ALS2 Rabbit pAb

CatalogNo: YN0436

Key Features

Host Species	Reactivity	Applications
• Rabbit	• Human,Mouse,Rat	• WB,ELISA

MW	Isotype
• 182kD (Observed)	• IgG

Storage

Storage*	-15°C to -25°C/1 year(Do not lower than -25°C)
Formulation	Liquid in PBS containing 50% glycerol,0.5% BSA and 0.02% sodium azide.

Recommended Dilution Ratios

WB 1:500-2000
ELISA 1:5000-20000

Basic Information

Clonality	Polyclonal

Immunogen Information

Immunogen	Synthesized peptide derived from human protein . at AA range: 390-470
Specificity	ALS2 Polyclonal Antibody detects endogenous levels of protein.

Target Information

Gene name	ALS2 ALS2CR6 KIAA1563

Protein Name Alsin (Amyotrophic lateral sclerosis 2 chromosomal region candidate gene 6 protein) (Amyotrophic lateral sclerosis 2 protein)

Organism	Gene ID	UniProt ID
Human	57679;	Q96Q42;
Mouse		Q920R0;
Rat		P0C5Y8;

Cellular Localization ruffle,early endosome,centrosome,cytosol,postsynaptic density,membrane,lamellipodium,axon,dendrite,growth cone,vesicle,neuronal cell body,dendritic spine,intracellular membra

Tissue specificity Brain,Colon,Kidney,Placenta,

Function Disease:Defects in ALS2 are the cause of amyotrophic lateral sclerosis type 2 (ALS2) [MIM:205100]. ALS2 is a familial form of amyotrophic lateral sclerosis, a neurodegenerative disorder affecting upper and lower motor neurons and resulting in fatal paralysis. Sensory abnormalities are absent. Death usually occurs within 2 to 5 years. The etiology of amyotrophic lateral sclerosis is likely to be multifactorial, involving both genetic and environmental factors. The disease is inherited in 5-10% of cases leading to familial forms.,Disease:Defects in ALS2 are the cause of infantile-onset ascending spastic paralysis (IAHSP) [MIM:607225]. IAHSP is characterized by progressive spasticity and weakness of limbs.,Disease:Defects in ALS2 are the cause of juvenile primary lateral sclerosis (JPLS) [MIM:606353]. JPLS is a neurodegenerative disorder which is closely related to but clinically distinct from amyotrophic lateral sclerosis. It is a progressive paralytic disorder which results from dysfunction of the upper motor neurons of the motor cortex while the lower neurons are unaffected.,Function:May act as a GTPase regulator. Controls survival and growth of spinal motoneurons.,online information:ALS genetic mutations db,PTM:Phosphorylated upon DNA damage, probably by ATM or ATR.,similarity:Contains 1 DH (DBL-homology) domain.,similarity:Contains 1 PH domain.,similarity:Contains 1 VPS9 domain.,similarity:Contains 5 RCC1 repeats.,similarity:Contains 8 MORN repeats.,subunit:Forms a heteromeric complex with ALS2CL. Interacts with ALS2CL.,

Validation Data

Contact information

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Please scan the QR code to access additional product information:
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