

DESCRIPTION

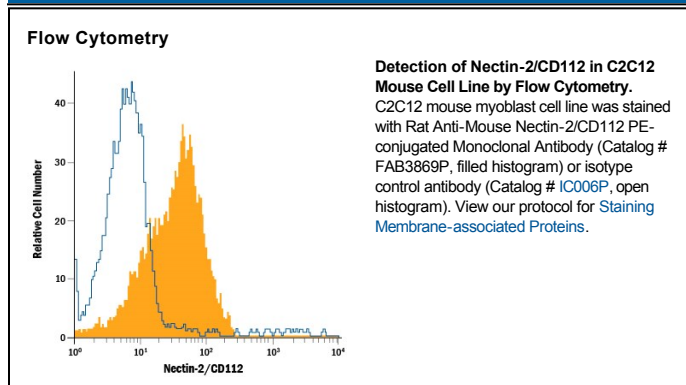
Species Reactivity	Mouse
Specificity	Detects mouse Nectin-2/CD112 in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant human Nectin-2 or recombinant mouse CD155/PVR is observed.
Source	Monoclonal Rat IgG _{2A} Clone # 829038
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse Nectin-2/CD112 Gln32-Gly351 (predicted) Accession # P32507
Conjugate	Phycoerythrin Excitation Wavelength: 488 nm Emission Wavelength: 565-605 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



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

Nectins are a small family of Ca⁺⁺-independent immunoglobulin (Ig)-like Cell Adhesion Molecules (CAMs) that control cell adhesion, proliferation, and migration (1, 2, 3). The name Nectin derives from the Latin word *necto*, which means "to connect". The Nectin family contains four members (Nectin-1 to -4), all of which show alternate splicing, a transmembrane (TM) region (except for Nectin-1 γ which is secreted), and three extracellular Ig-domains. Nectins are highly homologous to the human receptor for poliovirus, and as such have been given the alternate name of poliovirus receptor-related proteins. They do not, however, appear to bind poliovirus (1). Mouse Nectin-2 is a 70 to 78 kDa type I TM glycoprotein that is found on a variety of cell types (4, 5). It has two splice forms (4, 6, 7). Nectin-2 α /PRR2 is a 65 kDa short form and is synthesized as a 467 amino acid precursor. It contains a 31 aa signal sequence, a 315 aa extracellular domain (ECD), a 28 aa TM segment, and a 93 aa cytoplasmic region. The ECD contains one N-terminal V-type Ig domain and two 85-95 aa C2-type Ig-like domains (aa 153-337) (8). The V-domain is believed to mediate Nectin binding to its ligands (9). A long, 78 kDa, 530 aa isoform of mouse Nectin-2 (Nectin-2 δ) also exists. It has the same signal sequence and extracellular domain as Nectin-2 α (aa 1-338), but differs in the TM segment (21 aa in length) and cytoplasmic region (159 aa in length) (4, 6, 7). Mouse Nectin-2 ECD (aa 32-338) shares 72%, 77% and 95% aa identity with the ECD in human, canine and rat Nectin-2, respectively. Nectin-2 is known to bind pseudorabies virus, and herpes simplex virus-2 (HSV-2). It also binds select HSV-1 strains. It does not bind poliovirus (1, 10, 11). As a cell adhesion molecule, Nectin-2 will form cis-homodimers (same cell) and trans-homodimers (across cells). Nectin-2 will not cis-dimerize with other Nectins, but will trans-heterodimerize with Nectin-3 and CD266/DNAM-1 (1, 3, 11, 12, 13). Nectin-2 is found concentrated at cell-to-cell interfaces, and is presumed to contribute to tight and adherens junction formation (14). Through its interaction with NK and T cell expressed DNAM-1, it also promotes lymphocyte cytotoxicity and cytokine secretion against both tumors and dendritic cells (DC) expressing Nectin-2 (15, 16). In the case of DC, this may be a mechanism whereby the immune system eliminates DC that are inefficient at antigen presentation. Nectin-2 is expressed on epithelium, endothelial cells, Sertoli cells, monocytes, dendritic cells, granulosa cells, mast cells, eosinophils and fibroblasts.

References:

1. Takai, Y. and H. Nakanishi (2003) *J. Cell Sci.* **116**:17.
2. Rikitake, Y. and Y. Takai (2008) *Cell. Mol. Life Sci.* **65**:253.
3. Sasisaka, T. *et al.* (2007) *Curr. Opin. Cell Biol.* **19**:1.
4. Aoki, J. *et al.* (1994) *J. Biol. Chem.* **269**:8431.
5. Takahashi, K. *et al.* (1999) *J. Cell Biol.* **145**:539.
6. Aoki, J. *et al.* (1997) *Exp. Cell Res.* **235**:374.
7. Lopez, M. *et al.* (1998) *Blood* **92**:4602.
8. Morrison, M.E. and V.R. Racaniello (1992) *J. Virol.* **66**:2807.
9. Struyf, F. *et al.* (2002) *J. Virol.* **76**:12940.
10. Delboy, M.G. *et al.* (2006) *Virology J.* **3**:105.
11. Irie, K. *et al.* (2004) *Semin. Cell Dev. Biol.* **15**:643.
12. Tahara-Hanaoka, S. *et al.* (2004) *Int. Immunol.* **16**:533.
13. Satoh-Horikawa, K. *et al.* (2000) *J. Biol. Chem.* **275**:10291.
14. Nakanishi, H. and Y. Takai (2004) *Biol. Chem.* **385**:885.
15. Tahara-Hanaoka, S. *et al.* (2006) *Blood* **107**:1491.
16. Pende, D. *et al.* (2006) *Blood* **107**:2030.