

**JNK1 Blocking Peptide (Thr183/Tyr185)**

Synthetic peptide

Catalog # BP22143a

**Specification**

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**JNK1 Blocking Peptide (Thr183/Tyr185) - Product Information**

Primary Accession

[P45983](#)

Other Accession

[O91Y86](#), [P49185](#), [Q8QHK8](#), [P53779](#), [O61831](#),  
[P49187](#)**JNK1 Blocking Peptide (Thr183/Tyr185) - Additional Information****Gene ID** 5599**Other Names**

Mitogen-activated protein kinase 8, MAP kinase 8, MAPK 8, 2.7.11.24, JNK-46, Stress-activated protein kinase 1c, SAPK1c, Stress-activated protein kinase JNK1, c-Jun N-terminal kinase 1, MAPK8, JNK1, PRKM8, SAPK1, SAPK1C

**Target/Specificity**

The synthetic peptide sequence is selected from aa 177-189 of HUMAN MAPK8

**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.

**JNK1 Blocking Peptide (Thr183/Tyr185) - Protein Information****Name** MAPK8**Function**

Serine/threonine-protein kinase involved in various processes such as cell proliferation, differentiation, migration, transformation and programmed cell death. Extracellular stimuli such as pro- inflammatory cytokines or physical stress stimulate the stress- activated protein kinase/c-Jun N-terminal kinase (SAP/JNK) signaling pathway (PubMed:<a href="http://www.uniprot.org/citations/28943315" target="\_blank">28943315</a>). In this cascade, two dual specificity kinases MAP2K4/MKK4 and MAP2K7/MKK7 phosphorylate and activate MAPK8/JNK1. In turn, MAPK8/JNK1 phosphorylates a number of transcription factors, primarily components of AP-1 such as JUN, JDP2 and ATF2 and thus regulates AP-1 transcriptional activity (PubMed:<a href="http://www.uniprot.org/citations/18307971" target="\_blank">18307971</a>). Phosphorylates the replication licensing factor CDT1, inhibiting the interaction between CDT1 and the histone H4 acetylase HBO1 to replication origins (PubMed:<a

<http://www.uniprot.org/citations/21856198> target="\_blank">21856198</a>). Loss of this interaction abrogates the acetylation required for replication initiation (PubMed:<a href="http://www.uniprot.org/citations/21856198" target="\_blank">21856198</a>). Promotes stressed cell apoptosis by phosphorylating key regulatory factors including p53/TP53 and Yes-associated protein YAP1 (PubMed:<a href="http://www.uniprot.org/citations/21364637" target="\_blank">21364637</a>). In T-cells, MAPK8 and MAPK9 are required for polarized differentiation of T-helper cells into Th1 cells. Contributes to the survival of erythroid cells by phosphorylating the antagonist of cell death BAD upon EPO stimulation (PubMed:<a href="http://www.uniprot.org/citations/21095239" target="\_blank">21095239</a>). Mediates starvation-induced BCL2 phosphorylation, BCL2 dissociation from BECN1, and thus activation of autophagy (PubMed:<a href="http://www.uniprot.org/citations/18570871" target="\_blank">18570871</a>). Phosphorylates STMN2 and hence regulates microtubule dynamics, controlling neurite elongation in cortical neurons (By similarity). In the developing brain, through its cytoplasmic activity on STMN2, negatively regulates the rate of exit from multipolar stage and of radial migration from the ventricular zone (By similarity). Phosphorylates several other substrates including heat shock factor protein 4 (HSF4), the deacetylase SIRT1, ELK1, or the E3 ligase ITCH (PubMed:<a href="http://www.uniprot.org/citations/16581800" target="\_blank">16581800</a>, PubMed:<a href="http://www.uniprot.org/citations/17296730" target="\_blank">17296730</a>, PubMed:<a href="http://www.uniprot.org/citations/20027304" target="\_blank">20027304</a>). Phosphorylates the CLOCK-BMAL1 heterodimer and plays a role in the regulation of the circadian clock (PubMed:<a href="http://www.uniprot.org/citations/22441692" target="\_blank">22441692</a>). Phosphorylates the heat shock transcription factor HSF1, suppressing HSF1-induced transcriptional activity (PubMed:<a href="http://www.uniprot.org/citations/10747973" target="\_blank">10747973</a>). Phosphorylates POU5F1, which results in the inhibition of POU5F1's transcriptional activity and enhances its proteasomal degradation (By similarity). Phosphorylates JUND and this phosphorylation is inhibited in the presence of MEN1 (PubMed:<a href="http://www.uniprot.org/citations/22327296" target="\_blank">22327296</a>). In neurons, phosphorylates SYT4 which captures neuronal dense core vesicles at synapses (By similarity). Phosphorylates EIF4ENIF1/4-ET in response to oxidative stress, promoting P-body assembly (PubMed:<a href="http://www.uniprot.org/citations/22966201" target="\_blank">22966201</a>). Phosphorylates SIRT6 in response to oxidative stress, stimulating its mono-ADP-ribosyltransferase activity (PubMed:<a href="http://www.uniprot.org/citations/27568560" target="\_blank">27568560</a>). Phosphorylates NLRP3, promoting assembly of the NLRP3 inflammasome (PubMed:<a href="http://www.uniprot.org/citations/28943315" target="\_blank">28943315</a>). Phosphorylates ALKBH5 in response to reactive oxygen species (ROS), promoting ALKBH5 sumoylation and inactivation (PubMed:<a href="http://www.uniprot.org/citations/34048572" target="\_blank">34048572</a>).

### Cellular Location

Cytoplasm. Nucleus. Synapse {ECO:0000250|UniProtKB:P49185}. Note=In the cortical neurons, predominantly cytoplasmic and associated with the Golgi apparatus and endosomal fraction. Increased neuronal activity increases phosphorylated form at synapses (By similarity). Colocalizes with POU5F1 in the nucleus. {ECO:0000250|UniProtKB:P49185, ECO:0000250|UniProtKB:Q91Y86}

## JNK1 Blocking Peptide (Thr183/Tyr185) - Protocols

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

- [Blocking Peptides](#)

## JNK1 Blocking Peptide (Thr183/Tyr185) - Images

## JNK1 Blocking Peptide (Thr183/Tyr185) - Background

Serine/threonine-protein kinase involved in various processes such as cell proliferation, differentiation, migration, transformation and programmed cell death. Extracellular stimuli such as

proinflammatory cytokines or physical stress stimulate the stress-activated protein kinase/c-Jun N-terminal kinase (SAP/JNK) signaling pathway. In this cascade, two dual specificity kinases MAP2K4/MKK4 and MAP2K7/MKK7 phosphorylate and activate MAPK8/JNK1. In turn, MAPK8/JNK1 phosphorylates a number of transcription factors, primarily components of AP-1 such as JUN, JDP2 and ATF2 and thus regulates AP-1 transcriptional activity. Phosphorylates the replication licensing factor CDT1, inhibiting the interaction between CDT1 and the histone H4 acetylase HBO1 to replication origins. Loss of this interaction abrogates the acetylation required for replication initiation. Promotes stressed cell apoptosis by phosphorylating key regulatory factors including p53/TP53 and Yes-associates protein YAP1. In T-cells, MAPK8 and MAPK9 are required for polarized differentiation of T-helper cells into Th1 cells. Contributes to the survival of erythroid cells by phosphorylating the antagonist of cell death BAD upon EPO stimulation. Mediates starvation-induced BCL2 phosphorylation, BCL2 dissociation from BECN1, and thus activation of autophagy. Phosphorylates STMN2 and hence regulates microtubule dynamics, controlling neurite elongation in cortical neurons. In the developing brain, through its cytoplasmic activity on STMN2, negatively regulates the rate of exit from multipolar stage and of radial migration from the ventricular zone. Phosphorylates several other substrates including heat shock factor protein 4 (HSF4), the deacetylase SIRT1, ELK1, or the E3 ligase ITCH. Phosphorylates the CLOCK-ARNTL/BMAL1 heterodimer and plays a role in the regulation of the circadian clock (PubMed:22441692).

#### **JNK1 Blocking Peptide (Thr183/Tyr185) - References**

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Deloukas P., et al. Nature 429:375-381(2004).  
Goshima N., et al. Nat. Methods 5:1011-1017(2008).