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TAX1BP1 Antibody (Center)

Affinity Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP16130C

Specification

TAX1BP1 Antibody (Center) - Product Information

Application	**WB,E**
Primary Accession	Q86VP1
Other Accession	NP_006015.4, NP_001073333.1
Reactivity	**Human**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Calculated MW	**90877**
Antigen Region	**400-429**

TAX1BP1 Antibody (Center) - Additional Information

Gene ID 8887

Other Names
Tax1-binding protein 1, TRAF6-binding protein, TAX1BP1, T6BP

Target/Specificity
This TAX1BP1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 400-429 amino acids from the Central region of human TAX1BP1.

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
TAX1BP1 Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

TAX1BP1 Antibody (Center) - Protein Information

Name TAX1BP1

Synonyms T6BP

Function Ubiquitin-binding adapter that participates in inflammatory, antiviral and innate immune processes as well as selective autophagy regulation (PubMed:30459273, PubMed:29940186, PubMed:30909570). Plays a key role in the negative regulation of NF-kappa-B and IRF3 signalings by acting as an adapter for the ubiquitin-editing enzyme A20/TNFAIP3 to bind and inactivate its substrates (PubMed:17703191). Disrupts the interactions between the E3 ubiquitin ligase TRAF3 and TBK1/IKBKE to attenuate 'Lys63'-linked polyubiquitination of TBK1 and thereby IFN- beta production (PubMed:21885437). Recruits also A20/TNFAIP3 to ubiquitinated signaling proteins TRAF6 and RIPK1, leading to their deubiquitination and disruption of IL-1 and TNF-induced NF-kappa-B signaling pathways (PubMed:17703191). Inhibits virus-induced apoptosis by inducing the 'Lys-48'-linked polyubiquitination and degradation of MAVS via recruitment of the E3 ligase ITCH, thereby attenuating MAVS- mediated apoptosis signaling (PubMed:27736772). As a macroautophagy/autophagy receptor, facilitates the xenophagic clearance of pathogenic bacteria such as Salmonella typhimurium and Mycobacterium tuberculosis (PubMed:26451915). Upon NBR1 recruitment to the SQSTM1- ubiquitin condensates, acts as the major recruiter of RB1CC1 to these ubiquitin condensates to promote their autophagic degradation (PubMed:33226137, PubMed:34471133). Mediates the autophagic degradation of other substrates including TICAM1 (PubMed:28898289).

Cellular Location
Cytoplasm. Mitochondrion. Preautophagosomal structure Cytoplasmic vesicle, autophagosome

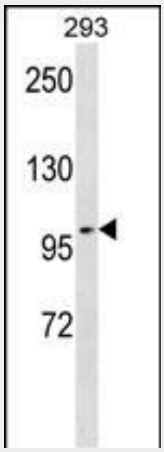
Tissue Location
Expressed in all tissues tested.

TAX1BP1 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 Cytomety
- Cell Culture

TAX1BP1 Antibody (Center) - Images



TAX1BP1 Antibody (Center) (Cat. #AP16130c) western blot analysis in 293 cell line lysates

(35ug/lane).This demonstrates the TAX1BP1 antibody detected the TAX1BP1 protein (arrow).

TAX1BP1 Antibody (Center) - Background

The HTLV-1 Tax protein transcriptionally activates the HTLV-1 promoter. Tax also binds to and stimulates the expression of cellular genes, including transcription factors and other proteins (Gachon et al., 1998 [PubMed 9733879]).

TAX1BP1 Antibody (Center) - References

Parvatiyar, K., et al. J. Biol. Chem. 285(20):14999-15009(2010)
Ruiz, M.T., et al. Braz J Otorhinolaryngol 76(2):193-198(2010)
Shembade, N., et al. Science 327(5969):1135-1139(2010)
Dieguez-Gonzalez, R., et al. Ann. Rheum. Dis. 68(4):579-583(2009)
Shembade, N., et al. EMBO J. 28(5):513-522(2009)

TAX1BP1 Antibody (Center) - Citations

- Elevated p62/SQSTM1 determines the fate of autophagy-deficient neural stem cells by increasing superoxide.