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PRODUCT INFORMATION

Ninjurin-1 Extracellular Domain (rat, recombinant)

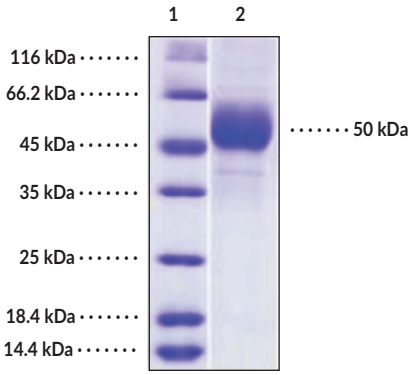
Item No. 41069

Overview and Properties

Synonyms:	Nerve Injury-induced Protein 1, NINJ1
Source:	Recombinant N-terminal human IgG1 Fc-tagged rat ninjurin-1 extracellular domain expressed in HEK293 cells
Amino Acids:	1-79
Uniprot No.:	P70617
Molecular Weight:	37 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	≥95% estimated by SDS-PAGE
Supplied in:	Lyophilized from sterile PBS, pH 7.4
Endotoxin Testing:	<1.0 EU/µg, determined by the LAL endotoxin assay
Protein Concentration:	***batch specific*** mg/ml

Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Image



Lane 1: MW Markers
Lane 2: Ninjurin-1 Extracellular Domain (rat, recombinant)

SDS-PAGE Analysis of Ninjurin-1 Extracellular Domain (rat, recombinant). This protein has an apparent molecular weight of 50 kDa by SDS-PAGE under reducing conditions due to glycosylation.

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PRODUCT INFORMATION

Description

Ninjurin-1 is a type 3b transmembrane protein and adhesion molecule.[1] It is composed of an N-terminal extracellular domain containing a region required for homophilic binding, two transmembrane domains, a short intracellular region, and a second extracellular domain at the C-terminal.[1,2] The N-terminal extracellular domain can be cleaved by matrix metalloprotease-9 (MMP-9) to release soluble ninjurin-1 (sNinj1).[3] Ninjurin-1 is expressed in Schwann cells, neurons, and certain activated immune cells, including macrophages and T cells, as well as embryonic and adult epithelial tissues.[1,4] It is involved in cell adhesion, axonal growth, and chemotaxis, as well as cell lysis and damage-associated molecular pattern (DAMP) molecule release during pyroptosis, among other activities.[2,4] *NINJ1* knockout reduces disease severity in a mouse model of experimental autoimmune encephalomyelitis (EAE).[5] The expression of *Ninj1* is increased in Schwann cells and dorsal root ganglion neurons after nerve injury in rats, and ninjurin-1 is involved in peripheral nerve regeneration after sciatic nerve injury in mice.[4,6] An A-C reversal in the third exon of *NINJ1*, which results in an aspartate-to-alanine mutation, increases the likelihood of developing nerve damage in patients with leprosy.[4] Serum levels of sNinj1 are increased in patients with hepatocellular carcinoma and are positively correlated with tumor size, cancer stage, and metastasis.[3] Cayman's Ninjurin-1 Extracellular Domain (rat, recombinant) protein is a disulfide-linked homodimer. The reduced monomer, composed of ninjurin-1 (amino acids 1-79) fused to human IgG1 Fc at its N-terminus, consists of 340 amino acids and has a calculated molecular weight of 37 kDa. As a result of glycosylation, the monomer migrates at approximately 50 kDa by SDS-PAGE under reducing conditions.

References

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3. Yan, L., Su, W., Gan, D., *et al.* Circulating sNinj1 as a novel predictor of prognosis and severity in hepatocellular carcinoma. *Clin. Chim. Acta* **550,** 117581 (2023).
4. Liu, K., Wang, Y., and Li, H. The role of Ninjurin1 and its impact beyond the nervous system. *Dev. Neurosci.* **42(5-6),** 159-169 (2020).
5. Ahn, B.J., Le, H., Shin, M.W., *et al.* Ninjurin1 deficiency attenuates susceptibility of experimental autoimmune encephalomyelitis in mice. *J. Biol. Chem.* **289(6),** 3328-3338 (2014).
6. Tomita, Y., Horiuchi, K., Kano, K., *et al.* Ninjurin 1 mediates peripheral nerve regeneration through Schwann cell maturation of NG2-positive cells. *Biochem. Biophys. Res. Commun.* **519(3),** 462-468 (2019).

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