

**STAT2 Antibody (N-term) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP13749a****Specification**

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**STAT2 Antibody (N-term) Blocking peptide - Product Information**Primary Accession [P52630](#)**STAT2 Antibody (N-term) Blocking peptide - Additional Information****Gene ID** 6773**Other Names**

Signal transducer and activator of transcription 2, p113, STAT2

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody AP13749a was selected from the N-term region of STAT2. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**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.

**STAT2 Antibody (N-term) Blocking peptide - Protein Information****Name** STAT2**Function**

Signal transducer and activator of transcription that mediates signaling by type I interferons (IFN-alpha and IFN-beta). Following type I IFN binding to cell surface receptors, Jak kinases (TYK2 and JAK1) are activated, leading to tyrosine phosphorylation of STAT1 and STAT2. The phosphorylated STATs dimerize, associate with IRF9/ISGF3G to form a complex termed ISGF3 transcription factor, that enters the nucleus. ISGF3 binds to the IFN stimulated response element (ISRE) to activate the transcription of interferon stimulated genes, which drive the cell in an antiviral state (PubMed: [9020188](http://www.uniprot.org/citations/9020188), PubMed: [23391734](http://www.uniprot.org/citations/23391734)). In addition, has also a negative feedback regulatory role in the type I interferon signaling by recruiting USP18 to the type I IFN receptor subunit IFNAR2 thereby mitigating the response to type I IFNs (PubMed: [28165510](http://www.uniprot.org/citations/28165510)). Acts as a regulator of mitochondrial fission by modulating the phosphorylation of DNMT1L at 'Ser-616' and

'Ser-637' which activate and inactivate the GTPase activity of DNML1 respectively (PubMed:<a href="http://www.uniprot.org/citations/26122121" target="\_blank">26122121</a>, PubMed:<a href="http://www.uniprot.org/citations/23391734" target="\_blank">23391734</a>, PubMed:<a href="http://www.uniprot.org/citations/9020188" target="\_blank">9020188</a>).

#### **Cellular Location**

Cytoplasm. Nucleus Note=Translocated into the nucleus upon activation by IFN-alpha/beta

#### **STAT2 Antibody (N-term) Blocking peptide - Protocols**

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

- [Blocking Peptides](#)

#### **STAT2 Antibody (N-term) Blocking peptide - Images**

#### **STAT2 Antibody (N-term) Blocking peptide - Background**

The protein encoded by this gene is a member of the STATprotein family. In response to cytokines and growth factors, STATfamily members are phosphorylated by the receptor associatedkinases, and then form homo- or heterodimers that translocate tothe cell nucleus where they act as transcription activators. Inresponse to interferon (IFN), this protein forms a complex withSTAT1 and IFN regulatory factor family protein p48 (ISGF3G), inwhich this protein acts as a transactivator, but lacks the abilityto bind DNA directly. Transcription adaptor P300/CBP (EP300/CREBBP)has been shown to interact specifically with this protein, which isthought to be involved in the process of blocking IFN-alpharesponse by adenovirus. Multiple transcript variants encodingdifferent isoforms have been found for this gene. [provided byRefSeq].

#### **STAT2 Antibody (N-term) Blocking peptide - References**

Silva, L.K., et al. Eur. J. Hum. Genet. 18(11):1221-1227(2010)Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010)Han, S., et al. Hum. Immunol. 71(7):727-730(2010)Lou, Y.J., et al. Zhonghua Yi Xue Yi Chuan Xue Za Zhi 27(3):255-258(2010)Rosas-Murrieta, N.H., et al. Virol. J. 7, 263 (2010) :