abcam

# Product datasheet

# Recombinant Human 4E-BP2 protein ab172157

1 Image

## Description

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| **Product name** | Recombinant Human 4E-BP2 protein |
| **Purity** | > 95 % SDS-PAGE. |
| **Endotoxin level** | 1.000 Eu/µg |
| **Expression system** | Escherichia coli |
| **Accession** | Q13542 |
| **Protein length** | Full length protein |
| **Animal free** | No |
| **Nature** | Recombinant |
| **Species** | Human |
| **Sequence** | MSSSAGSGHQPSQSRAIPTRTVAISDAAQLPHDYCTTPGG TLFSTTPGGT RIIYDRKFLLDRRNSPMAQTPPCHLPNIPGVTSPGTLIEDSK VEVNNLNN LNNHDRKHAVGDDAQFEMDI |
| **Predicted molecular weight** | 12 kDa |
| **Amino acids** | 1 to 120 |
| **Tags** | His tag N-Terminus |

## Specifications

Our Abpromise guarantee covers the use of **ab172157** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

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| **Applications** | SDS-PAGE |
| **Form** | Lyophilized |

## Preparation and Storage

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| **Stability and Storage** | Shipped at 4°C. Store at -20°C or -80°C. Avoid freeze / thaw cycle.<br>pH: 8.00<br>Constituents: 0.88% Sodium chloride, 99% Tris-HCl buffer |

## General Info

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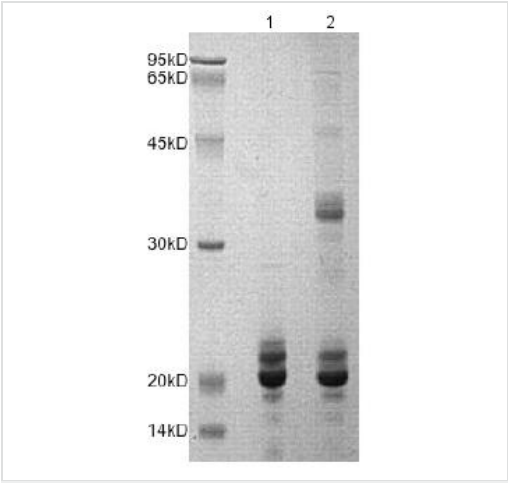
| **Function** | Repressor of translation initiation involved in synaptic plasticity, learning and memory formation (By similarity). Regulates EIF4E activity by preventing its assembly into the eIF4F complex: hypophosphorylated form of EIF4EBP2 competes with EIF4G1/EIF4G3 and strongly binds to EIF4E, leading to repress translation. In contrast, hyperphosphorylated form dissociates from EIF4E, allowing interaction between EIF4G1/EIF4G3 and EIF4E, leading to initiation of translation (PubMed:25533957). EIF4EBP2 is enriched in brain and acts as a regulator of synapse activity and neuronal stem cell renewal via its ability to repress translation initiation (By similarity). Mediates the regulation of protein translation by hormones, growth factors and other stimuli that signal through the MAP kinase and mTORC1 pathways. |
| **Sequence similarities** | Belongs to the eIF4E-binding protein family. |
| **Domain** | The TOS motif mediates interaction with RPTOR, leading to promote phosphorylation by mTORC1 complex.<br>Intrinsically disordered protein that undergoes folding upon phosphorylation (PubMed:25533957). Hypophosphorylated form interacts strongly with EIF4E using (1) the YXXXXLPhi motif, that undergoes a disorder-to-helix transition upon binding and (2) the secondary EIF4E binding sites (residues 78-82) (PubMed:24207126, PubMed:25533957). Phosphorylation at Thr-37 and Thr-46 induces folding of region encompassing residues from Pro-18 to Arg-62 of into a four-stranded beta-domain that sequesters the helical YXXXXLPhi motif into a buried beta-strand, blocking accessibility to EIF4E. Protein phosphorylated at Thr-37 and Thr-46 is however unstable and subsequent phosphorylation at Ser-65, Thr-70 and Ser-83 is required to stabilize the fold, decreasing affinity for EIF4E by a factor of 4000 (PubMed:24207126, PubMed:25533957). |
| **Post-translational modifications** | Phosphorylation at Thr-37, Thr-46, Ser-65, Thr-70 and Ser-83 is mediated by MTOR and corresponds to the hyperphosphorylated form: it abolishes binding to EIF4E by inducing folding of intrinsically disordered regions (PubMed:24207126, PubMed:25533957). First phosphorylated at Thr-37 and Thr-46 by MTOR, inducing folding of region encompassing residues from Pro-18 to Arg-62 of into a four-stranded beta-domain that sequesters the helical YXXXXLPhi motif into a partly buried beta-strand, blocking accessibility to EIF4E. Protein phosphorylated at Thr-37 and Thr-46 is however unstable and subsequent phosphorylation at Ser-65, Thr-70 and Ser-83 is required to stabilize the fold, decreasing affinity for EIF4E by a factor of 4000 (PubMed:24207126, PubMed:25533957). Phosphorylated in response to insulin, EGF and PDGF.<br>Deamidated at Asn-99 and Asn-102 to aspartate (Asp) in brain. Deamidation promotes interaction with RPTOR, subsequent phosphorylation by mTORC1 and increased translation, leading to impair kinetics of excitatory synaptic transmission. Deamidation takes place during postnatal development, when the PI3K-Akt-mTOR signaling is reduced, suggesting it acts as a compensatory mechanism to promote translation despite attenuated PI3K-Akt-mTOR signaling in neuron development. Deamidation converts Asn residues into a mixture of Asp and isoaspartate; interactions with PCMT1 is required to prevent isoaspartate accumulation and convert isoaspartate to Asp. |

## Images




SDS-PAGE - Recombinant Human 4E-BP2 protein (ab172157)

SDS-PAGE analysis of ab172157 in 1) Non reducing and 2) Reducing conditions.

**Please note:** All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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