

ANP32A Antibody (Center) Blocking peptide
Synthetic peptide
Catalog # BP13728c**Specification**

ANP32A Antibody (Center) Blocking peptide - Product InformationPrimary Accession [P39687](#)**ANP32A Antibody (Center) Blocking peptide - Additional Information****Gene ID** 8125**Other Names**

Acidic leucine-rich nuclear phosphoprotein 32 family member A, Acidic nuclear phosphoprotein pp32, pp32, Leucine-rich acidic nuclear protein, LANP, Mapmodulin, Potent heat-stable protein phosphatase 2A inhibitor I1PP2A, Putative HLA-DR-associated protein I, PHAPI, ANP32A, C15orf1, LANP, MAPM, PHAP1

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP13728c was selected from the Center region of ANP32A. 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.

ANP32A Antibody (Center) Blocking peptide - Protein Information**Name** ANP32A**Synonyms** C15orf1, LANP, MAPM, PHAP1**Function**

Multifunctional protein that is involved in the regulation of many processes including tumor suppression, apoptosis, cell cycle progression or transcription (PubMed:16341127, PubMed:11360199, PubMed:18439902, PubMed:10400610). Promotes apoptosis by favouring the activation of caspase-9/CASP9 and allowing apoptosome formation (PubMed:18439902).

In addition, plays a role in the modulation of histone acetylation and transcription as part of the INHAT (inhibitor of histone acetyltransferases) complex. Inhibits the histone- acetyltransferase activity of EP300/CREBBP (CREB-binding protein) and EP300/CREBBP-associated factor by histone masking (PubMed:11830591). Preferentially binds to unmodified histone H3 and sterically inhibiting its acetylation and phosphorylation leading to cell growth inhibition (PubMed:16341127). Participates in other biochemical processes such as regulation of mRNA nuclear-to-cytoplasmic translocation and stability by its association with ELAVL1 (Hu-antigen R) (PubMed:18180367). Plays a role in E4F1-mediated transcriptional repression as well as inhibition of protein phosphatase 2A (PubMed:15642345, PubMed:17557114).

Cellular Location

Nucleus. Cytoplasm Endoplasmic reticulum. Note=Translocates to the cytoplasm during the process of neuritogenesis (By similarity). Shuttles between nucleus and cytoplasm. {ECO:0000250, ECO:0000269|PubMed:18180367}

Tissue Location

Expressed in all tissues tested. Highly expressed in kidney and skeletal muscle, moderate levels of expression in brain, placenta and pancreas, and weakly expressed in lung. Found in all regions of the brain examined (amygdala, caudate nucleus, corpus callosum, hippocampus and thalamus), with highest levels in amygdala

ANP32A Antibody (Center) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

ANP32A Antibody (Center) Blocking peptide - Images

ANP32A Antibody (Center) Blocking peptide - Background

Implicated in a number of cellular processes, including proliferation, differentiation, caspase-dependent and caspase-independent apoptosis, suppression of transformation (tumor suppressor), inhibition of protein phosphatase 2A, regulation of mRNA trafficking and stability in association with ELAVL1, and inhibition of acetyltransferases as part of the INHAT (inhibitor of histone acetyltransferases) complex. Plays a role in E4F1-mediated transcriptional repression.

ANP32A Antibody (Center) Blocking peptide - References

Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010)Habrukowich, C., et al. J. Biol. Chem. 285(35):26825-26831(2010)Talmud, P.J., et al. Am. J. Hum. Genet. 85(5):628-642(2009)Valdes, A.M., et al. Arthritis Rheum. 60(7):2046-2054(2009)Matilla, A., et al. Cerebellum 4(1):7-18(2005)