

# **BTK Antibody (Center) Blocking Peptide**

Synthetic peptide Catalog # BP7699c

## **Specification**

# BTK Antibody (Center) Blocking Peptide - Product Information

**Primary Accession** 

Q06187

# BTK Antibody (Center) Blocking Peptide - Additional Information

Gene ID 695

#### **Other Names**

Tyrosine-protein kinase BTK, Agammaglobulinemia tyrosine kinase, ATK, B-cell progenitor kinase, BPK, Bruton tyrosine kinase, BTK, AGMX1, ATK, BPK

# **Target/Specificity**

The synthetic peptide sequence used to generate the antibody <a href=/product/products/AP7699c>AP7699c</a> was selected from the Center region of human BTK . 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.

### BTK Antibody (Center) Blocking Peptide - Protein Information

#### **Name BTK**

Synonyms AGMX1, ATK, BPK

### **Function**

Non-receptor tyrosine kinase indispensable for B lymphocyte development, differentiation and signaling (PubMed:<a href="http://www.uniprot.org/citations/19290921" http://www.uniprot.org/citations/19290921"

target="\_blank">19290921</a>). Binding of antigen to the B-cell antigen receptor (BCR) triggers signaling that ultimately leads to B-cell activation (PubMed:<a

href="http://www.uniprot.org/citations/19290921" target="\_blank">19290921</a>). After BCR engagement and activation at the plasma membrane, phosphorylates PLCG2 at several sites, igniting the downstream signaling pathway through calcium mobilization, followed by activation of the protein kinase C (PKC) family members (PubMed:<a

href="http://www.uniprot.org/citations/11606584" target="\_blank">11606584</a>). PLCG2



phosphorylation is performed in close cooperation with the adapter protein B-cell linker protein BLNK (PubMed: <a href="http://www.uniprot.org/citations/11606584" target=" blank">11606584</a>). BTK acts as a platform to bring together a diverse array of signaling proteins and is implicated in cytokine receptor signaling pathways (PubMed: <a href="http://www.uniprot.org/citations/16517732" target=" blank">16517732</a>, PubMed:<a href="http://www.uniprot.org/citations/17932028" target="blank">17932028</a>). Plays an important role in the function of immune cells of innate as well as adaptive immunity, as a component of the Toll-like receptors (TLR) pathway (PubMed: <a href="http://www.uniprot.org/citations/16517732" target="\_blank">16517732</a>). The TLR pathway acts as a primary surveillance system for the detection of pathogens and are crucial to the activation of host defense (PubMed: <a href="http://www.uniprot.org/citations/16517732" target=" blank">16517732</a>). Especially, is a critical molecule in regulating TLR9 activation in splenic B-cells (PubMed: <a href="http://www.uniprot.org/citations/16517732" target=" blank">16517732</a>, PubMed:<a href="http://www.uniprot.org/citations/17932028" target="blank">17932028</a>). Within the TLR pathway, induces tyrosine phosphorylation of TIRAP which leads to TIRAP degradation (PubMed:<a href="http://www.uniprot.org/citations/16415872" target=" blank">16415872</a>). BTK also plays a critical role in transcription regulation (PubMed:<a href="http://www.uniprot.org/citations/19290921" target=" blank">19290921</a>). Induces the activity of NF- kappa-B, which is involved in regulating the expression of hundreds of genes (PubMed:<a href="http://www.uniprot.org/citations/19290921" target=" blank">19290921</a>). BTK is involved on the signaling pathway linking TLR8 and TLR9 to NF-kappa-B (PubMed: <a href="http://www.uniprot.org/citations/19290921" target=" blank">19290921</a>). Acts as an activator of NLRP3 inflammasome assembly by mediating phosphorylation of NLRP3 (PubMed: <a href="http://www.uniprot.org/citations/34554188" target=" blank">34554188</a>). Transiently phosphorylates transcription factor GTF2I on tyrosine residues in response to BCR (PubMed:<a href="http://www.uniprot.org/citations/9012831" target=" blank">9012831</a>). GTF2I then translocates to the nucleus to bind regulatory enhancer elements to modulate gene expression (PubMed:<a href="http://www.uniprot.org/citations/9012831" target=" blank">9012831</a>). ARID3A and NFAT are other transcriptional target of BTK (PubMed: <a href="http://www.uniprot.org/citations/16738337" target=" blank">16738337</a>). BTK is required for the formation of functional ARID3A DNA-binding complexes (PubMed: <a href="http://www.uniprot.org/citations/16738337" target=" blank">16738337</a>). There is however no evidence that BTK itself binds directly to DNA (PubMed:<a href="http://www.uniprot.org/citations/16738337" target=" blank">16738337</a>). BTK has a dual role in the regulation of apoptosis (PubMed:<a href="http://www.uniprot.org/citations/9751072" target=" blank">9751072</a>).

# **Cellular Location**

Cytoplasm. Cell membrane; Peripheral membrane protein. Nucleus Membrane raft {ECO:0000250|UniProtKB:P35991}. Note=In steady state, BTK is predominantly cytosolic. Following B-cell receptor (BCR) engagement by antigen, translocates to the plasma membrane through its PH domain Plasma membrane localization is a critical step in the activation of BTK. A fraction of BTK also shuttles between the nucleus and the cytoplasm, and nuclear export is mediated by the nuclear export receptor CRM1.

### **Tissue Location**

Predominantly expressed in B-lymphocytes.

# BTK Antibody (Center) Blocking Peptide - Protocols

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

Blocking Peptides

BTK Antibody (Center) Blocking Peptide - Images



# BTK Antibody (Center) Blocking Peptide - Background

BTK plays a crucial role in B-cell ontogeny. This protein transiently phosphorylates GTF2I on tyrosine residues in response to B-cell receptor cross-linking. Defects in BTK are the cause of X-linked agammaglobulinemia type 1 (XLA). XLA is a humoral immunodeficiency disease which results in developmental defects in the maturation pathway of B-cells. Affected boys have normal levels of pre-B-cells in their bone marrow but virtually no circulating mature B-lymphocytes. This results in a lack of immunoglobulins of all classes and leads to recurrent bacterial infections like otitis, conjunctivitis, dermatitis, sinusitis or fatal sepsis or meningitis within the first years of life.

## BTK Antibody (Center) Blocking Peptide - References

Marquez, J.A., et al., EMBO J. 22(18):4616-4624 (2003). Jefferies, C.A., et al., J. Biol. Chem. 278(28):26258-26264 (2003). Horwood, N.J., et al., J. Exp. Med. 197(12):1603-1611 (2003). Goodman, P.A., et al., Leuk. Lymphoma 44(6):1011-1018 (2003). Noordzij, J.G., et al., J. Clin. Immunol. 22(5):306-318 (2002).