

DESCRIPTION

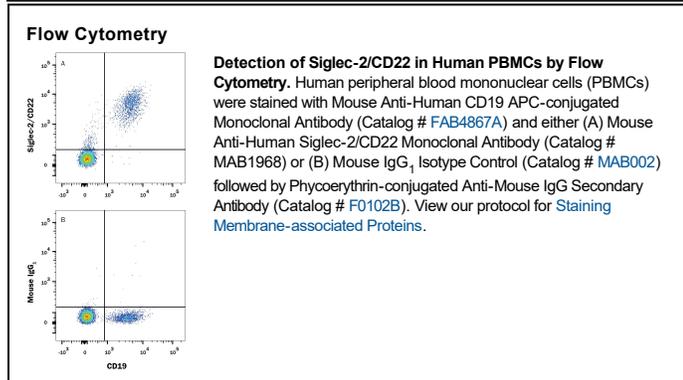
Species Reactivity	Human
Specificity	Detects human Siglec-2 in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant human Siglec-3, -5, -7, -9, -10, -F, or recombinant mouse Siglec-11 is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 219934
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Siglec-2/CD22 Asp20-Arg687 Accession # CAA42006
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
Flow Cytometry	0.25 µg/10 ⁶ cells	See Below
Immunocytochemistry	8-25 µg/mL	Immersion fixed human peripheral blood mononuclear cells
CyTOF-ready	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 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

Siglecs (Sialic acid binding Ig-like Lectins) are I-type (Ig-type) lectins belonging to the Ig superfamily. They are characterized by an N-terminal V-type Ig-like domain which mediates sialic acid binding, followed by varying numbers of C2-type Ig-like domains (1, 2). Fourteen human Siglecs have been cloned and characterized. They are Sialoadhesin/CD169/Siglec-1, CD22/Siglec-2, CD33/Siglec-3, Myelin-Associated Glycoprotein (MAG/Siglec-4a), and the identified Siglecs 5 to 11, plus 14 to 16 (1-3). To date, no Siglec has been shown to recognize any cell surface ligand other than sialic acid, suggesting that interactions with glycans containing this carbohydrate are important in mediating the biological functions of Siglecs. Human Siglec-2, also known as B-cell antigen CD22 or B lymphocyte cell adhesion molecule (BL-CAM), is a B cell restricted glycoprotein that is expressed in the cytoplasm of progenitor B and pre-B cells and on the surface of mature B cells and intestinal eosinophils (3,4). Two distinct human Siglec-2/CD22 cDNAs that arise from differential RNA processing of the same gene have been isolated. The predominant Siglec-2/CD22 β encodes an 847 amino acid (aa) polypeptide with a hydrophobic signal peptide, an V-type N-terminal Ig-like domain, six C2-type Ig-like domains, a transmembrane region and a cytoplasmic tail with 4 immunoreceptor tyrosine-based inhibition motifs (ITIMs) (5). The variant Siglec-2/CD22 α encodes a 647 aa polypeptide missing two C2-type Ig-like domains and has a truncated (23 aa) cytoplasmic tail (6). Siglec-2/CD22 is an adhesion molecule that preferentially binds α 2,6- linked sialic acid on the same (cis) or adjacent (trans) cells. Besides its role as an adhesion molecule, Siglec-2/CD22 is a coreceptor that physically interacts with B cell receptor (BCR) and is rapidly phosphorylated upon BCR ligation (3). It negatively regulates BCR signals by recruiting tyrosine phosphatase SHP-1 to its ITIMs, likely within large oligomeric complexes. Over aa 20-687, human and mouse share 59% aa sequence identity.

References:

1. Magesh, S. *et al.* (2011) *Curr. Med. Chem.* **18**:3537.
2. Bocher, B.S. and N. Zimmermann (2015) *J. Allergy Clin. Immunol.* **135**:598.
3. Nitschke, L. (2014) *Glycobiology* **24**:807.
4. Wen, T. *et al.* (2012) *J. Immunol.* **188**:1075.
5. Wilson, G.L *et al.* (1991) *J. Exp. Med.* **173**:137.
6. Stamenkovic, I. and B. Seed (1990) *Nature* **345**:74.