

### MAP3K7IP1 Antibody

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP6861c

# **Specification**

### MAP3K7IP1 Antibody - Product Information

**Application** WB,E **Primary Accession** 015750 Other Accession **Q8CF89** Reactivity Human Predicted Mouse Host **Rabbit** Clonality **Polyclonal** Isotype Rabbit IgG Calculated MW 54644 Antigen Region 416-445

# MAP3K7IP1 Antibody - Additional Information

#### **Gene ID** 10454

### **Other Names**

TGF-beta-activated kinase 1 and MAP3K7-binding protein 1, Mitogen-activated protein kinase kinase 7-interacting protein 1, TGF-beta-activated kinase 1-binding protein 1, TAK1-binding protein 1, TAB1, MAP3K7IP1

### Target/Specificity

This MAP3K7IP1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 416-445 amino acids from human MAP3K7IP1.

### **Dilution**

WB~~1:1000

E~~Use at an assay dependent concentration.

#### **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**

MAP3K7IP1 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

#### MAP3K7IP1 Antibody - Protein Information



#### Name TAB1

#### Synonyms MAP3K7IP1

**Function** Key adapter protein that plays an essential role in JNK and NF-kappa-B activation and proinflammatory cytokines production in response to stimulation with TLRs and cytokines (PubMed:22307082, PubMed:24403530). Mechanistically, associates with the catalytic domain of MAP3K7/TAK1 to trigger MAP3K7/TAK1 autophosphorylation leading to its full activation (PubMed:10838074, PubMed:25260751, PubMed:37832545). Similarly, associates with MAPK14 and triggers its autophosphorylation and subsequent activation (PubMed:11847341, PubMed:29229647). In turn, MAPK14 phosphorylates TAB1 and inhibits MAP3K7/TAK1 activation in a feedback control mechanism (PubMed:14592977). Also plays a role in recruiting MAPK14 to the TAK1 complex for the phosphorylation of the TAB2 and TAB3 regulatory subunits (PubMed:18021073).

#### **Cellular Location**

Cytoplasm, cytosol. Endoplasmic reticulum membrane; Peripheral membrane protein; Cytoplasmic side. Note=Recruited to the endoplasmic reticulum following interaction with STING1

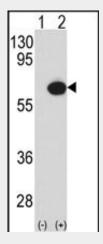
Tissue Location Ubiquitous..

## MAP3K7IP1 Antibody - 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

#### MAP3K7IP1 Antibody - Images



Western blot analysis of MAP3K7IP1 (arrow) using rabbit polyclonal MAP3K7IP1-pS438 (Cat. #AP6861c). 293 cell lysates (2 ug/lane) either nontransfected (Lane 1) or transiently transfected with the MAP3K7IP1 gene (Lane 2) (Origene Technologies).



# MAP3K7IP1 Antibody - Background

MAP3K7IP1 was identified as a regulator of the MAP kinase kinase kinase MAP3K7/TAK1, which is known to mediate various intracellular signaling pathways, such as those induced by TGF beta, interleukin 1, and WNT-1. This protein interacts and thus activates TAK1 kinase. It has been shown that the C-terminal portion of this protein is sufficient for binding and activation of TAK1, while a portion of the N-terminus acts as a dominant-negative inhibitor of TGF beta, suggesting that this protein may function as a mediator between TGF beta receptors and TAK1. This protein can also interact with and activate the mitogen-activated protein kinase 14 (MAPK14/p38alpha), and thus represents an alternative activation pathway, in addition to the MAPKK pathways, which contributes to the biological responses of MAPK14 to various stimuli.

# MAP3K7IP1 Antibody - References

Arch,R.H., et.al., Genes Dev. 12 (18), 2821-2830 (1998) Yamaguchi,K., et.al., EMBO J. 18 (1), 179-187 (1999)