

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse SIGNR1 in direct ELISAs and Western blots. In direct ELISAs, approximately 20% cross-reactivity with recombinant mouse (rm) SIGNR3 and rmSIGNR4 is observed, and approximately 10% cross-reactivity with recombinant human DC-SIGNR and rmDC-SIGN is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse SIGNR1 Ser76-Gly325 Accession # Q8CJ91
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

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.

	Recommended Concentration	Sample
Western Blot	0.1 µg/mL	Recombinant Mouse SIGNR1/CD209 Fc Chimera (Catalog # 1836-SR)
Adhesion Blockade	The adhesion of human ICAM-3 expressing CHO Chinese hamster ovary cells (5 x 10 ⁴ cells/well) to immobilized Recombinant Mouse SIGNR1/CD209b Fc Chimera (Catalog # 1836-SR , 10 µg/mL, 100 µL/well) was maximally inhibited (80-100%) by 10 µg/mL of the antibody.	

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

SIGNR1 belongs to the family of C-type lectins that participate in innate immune responses by binding and clearing pathogens (1-3). SIGNR1 is one of four mouse proteins, encoded by separate genes, with significant homology to DC-SIGN and DC-SIGNR (4). Mouse SIGNR1 consists of a 53 amino acid (aa) cytoplasmic domain, a 20 aa transmembrane segment, and a 252 aa extracellular domain that contains a juxtamembrane neck region and a carbohydrate recognition domain (CRD) (4, 5). SIGNR1 is a 50kDa type II protein that forms high molecular weight aggregates as has been shown for DC-SIGN and DC-SIGNR (6, 7). The mouse SIGNR1 mRNA is extensively spliced, giving rise to multiple isoforms in addition to variability in the 5' and 3' untranslated regions. The α isoform is known as SIGNR1. The β and γ isoforms lack the transmembrane segment, with the γ isoform also showing a 21 aa insertion in the CRD. The δ isoform lacks almost the entire CRD (5). Functional differences between the isoforms have not been described. Within the CRD, mouse SIGNR1 shares 67%-70% aa sequence identity with mouse DC-SIGN, SIGNR2, 3, and 4, and 64% - 68% aa sequence identity with human DC-SIGN, human L-SIGN, and rat DC-SIGN. The CRD binds mannose, GlcNAc, and fucose structures on some bacterial strains, mycobacteria, and yeast (8). SIGNR1 is expressed on macrophages found in the splenic marginal zone, lymph node medulla, and peritoneum (6, 9, 10). It plays a dominant role in the clearance of circulating *S. pneumoniae* (11 - 13), enhances TLR4 signaling (14), and cooperates with Dectin-1 in the nonopsonic clearance of fungal pathogens (9, 15). SIGNR1 also binds mouse ICAM-2, human ICAM-2 and -3, and HIV gp120 (10).

References:

1. Marshall, A.S.J. and S. Gordon (2004) *Eur. J. Immunol.* **34**:18.
2. Cambi, A. and C.G. Figdor (2005) *Curr. Opin. Immunol.* **17**:345.
3. McGreal, E.P. *et al.* (2005) *Curr. Opin. Immunol.* **17**:18.
4. Park, C.G. *et al.* (2001) *Int. Immunol.* **13**:1283.
5. Parent, S.A. *et al.* (2002) *Gene* **293**:33.
6. Kang, Y.-S. *et al.* (2003) *Int. Immunol.* **15**:177.
7. Feinberg, H. *et al.* (2005) *J. Biol. Chem.* **280**:1327.
8. Galustian, C. *et al.* (2003) *Int. Immunol.* **16**:853.
9. Taylor, P.R. *et al.* (2004) *J. Immunol.* **172**:1157.
10. Geijtenbeek, T.B.H. *et al.* (2002) *Blood* **100**:2908.
11. Koppel, E.A. *et al.* (2005) *Eur. J. Immunol.* **35**:2962.
12. Lanoue, A. *et al.* (2004) *J. Exp. Med.* **200**:1383.
13. Kang, Y.-S. *et al.* (2004) *Proc. Natl. Acad. Sci.* **101**:215.
14. Nagaoka, K. *et al.* (2005) *Int. Immunol.* **17**:827.
15. Takahara, K. *et al.* (2003) *Int. Immunol.* **16**:819.