

**BTK Blocking Peptide (Center)**  
Synthetic peptide  
Catalog # BP20992a**Specification****BTK Blocking Peptide (Center) - Product Information**

Primary Accession [Q06187](#)  
Other Accession [P35991](#), [Q8JH64](#)

**BTK Blocking Peptide (Center) - 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 is selected  
from aa 396-410 of HUMAN BTK

**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 Blocking Peptide (Center) - Protein Information**

**Name** BTK

**Synonyms** AGMX1, ATK, BPK

**Function**

Non-receptor tyrosine kinase indispensable

**BTK Blocking Peptide (Center) - Background**

Non-receptor tyrosine kinase indispensable  
for B lymphocyte development, differentiation  
and signaling. Binding of antigen to the B-cell  
antigen receptor (BCR) triggers signaling that  
ultimately leads to B-cell activation. 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. PLCG2 phosphorylation  
is performed in close cooperation with the  
adapter protein B-cell linker protein BLNK. BTK  
acts as a platform to bring together a diverse  
array of signaling proteins and is implicated in  
cytokine receptor signaling pathways. 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. The TLR pathway acts as a primary  
surveillance system for the detection of  
pathogens and are crucial to the activation of  
host defense. Especially, is a critical molecule  
in regulating TLR9 activation in splenic B-cells.  
Within the TLR pathway, induces tyrosine  
phosphorylation of TIRAP which leads to TIRAP  
degradation. BTK plays also a critical role in  
transcription regulation. Induces the activity of  
NF-kappa-B, which is involved in regulating the  
expression of hundreds of genes. BTK is  
involved on the signaling pathway linking TLR8  
and TLR9 to NF-kappa-B. Transiently  
phosphorylates transcription factor GTF2I on  
tyrosine residues in response to BCR. GTF2I  
then translocates to the nucleus to bind  
regulatory enhancer elements to modulate  
gene expression. ARID3A and NFAT are other  
transcriptional target of BTK. BTK is required  
for the formation of functional ARID3A  
DNA-binding complexes. There is however no  
evidence that BTK itself binds directly to DNA.  
BTK has a dual role in the regulation of  
apoptosis.

**BTK Blocking Peptide (Center) -**

for B lymphocyte development, differentiation and signaling. Binding of antigen to the B-cell antigen receptor (BCR) triggers signaling that ultimately leads to B-cell activation. 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. PLCG2 phosphorylation is performed in close cooperation with the adapter protein B-cell linker protein BLNK. BTK acts as a platform to bring together a diverse array of signaling proteins and is implicated in cytokine receptor signaling pathways. 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. The TLR pathway acts as a primary surveillance system for the detection of pathogens and are crucial to the activation of host defense. Especially, is a critical molecule in regulating TLR9 activation in splenic B-cells. Within the TLR pathway, induces tyrosine phosphorylation of TIRAP which leads to TIRAP degradation. BTK plays also a critical role in transcription regulation. Induces the activity of NF-kappa-B, which is involved in regulating the expression of hundreds of genes. BTK is involved on the signaling pathway linking TLR8 and TLR9 to NF-kappa-B. Transiently phosphorylates transcription factor GTF2I on tyrosine residues in response to BCR. GTF2I then translocates to the nucleus to bind regulatory enhancer elements to modulate gene expression. ARID3A and NFAT are other transcriptional target of BTK. BTK is required for the formation of functional ARID3A DNA-binding complexes. There is however no evidence that BTK itself binds directly to DNA. BTK has a dual role in the regulation of apoptosis.

### **Cellular Location**

Cytoplasm. Cell membrane; Peripheral membrane protein. Nucleus. 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

### **References**

- Vetrie D.,et al.Nature 361:226-233(1993).  
Vetrie D.,et al.Nature 364:362-362(1993).  
Ohta Y.,et al.Proc. Natl. Acad. Sci. U.S.A. 91:9062-9066(1994).  
Rohrer J.,et al.Immunogenetics 40:319-324(1994).  
Hagemann T.L.,et al.Hum. Mol. Genet. 3:1743-1749(1994).

by the nuclear export receptor CRM1

**Tissue Location**

Predominantly expressed in B-lymphocytes.

**BTK Blocking Peptide (Center) - Protocols**

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

- [Blocking Peptides](#)