

Mouse Irf3 Blocking Peptide (N-term)

Synthetic peptide Catalog # BP20073a

Specification

Mouse Irf3 Blocking Peptide (N-term) - Product Information

Primary Accession
Other Accession

<u>Q764M6</u>, <u>Q4IF28</u>, <u>NP 058545.1</u>

P70671

Mouse Irf3 Blocking Peptide (N-term) - Additional Information

Gene ID 54131

Other Names

Interferon regulatory factor 3, IRF-3, Irf3

Target/Specificity

The synthetic peptide sequence is selected from aa 61-72 of MOUSE Irf3

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.

Mouse Irf3 Blocking Peptide (N-term) - Protein Information

Name Irf3

Function

Key transcriptional regulator of type I interferon (IFN)- dependent immune responses which plays a critical role in the innate immune response against DNA and RNA viruses (PubMed:15800576). Regulates the transcription of type I IFN genes (IFN-alpha and IFN- beta) and IFN-stimulated genes (ISG) by binding to an interferon- stimulated response element (ISRE) in their promoters (PubMed:15800576). Acts as a more potent activator of the IFN-beta (IFNB) gene than the IFN-alpha (IFNA) gene and plays a critical role in both the early and late phases of the IFNA/B gene induction (PubMed:16846591/, PubMed:20049431/a>). Found in an inactive form in the cytoplasm of uninfected cells and following viral infection, double-stranded RNA (dsRNA), or toll-like receptor (TLR) signaling, is phosphorylated by IKBKE and TBK1 kinases (PubMed:16846591 | blank">16846591/a>,



PubMed:16979567, PubMed:20049431). This induces a conformational change, leading to its dimerization and nuclear localization and association with CREB binding protein (CREBBP) to form dsRNA-activated factor 1 (DRAF1), a complex which activates the transcription of the type I IFN and ISG genes (PubMed:16846591, PubMed:20049431, PubMed:20049431). Can activate distinct gene expression programs in macrophages and can induce significant apoptosis in primary macrophages (PubMed:16846591" target="_blank">16846591, PubMed:16979567, PubMed:16979567, PubMed:20049431 href="http://www.uniprot.org/citations/20049431" target="_blank">20049431

Cellular Location

Cytoplasm {ECO:0000250|UniProtKB:Q14653}. Nucleus {ECO:0000250|UniProtKB:Q14653}. Mitochondrion {ECO:0000250|UniProtKB:Q14653}. Note=Shuttles between cytoplasmic and nuclear compartments, with export being the prevailing effect. When activated, IRF3 interaction with CREBBP prevents its export to the cytoplasm. Recruited to mitochondria via TOMM70:HSP90AA1 upon Sendai virus infection. {ECO:0000250|UniProtKB:Q14653}

Mouse Irf3 Blocking Peptide (N-term) - Protocols

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

• Blocking Peptides

Mouse Irf3 Blocking Peptide (N-term) - Images

Mouse Irf3 Blocking Peptide (N-term) - Background

Mediates interferon-stimulated response element (ISRE) promoter activation. Functions as a molecular switch for antiviral activity. DsRNA generated during the course of an viral infection leads to IRF3 phosphorylation on the C-terminal serine/threonine cluster. This induces a conformational change, leading to its dimerization, nuclear localization and association with CREB binding protein (CREBBP) to form dsRNA-activated factor 1 (DRAF1), a complex which activates the transcription of genes under the control of ISRE. The complex binds to the IE and PRDIII regions on the IFN-alpha and IFN-beta promoters respectively. IRF-3 does not have any transcription activation domains (By similarity).

Mouse Irf3 Blocking Peptide (N-term) - References

Marichal, T., et al. J. Allergy Clin. Immunol. 126(4):836-844(2010) Menachery, V.D., et al. J. Virol. 84(19):9685-9694(2010) Carrigan, S.O., et al. J. Immunol. 185(6):3602-3609(2010) Wang, J., et al. J. Immunol. 185(3):1720-1729(2010) Farlik, M., et al. Immunity 33(1):25-34(2010)