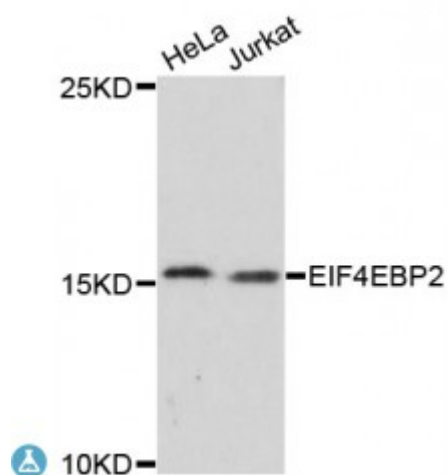


Anti-EIF4EBP2 Antibody



Description

This gene encodes a member of the eukaryotic translation initiation factor 4E binding protein family. The gene products of this family bind eIF4E and inhibit translation initiation. However, insulin and other growth factors can release this inhibition via a phosphorylation-dependent disruption of their binding to eIF4E. Regulation of protein production through these gene products have been implicated in cell proliferation, cell differentiation and viral infection.

Model	STJ115646
Host	Rabbit
Reactivity	Human
Applications	WB
Immunogen	Recombinant fusion protein containing a sequence corresponding to amino acids 61-120 of human EIF4EBP2 (NP_004087.1).
Gene ID	1979
Gene Symbol	EIF4EBP2
Dilution range	WB 1:500 - 1:2000
Purification	Affinity purification
Note	For Research Use Only (RUO).
Protein Name	Eukaryotic translation initiation factor 4E-binding protein 2 4E-BP2 eIF4E-binding protein 2
Molecular Weight	12.939 kDa

Clonality	Polyclonal
Conjugation	Unconjugated
Isotype	IgG
Formulation	PBS with 0.02% sodium azide, 50% glycerol, pH7.3.
Storage Instruction	Store at -20C. Avoid freeze / thaw cycles.
Database Links	HGNC:32890MIM:602224
Alternative Names	Eukaryotic translation initiation factor 4E-binding protein 2 4E-BP2 eIF4E-binding protein 2
Function	Repressor of translation initiation involved in synaptic plasticity, learning and memory formation , 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 ,
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 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