

MAPK3 Blocking Peptide (N-term) Synthetic peptide Catalog # BP21753a

Specification

MAPK3 Blocking Peptide (N-term) - Product Information

Primary Accession

<u>P27361</u>

MAPK3 Blocking Peptide (N-term) - Additional Information

Gene ID 5595

Other Names

Mitogen-activated protein kinase 3, MAP kinase 3, MAPK 3, ERT2, Extracellular signal-regulated kinase 1, ERK-1, Insulin-stimulated MAP2 kinase, MAP kinase isoform p44, p44-MAPK, Microtubule-associated protein 2 kinase, p44-ERK1, MAPK3, ERK1, PRKM3

Target/Specificity

The synthetic peptide sequence is selected from aa 23-37 of HUMAN MAPK3

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.

MAPK3 Blocking Peptide (N-term) - Protein Information

Name MAPK3

Synonyms ERK1, PRKM3

Function

Serine/threonine kinase which acts as an essential component of the MAP kinase signal transduction pathway (PubMed:34497368). MAPK1/ERK2 and MAPK3/ERK1 are the 2 MAPKs which play an important role in the MAPK/ERK cascade. They participate also in a signaling cascade initiated by activated KIT and KITLG/SCF. Depending on the cellular context, the MAPK/ERK cascade mediates diverse biological functions such as cell growth, adhesion, survival and differentiation through the regulation of transcription, translation, cytoskeletal rearrangements. The MAPK/ERK cascade also plays a role in initiation and regulation of meiosis, mitosis, and postmitotic functions in differentiated cells by phosphorylating a number of transcription factors. About 160 substrates have already been discovered for ERKs. Many of these substrates are localized in the nucleus, and seem to participate in the regulation of transcription upon stimulation. However, other substrates



are found in the cytosol as well as in other cellular organelles, and those are responsible for processes such as translation, mitosis and apoptosis. Moreover, the MAPK/ERK cascade is also involved in the regulation of the endosomal dynamics, including lysosome processing and endosome cycling through the perinuclear recycling compartment (PNRC); as well as in the fragmentation of the Golgi apparatus during mitosis. The substrates include transcription factors (such as ATF2, BCL6, ELK1, ERF, FOS, HSF4 or SPZ1), cytoskeletal elements (such as CANX, CTTN, GJA1, MAP2, MAPT, PXN, SORBS3 or STMN1), regulators of apoptosis (such as BAD, BTG2, CASP9, DAPK1, IER3, MCL1 or PPARG), regulators of translation (such as EIF4EBP1) and a variety of other signaling-related molecules (like ARHGEF2, DEPTOR, FRS2 or GRB10) (PubMed:>35216969). Protein kinases (such as RAF1, RPS6KA1/RSK1, RPS6KA3/RSK2, RPS6KA2/RSK3, RPS6KA6/RSK4, SYK, MKNK1/MNK1, MKNK2/MNK2, RPS6KA5/MSK1, RPS6KA4/MSK2, MAPKAPK3 or MAPKAPK5) and phosphatases (such as DUSP1, DUSP4, DUSP6 or DUSP16) are other substrates which enable the propagation the MAPK/ERK signal to additional cytosolic and nuclear targets, thereby extending the specificity of the cascade.

Cellular Location

Cytoplasm {ECO:0000250|UniProtKB:P21708}. Nucleus. Membrane, caveola {ECO:0000250|UniProtKB:P21708}. Cell junction, focal adhesion {ECO:0000250|UniProtKB:Q63844} Note=Autophosphorylation at Thr-207 promotes nuclear localization (PubMed:19060905). PEA15-binding redirects the biological outcome of MAPK3 kinase-signaling by sequestering MAPK3 into the cytoplasm (By similarity). {ECO:0000250|UniProtKB:Q63844, ECO:0000269|PubMed:19060905}

MAPK3 Blocking Peptide (N-term) - Protocols

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

Blocking Peptides

MAPK3 Blocking Peptide (N-term) - Images

MAPK3 Blocking Peptide (N-term) - Background

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MAPK3 Blocking Peptide (N-term) - References

Charest D.L., et al.Mol. Cell. Biol. 13:4679-4690(1993). Aebersold D.M., et al.Submitted (APR-2001) to the EMBL/GenBank/DDBJ databases. Cheng H., et al.Submitted (FEB-2006) to the EMBL/GenBank/DDBJ databases. Martin J., et al.Nature 432:988-994(2004). Mural R.J., et al.Submitted (JUL-2005) to the EMBL/GenBank/DDBJ databases.