

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human CLEC9a in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant human (rh) CLEC1, 2, 2A, 3B, 10A, 12B, 14A, rhCD302/CLEC13a, rhMICL, or recombinant mouse CLEC9a is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 683409
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human CLEC9a Lys57-Val241 Accession # Q6UXN8
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
Flow Cytometry	2.5 µg/10 ⁶ cells	Human HEK293 cells transfected with human CLEC9a.
CyTOF-ready	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	
Binding Inhibition	The binding of Recombinant Human CLEC9a Fc Chimera (Catalog # 6049-CL, 100 ng/mL) to a ligand expressed in Fas Ligand-treated A20 mouse B cell lymphoma cell line was maximally inhibited (>90%) by 1 µg/mL of the antibody, as detected by flow cytometry.	

PREPARATION AND STORAGE

Reconstitution	Sterile PBS to a final concentration of 0.5 mg/mL.
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

CLEC9a (C-type lectin domain family 9 member A), also known as DNGR-1, is a type II transmembrane glycoprotein member of the C-type lectin superfamily (1, 2). Mature human CLEC9a consists of a 35 amino acid (aa) cytoplasmic domain with an ITAM-like motif, a 21 aa transmembrane segment, and a 185 extracellular domain (ECD) that contains a stalk region and one C-type lectin domain (CTLD) (3-5). Within the ECD, human CLEC9a shares 57% aa sequence identity with mouse and rat CLEC9a. Although the CTLD of CLEC9a structurally resembles that of other C-type lectins, it lacks the conserved residues that typically mediate calcium and carbohydrate binding. CLEC9a is expressed as a disulfide-linked homodimer of approximately 50 kDa N-glycosylated subunits (3, 5). Human CLEC9a expression is restricted to a subpopulation of BDCA-3⁺ conventional dendritic cells (cDC) and CD16⁺ monocytes (3-5). BDCA-3⁺ cDC are analogous to mouse CD8⁺ cDC which are specialized in antigenic cross-presentation in complex with MHC class I molecules (6). In mouse, CLEC9a is additionally expressed on plasmacytoid dendritic cells (4, 5). CLEC9a ligation enhances antigen uptake and processing, leading to presentation on MHC class I and cytotoxic T cell (CTL) priming (3-5). In mouse, CLEC9a recognizes normally intracellular determinant(s) of necrotic cells and mediates their uptake by the dendritic cell (7). The subsequent antigenic cross-presentation to CTL is important for clearing necrotic cellular debris (7). CLEC9a signaling triggers activation of the tyrosine kinase Syk (3, 7).

References:

1. Huysamen, C. and G.D. Brown (2009) *FEMS Microbiol. Lett.* **290**:121.
2. Geijtenbeek, T.B.H. *et al.* (2004) *Annu. Rev. Immunol.* **22**:33.
3. Huysamen, C. *et al.* (2008) *J. Biol. Chem.* **283**:16693.
4. Caminschi, I. *et al.* (2008) *Blood* **112**:3264.
5. Sancho, D. *et al.* (2008) *J. Clin. Invest.* **118**:2098.
6. Dudziak, D. *et al.* (2007) *Science* **315**:107.
7. Sancho, D. *et al.* (2009) *Nature* **458**:899.