

Phospho-BTK (Y223) Antibody

Rabbit mAb

Catalog # AP90561

Product Information

Application	WB, IP
Primary Accession	Q06187
Reactivity	Rat, Human, Mouse
Clonality	Monoclonal
Other Names	Agammaglobulinaemia tyrosine kinase; AGMX1; ATK; B cell progenitor kinase; BPK; Bruton's tyrosine kinase; EC 2.7.10.2; kinase Btk; Kinase EMB;
Isotype	Rabbit IgG
Host	Rabbit
Calculated MW	76281

Additional Information

Dilution	WB 1:1000~1:2000 IP 1:20
Purification	Affinity-chromatography
Immunogen	A synthesized peptide derived from human Phospho-BTK (Y223)
Description	Defects in the Bruton tyrosine kinase (BTK) gene cause Agammaglobulinemia. Agammaglobulinemia is an X-linked immunodeficiency characterized by failure to produce mature B lymphocyte cells and associated with a failure of Ig heavy chain rearrangement.
Storage Condition and Buffer	Rabbit IgG in phosphate buffered saline , pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol. Store at +4°C short term. Store at -20°C long term. Avoid freeze / thaw cycle.

Protein Information

Name	BTK
Synonyms	AGMX1, ATK, BPK
Function	Non-receptor tyrosine kinase indispensable for B lymphocyte development, differentiation and signaling (PubMed: 19290921). Binding of antigen to the B-cell antigen receptor (BCR) triggers signaling that ultimately leads to B-cell activation (PubMed: 19290921). 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: 11606584). PLCG2 phosphorylation is performed in close cooperation with the adapter protein B-cell linker protein BLNK (PubMed: 11606584). BTK acts as a platform to bring together a diverse array of signaling proteins and is implicated in cytokine receptor signaling pathways (PubMed: 16517732 , PubMed: 17932028). 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:[16517732](#)). The TLR pathway acts as a primary surveillance system for the detection of pathogens and are crucial to the activation of host defense (PubMed:[16517732](#)). Especially, is a critical molecule in regulating TLR9 activation in splenic B-cells (PubMed:[16517732](#), PubMed:[17932028](#)). Within the TLR pathway, induces tyrosine phosphorylation of TIRAP which leads to TIRAP degradation (PubMed:[16415872](#)). BTK also plays a critical role in transcription regulation (PubMed:[19290921](#)). Induces the activity of NF-kappa-B, which is involved in regulating the expression of hundreds of genes (PubMed:[19290921](#)). BTK is involved on the signaling pathway linking TLR8 and TLR9 to NF-kappa-B (PubMed:[19290921](#)). Acts as an activator of NLRP3 inflammasome assembly by mediating phosphorylation of NLRP3 (PubMed:[34554188](#)). Transiently phosphorylates transcription factor GTF2I on tyrosine residues in response to BCR (PubMed:[9012831](#)). GTF2I then translocates to the nucleus to bind regulatory enhancer elements to modulate gene expression (PubMed:[9012831](#)). ARID3A and NFAT are other transcriptional target of BTK (PubMed:[16738337](#)). BTK is required for the formation of functional ARID3A DNA-binding complexes (PubMed:[16738337](#)). There is however no evidence that BTK itself binds directly to DNA (PubMed:[16738337](#)). BTK has a dual role in the regulation of apoptosis (PubMed:[9751072](#)). Plays a role in STING1- mediated induction of type I interferon (IFN) response by phosphorylating DDX41 (PubMed:[25704810](#)).

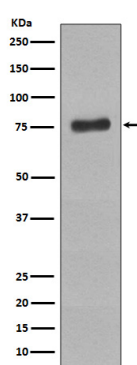
Cellular Location

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.

Images



Western blot analysis of Phospho-BTK (Y223) expression in Raji cell lysate treated with pervanadate.

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