniprot.org/citations/12646134), PubMed: [14722070](http://www.uniprot.org/citations/14722070)). Has also been shown to dephosphorylate phosphotyrosine- and phosphoserine-containing peptides (PubMed: [9537414](http://www.uniprot.org/citations/9537414)). Negatively regulates EGFR degradation through regulation of EGFR trafficking from the late

endosome to the lysosome (PubMed:14722070). Plays a role in vacuolar formation and morphology. Regulates desmin intermediate filament assembly and architecture (PubMed:21135508). Plays a role in mitochondrial morphology and positioning (PubMed:21135508). Required for skeletal muscle maintenance but not for myogenesis (PubMed:21135508). In skeletal muscles, stabilizes MTMR12 protein levels (PubMed:23818870).

Cellular Location

Cytoplasm. Cell membrane; Peripheral membrane protein. Cell projection, filopodium. Cell projection, ruffle. Late endosome. Cytoplasm, myofibril, sarcomere {ECO:0000250|UniProtKB:Q9Z2C5}. Note=Localizes as a dense cytoplasmic network (PubMed:11001925). Also localizes to the plasma membrane, including plasma membrane extensions such as filopodia and ruffles (PubMed:12118066). Predominantly located in the cytoplasm following interaction with MTMR12 (PubMed:12847286). Recruited to the late endosome following EGF stimulation (PubMed:14722070). In skeletal muscles, co-localizes with MTMR12 in the sarcomere (By similarity) {ECO:0000250|UniProtKB:Q9Z2C5, ECO:0000269|PubMed:11001925, ECO:0000269|PubMed:12118066, ECO:0000269|PubMed:12847286, ECO:0000269|PubMed:14722070}

MTM1 Antibody (N-term) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

MTM1 Antibody (N-term) Blocking Peptide - Images

MTM1 Antibody (N-term) Blocking Peptide - Background

MTM1 is a member of a protein family that encodes tyrosine phosphatases. Myotubularin is required for muscle cell differentiation and mutations in MTM1 have been identified as being responsible for X-linked myotubular myopathy. MTM1 is a potent phosphatidylinositol 3-phosphate phosphatase (PI(3)P). Mutations in the MTM1 gene that cause human myotubular myopathy dramatically reduce the ability of the phosphatase to dephosphorylate PI(3)P. The findings provided evidence that myotubularin exerts its effects during myogenesis by regulating the cellular levels of the inositol lipid PI(3)P.

MTM1 Antibody (N-term) Blocking Peptide - References

Nandurkar, H.H., et al., Proc. Natl. Acad. Sci. U.S.A. 100(15):8660-8665 (2003).Biancalana, V., et al., Hum. Genet. 112(2):135-142 (2003).Wishart, M.J., et al., Trends Cell Biol. 12(12):579-585 (2002).Herman, G.E., et al., Hum. Mutat. 19(2):114-121 (2002).Sutton, I.J., et al., Neurology 57(5):900-902 (2001).