

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Nectin-2/CD112 in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant human (rh) Nectin-1, rhCD155/PVR, or recombinant mouse Nectin-2/CD112 is observed.
Source	Monoclonal Mouse IgG _{2B} Clone # 610615
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Nectin-2/CD112 Gln32-Gly360 Accession # Q92692
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.

Neutralization	In a functional ELISA, 0.01-0.06 µg/mL of this antibody will block 50% of the binding of 0.5 µg/mL of Recombinant Human DNAM-1/CD226 Fc Chimera (Catalog # 666-DN) to immobilized Recombinant Human Nectin-2/CD112 Fc Chimera (Catalog # 9317-N2) coated at 0.5 µg/mL (100 µL/well).
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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

Nectins are a small family of Ca⁺⁺-independent immunoglobulin (Ig)-like Cell Adhesion Molecules (CAMs) that organize intercellular junctions (1). The Nectin family has at least four members (Nectin-1-4), all of which show alternate splicing (except for Nectin-4), a transmembrane (TM) region (except for Nectin-1γ), and three extracellular Ig-domains. Nectins are highly homologous to the human receptor for poliovirus, and as such have been alternately named poliovirus receptor-related proteins. They do not, however, appear to bind poliovirus (1). Nectin-2 is a 60 or 65 kDa type I TM glycoprotein that is found on a variety of cell types (2, 3). It has two splice forms (4, 5). Nectin-2δ is a 65 kDa long form and is synthesized as a 538 amino acid precursor. It contains a 31 amino acid (aa) signal sequence, a 329 aa extracellular region, a 21 aa TM segment, and a 157 aa cytoplasmic domain. The extracellular region contains one N-terminal 85 aa V-type Ig domain and two 45-55 aa C2-type Ig domains. The V-domain is believed to mediate Nectin binding to its ligands (6). The short, 60 kDa isoform of Nectin-2 (Nectin-2α) has the same signal sequence and extracellular domain as nectin-2δ, but differs in the TM and cytoplasmic region (4, 5). In this case, the cytoplasmic tail is only 94 aa in length. The human extracellular region shows 72% aa sequence identity with the equivalent region in mouse. Nectin-2 is known to bind the pseudorabies virus, and herpes simplex virus-2 (HSV-2), but not HSV-1. It does not bind poliovirus. As a cell adhesion molecule, Nectin-2 will form *cis*-homodimers (same cell), followed by trans-dimers (across cells). Nectin-2 will not *cis*-dimerize with other Nectins, but will *cis*-dimerize with its two splice forms. Notably, a Nectin-2 *cis*-dimer on one cell will heterodimerize with a Nectin-3 *cis*-dimer on another cell (1). Nectin-2 is found concentrated in adherens junctions, and exists on neurons, endothelial cells, epithelial cells and fibroblasts.

References:

1. Takai, Y. and H. Nakanishi, 2003. *J. Cell Sci.* **116**:17.
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3. Pende, D. *et al.* (2005) *Mol. Immunol.* **42**:463.
4. Eberle, F. *et al.* (1995) *Gene* **159**:267.
5. Warner, M.S. *et al.* (1998) *Virology* **246**:179.
6. Struyf, F. *et al.* (2002) *J. Virol.* **76**:12940.