

DESCRIPTION

Source Human embryonic kidney cell, HEK293-derived
Trp49-Leu174 (Gly136Asp), with an N-terminal HA tag
Accession # Q6UUVW9

N-terminal Sequence Analysis Tyr

Predicted Molecular Mass 16 kDa

SPECIFICATIONS

SDS-PAGE 19-30 kDa, reducing conditions

Activity Measured by its ability to induce IFN- γ secretion by mouse splenocytes.
The ED₅₀ for this effect is 0.4-2 μ g/mL.

Endotoxin Level <0.10 EU per 1 μ g of the protein by the LAL method.

Purity >95%, by SDS-PAGE with silver staining.

Formulation Lyophilized from a 0.2 μ m filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 μ g/mL in PBS.

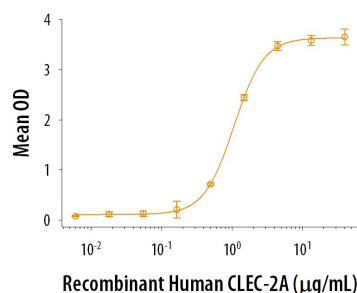
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

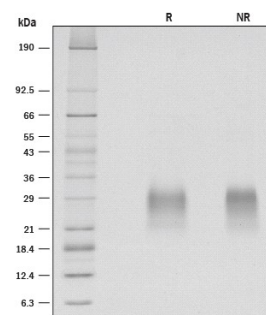
DATA

Bioactivity



Recombinant Human CLEC-2A (Catalog # 8435-CL) induces IFN- γ secretion by mouse splenocytes. The ED₅₀ for this effect is 0.4-2 μ g/mL.

SDS-PAGE



1 μ g/lane of Recombinant Human CLEC-2A was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by silver staining, showing R bands at 22.3, 28.4 kDa and NR bands at 21.8, 29.1 kDa.

BACKGROUND

CLEC-2A, also called keratinocyte-associated C-type lectin (KACL), is a type 2 transmembrane glycoprotein member of the C-type lectin-like receptor (CTLR) family. CLEC-2A is part of the subgroup of CLEC-2 proteins that also includes CLEC-2B/AICL, CLEC-2D/LLT, and CD69/CLEC-2C, all of which are encoded by the natural killer gene complex (NKG) (1, 2). CLEC-2A is composed of a 126 amino acid (aa) carboxy terminal extracellular C-type lectin-like domain (CTLD), a 21 aa transmembrane domain and a 27 aa amino terminal cytoplasmic domain. The CTLD folds into a compact structure with two α -helices and two antiparallel β -sheets stabilized by three conserved intramolecular disulfide bonds (3). CLEC-2 proteins act as ligands and interact with NKRP1 receptors which also are CTLR and encoded by the NKG. The crystal structure of the complex shows CLEC-2A as a non-disulfide linked homodimer that symmetrically binds two NKp65 monomers. Interaction occurs via the membrane-distal surface of the CTLD in a head-to-head orientation (4). Five key residues for CLEC-2A binding to NKp65 are conserved or conservatively substituted among CLEC-2 proteins. Thus, the CLEC-2A-NKp65 interaction is proposed as a model for all NKRP1-CLEC-2 complexes (4). Orthologs for NKp65 and CLEC-2A are present in chimpanzee, rhesus macaque and cow but have not been described in rodents (5). Whereas CLEC-2B/AICL, CLEC-2D/LLT, and CD69/CLEC-2C are generally expressed on hematopoietic cells, CLEC-2A expression is restricted to keratinocytes. In vitro, the NKp65-CLEC-2A interaction will trigger natural killer cell cytotoxicity and the release of proinflammatory cytokines (6). Thus, CLEC-2A facilitates NKp65-mediated immunosurveillance of keratinocytes (6).

References:

1. Spreu, J. *et al.* (2007) Immunogenetics **59**:903.
2. Yokohama, W.M. *et al.* (2003) Nat.Rev.Immunol. **3**:304.
3. Zelensky, A.N. *et al.* (2005) FEBS J. **272**:6179.
4. Li, Y. *et al.* (2013) Proc. Natl. Acad. Sci. USA **110**:11505.
5. Vogler, I. *et al.* (2011) J. Innate Immun. **3**:227.
6. Spreu, J. *et al.* (2010) Proc. Natl. Acad. Sci. USA **107**:5100.