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BTK Antibody (Center)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7699c

Specification

BTK Antibody (Center) - Product Information

Application	**IF, WB, IHC-P,E**
Primary Accession	Q06187
Reactivity	**Human**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Calculated MW	**76281**
Antigen Region	**209-239**

BTK Antibody (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
This BTK antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 209-239 amino acids from the Central region of human BTK.

Dilution
IF~~1:10~50
WB~~1:1000
IHC-P~~1:50~100

Format
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage
Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
BTK Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

BTK Antibody (Center) - 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).

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.

BTK Antibody (Center) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

BTK Antibody (Center) - Images

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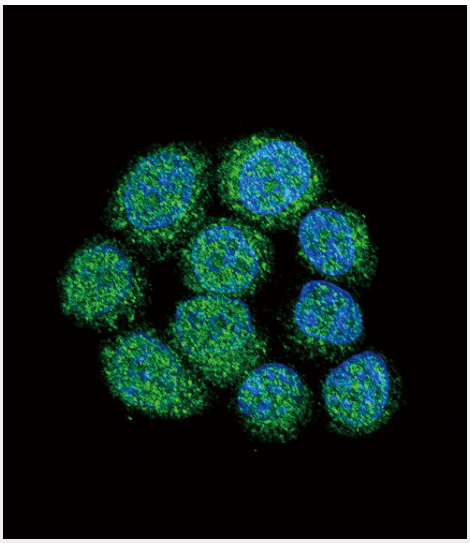
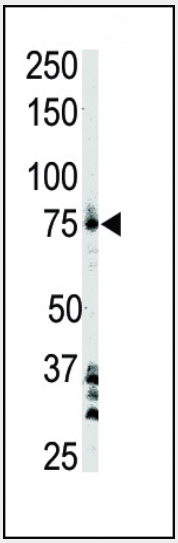
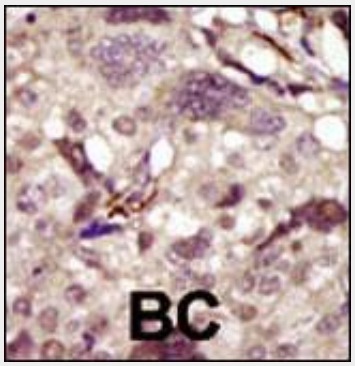


Confocal immunofluorescent analysis of BTK Antibody (Center)(Cat#AP7699c) with 293 cell followed by Alexa Fluor 488-conjugated goat anti-rabbit lgG (green).DAPI was used to stain the cell nuclear (blue).



The anti-BTK Pab (Cat. #AP7699c) is used in Western blot to detect BTK in Ramos cell lysate.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

BTK Antibody (Center) - 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) - References

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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).