


Anti-Myelin Protein Zero antibody ab31851

★★★★★ [14 Abreviews](#) [47 References](#) [1 Image](#)

Overview

Product name	Anti-Myelin Protein Zero antibody
Description	Rabbit polyclonal to Myelin Protein Zero
Host species	Rabbit
Tested applications	Suitable for: WB Unsuitable for: IHC-Fr
Species reactivity	Reacts with: Mouse Predicted to work with: Rat, Human 
Immunogen	Synthetic peptide conjugated to KLH derived from within residues 200 to the C-terminus of Rat Myelin Protein Zero. Read Abcam's proprietary immunogen policy (Peptide available as ab31869 .)
General notes	<p>The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets your needs before purchasing.</p> <p>If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, along with publications, customer reviews and Q&As</p>

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.
Storage buffer	pH: 7.40 Preservative: 0.02% Sodium azide Constituent: PBS
Purity	<p>Batches of this product that have a concentration < 1mg/ml may have BSA added as a stabilising agent. If you would like information about the formulation of a specific lot, please contact our scientific support team who will be happy to help.</p> <p>Immunogen affinity purified</p>

Clonality	Polyclonal
Isotype	IgG

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab31851 in the following tested applications. The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
WB	★★★★★ (5)	Use a concentration of 1 µg/ml. Detects a band of approximately 25 kDa (predicted molecular weight: 27 kDa).

Application notes Is unsuitable for IHC-Fr.

Target

Function Creation of an extracellular membrane face which guides the wrapping process and ultimately compacts adjacent lamellae.

Tissue specificity Found only in peripheral nervous system Schwann cells.

Involvement in 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.

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).

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.

Defects in MPZ are the cause of Adie pupil (ADIEP) [MIM:103100]. A stationary, benign disorder characterized by tonic, sluggishly reacting pupil and hypoactive or absent tendon reflexes. Adie pupil is a characteristic of Charcot-Marie-Tooth disease type 2J.

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.

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.

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.

Defects in MPZ are a cause of Roussy-Levy syndrome (ROULS) [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.

Sequence similarities

Belongs to the myelin P0 protein family.

Contains 1 Ig-like V-type (immunoglobulin-like) domain.

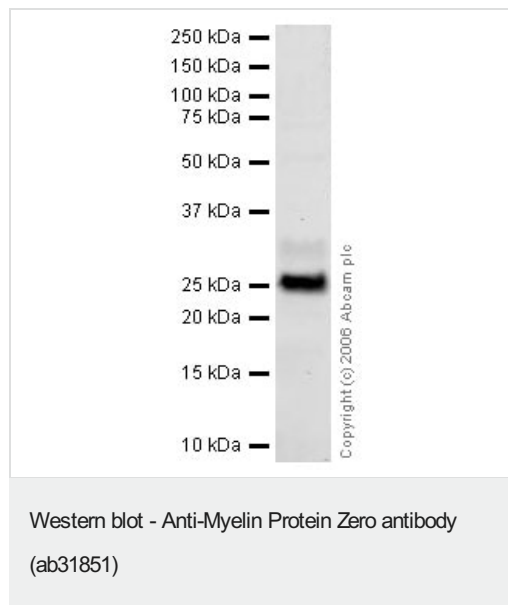
Post-translational modifications

N-glycosylated; contains sulfate-substituted glycan.

Cellular localization

Membrane.

Images



Anti-Myelin Protein Zero antibody (ab31851) at 1 µg/ml + Mouse Sciatic Nerve Whole Tissue Lysate at 20 µg

Secondary

IRDye 680 Conjugated Goat Anti-Rabbit IgG (H+L) at 1/15000 dilution

Performed under reducing conditions.

Predicted band size: 27 kDa

Observed band size: 25 kDa

We also see a similar banding pattern in Rat Sciatic Nerve lysate although the band is more smeared than observed in the Western Blot shown here. We believe the smearing is caused by glycosylation of the target protein.

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