

Recombinant Mouse MAG/Siglec-4a Protein (His Tag)

Catalog Number:PKSM040311



Note: Centrifuge before opening to ensure complete recovery of vial contents.

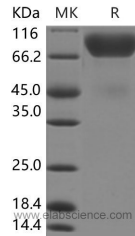
Description

Synonyms	Myelin-Associated Glycoprotein;MAG;Siglec-4a
Species	Mouse
Expression Host	HEK293 Cells
Sequence	Met1-Pro516
Accession	NP_034888.1
Calculated Molecular Weight	56.3 kDa
Tag	C-His

Properties

Purity	> 95 % as determined by SDS-PAGE
Endotoxin	< 1.0 EU per µg of the protein as determined by the LAL method.
Storage	Lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile PBS, pH 7.4
Reconstitution	Please refer to the printed manual for detailed information.

Data



Background

The myelin-associated glycoprotein (MAG) contains five immunoglobulin-like domains and belongs to the sialic-acid-binding subgroup of the Ig superfamily. MAG is a transmembrane glycoprotein of 100kDa localized in myelin sheaths of periaxonal Schwann cell and oligodendroglial membranes where it functions in glia-axon interactions. It appears to function both as a receptor for an axonal signal that promotes the differentiation, maintenance and survival of oligodendrocytes and as a ligand for an axonal receptor that is needed for the maintenance of myelinated axons. MAG contains a carbohydrate epitope shared with other glycoconjugates that is a target antigen in autoimmune peripheral neuropathy associated with IgM gammopathy and has been implicated in a dying back oligodendroglialopathy in multiple sclerosis. MAG is considered as a transmembrane protein of both CNS and PNS myelin and it strongly inhibits neurite outgrowth in both developing cerebellar and adult dorsal root ganglion neurons. In contrast, MAG promotes neurite outgrowth from newborn DRG neurons. Thus, MAG may be responsible for the lack of CNS nerve regeneration and may influence both temporally and spatially regeneration in the PNS.

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