

TRRAP Antibody (C-term) Blocking Peptide

Synthetic peptide Catalog # BP8056b

Specification

TRRAP Antibody (C-term) Blocking Peptide - Product Information

Primary Accession Other Accession <u>Q9Y4A5</u> <u>Q9Y6H4</u>

TRRAP Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 8295

Other Names Transformation/transcription domain-associated protein, 350/400 kDa PCAF-associated factor, PAF350/400, STAF40, Tra1 homolog, TRRAP, PAF400

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP8056b was selected from the C-term region of human TRRAP . 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.

TRRAP Antibody (C-term) Blocking Peptide - Protein Information

Name TRRAP

Synonyms PAF400

Function

Adapter protein, which is found in various multiprotein chromatin complexes with histone acetyltransferase activity (HAT), which gives a specific tag for epigenetic transcription activation. Component of the NuA4 histone acetyltransferase complex which is responsible for acetylation of nucleosomal histones H4 and H2A. Plays a central role in MYC transcription activation, and also participates in cell transformation by MYC. Required for p53/TP53-, E2F1- and E2F4- mediated transcription activation. Also involved in transcription activation mediated by the adenovirus E1A, a viral oncoprotein that deregulates transcription of key genes. Probably acts by linking transcription factors such as E1A, MYC or E2F1 to HAT complexes such as STAGA thereby allowing



transcription activation. Probably not required in the steps following histone acetylation in processes of transcription activation. May be required for the mitotic checkpoint and normal cell cycle progression. Component of a SWR1-like complex that specifically mediates the removal of histone H2A.Z/H2AZ1 from the nucleosome. May play a role in the formation and maintenance of the auditory system (By similarity).

Cellular Location Nucleus

TRRAP Antibody (C-term) Blocking Peptide - Protocols

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

Blocking Peptides

TRRAP Antibody (C-term) Blocking Peptide - Images

TRRAP Antibody (C-term) Blocking Peptide - Background

TRRAP, a member of the TRA1 subfamily of the PI3/PI4 kinase family, is an adapter protein found in various multiprotein chromatin complexes with histone acetyltransferase activity (HAT), which gives a specific tag for epigenetic transcription activation. It plays a central role in MYC (c-Myc) transcription activation, and also participates in cell transformation by MYC. TRRAP is required for p53/TP53-, E2F1- and E2F4-mediated transcription activation, and is also involved in transcription activation mediated by the adenovirus E1A, a viral oncoprotein that deregulates transcription of key genes. It likely performs by linking transcription factors such as E1A, MYC or E2F1 to HAT complexes such as STAGA thereby allowing transcription activation. Although probably not required in the steps following histone acetylation in processes of transcription activation, it may be required for the mitotic checkpoint and normal cell cycle progression. It is a component of various HAT complexes, including the PCAF, TFTC, TIP60 STAGA and BAF53 complexes. The PI3K/PI4K domain of this nuclear protein is required for the recruitment of HAT complexes, and the MYC-dependent transactivation. Although it is strongly related to the PI3/PI4-kinase family, it lacks the typical motifs that constitute the catalytic site of PI3/PI4-kinase proteins, and lacks such activity.

TRRAP Antibody (C-term) Blocking Peptide - References

Liu, X., et al., J. Biol. Chem. 278(22):20405-20412 (2003).Lang, S.E., et al., Oncogene 22(18):2836-2841 (2003).Hillier, L.W., et al., Nature 424(6945):157-164 (2003).Ard, P.G., et al., Mol. Cell. Biol. 22(16):5650-5661 (2002).Park, J., et al., Mol. Cell. Biol. 22(5):1307-1316 (2002).