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# TAX1BP1 Antibody (Center)

Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP16130C

## Product Information

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|---|---|
| **Application** | WB, E |
| **Primary Accession** | Q86VP1 |
| **Other Accession** | NP_006015.4, NP_001073333.1 |
| **Reactivity** | Human |
| **Predicted** | Mouse, Canine, Rabbit |
| **Host** | Rabbit |
| **Clonality** | Polyclonal |
| **Isotype** | Rabbit IgG |
| **Clone Names** | RB35312 |
| **Calculated MW** | 90877 |
| **Antigen Region** | 400-429 |

## Additional Information

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

## Protein Information

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| **Name** | TAX1BP1 |
| **Synonyms** | T6BP |
| **Function** | Ubiquitin-binding adapter that participates in inflammatory, antiviral and |

innate immune processes as well as selective autophagy regulation (PubMed:29940186, PubMed:30459273, 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). Also recruits 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.

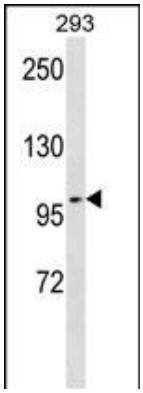
## Background

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]).

## 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)

## 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).

## Citations

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

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.