

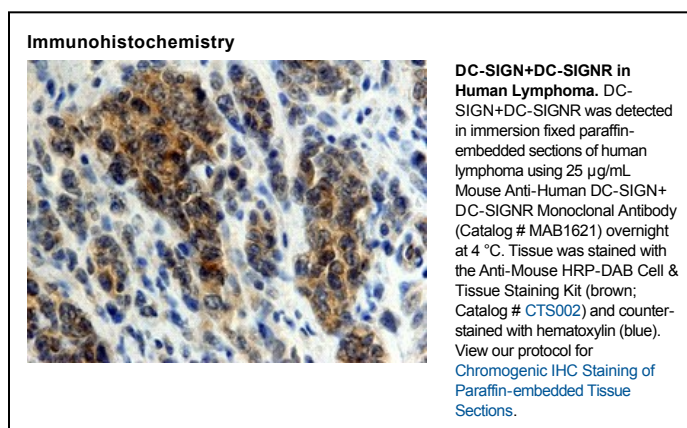
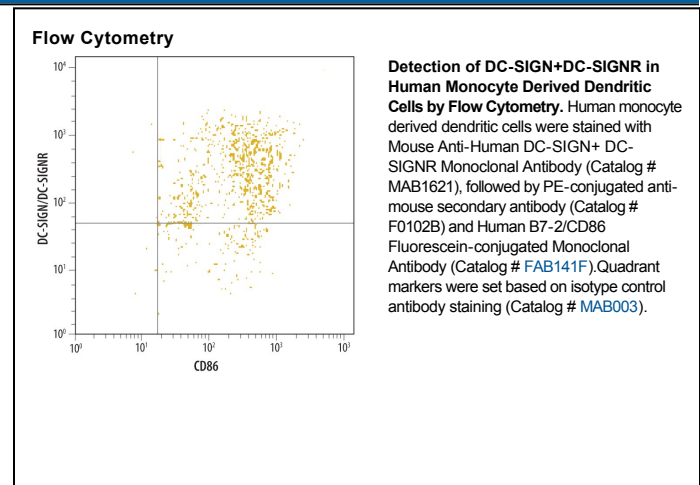
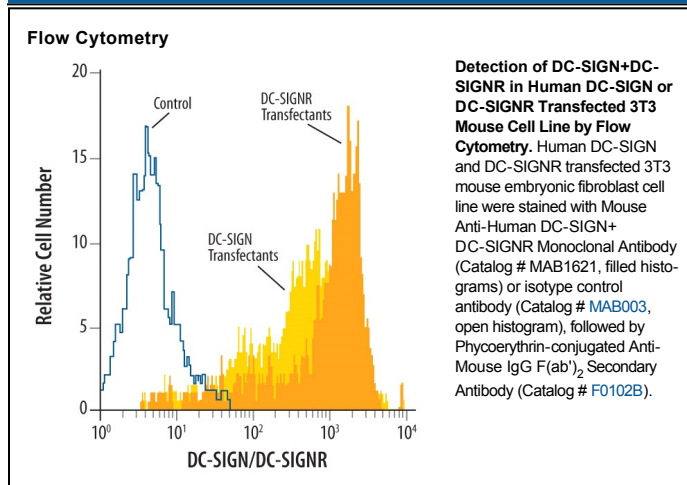
| DESCRIPTION | |
|---------------------------|---|
| Species Reactivity | Human |
| Specificity | Recognizes both human DC-SIGN and human DC-SIGNR on transfected cells. Does not react with parental mouse cells or irrelevant transfectants. |
| Source | Monoclonal Mouse IgG _{2A} Clone # 120612 |
| Purification | Protein A or G purified from hybridoma culture supernatant |
| Immunogen | NIH-3T3 mouse embryonic fibroblast cell line transfected with human DC-SIGNR Accession # Q9H2X3 |
| 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 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 | See Below |
| Immunohistochemistry | 8-25 µg/mL | See Below |
| 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

DC-SIGN (Dendritic Cell- Specific ICAM-3 Grabbing Non-Integrin) has been shown to play an important role in regulating dendritic cell (DC) and T cell interactions, including antigen presentation to T cells and enhancement of transinfection of CD4+ T cells by HIV-1 (1, 2). Efforts to identify additional type II membrane proteins resulted in the isolation of a molecule related in sequence to DC-SIGN known as DC-SIGNR (DC-SIGN Related) (3, 4). DC-SIGNR shares 73 - 80% amino acid homology with DC-SIGN and is located on human chromosome 19p13.3. Its structure is similar to DC-SIGN and therefore binds mannose residues in a calcium dependent fashion, including ICAM-3 and HIV-1 gp120 (5). DC-SIGNR, also known as L-SIGN (Liver/Lymph node-Specific ICAM-3-Grabbing Non-integrin) and DC-SIGNR, is polymorphic since allelic variations of the exon 4 encoded sequence have been isolated (5). This is further supported by a study demonstrating the ability to isolate a large repertoire of DC-SIGNR transcripts largely the result of alternative splicing of the 7 coding exons (6). L-SIGN/DC-SIGNR is primarily transcribed in the liver and lymph nodes but not in monocyte derived DC (5). Expression of L-SIGN/DC-SIGNR is restricted to endothelial cells derived from liver sinusoids, lymph nodes sinuses and capillaries (7) although variable expression in placenta and some monocytic cell lines has also been reported, including both membrane and soluble isoforms of the protein (6). Expression of DC-SIGN is induced during the in-vitro generation of DC from either monocytes or bone marrow progenitors, with maximal surface expression at day 7 of culture (1). Immature DC in the skin and mature DC in the tonsil have been demonstrated to express DC-SIGN (8). Analysis of various tissues and cell lines suggests that DC-SIGN expression is restricted to DC (1) although a more recent report finds evidence of expression in placenta, resting monocytes and monocytic cell lines (6). This discrepancy may be partially related to the multiple isoforms of DC-SIGN transcripts, including both membrane and soluble forms, as well as exon splice variants reported in the latter study (6).

References:

1. Geijtenbeek, T.B.H. *et al.* (2000) *Cell* **100**:575.
2. Geijtenbeek, T.B.H. *et al.* (2000) *Cell* **100**:587.
3. Yokoyama-Kobayashi, M.T. *et al.* (1999) *Gene* **228**:161.
4. Soilleux, E.J. *et al.* (2000) *J. Immunol.* **165**:2937.
5. Bashirova, A.A. *et al.* (2001) *J. Exp. Med.* **193**:671.
6. Mummidi, S. *et al.* (2001) *J. Biol. Chem.* **276**:33196.
7. Pohlman, S. *et al.* (2001) *Proc. Natl. Acad. Sci. USA* **98**:2670.
8. Geijtenbeek, T.B.H. *et al.* (2000) *Nature Immunol.* **1**:353.