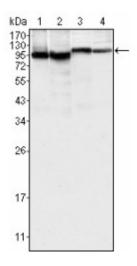


## Anti-Btk antibody



4

**Description** Mouse monoclonal to Btk.

Model STJ97885

**Host** Mouse

**Reactivity** Human, Simian

**Applications** ELISA, IF, IHC, WB

**Immunogen** Purified recombinant fragment of Btk expressed in E. Coli.

**Gene ID** 695

Gene Symbol BTK

**Dilution range** WB 1:500-1:2000IHC 1:200-1:1000IF 1:200-1:1000ELISA 1:10000

**Specificity** Btk Monoclonal Antibody detects endogenous levels of Btk protein.

**Tissue Specificity** Predominantly expressed in B-lymphocytes.

**Purification** Affinity purification

Clone ID 7F12H4

**Note** For Research Use Only (RUO).

Protein Name Tyrosine-protein kinase BTK Agammaglobulinemia tyrosine kinase ATK B-

cell progenitor kinase BPK Bruton tyrosine kinase

**Clonality** Monoclonal

**Conjugation** Unconjugated

**Isotype** IgG1

**Formulation** Ascitic fluid containing 0.03% sodium azide.

**Storage Instruction** Store at -20°C, and avoid repeat freeze-thaw cycles.

Database Links <u>HGNC:1133OMIM:300300</u>

Alternative Names Tyrosine-protein kinase BTK Agammaglobulinemia tyrosine kinase ATK B-

cell progenitor kinase BPK Bruton tyrosine kinase

**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 NFkappa-B, which is involved in regulating the expression of hundreds of genes. BTK is involved on the signaling pathway linking TLR8 and TLR9 to NFkappa-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.

**Sequence and Domain Family** The PH d

The PH domain mediates the binding to inositol polyphosphate and phosphoinositides, leading to its targeting to the plasma membrane. It is extended in the BTK kinase family by a region designated the TH (Tec homology) domain, which consists of about 80 residues preceding the SH3

domain.

**Cellular Localization** 

Cytoplasm. Cell membrane. Peripheral membrane protein. Nucleus. 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.

Post-translational Modifications Following B-cell receptor (BCR) engagement, translocates to the plasma membrane where it gets phosphorylated at Tyr-551 by LYN and SYK. Phosphorylation at Tyr-551 is followed by autophosphorylation of Tyr-223 which may create a docking site for a SH2 containing protein. Phosphorylation at Ser-180 by PRKCB, leads in translocation of BTK back to the cytoplasmic fraction. Phosphorylation at Ser-21 and Ser-115 creates a binding site for PIN1 at these Ser-Pro motifs, and promotes it's recruitment.

St John's Laboratory Ltd

**F** +44 (0)207 681 2580 **T** +44 (0)208 223 3081

W http://www.stjohnslabs.com/
E info@stjohnslabs.com