

BLNK Antibody (Ascites)
Mouse Monoclonal Antibody (Mab)
Catalog # AM2071a**Specification**

BLNK Antibody (Ascites) - Product Information

Application	WB,E
Primary Accession	Q8WV28
Other Accession	NP_001107566.1
Reactivity	Human
Host	Mouse
Clonality	Monoclonal
Isotype	IgM
Calculated MW	50466
Antigen Region	150-178

BLNK Antibody (Ascites) - Additional Information**Gene ID** 29760**Other Names**

B-cell linker protein, B-cell adapter containing a SH2 domain protein, B-cell adapter containing a Src homology 2 domain protein, Cytoplasmic adapter protein, Src homology 2 domain-containing leukocyte protein of 65 kDa, SLP-65, BLNK, BASH, SLP65

Target/Specificity

This BLNK antibody is generated from mice immunized with a KLH conjugated synthetic peptide between 150-178 amino acids from human BLNK.

Dilution

WB~~1:500~8000

E~~Use at an assay dependent concentration.

Format

Mouse monoclonal antibody supplied in crude ascites with 0.09% (W/V) sodium azide.

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

BLNK Antibody (Ascites) is for research use only and not for use in diagnostic or therapeutic procedures.

BLNK Antibody (Ascites) - Protein Information**Name** BLNK

Synonyms BASH, SLP65

Function Functions as a central linker protein, downstream of the B- cell receptor (BCR), bridging the SYK kinase to a multitude of signaling pathways and regulating biological outcomes of B-cell function and development. Plays a role in the activation of ERK/EPHB2, MAP kinase p38 and JNK. Modulates AP1 activation. Important for the activation of NF-kappa-B and NFAT. Plays an important role in BCR- mediated PLCG1 and PLCG2 activation and Ca(2+) mobilization and is required for trafficking of the BCR to late endosomes. However, does not seem to be required for pre-BCR-mediated activation of MAP kinase and phosphatidyl-inositol 3 (PI3) kinase signaling. May be required for the RAC1-JNK pathway. Plays a critical role in orchestrating the pro-B cell to pre-B cell transition. May play an important role in BCR- induced B-cell apoptosis.

Cellular Location

Cytoplasm. Cell membrane. Note=BCR activation results in the translocation to membrane fraction

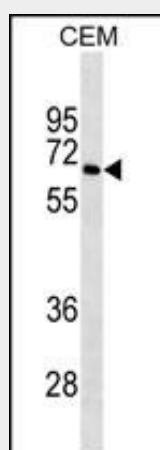
Tissue Location

Expressed in B-cell lineage and fibroblast cell lines (at protein level). Highest levels of expression in the spleen, with lower levels in the liver, kidney, pancreas, small intestines and colon

BLNK Antibody (Ascites) - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

BLNK Antibody (Ascites) - Images

BLNK Antibody (Cat. #AM2071a) western blot analysis in CEM cell line lysates (35µg/lane). This demonstrates the BLNK antibody detected the BLNK protein (arrow).

BLNK Antibody (Ascites) - Background

This gene encodes a cytoplasmic linker or adaptor protein

that plays a critical role in B cell development. This protein bridges B cell receptor-associated kinase activation with downstream signaling pathways, thereby affecting various biological functions. The phosphorylation of five tyrosine residues is necessary for this protein to nucleate distinct signaling effectors following B cell receptor activation. Mutations in this gene cause hypoglobulinemia and absent B cells, a disease in which the pro- to pre-B-cell transition is developmentally blocked. Deficiency in this protein has also been shown in some cases of pre-B acute lymphoblastic leukemia. Alternatively spliced transcript variants encoding different isoforms have been found for this gene.

BLNK Antibody (Ascites) - References

Rose, J.E., et al. Mol. Med. 16 (7-8), 247-253 (2010) :
Davila, S., et al. Genes Immun. 11(3):232-238(2010)
Oellerich, T., et al. Mol. Cell Proteomics 8(7):1738-1750(2009)
Imamura, Y., et al. J. Biol. Chem. 284(15):9804-9813(2009)
Li, H., et al. PLoS ONE 4 (7), E6410 (2009) :