

STAT1 Blocking Peptide (C-term)
Synthetic peptide
Catalog # BP19835b**Specification**

STAT1 Blocking Peptide (C-term) - Product Information

Primary Accession [P42224](#)
Other Accession [NP_009330.1](#)

STAT1 Blocking Peptide (C-term) - Additional Information

Gene ID 6772

Other Names

Signal transducer and activator of transcription 1-alpha/beta, Transcription factor ISGF-3 components p91/p84, STAT1

Target/Specificity

The synthetic peptide sequence is selected from aa 732-745 of HUMAN STAT1

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.

STAT1 Blocking Peptide (C-term) - Protein Information

Name STAT1

Function

Signal transducer and transcription activator that mediates cellular responses to interferons (IFNs), cytokine KITLG/SCF and other cytokines and other growth factors (PubMed:9724754, PubMed:12855578, PubMed:12764129, PubMed:15322115, PubMed:34508746, PubMed:35568036, PubMed:23940278). Following type I IFN (IFN-alpha and IFN-beta) binding to cell surface receptors, signaling via protein kinases leads to activation of Jak kinases (TYK2 and JAK1) and to tyrosine phosphorylation of STAT1 and STAT2. The phosphorylated STATs dimerize and associate with ISGF3G/IRF-9 to form a complex termed ISGF3 transcription factor, that enters the nucleus (PubMed:28753426, PubMed:35568036). ISGF3 binds to the IFN stimulated response element (ISRE) to activate the transcription of IFN-stimulated genes (ISG), which drive the cell in an antiviral state (PubMed:28753426, PubMed:35568036). In response to type II IFN (IFN-gamma), STAT1 is tyrosine- and serine-phosphorylated (PubMed:26479788). It then forms a homodimer termed IFN-gamma-activated factor (GAF), migrates into the nucleus and binds to the IFN gamma activated sequence (GAS) to drive the expression of the target genes, inducing a cellular antiviral state (PubMed:8156998). Becomes activated in response to KITLG/SCF and KIT signaling (PubMed:15526160). May mediate cellular responses to activated FGFR1, FGFR2, FGFR3 and FGFR4 (PubMed:19088846). Involved in food tolerance in small intestine: associates with the Gasdermin-D, p13 cleavage product (13 kDa GSDMD) and promotes transcription of CIITA, inducing type 1 regulatory T (Tr1) cells in upper small intestine (By similarity).

Cellular Location

Cytoplasm. Nucleus Note=Translocated into the nucleus upon tyrosine phosphorylation and dimerization, in response to IFN-gamma and signaling by activated FGFR1, FGFR2, FGFR3 or FGFR4 (PubMed:15322115). Monomethylation at Lys- 525 is required for phosphorylation at Tyr-701 and translocation into the nucleus (PubMed:28753426). Translocates into the nucleus in response to interferon-beta stimulation (PubMed:26479788)

STAT1 Blocking Peptide (C-term) - Protocols

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

- [Blocking Peptides](#)

STAT1 Blocking Peptide (C-term) - Images

STAT1 Blocking Peptide (C-term) - Background

The protein encoded by this gene is a member of the STAT protein family. In response to cytokines and growth factors, STAT family members are phosphorylated by the receptor associated kinases, and then form homo- or heterodimers that translocate to the cell nucleus where they act as transcription activators. This protein can be activated by various ligands including interferon-alpha, interferon-gamma, EGF, PDGF and IL6. This protein mediates the expression of a variety of genes, which is thought to be important for cell viability in response to different cell stimuli and pathogens. Two alternatively spliced transcript variants encoding distinct isoforms have been described. [provided by RefSeq].

STAT1 Blocking Peptide (C-term) - References

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Rosas-Murrieta, N.H., et al. Virol. J. 7, 263 (2010) :
DeVries, T.A., et al. J. Biol. Chem. 279(44):45603-45612(2004)
Zhang, Y., et al. Carcinogenesis 25(7):1165-1175(2004)
Sakamoto, S., et al. J. Biol. Chem. 279(5):3245-3253(2004)