

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Nogo Receptor (NgR) in direct ELISAs and Western blots.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	recombinant human NgR Cys27-Ser447 Accession # Q9BZR6
Conjugate	Alexa Fluor 532 Excitation Wavelength: 534 nm Emission Wavelength: 553 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide
*Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.	

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

Western Blot	Optimal dilution of this antibody should be experimentally determined.
Blockade of Receptor-ligand Interaction	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

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 residues (aa) 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^{NTR}), 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^{NTR}. The activated p75^{NTR} 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).

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