

TRIM32 Antibody (N-term) Blocking peptide
Synthetic peptide
Catalog # BP13287a**Specification**

TRIM32 Antibody (N-term) Blocking peptide - Product InformationPrimary Accession [Q13049](#)**TRIM32 Antibody (N-term) Blocking peptide - Additional Information****Gene ID** 22954**Other Names**

E3 ubiquitin-protein ligase TRIM32, 632-, 72 kDa Tat-interacting protein, Tripartite motif-containing protein 32, Zinc finger protein HT2A, TRIM32, HT2A

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP13287a was selected from the N-term region of TRIM32. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

TRIM32 Antibody (N-term) Blocking peptide - Protein Information**Name** TRIM32**Synonyms** HT2A**Function**

E3 ubiquitin ligase that plays a role in various biological processes including neural stem cell differentiation, innate immunity, inflammatory response and autophagy (PubMed:19349376, PubMed:31123703). Plays a role in virus-triggered induction of IFN-beta and TNF-alpha by mediating the ubiquitination of STING1. Mechanistically, targets STING1 for 'Lys-63'-linked ubiquitination which promotes the interaction of STING1 with TBK1 (PubMed:22745133). Regulates bacterial clearance and promotes autophagy in Mycobacterium tuberculosis-infected macrophages (PubMed:37543647). Negatively

regulates TLR3/4-mediated innate immune and inflammatory response by triggering the autophagic degradation of TICAM1 in an E3 activity-independent manner (PubMed:28898289). Plays an essential role in oxidative stress induced cell death by inducing loss of transmembrane potential and enhancing mitochondrial reactive oxygen species (ROS) production during oxidative stress conditions (PubMed:32918979). Ubiquitinates XIAP and targets it for proteasomal degradation (PubMed:21628460). Ubiquitinates DTNBP1 (dysbindin) and promotes its degradation (PubMed:19349376). May ubiquitinate BBS2 (PubMed:22500027). Ubiquitinates PIAS4/PIASY and promotes its degradation in keratinocytes treated with UVB and TNF-alpha (By similarity). Also acts as a regulator of autophagy by mediating formation of unanchored 'Lys-63'-linked polyubiquitin chains that activate ULK1: interaction with AMBRA1 is required for ULK1 activation (PubMed:31123703). Positively regulates dendritic branching by promoting ubiquitination and subsequent degradation of the epigenetic factor CDYL (PubMed:34888944).

Cellular Location

Cytoplasm. Mitochondrion. Endoplasmic reticulum. Note=Localized in cytoplasmic bodies, often located around the nucleus

Tissue Location

Spleen, thymus, prostate, testis, ovary, intestine, colon and skeletal muscle.

TRIM32 Antibody (N-term) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

TRIM32 Antibody (N-term) Blocking peptide - Images

TRIM32 Antibody (N-term) Blocking peptide - Background

The protein encoded by this gene is a member of the tripartite motif (TRIM) family. The TRIM motif includes three zinc-binding domains, a RING, a B-box type 1 and a B-box type 2, and a coiled-coil region. The protein localizes to cytoplasmic bodies. The protein has also been localized to the nucleus, where it interacts with the activation domain of the HIV-1 Tat protein. The Tat protein activates transcription of HIV-1 genes. [provided by RefSeq].

TRIM32 Antibody (N-term) Blocking peptide - References

Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010) Liu, Y., et al. J. Invest. Dermatol. 130(5):1384-1390(2010) Talmud, P.J., et al. Am. J. Hum. Genet. 85(5):628-642(2009) Markson, G., et al. Genome Res. 19(10):1905-1911(2009) van Wijk, S.J., et al. Mol. Syst. Biol. 5, 295 (2009) :