

DESCRIPTION

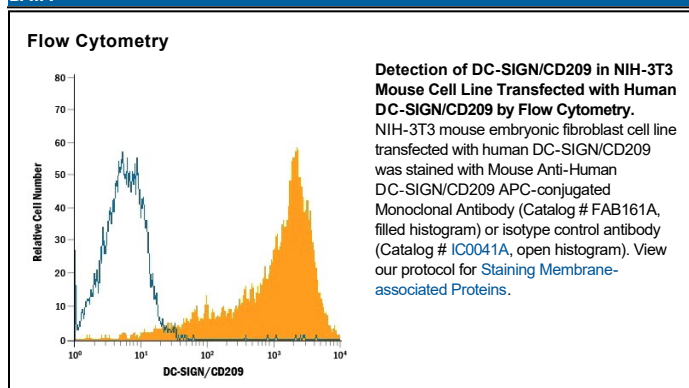
Species Reactivity	Human
Specificity	Detects human DC-SIGN/CD209 on transfected NIH/3T3 cells and on monocyte derived dendritic cells. Does not react with parental mouse cells or irrelevant transfectants, such as human DC-SIGN2.
Source	Monoclonal Mouse IgG _{2B} Clone # 120507
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	NIH-3T3 mouse embryonic fibroblast cell line transfected with human DC-SIGN/CD209
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 µL/10 ⁶ cells	See Below

DATA



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. ● 12 months from date of receipt, 2 to 8 °C as supplied.

BACKGROUND

Human DC-SIGN (Dendritic Cell-Specific ICAM-3 Grabbing Non-integrin) also known as CD209 is a member of the chromosome 19 C-type lectin family that includes DC-SIGN, DC-SIGN-related protein, CD23 and LSECtin (1). DC-SIGN was initially reported to be a 46 kDa, 404 amino acid (aa) type II transmembrane protein that contained a 40 aa cytoplasmic N-terminus, a 21 aa transmembrane segment, and a 343 aa extracellular C-terminus (2). The extracellular region contains a distal, 115 aa Ca⁺⁺-dependent carbohydrate-binding lectin domain and a membrane-proximal linker segment that is composed of seven 23 aa repeats (2, 3). The lectin domain is believed to preferably bind mannose, either within the context of ICAM-3 (on T cells) or ICAM-2 (on endothelial cells) (2, 4, 5). DC-SIGN expression appears to be limited to dendritic cells (DC) and macrophages (6), and DC interaction with the ICAMs both aids DC cell trafficking and immunological synapse formation (7). Since the original report on DC-SIGN, multiple splice forms have been discovered, generating both membrane-bound and soluble forms (3). There are eight type A isoforms, all of which begin with the same 15 aa of exon 1a. Four contain the transmembrane region of exon II, and four do not (*i.e.*, are soluble). Among these eight type A isoforms, only three retain the entire 343 aa found in the full length form described in reference #2 (the full length form is referred to as Type I mDC-SIGN1A) (3). Five additional isoforms utilize an alternate start site, and these are referred to as type B isoforms. These all show a 35 aa cytoplasmic domain. One also has a transmembrane segment; four do not. Two of the five contain full, unspliced extracellular regions (3). All of this suggests enormous complexity in DC-SIGN biology. DC-SIGN is not well conserved across species. Human and mouse show little overall aa identity. In the lectin domain, however, human DC-SIGN shares 68% aa identity with mouse DC-SIGN (8). Human and rhesus monkey DC-SIGN share 91% aa identity over the entire extracellular region (8). A detailed description of the additional properties of this monoclonal antibody (MAB161) have been published (9, 10).

References:

1. Liu, W. *et al.* (2004) J. Biol. Chem. **279**:18748.
2. Curtis, B.M. *et al.* (1992) Proc. Natl. Acad. Sci. USA **89**:8356.
3. Mummidi, S. *et al.* (2001) J. Biol. Chem. **276**:33196.
4. Su, S.V. *et al.* (2004) J. Biol. Chem. **279**:19122.
5. Cambi, A. *et al.* (2005) Cell. Microbiol. **7**:481.
6. Serrano-Gomez, D. *et al.* (2004) J. Immunol. **173**:5635.
7. Geijtenbeek, T.B.H. and Y. van Kooyk (2003) Curr. Top. Microbiol. Immunol. **276**:32.
8. Baribaud, F. *et al.* (2001) J. Virol. **75**:10281.
9. Wu, L. *et al.* (2002) J. Virol. **76**:5905.
10. Baribaud, F. *et al.* (2002) J. Virol. **76**:9135.