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EIF2A Antibody

Purified Mouse Monoclonal Antibody (Mab)
Catalog # AM8703b

Product Information

Application	WB, E
Primary Accession	P05198
Reactivity	Human, Mouse, Rat
Predicted	Bovine, Canine, Rabbit, Chicken
Host	Mouse
Clonality	monoclonal
Isotype	IgG2b,k
Clone Names	1763CT191.40.42
Calculated MW	36112

Additional Information

Gene ID	1965
Other Names	Eukaryotic translation initiation factor 2 subunit 1, Eukaryotic translation initiation factor 2 subunit alpha, eIF-2-alpha, eIF-2A, eIF-2alpha, EIF2S1, EIF2A
Target/Specificity	This EIF2A antibody is generated from a mouse immunized with a recombinant protein of human EIF2A.
Dilution	WB~~1:2000 E~~Use at an assay dependent concentration.
Format	Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	EIF2A Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	EIF2S1 (HGNC:3265)
Synonyms	EIF2A
Function	Member of the eIF2 complex that functions in the early steps of protein synthesis by forming a ternary complex with GTP and initiator tRNA (PubMed:16289705, PubMed:38340717). This complex binds to a 40S

	ribosomal subunit, followed by mRNA binding to form a 43S pre- initiation complex (43S PIC) (PubMed:16289705). Junction of the 60S ribosomal subunit to form the 80S initiation complex is preceded by hydrolysis of the GTP bound to eIF2 and release of an eIF2-GDP binary complex (PubMed:16289705). In order for eIF2 to recycle and catalyze another round of initiation, the GDP bound to eIF2 must exchange with GTP by way of a reaction catalyzed by eIF2B (PubMed:16289705). EIF2S1/eIF2-alpha is a key component of the integrated stress response (ISR), required for adaptation to various stress: phosphorylation by metabolic-stress sensing protein kinases (EIF2AK1/HRI, EIF2AK2/PKR, EIF2AK3/PERK and EIF2AK4/GCN2) in response to stress converts EIF2S1/eIF2-alpha in a global protein synthesis inhibitor, leading to an attenuation of cap-dependent translation, while concomitantly initiating the preferential translation of ISR-specific mRNAs, such as the transcriptional activators ATF4 and QRICH1, and hence allowing ATF4- and QRICH1-mediated reprogramming (PubMed:19131336, PubMed:33384352, PubMed:38340717). EIF2S1/eIF2-alpha also acts as an activator of mitophagy in response to mitochondrial damage: phosphorylation by EIF2AK1/HRI promotes relocalization to the mitochondrial surface, thereby triggering PRKN-independent mitophagy (PubMed:38340717).
Cellular Location	Cytoplasm, Stress granule {ECO:0000250|UniProtKB:Q6ZWX6}. Cytoplasm, cytosol {ECO:0000250|UniProtKB:P56286}. Mitochondrion. Note=Colocalizes with NANOS3 in the stress granules (By similarity). Relocalizes to the surface of mitochondria in response to mitochondrial damage and phosphorylation by EIF2AK1/HRI (PubMed:38340717). {ECO:0000250|UniProtKB:Q6ZWX6, ECO:0000269|PubMed:38340717}

Background

Functions in the early steps of protein synthesis by forming a ternary complex with GTP and initiator tRNA. This complex binds to a 40S ribosomal subunit, followed by mRNA binding to form a 43S preinitiation complex. Junction of the 60S ribosomal subunit to form the 80S initiation complex is preceded by hydrolysis of the GTP bound to eIF-2 and release of an eIF-2-GDP binary complex. In order for eIF-2 to recycle and catalyze another round of initiation, the GDP bound to eIF-2 must exchange with GTP by way of a reaction catalyzed by eIF-2B.

References

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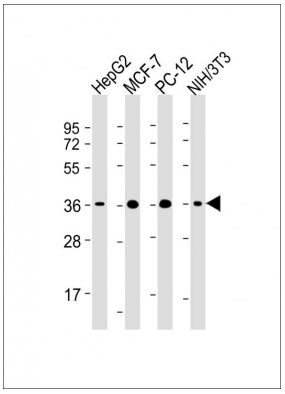
Images

All lanes : Anti-EIF2A Antibody at 1:2000 dilution Lane 1: HepG2 whole cell lysate Lane 2: MCF-7 whole cell lysate Lane 3: PC-12 whole cell lysate Lane 4: NIH/3T3 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-mouse IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 36 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



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