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eIF2α Antibody

Purified Mouse Monoclonal Antibody (Mab)
Catalog # AP52859

Product Information

Application	WB
Primary Accession	P05198
Reactivity	Human
Host	Mouse
Clonality	Monoclonal
Isotype	IgG2b
Calculated MW	36112

Additional Information

Gene ID	1965
Other Names	Dmel\CG6376 ;Dmel_CG6376 ;drosE2F1 ;E(Sev-CycE)3A ;E(var)3-93E ;E2-promoter binding facto ;E2F 1 ;E2F transcription factor 1 ;E2F-1 ;E2f-PA ;E2f-PB ;E2f-PC ;E2F1 ;E2f1 E2F transcription factor 1 ;E2F1_HUMAN ;Evar(3)164 ;KIAA4009 ;l(3)07172 ;l(3)j3B1 ;l(3)j3C2 ;l(3)rM729 ;mKIAA4009 ;OTTHUMP00000030661 ;PBR3 ;PRB binding protein E2F 1 ;PRB-binding protein E2F-1 ;RBAP 1 ;RBAP-1 ;RBAP1 ;RBBP-3 ;RBBP3 ;RBP 3 ;RBP3 ;Retinoblastoma-associated protein 1 ;Retinoblastoma-binding protein 3 ;Transcription factor E2F1.
Dilution	WB~~1:1000
Format	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide, pH 7.3.
Storage	Store at -20 °C.Stable for 12 months from date of receipt

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

Ernst H.,et al.J. Biol. Chem. 262:1206-1212(1987).
Langland J.O.,et al.Virology 324:419-429(2004).
Paytubi S.,et al.Biochem. J. 409:223-231(2008).
Montero H.,et al.J. Virol. 82:1496-1504(2008).
Mayya V.,et al.Sci. Signal. 2:RA46-RA46(2009).

Images

Image not found : 201506/AP52859_WB_1.jpg	Western blot detection of eIF2α in C2C12,MCF7,C6 and Hela cell lysates using eIF2α mouse mAb (1:1000 diluted).Predicted band size:38KDa.Observed band size:38KDa.