

## SH3BP1 Antibody (Center)

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP16942c

## **Specification**

#### SH3BP1 Antibody (Center) - Product Information

Application WB,E
Primary Accession Q9Y3L3

Other Accession <u>P55194</u>, <u>NP\_061830.3</u>, <u>D3ZFJ3</u>, <u>Q6ZT62</u>

Reactivity
Predicted
Host
Clonality
Isotype
Calculated MW
Antigen Region

Human
Mouse, Rat
Rabbit
Polyclonal
Rabbit IgG
75713
419-446

# SH3BP1 Antibody (Center) - Additional Information

#### **Gene ID 23616**

#### **Other Names**

SH3 domain-binding protein 1, 3BP-1, SH3BP1

### Target/Specificity

This SH3BP1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 419-446 amino acids from the Central region of human SH3BP1.

## **Dilution**

WB~~1:1000

#### **Format**

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

#### **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**

SH3BP1 Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

## SH3BP1 Antibody (Center) - Protein Information

## Name SH3BP1 (HGNC:10824)

Function GTPase activating protein (GAP) which specifically converts GTP-bound Rho-type



GTPases including RAC1 and CDC42 in their inactive GDP-bound form. By specifically inactivating RAC1 at the leading edge of migrating cells, it regulates the spatiotemporal organization of cell protrusions which is important for proper cell migration (PubMed:21658605). Also negatively regulates CDC42 in the process of actin remodeling and the formation of epithelial cell junctions (PubMed:22891260). Through its GAP activity toward RAC1 and/or CDC42 plays a specific role in phagocytosis of large particles. Specifically recruited by a PI3 kinase/PI3K-dependent mechanism to sites of large particles engagement, inactivates RAC1 and/or CDC42 allowing the reorganization of the underlying actin cytoskeleton required for engulfment (PubMed:26465210). It also plays a role in angiogenesis and the process of repulsive guidance as part of a semaphorin-plexin signaling pathway. Following the binding of PLXND1 to extracellular SEMA3E it dissociates from PLXND1 and inactivates RAC1, inducing the intracellular reorganization of the actin cytoskeleton and the collapse of cells (PubMed:24841563).

#### **Cellular Location**

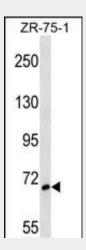
Cell projection. Cell junction, tight junction. Cell junction, adherens junction. Cell projection, phagocytic cup. Nucleus Cytoplasm, cytosol. Note=Localizes at the leading edge of migrating cells (PubMed:21658605, PubMed:24841563) Accumulation at forming phagocytic cups is PI3 kinase/PI3K-dependent and is specific for sites of large particles engagement and their phosphatidylinositol 3,4,5-triphosphate membrane content (PubMed:26465210).

#### SH3BP1 Antibody (Center) - Protocols

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

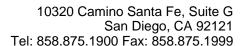
- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cvtometv
- Cell Culture

# SH3BP1 Antibody (Center) - Images



SH3BP1 Antibody (Center) (Cat. #AP16942c) western blot analysis in ZR-75-1 cell line lysates (35ug/lane). This demonstrates the SH3BP1 antibody detected the SH3BP1 protein (arrow).

## SH3BP1 Antibody (Center) - Background





SH3BP1 binds differentially to the SH3 domains of certain proteins of signal transduction pathways. This protein binds preferentially to the ABL1 proto-oncogene, SRC and GRB2. Shows GAP activity for Rac-related proteins but not for Rho-or Ras-related proteins. It inhibits PDGF-induced membrane ruffling mediated by Rac (By similarity).

# SH3BP1 Antibody (Center) - References

Collins, J.E., et al. Genome Biol. 5 (10), R84 (2004): Scott, M.P., et al. J. Biol. Chem. 277(31):28238-28246(2002) Dunham, I., et al. Nature 402(6761):489-495(1999)