

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

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**BLNK Antibody (Ascites) - Product Information**

Application	WB,E
Primary Accession	<a href="#">O8WV28</a>
Other Accession	<a href="#">NP_001107566.1</a>
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

**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**

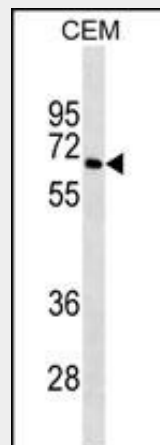
Expressed in B-cell lineage and fibroblast cell lines (at protein level). Highest levels of expression in the spleen, with lower levels in the liver, kidney, pancreas, small intestines and colon

### **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) :