

DESCRIPTION

Source	Chinese Hamster Ovary cell line, CHO-derived rat Siglec-2/CD22 protein		
	Rat Siglec-2/CD22 (Trp24-Gly690) Accession # NP_001100973.1	IEGRMDP	Mouse IgG _{2a} (Glu98-Lys330)
	N-terminus		C-terminus
N-terminal Sequence Analysis	Trp24		
Predicted Molecular Mass	102 kDa		

SPECIFICATIONS

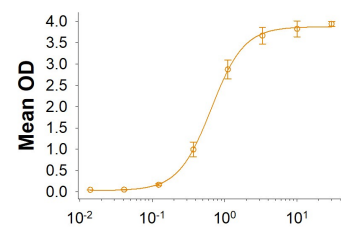
SDS-PAGE	112-138 kDa, under reducing conditions
Activity	Measured by the ability of the immobilized protein to support the adhesion of human red blood cells. The ED ₅₀ for this effect is 0.15-1.8 µg/mL.
Endotoxin Level	<1.0 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 500 µg/mL in PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
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. • 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA

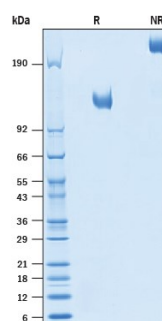
Bioactivity



Recombinant Rat Siglec-2/CD22 (µg/mL)

Recombinant Rat Siglec-2/CD22 Fc Chimera (Catalog # 10572-SL) supports the adhesion of human red blood cells. The ED₅₀ for this effect is 0.15-1.8 µg/mL.

SDS-PAGE



2 µg/lane of Recombinant Rat Siglec-2/CD22 Fc Chimera Protein (Catalog # 10572-SL) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 112-138 kDa and 220-280 kDa, respectively.

BACKGROUND

Sialic acid-binding immunoglobulin-like lectin 2 (Siglec-2), also known as B-cell receptor CD22 or B-lymphocyte cell adhesion molecule (BL-CAM), is a I-type (Ig-type) lectin belonging to the sialoadhesin subclass of the immunoglobulin superfamily (1). Fourteen human and nine mouse Siglecs have been characterized and are divided into 2 families: CD33 related and evolutionarily conserved (2, 3). The extracellular domain (ECD) of Siglecs are characterized by an N-terminal Ig-like V-type domain, which mediates sialic acid binding, followed by varying numbers of Ig-like C2-type domains (1-3). The predominant form of human Siglec-2 contains a N-terminal Ig-like V-type domain, six Ig-like C2-type domains, a transmembrane region and a cytoplasmic tail with six tyrosine residues and four immunoreceptor tyrosine-based inhibition motifs (ITIMs) (1-3). A variant form of Siglec-2 missing two Ig-like C2-type domains along with a truncated cytoplasmic tail has also been identified (4). The mature ECD of rat Siglec-2 shares 58% and 76% amino acid sequence identity with human and mouse Siglec-2, respectively. Siglec-2 is an adhesion molecule that preferentially binds alpha 2,6- linked sialic acid on the same (cis) or adjacent (trans) cells (5). Besides its role as an adhesion molecule, Siglec-2 is a coreceptor that physically interacts with B-cell receptor (BCR), negatively regulating BCR signals by recruiting tyrosine phosphatase SHP-1 to its ITIMs. Phosphorylated Siglec-2 can also interact with other intracellular effector proteins such as Syk, PLC gamma, PI3 kinase and Grb-2, suggesting it may play a role in positive signaling (2). Another function of Siglec-2 is that it mediates the anti-phagocytic effect of alpha 2,6-linked sialic acid, and inhibition of Siglec-2 promotes the clearance of myelin debris, amyloid-beta oligomers and alpha -synuclein fibrils in vivo (6). Siglec-2 also plays a role in autoimmunity and has great potential for Siglec-2-based immunotherapeutics for the treatment of autoimmune diseases such as systemic lupus erythematosus (SLE) (7).

References:

1. Sato, S. *et al.* (1996) *Immunity*. **5**:551.
2. Crocker, P.R. and A. Varki (2001) *Trends Immunol.* **22**:337.
3. Macauley, M.S. *et al.* (2014) *Nature Rev Imm.* **14**:653.
4. Stamenkovic, I. and B. Seed (1990) *Nature* **345**:74.
5. Collins, B.E. *et al.* (2004) *Proc. Natl. Acad. Sci.* **101**:6104.
6. Pluvinaige, J.V. *et al.* (2019) *Nature*. **568**:7751.
7. Clark, E.A. *et al.* (2018) *Front Immunol.* **9**:2235.