

**ISG15 Antibody (C-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP1150b****Specification**

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**ISG15 Antibody (C-term) Blocking Peptide - Product Information**Primary Accession [P05161](#)**ISG15 Antibody (C-term) Blocking Peptide - Additional Information**

Gene ID 9636

**Other Names**

Ubiquitin-like protein ISG15, Interferon-induced 15 kDa protein, Interferon-induced 17 kDa protein, IP17, Ubiquitin cross-reactive protein, hUCRP, ISG15, G1P2, UCRP

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP1150b](/product/products/AP1150b) was selected from the C-term region of human ISG15. 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.

**ISG15 Antibody (C-term) Blocking Peptide - Protein Information**Name ISG15 ([HGNC:4053](#))

Synonyms G1P2, UCRP

**Function**

Ubiquitin-like protein which plays a key role in the innate immune response to viral infection either via its conjugation to a target protein (ISGylation) or via its action as a free or unconjugated protein (PubMed: <http://www.uniprot.org/citations/27564865> target="\_blank">27564865</a>). ISGylation involves a cascade of enzymatic reactions involving E1, E2, and E3 enzymes which catalyze the conjugation of ISG15 to a lysine residue in the target protein (PubMed: <http://www.uniprot.org/citations/33727702> target="\_blank">33727702</a>). Its target proteins include IFIT1, MX1/MxA, PPM1B, UBE2L6, UBA7, CHMP5, CHMP2A, CHMP4B and CHMP6. Isylation of the viral sensor IFIH1/MDA5 promotes IFIH1/MDA5 oligomerization and triggers activation of innate immunity against a range of viruses,

including coronaviruses, flaviviruses and picornaviruses (PubMed:<a href="http://www.uniprot.org/citations/33727702" target="\_blank">33727702</a>). Can also isgylate: EIF2AK2/PKR which results in its activation, RIGI which inhibits its function in antiviral signaling response, EIF4E2 which enhances its cap structure-binding activity and translation-inhibition activity, UBE2N and UBE2E1 which negatively regulates their activity, IRF3 which inhibits its ubiquitination and degradation and FLNB which prevents its ability to interact with the upstream activators of the JNK cascade thereby inhibiting IFNA-induced JNK signaling. Exhibits antiviral activity towards both DNA and RNA viruses, including influenza A, HIV-1 and Ebola virus. Restricts HIV-1 and ebola virus via disruption of viral budding. Inhibits the ubiquitination of HIV-1 Gag and host TSG101 and disrupts their interaction, thereby preventing assembly and release of virions from infected cells. Inhibits Ebola virus budding mediated by the VP40 protein by disrupting ubiquitin ligase activity of NEDD4 and its ability to ubiquitinate VP40. ISGylates influenza A virus NS1 protein which causes a loss of function of the protein and the inhibition of virus replication. The secreted form of ISG15 can: induce natural killer cell proliferation, act as a chemotactic factor for neutrophils and act as a IFN-gamma-inducing cytokine playing an essential role in antimycobacterial immunity. The secreted form acts through the integrin ITGAL/ITGB2 receptor to initiate activation of SRC family tyrosine kinases including LYN, HCK and FGR which leads to secretion of IFNG and IL10; the interaction is mediated by ITGAL (PubMed:<a href="http://www.uniprot.org/citations/29100055" target="\_blank">29100055</a>).

### **Cellular Location**

Cytoplasm. Secreted. Note=Exists in three distinct states: free within the cell, released into the extracellular space, or conjugated to target proteins

### **Tissue Location**

Detected in lymphoid cells, striated and smooth muscle, several epithelia and neurons. Expressed in neutrophils, monocytes and lymphocytes. Enhanced expression seen in pancreatic adenocarcinoma, endometrial cancer, and bladder cancer, as compared to non-cancerous tissue. In bladder cancer, the increase in expression exhibits a striking positive correlation with more advanced stages of the disease.

## **ISG15 Antibody (C-term) Blocking Peptide - Protocols**

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

- [Blocking Peptides](#)

## **ISG15 Antibody (C-term) Blocking Peptide - Images**

## **ISG15 Antibody (C-term) Blocking Peptide - Background**

ISG15 is secreted from monocytes in response to type I interferons and causes natural killer (NK)-cell proliferation and an augmentation of non-MCH (major histocompatibility complex)-restricted cytotoxicity. Synthesis is stimulated by IFN-alpha or IFN-beta or IFN-omega , but not IFN-gamma . ISG15 expression is also induced by overexpression of interferon regulatory factors that participate in transcriptional regulation of IFN genes, and by influenza B virus. ISG15 is secreted also by cell lines of monocyte, T-lymphocyte, B-lymphocyte, human fibroblasts, and epithelial origins. The induction of terminal differentiation in human melanoma cells is associated with alterations in ISG15 expression. Enhancement of NK cell proliferation, augmentation of non-major histocompatibility complex-restricted cytotoxicity, and induction of IFN-gamma from T cells identify ISG15 as a member of the cytokine cascade and suggest that it may be responsible for amplifying and directing some of the immunomodulatory effects of IFN-alpha or IFN-beta. ISG15 has also been shown to function intracellularly as a ubiquitin homolog.

## **ISG15 Antibody (C-term) Blocking Peptide - References**

Padovan, E., et al., Cancer Res. 62(12):3453-3458 (2002). Meraro, D., et al., J. Immunol. 168(12):6224-6231 (2002). Reich, N., et al., Proc. Natl. Acad. Sci. U.S.A. 84(18):6394-6398 (1987). Blomstrom, D.C., et al., J. Biol. Chem. 261(19):8811-8816 (1986). Clauss, I.M., et al., Cytogenet. Cell Genet. 53 (2-3), 166-168 (1990).