

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human CLEC-2 in direct ELISAs and Western blots. In direct ELISAs, does not cross-react with recombinant human CLEC-1.
Source	Monoclonal Mouse IgG _{2A} Clone # 219133
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	NS0-derived rhCLEC-2 extracellular domain Gln58-Pro229 Accession # AAF36777
Conjugate	Allophycocyanin Excitation Wavelength: 620-650 nm Emission Wavelength: 660-670 nm
Formulation	Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *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.

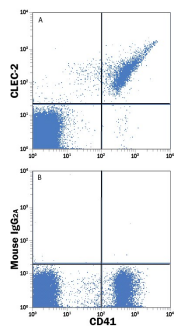
APPLICATIONS

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.

	Recommended Concentration	Sample
Flow Cytometry	10 μ L/10 ⁶ cells	See Below

DATA

Flow Cytometry



Detection of CLEC-2 in Human Whole Blood by Flow Cytometry. Human whole blood was stained with Mouse Anti-Human CD41 FITC-conjugated Monoclonal Antibody and either (A) Mouse Anti-Human CLEC-2 APC-conjugated Monoclonal Antibody (Catalog # FAB1718A) or (B) Mouse IgG_{2A} Allophycocyanin Isotype Control (Catalog # IC0041A). View our protocol for [Staining Membrane-associated Proteins](#).

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. <ul style="list-style-type: none"> 12 months from date of receipt, 2 to 8 °C as supplied.

BACKGROUND

C-type lectin-like receptor 2 (CLEC-2) is a 32 kDa type II transmembrane glycoprotein and member of the C-type lectin-like family of receptors (1-4). CLEC-2 consists of a 33 amino acid (aa) cytoplasmic domain, a 21 aa transmembrane region, and a 175 aa extracellular domain. The cytoplasmic domain contains multiple threonine and serine residues which are sites of potential phosphorylation, and a YXXL (Tyr-Xaa-Xaa-Leu) motif through which CLEC-2 does its signaling (2, 4-5). Ligand binding and cross-linking of CLEC-2 induces Src kinase-dependent tyrosine phosphorylation of the YXXL sequence, inducing activation of the tyrosine kinase Syk and initiation of a signaling pathway that culminates in activation of phospholipase C γ 2 (2, 5). The extracellular domain contains three potential sites of N-linked glycosylation, and a single carbohydrate recognition domain (CRD) which shows conservation of six cysteine residues (1, 6). Unlike most other members of the C-type lectin-like family of receptors, CLEC-2's CRD lacks the amino acid residues that are crucial for Ca²⁺-dependent carbohydrate binding, making it a non-classical C-type lectin receptor (1, 6). A splicing variant at aa 22-55 produces two isoforms for CLEC-2. Isoform 1 is the longer protein, and in isoform 2, an alanine residue is substituted for aa 22-55. Human CLEC-2 shares 63% aa sequence identity with mouse CLEC-2. CLEC-2 is expressed preferentially in liver, and is also detected in myeloid cells (monocytes, dendritic cells, and granulocytes) (1), platelets, and megakaryocytes (4). CLEC-2 is the receptor for the platelet-aggregating snake venom protein rhodocytin (3-4) and the molecule podoplanin, a transmembrane sialoglycoprotein that, when bound to CLEC-2, is involved in platelet aggregation, tumor metastasis, and lymphatic vessel formation (2, 7). CLEC-2 has also been shown to enhance infectivity of HIV-1 by mediating HIV-1 attachment and transfer by CLEC-2 transfected cells and platelets (8).

References:

1. Colonna, M. *et al.* (2000) *Eur. J. Immunol.* **30**:697.
2. Christou, C.M. *et al.* (2008) *Biochem. J.* **411**:133.
3. Watson, A.A. *et al.* (2007) *J. Biol. Chem.* **282**:3165.
4. Suzuki-Inoue, K. *et al.* (2006) *Blood* **107**:542.
5. Fuller, G.L. *et al.* (2007) *J. Biol. Chem.* **282**:12397.
6. Weis, W.I. *et al.* (1998) *Immunol. Rev.* **163**:19.
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8. Chaipan, C. *et al.* (2006) *J. Virol.* **80**:8951.