

**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 [AP7699c](/product/products/AP7699c) 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****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).

**Synonyms** AGMX1, ATK, BPK

**Function**

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.

**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 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](#)