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IRF3 Blocking Peptide (N-Term)

Synthetic peptide
Catalog # BP21782a

Specification

IRF3 Blocking Peptide (N-Term) - Product Information

Primary Accession Q14653

IRF3 Blocking Peptide (N-Term) - Additional Information

Gene ID 3661

Other Names
Interferon regulatory factor 3, IRF-3, IRF3

Target/Specificity
The synthetic peptide sequence is selected from aa 98-108 of HUMAN 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.

IRF3 Blocking Peptide (N-Term) - Protein Information

Name IRF3 {ECO:0000303|PubMed:9803267, ECO:0000312|HGNC:HGNC:6118}

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:8524823, PubMed:22394562, PubMed:25636800, PubMed:27302953, PubMed:24049179, PubMed:31340999, PubMed:36603579). 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:8524823, PubMed:11846977, PubMed:16846591, PubMed:16979567, PubMed:<a

href="http://www.uniprot.org/citations/20049431" target="_blank">20049431, PubMed:36603579, PubMed:32972995). 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:16979567, PubMed:20049431, PubMed:36603579). 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:22394562, PubMed:25636800, PubMed:36603579, PubMed:27302953). 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:16154084, PubMed:27302953, PubMed:33440148, PubMed:36603579). Can activate distinct gene expression programs in macrophages and can induce significant apoptosis in primary macrophages (PubMed:16846591). In response to Sendai virus infection, is recruited by TOMM70:HSP90AA1 to mitochondrion and forms an apoptosis complex TOMM70:HSP90AA1:IRF3:BAX inducing apoptosis (PubMed:25609812). Key transcription factor regulating the IFN response during SARS-CoV-2 infection (PubMed:33440148).

Cellular Location
Cytoplasm. Nucleus Mitochondrion. Note=Shuttles between cytoplasmic and nuclear compartments, with export being the prevailing effect (PubMed:10805757, PubMed:35922005). When activated, IRF3 interaction with CREBBP prevents its export to the cytoplasm (PubMed:10805757). Recruited to mitochondria via TOMM70:HSP90AA1 upon Sendai virus infection (PubMed:25609812).

Tissue Location
Expressed constitutively in a variety of tissues.

IRF3 Blocking Peptide (N-Term) - Protocols

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

- Blocking Peptides

IRF3 Blocking Peptide (N-Term) - Images

IRF3 Blocking Peptide (N-Term) - Background

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. 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. 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. 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. 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. Can activate distinct gene expression programs in macrophages and can induce significant apoptosis in primary macrophages.

IRF3 Blocking Peptide (N-Term) - References

Au W.W.-C.,et al.Proc. Natl. Acad. Sci. U.S.A. 92:11657-11661(1995).
Tabata Y.,et al.Submitted (FEB-2003) to the EMBL/GenBank/DDBJ databases.
Ota T.,et al.Nat. Genet. 36:40-45(2004).
Grimwood J.,et al.Nature 428:529-535(2004).
Mural R.J.,et al.Submitted (JUL-2005) to the EMBL/GenBank/DDBJ databases.