

**DESCRIPTION**

<b>Source</b>	Mouse myeloma cell line, NS0-derived		
	Mouse Nogo Receptor (Cys27 - Ser447) Accession # Q99PI8	IEGRMD	Human IgG <sub>1</sub> (Pro100 - Lys330)
	N-terminus		C-terminus

<b>N-terminal Sequence Analysis</b>	Cys27
<b>Structure / Form</b>	Disulfide-linked homodimer
<b>Predicted Molecular Mass</b>	72.1 kDa (monomer)

**SPECIFICATIONS**

<b>SDS-PAGE</b>	95-100 kDa, reducing conditions
<b>Activity</b>	Measured by its ability to bind rrMAG/Fc Chimera in a functional ELISA.
<b>Endotoxin Level</b>	<0.01 EU per 1 µg of the protein by the LAL method.
<b>Purity</b>	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 100 µg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> <li>• 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>• 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>• 3 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

**BACKGROUND**

Nogo Receptor (NgR), also named reticulon 4 receptor, is a glycosylphosphoinositol (GPI)-anchored protein that belongs to the family of leucine-rich repeat (LRR) proteins (1). It is expressed predominantly in the central nervous systems in neurons and their axons. NgR plays an essential role in mediating axon growth inhibition induced by the structurally distinct myelin-derived proteins Nogo, myelin-associated glycoprotein (MAG), and myelin oligodendrocyte glycoprotein (Omgp) (2, 3). Human NgR cDNA encodes a 473 amino acid (aa) residue precursor with a 26 aa putative signal peptide, an LRR-type N-terminal region, eight LRR repeats, a cysteine-rich LRR-type C-terminal region, a GPI linkage domain and a 26 aa C-terminal propeptide that is removed in the mature form (1). All of the LRR domains within NgR are required for ligand binding and receptor oligomerization (4). NgR mediates its inhibitory actions by interacting with the p75 neurotrophin receptor (p75<sup>NTR</sup>), a tumor necrosis factor receptor superfamily (TNFRSF) member also known for modulating the activities of the Trk family of receptor tyrosine kinases, and for inducing apoptosis in neurons and oligodendrocytes (5). Upon ligand binding, NgR binds to and activates the p75<sup>NTR</sup>. The activated p75<sup>NTR</sup> then sequesters the Rho guanine dissociation inhibitor (Rho-GDI) away from Rho and allows Rho to change into the active GTP-bound state which can interact with signaling proteins to suppress axonal growth and regeneration (4). The truncated extracellular domain of NgR has been shown to bind the myelin-derived inhibitors and block inhibition of axon growth by myelin (6).

**References:**

1. Fournier, A.E. *et al.* (2001) *Nature* **409**:341.
2. GrandPre, T. *et al.* (2002) *Nature* **417**:547.
3. Wang, K.C. *et al.* (2002) *Nature* **420**:74.
4. Barton, W.A. *et al.* (2003) *EMBO Journal* **22**:3291.
5. Yamashita, T. and M. Tohyama (2003) *Nature Neuroscience* **6**:461.
6. Fournier, A.S. *et al.* (2002) *J. Neurosci.* **22**:8876.