10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

AP2B1 Antibody (Center) Blocking Peptide

Synthetic peptide
Catalog # BP14961c

Specification

AP2B1 Antibody (Center) Blocking Peptide - Product Information

Primary Accession P63010

AP2B1 Antibody (Center) Blocking Peptide - Additional Information

Gene ID 163

Other Names
AP-2 complex subunit beta, AP105B, Adaptor protein complex AP-2 subunit beta, Adaptor-related protein complex 2 subunit beta, Beta-2-adaptin, Beta-adaptin, Clathrin assembly protein complex 2 beta large chain, Plasma membrane adaptor HA2/AP2 adaptin beta subunit, AP2B1, ADTB2, CLAPB1

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.

AP2B1 Antibody (Center) Blocking Peptide - Protein Information

Name AP2B1

Synonyms ADTB2, CLAPB1

Function
Component of the adaptor protein complex 2 (AP-2). Adaptor protein complexes function in protein transport via transport vesicles in different membrane traffic pathways. Adaptor protein complexes are vesicle coat components and appear to be involved in cargo selection and vesicle formation. AP-2 is involved in clathrin-dependent endocytosis in which cargo proteins are incorporated into vesicles surrounded by clathrin (clathrin-coated vesicles, CCVs) which are destined for fusion with the early endosome. The clathrin lattice serves as a mechanical scaffold but is itself unable to bind directly to membrane components. Clathrin-associated adaptor protein (AP) complexes which can bind directly to both the clathrin lattice and to the lipid and protein components of membranes are considered to be the major clathrin adaptors contributing the CCV formation. AP-2 also serves as a cargo receptor to selectively sort the membrane proteins involved in receptor-mediated endocytosis. AP-2 seems to play a role in the recycling of synaptic vesicle membranes from the presynaptic surface. AP-2 recognizes Y-X-X-[FILMV] (Y-X-X-Phi) and [ED]-X-X-X-L- [LI] endocytosis signal motifs within the cytosolic tails of transmembrane cargo

molecules. AP-2 may also play a role in maintaining normal post-endocytic trafficking through the ARF6-regulated, non- clathrin pathway. During long-term potentiation in hippocampal neurons, AP-2 is responsible for the endocytosis of ADAM10 (PubMed:23676497). The AP-2 beta subunit acts via its C-terminal appendage domain as a scaffolding platform for endocytic accessory proteins; at least some clathrin-associated sorting proteins (CLASPs) are recognized by their [DE]-X(1,2)-F-X-X-[FL]-X-X-X-R motif. The AP-2 beta subunit binds to clathrin heavy chain, promoting clathrin lattice assembly; clathrin displaces at least some CLASPs from AP2B1 which probably then can be positioned for further coat assembly.

Cellular Location
Cell membrane. Membrane, coated pit; Peripheral membrane protein; Cytoplasmic side. Note=AP-2 appears to be excluded from internalizing CCVs and to disengage from sites of endocytosis seconds before internalization of the nascent CCV

Tissue Location
Expressed in the brain (at protein level).

AP2B1 Antibody (Center) Blocking Peptide - Protocols

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

- Blocking Peptides

AP2B1 Antibody (Center) Blocking Peptide - Images

AP2B1 Antibody (Center) Blocking Peptide - Background

The protein encoded by this gene is one of two large chaincomponents of the assembly protein complex 2, which serves to linkclathrin to receptors in coated vesicles. The encoded protein isfound on the cytoplasmic face of coated vesicles in the plasmamembrane. Two transcript variants encoding different isoforms havebeen found for this gene.

AP2B1 Antibody (Center) Blocking Peptide - References

Kahlfeldt, N., et al. J. Biol. Chem. 285(4):2734-2749(2010)Hood, F.E., et al. J. Cell. Sci. 122 (PT 13), 2185-2190 (2009) :Grass, B., et al. Histopathology 54(7):873-879(2009)Keyel, P.A., et al. Mol. Biol. Cell 19(12):5309-5326(2008)Rikova, K., et al. Cell 131(6):1190-1203(2007)