MYP0 Rabbit pAb

CatalogNo: YN0890

Key Features

Host Species
- Rabbit

Reactivity
- Human,Mouse,Rat

Applications
- WB,ELISA

MW
- 27kD (Observed)

Isotype
- IgG

Recommended Dilution Ratios

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

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.

Basic Information

Clonality	Polyclonal

Immunogen Information

Immunogen	Synthesized peptide derived from human protein . at AA range: 50-130
Specificity	MYP0 Polyclonal Antibody detects endogenous levels of protein.

Target Information

Gene name	MPZ

Protein Name Myelin protein P0 (Myelin peripheral protein) (MPP) (Myelin protein zero)

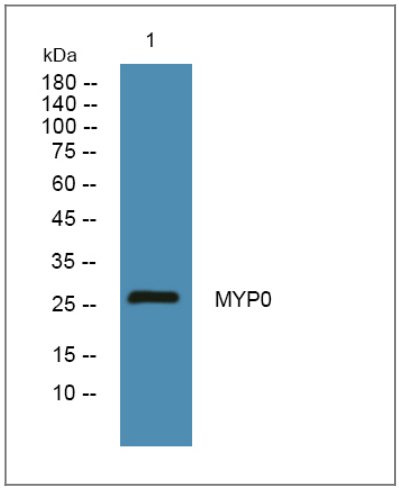
Organism	Gene ID	UniProt ID
Human	4359;	P25189;
Mouse		P27573;
Rat		P06907;

Cellular Localization Cell membrane ; Single-pass type I membrane protein.; [Isoform L-MPZ]: Myelin membrane ; Single-pass type I membrane protein .

Tissue specificity Found only in peripheral nervous system Schwann cells.

Function	Disease:Defects in MPZ are a cause of congenital hypomyelination neuropathy (CHN) [MIM:605253]. CHN is characterized clinically by early onset of hypotonia, areflexia, distal muscle weakness, and very slow nerve conduction velocities.,Disease:Defects in MPZ are a cause of Dejerine-Sottas syndrome (DSS) [MIM:145900]; also known as Dejerine-Sottas neuropathy (DSN) or hereditary motor and sensory neuropathy III (HMSN3). DSS is a severe degenerating neuropathy of the demyelinating Charcot-Marie-Tooth disease category, with onset by age 2 years. DSS is characterized by motor and sensory neuropathy with very slow nerve conduction velocities, increased cerebrospinal fluid protein concentrations, hypertrophic nerve changes, delayed age of walking as well as areflexia. There are both autosomal dominant and autosomal recessive forms of Dejerine-Sottas syndrome.,Disease:Defects in MPZ are a cause of Roussy-Levy syndrome [MIM:180800]; also known as Roussy-Levy hereditary areflexic dystasia. This autosomal dominant disorder resembles Charcot-Marie-Tooth disease type 1 in that it presents with foot deformity, weakness and atrophy of distal limb muscles, especially the peronei, and absent tendon reflexes. The phenotype differs, however, in that it includes static tremor of the upper limbs and gait ataxia.,Disease:Defects in MPZ are the cause of Adie pupil [MIM:103100]. Adie pupil is a feature of CMT2J. It is characterized by tonic, sluggishly reacting pupil and hypoactive or absent tendon reflexes.,Disease:Defects in MPZ are the cause of Charcot-Marie-Tooth disease type 1B (CMT1B) [MIM:118200]. CMT1B is a form of Charcot-Marie-Tooth disease, the most common inherited disorder of the peripheral nervous system. Charcot-Marie-Tooth disease is classified in two main groups on the basis of electrophysiologic properties and histopathology: primary peripheral demyelinating neuropathy or CMT1, and primary peripheral axonal neuropathy or CMT2. Neuropathies of the CMT1 group are characterized by severely reduced nerve conduction velocities (less than 38 m/sec), segmental demyelination and remyelination with onion bulb formations on nerve biopsy, slowly progressive distal muscle atrophy and weakness, absent deep tendon reflexes, and hollow feet.,Disease:Defects in MPZ are the cause of Charcot-Marie-Tooth disease type 2I (CMT2I) [MIM:607677]. CMT2I is a form of Charcot-Marie-Tooth disease, the most common inherited disorder of the peripheral nervous system. Charcot-Marie-Tooth disease is classified in two main groups on the basis of electrophysiologic properties and histopathology: primary peripheral demyelinating neuropathy or CMT1, and primary peripheral axonal neuropathy or CMT2. Neuropathies of the CMT2 group are characterized by signs of axonal regeneration in the absence of obvious myelin alterations, normal or slightly reduced nerve conduction velocities, and progressive distal muscle weakness and atrophy. CMT2I is characterized by late onset (range 47 to 60 years).,Disease:Defects in MPZ are the cause of Charcot-Marie-Tooth disease type 2J (CMT2J) [MIM:607736]. CMT2J is a form of Charcot-Marie-Tooth disease characterized by the association of axonal peripheral neuropathy with hearing loss and pupillary abnormalities such as Adie pupil. Inheritance is autosomal dominant.,Disease:Defects in MPZ may be the cause of Charcot-Marie-Tooth disease dominant intermediate type D (CMTDID) [MIM:607791]. CMTDID is a form of Charcot-Marie-Tooth disease characterized by features intermediate between demyelinating and axonal peripheral neuropathies, and motor median nerve conduction velocities ranging from 25 to 45 m/sec.,Function:Creation of an extracellular membrane face which guides the wrapping process and ultimately compacts adjacent lamellae.,PTM:N-glycosylated; contains sulfate-substituted glycan.,similarity:Belongs to the myelin P0 protein family.,similarity:Contains 1 Ig-like V-type (immunoglobulin-like) domain.,tissue specificity:Found only in peripheral nervous system Schwann cells.,

Validation Data



Western blot analysis of lysates from DU145 cells, primary antibody was diluted at 1:1000, 4°over night

Contact information

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