

DESCRIPTION

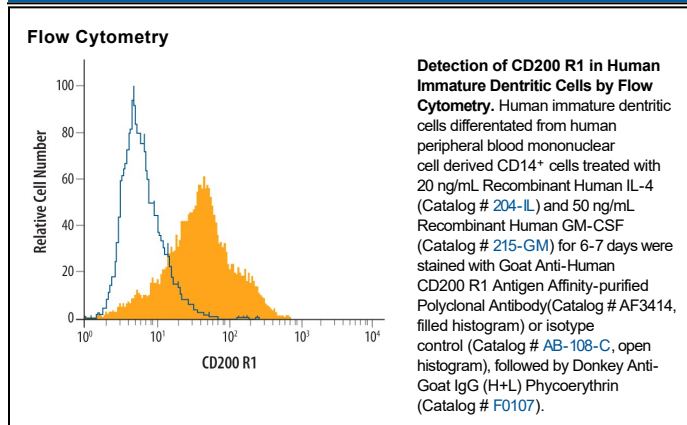
Species Reactivity	Human
Specificity	Detects human CD200 R1 in direct ELISAs and Western blots. In direct ELISAs and Western blots, less than 5% cross-reactivity with recombinant mouse CD200 R1 is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human CD200 R1 Ala27-Leu266 Accession # AAQ19772
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 Human CD200 R1 Fc Chimera (Catalog # 3414-CD)
Flow Cytometry	0.25 µg/10 ⁶ cells	See Below
CyTOF-ready	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	
Blockade of Receptor-ligand Interaction	In a functional ELISA, 3-15 µg/mL of this antibody will block 50% of the binding of 2.5 ng/mL of Recombinant Human CD200 Fc Chimera (Catalog # 2724-CD) to immobilized Recombinant Human CD200 R1 Fc Chimera coated at 2 µg/mL (100 µL/well). At 50 µg/mL, this antibody will block >90% of the binding.	

DATA



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

CD200 R1, also known as OX-2 receptor, is a 90 kDa transmembrane protein in the immunoglobulin superfamily (1-3). The standard human CD200 R1 cDNA encodes a 325 amino acid (aa) precursor that includes a 28 aa signal sequence, a 215 aa extracellular domain (ECD), a 21 aa transmembrane segment, and a 61 aa cytoplasmic domain. The ECD is composed of one Ig-like V-type domain and one Ig-like C2-type domain (4). Within the ECD, human CD200 R1 shares 56% aa sequence identity with mouse and rat CD200 R1. Alternate splicing of the human CD200 R1 mRNA generates four isoforms, two of which are truncated in the Ig-C2 domain and are likely secreted. The protein expressed here contains a mature region that is identical to that of the standard form. There is an N-terminal extension of 25 aa that, in the standard form, is part of the signal sequence. In human, a separate CD200 R12 gene encodes a protein that shares 81% ECD aa identity with CD200 R11. In mouse, at least four genes for CD200 R1-like molecules have been described (4 - 6). CD200 R1 expression is restricted primarily to mast cells, basophils, macrophages, and dendritic cells (7-9), while its ligand, CD200, is widely distributed (10). Disruption of this receptor-ligand system by knockout of the CD200 gene in mice leads to increased macrophage number and activation and predisposition to autoimmune disorders (11). Association of CD200 with CD200 R1 takes place between their respective N-terminal Ig-like domains (12). The capacity of CD200 R1-like molecules to interact with CD200 is controversial (5, 13). CD200 R1 propagates inhibitory signals despite its lacking a cytoplasmic ITIM (immunoreceptor tyrosine-based inhibitory motif) (8, 9, 14, 15) CD200 R1-like molecules, in contrast, are potentially activating receptors by means of their association with DAP12 (4, 6).

References:

1. Rosenblum, M.D. *et al.* (2006) *J. Dermatol. Sci.* **41**:165.
2. Gorczynski, R.M. (2005) *Curr. Opin. Invest. Drugs* **6**:483.
3. Barclay, A.N. *et al.* (2002) *Trends Immunol.* **23**:285.
4. Wright, G.J. *et al.* (2003) *J. Immunol.* **171**:3034.
5. Hatherley, D. *et al.* (2005) *J. Immunol.* **175**:2469.
6. Voehringer, D. *et al.* (2004) *J. Biol. Chem.* **279**:54117.
7. Shiratori, I. *et al.* (2005) *J. Immunol.* **175**:4441.
8. Cherwinski, H.M. *et al.* (2005) *J. Immunol.* **174**:1348.
9. Fallarino, F. *et al.* (2004) *J. Immunol.* **173**:3748.
10. Wright, G.J. *et al.* (2001) *Immunology* **102**:173.
11. Hoek, R.M. *et al.* (2000) *Science* **290**:1768.
12. Hatherley, D. and A.N. Barclay (2004) *Eur. J. Immunol.* **34**:1688.
13. Gorczynski, R. *et al.* (2004) *J. Immunol.* **172**:7744.
14. Jenmalm, M.C. *et al.* (2006) *J. Immunol.* **176**:191.
15. Zhang, S. *et al.* (2004) *J. Immunol.* **173**:6786.