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CKIP-1 Antibody (N-term) Blocking Peptide

Synthetic peptide
Catalog # BP8500a

Specification

CKIP-1 Antibody (N-term) Blocking Peptide - Product Information

Primary Accession Q53GL0

CKIP-1 Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 51177

Other Names
Pleckstrin homology domain-containing family O member 1, PH domain-containing family O member 1, C-Jun-binding protein, JBP, Casein kinase 2-interacting protein 1, CK2-interacting protein 1, CKIP-1, Osteoclast maturation-associated gene 120 protein, PLEKHO1, CKIP1, OC120

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP8500a was selected from the N-term region of human CKIP-1. 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.

CKIP-1 Antibody (N-term) Blocking Peptide - Protein Information

Name PLEKHO1

Synonyms CKIP1, OC120

Function
Plays a role in the regulation of the actin cytoskeleton through its interactions with actin capping protein (CP). May function to target CK2 to the plasma membrane thereby serving as an adapter to facilitate the phosphorylation of CP by protein kinase 2 (CK2). Appears to target ATM to the plasma membrane. Appears to also inhibit tumor cell growth by inhibiting AKT-mediated cell-survival. Also implicated in PI3K-regulated muscle differentiation, the regulation of AP-1 activity (plasma membrane bound AP-1 regulator that translocates to the nucleus) and the promotion of apoptosis induced by tumor necrosis factor TNF. When bound to PKB, it inhibits it probably by decreasing PKB level of phosphorylation.

Cellular Location
Cell membrane; Peripheral membrane protein. Nucleus. Cytoplasm Note=Predominantly localized to the plasma membrane through the binding to phosphatidylinositol 3-phosphate (PubMed:14729969). In C2C12 cells, with the absence of growth factor, it is found in the nucleus (PubMed:14729969). It rapidly translocates to the plasma membrane after insulin stimulation (PubMed:14729969). In response to TNF, it translocates from the plasma membrane to the cytoplasm and then to the nucleus accompanied by cleavage by caspase-3 (PubMed:15706351) However, the subcellular location is highly dependent of the cell type, and this explains why it is found exclusively at the plasma membrane, in some type of cells (Probable). {ECO:0000269|PubMed:14729969, ECO:0000269|PubMed:15706351, ECO:0000305}

Tissue Location
Abundantly expressed in skeletal muscle and heart, moderately in kidney, liver, brain and placenta and sparingly in the pancreas and lung. Easily detectable in cell lines such as MOLT-4, HEK293 and Jurkat.

CKIP-1 Antibody (N-term) Blocking Peptide - Protocols

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

- Blocking Peptides

CKIP-1 Antibody (N-term) Blocking Peptide - Images

CKIP-1 Antibody (N-term) Blocking Peptide - Background

CKIP-1 plays a role in the regulation of the actin cytoskeleton through its interactions with actin capping protein (CP). This protein may function to target CK2 to the plasma membrane thereby serving as an adapter to facilitate the phosphorylation of CP by protein kinase 2 (CK2). It appears to target ATM to the plasma membrane and appears to also inhibit tumor cell growth by inhibiting AKT-mediated cell-survival. Also implicated in PI3K-regulated muscle differentiation, the regulation of AP-1 activity (plasma membrane bound AP-1 regulator that translocates to the nucleus) and the promotion of apoptosis induced by tumor necrosis factor TNF. When bound to PKB, it inhibits it probably by decreasing PKB level of phosphorylation.

CKIP-1 Antibody (N-term) Blocking Peptide - References

Barrios-Rodiles,M., et.al., Science 307 (5715), 1621-1625 (2005)