

CPEB3 Antibody (Center) Blocking Peptide
Synthetic peptide
Catalog # BP16448c**Specification**

CPEB3 Antibody (Center) Blocking Peptide - Product InformationPrimary Accession [Q8NE35](#)**CPEB3 Antibody (Center) Blocking Peptide - Additional Information****Gene ID** 22849**Other Names**

Cytoplasmic polyadenylation element-binding protein 3, CPE-BP3, CPE-binding protein 3, hCPEB-3, CPEB3, KIAA0940

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.

CPEB3 Antibody (Center) Blocking Peptide - Protein Information**Name** CPEB3**Synonyms** KIAA0940**Function**

Sequence-specific RNA-binding protein which acts as a translational repressor in the basal unstimulated state but, following neuronal stimulation, acts as a translational activator (By similarity). In contrast to CPEB1, does not bind to the cytoplasmic polyadenylation element (CPE), a uridine-rich sequence element within the mRNA 3'-UTR, but binds to a U-rich loop within a stem-loop structure (By similarity). Required for the consolidation and maintenance of hippocampal-based long term memory (By similarity). In the basal state, binds to the mRNA 3'-UTR of the glutamate receptors GRIA2/GLUR2 mRNA and negatively regulates their translation (By similarity). Also represses the translation of DLG4, GRIN1, GRIN2A and GRIN2B (By similarity). When activated, acts as a translational activator of GRIA1 and GRIA2 (By similarity). In the basal state, suppresses SUMO2 translation but activates it following neuronal stimulation (By similarity). Binds to the 3'-UTR of TRPV1 mRNA and represses TRPV1 translation which is required to maintain normal thermoception (By similarity). Binds actin mRNA, leading to actin translational repression in the basal state and to translational activation following neuronal stimulation (By similarity). Negatively regulates target mRNA levels by binding to TOB1 which recruits CNOT7/CAF1 to a ternary complex and this leads to target mRNA deadenylation and decay (PubMed:21336257). In addition to its role in translation, binds to and inhibits the transcriptional activation activity of STAT5B without affecting its dimerization or DNA-binding activity. This, in turn, represses transcription of the STAT5B target gene EGFR which has been shown to play a role in enhancing learning and memory performance (PubMed:20639532). In contrast to CPEB1, CPEB2 and CPEB4, not required for cell cycle progression (PubMed:26398195).

Cellular Location

Cytoplasm. Nucleus. Synapse {ECO:0000250|UniProtKB:Q7TN99} Cell projection, dendrite {ECO:0000250|UniProtKB:Q7TN99}. Postsynaptic density {ECO:0000250|UniProtKB:Q7TN99}. Note=Predominantly cytoplasmic in unstimulated neurons but translocates to the nucleus following neuronal stimulation (PubMed:20639532, PubMed:22730302). Nuclear import is mediated by importin IPO5 (By similarity) {ECO:0000250|UniProtKB:Q7TN99, ECO:0000269|PubMed:20639532, ECO:0000269|PubMed:22730302}

CPEB3 Antibody (Center) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

CPEB3 Antibody (Center) Blocking Peptide - Images

CPEB3 Antibody (Center) Blocking Peptide - Background

Cytoplasmic polyadenylation element-binding (CPEB) proteins control polyadenylation-induced translation in early development. Studies in oocytes led to the delineation of *Xenopus* CPEB, the first member of the family to be identified, and its mouse homologue mCPEB-1. Recently, a second mouse family member, mCPEB-2, has been described in germ cells. Increasing evidence also implicates CPEB proteins as being important in the hippocampus, where these proteins are thought to regulate local protein synthesis and synaptic plasticity.

CPEB3 Antibody (Center) Blocking Peptide - References

Rose, J.E., et al. Mol. Med. 16 (7-8), 247-253 (2010) :Chadalavada, D.M., et al. Biochemistry 49(25):5321-5330(2010)Vogler, C., et al. Front Behav Neurosci 3, 4 (2009) :Salehi-Ashtiani, K., et al. Science 313(5794):1788-1792(2006)Grupe, A., et al. Am. J. Hum. Genet. 78(1):78-88(2006)