abcam

Product datasheet

Anti-Myelin Protein Zero antibody [EPR8883(2)] - BSA and Azide free ab250266

Recombinant

RabMAb

2 Images

Overview

Product name Anti-Myelin Protein Zero antibody [EPR8883(2)] - BSA and Azide free

Description Rabbit monoclonal [EPR8883(2)] to Myelin Protein Zero - BSA and Azide free

Host species Rabbit

Tested applications Suitable for: WB

Reacts with: Mouse **Species reactivity**

Predicted to work with: Rat, Horse, Guinea pig, Cow, Human, Pig, Chinese hamster

Immunogen Synthetic peptide. This information is proprietary to Abcam and/or its suppliers.

General notes ab250266 is the carrier-free version of ab180933.

> Our carrier-free antibodies are typically supplied in a PBS-only formulation, purified and free of BSA, sodium azide and glycerol. The carrier-free buffer and high concentration allow for increased conjugation efficiency.

This conjugation-ready format is designed for use with fluorochromes, metal isotopes, oligonucleotides, and enzymes, which makes them ideal for antibody labelling, functional and cellbased assays, flow-based assays (e.g. mass cytometry) and Multiplex Imaging applications.

Use our conjugation kits for antibody conjugates that are ready-to-use in as little as 20 minutes with <1 minute hands-on-time and 100% antibody recovery: available for fluorescent dyes, HRP, biotin and gold.

This product is compatible with the Maxpar® Antibody Labeling Kit from Fluidigm, without the need for antibody preparation. Maxpar[®] is a trademark of Fluidigm Canada Inc.

This product is a recombinant monoclonal antibody, which offers several advantages including:

- High batch-to-batch consistency and reproducibility
- Improved sensitivity and specificity
- Long-term security of supply
- Animal-free production

For more information see here.

Our RabMAb® technology is a patented hybridoma-based technology for making rabbit monoclonal antibodies. For details on our patents, please refer to **RabMAb**® **patents**.

Properties

Form Liquid

Storage instructions Shipped at 4°C. Store at +4°C. Do Not Freeze.

Storage buffer pH: 7.2

Constituent: PBS

Carrier free Yes

Purity Protein A purified

Clonality Monoclonal
Clone number EPR8883(2)

Isotype IgG

Applications

The Abpromise guarantee

Our **Abpromise guarantee** covers the use of ab250266 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		Use at an assay dependent concentration. Detects a band of approximately 25 kDa (predicted molecular weight: 28 kDa).

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 lg-like V-type (immunoglobulin-like) domain.

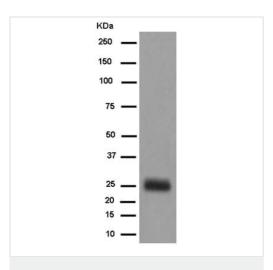
Post-translational modifications

N-glycosylated; contains sulfate-substituted glycan.

Cellular localization

Membrane.

Images



Western blot - Anti-Myelin Protein Zero antibody [EPR8883(2)] - BSA and Azide free (ab250266)

Anti-Myelin Protein Zero antibody [EPR8883(2)] ($\underline{ab180933}$) at 1/1000 dilution + Mouse spinal cord at 10 μg

Secondary

Goat Anti-Rabbit lgG, (H+L), Peroxidase conjugated at 1/1000 dilution

Predicted band size: 28 kDa

This data was developed using <u>ab180933</u>, the same antibody clone in a different buffer formulation.

Blocking and dilution buffer: 5% NFDM/TBST.



Research with confidence Consistent and reproducible results

Long-term and scalable supply Recombinant technology



specificity



Anti-Myelin Protein Zero antibody [EPR8883(2)] -

BSA and Azide free (ab250266)

Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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