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BTK Blocking Peptide (Center)
Synthetic peptide
Catalog # BP20992a

Specification

BTK Blocking Peptide (Center) - Product Information

Primary Accession	Q06187
Other Accession	P35991, Q8JH64

BTK Blocking Peptide (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
The synthetic peptide sequence is selected from aa 396-410 of HUMAN BTK

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 Blocking Peptide (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:<a href="http://www.uniprot.org/citations/11606584"

target="_blank">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 Blocking Peptide (Center) - Protocols

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

- Blocking Peptides

BTK Blocking Peptide (Center) - Images

BTK Blocking Peptide (Center) - Background

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.

BTK Blocking Peptide (Center) - References

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Hagemann T.L.,et al.Hum. Mol. Genet. 3:1743-1749(1994).