

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;

IsotypeRabbit IgGHostRabbitCalculated MW76281

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:<u>19290921</u>). Binding of antigen to the B-cell antigen receptor (BCR) triggers signaling that ultimately leads to B-cell activation (PubMed:<u>19290921</u>). 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:<u>11606584</u>). PLCG2 phosphorylation is performed in close cooperation with the adapter protein B-cell linker protein BLNK (PubMed:<u>11606584</u>). BTK acts as a platform to bring together a diverse array of signaling proteins and is implicated in cytokine receptor signaling pathways (PubMed:<u>16517732</u>, PubMed:<u>17932028</u>). 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 NFkappa-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:<u>16738337</u>). 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).

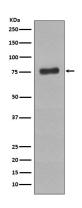
Cellular Location

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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