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[Primary Antibody]

Rabbit Anti-eIF2B epsilon antibody

Catalog Number: SL14533R

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	eIF2B epsilon
Immunogen:	KLH conjugated synthetic peptide derived from human eIF2B epsilon: 101-200/721
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
Storage:	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4℃. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.
Applications:	WB=1:500-2000 IHC-P=1:100-500 IHC-F=1:100-500 ICC/IF=1:100-500 IF=1:100-500 ELISA=1:5000-10000 （Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	(predicted: Human, Mouse, Rat, Dog, Pig, Cow, Horse, Rabbit, Sheep)
Predicted MW.:	80kDa
Subcellular Location:	The nucleus cytoplasmic
Background:	This gene encodes one of five subunits of eukaryotic translation initiation factor 2B (EIF2B), a GTP exchange factor for eukaryotic initiation factor 2 and an essential regulator for protein synthesis. Mutations in this gene and the genes encoding other EIF2B subunits have been associated with leukoencephalopathy with vanishing white matter. [provided by RefSeq, Nov 2009] **Function:** Catalyzes the exchange of eukaryotic initiation factor 2-bound GDP for GTP. **Subunit:** Complex of five different subunits; alpha, beta, gamma, delta and

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	epsilon. Interacts with RGS2. **Post-translational modifications:** Phosphorylated at Ser-544 by DYRK2; this is required for subsequent phosphorylation by GSK3B (By similarity). Phosphorylated on serine and threonine residues by GSK3B; phosphorylation inhibits its function. Polyubiquitinated, probably by NEDD4 (By similarity). DISEASE: Defects in EIF2B5 are a cause of leukodystrophy with vanishing white matter (VWM) [MIM:603896]. VWM is a leukodystrophy that occurs mainly in children. Neurological signs include progressive cerebellar ataxia, spasticity, inconstant optic atrophy and relatively preserved mental abilities. The disease is chronic-progressive with, in most individuals, additional episodes of rapid deterioration following febrile infections or minor head trauma. While childhood onset is the most common form of the disorder, some severe forms are apparent at birth. A severe, early-onset form seen among the Cree and Chippewayan populations of Quebec and Manitoba is called Cree leukoencephalopathy. Milder forms may not become evident until adolescence or adulthood. Some females with milder forms of the disease who survive to adolescence exhibit ovarian dysfunction. This variant of the disorder is called ovarioleukodystrophy. **Similarity:** Belongs to the eIF-2B gamma/epsilon subunits family. Contains 1 W2 domain. **Database links:** **Entrez Gene:** 8893 Human **Entrez Gene:** 224045 Mouse **Entrez Gene:** 192234 Rat Omim: 603945 Human **SwissProt:** Q13144 Human **SwissProt:** Q8CHW4 Mouse **SwissProt:** Q64350 Rat **Unigene:** 283551 Human **Unigene:** 233855 Mouse **Unigene:** 10607 Rat

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