Recombinant Human CD302/CLEC13A Fc Chimera

Catalog Number: 10203-CL

DESCRIPTION

Source

Human embryonic kidney cell, HEK293-derived human CD302/CLEC13A protein

Human CD302/CLEC13A (Asp23-Asn167) Accession # Q8IX05-1	IEGRMD	Human IgG_1 (Pro100-Lys330)

N-terminus C-terminus

N-terminal Sequence Analysis	Asp23
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	43 kDa

SPECIFICATIONS

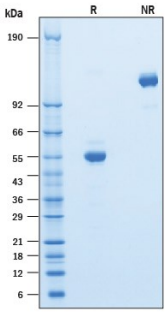
SDS-PAGE	51-59 kDa, under reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human CD302/CLEC13A Fc Chimer (Catalog # 10203-CL) is immobilized at 5 µg/mL, 100 µL/well, the concentration of Recombinant Human DEC-205/CD205 Fc Chimera (Catalog # 10205-DE) that produces 50% of the optimal binding response is 4-24 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>90%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 500 µg/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

SDS-PAGE



2 µg/lane of Recombinant Human CD302/CLEC13A Fc Chimera was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 51-59 kDa and 100-120 kDa, respectively.

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BACKGROUND

CD302, also known as CLEC13A and DCL1, is a type I transmembrane C-type lectin receptor. It was identified while cloning human DEC-205 and was termed DCL1 (DEC-205-associated C-type lectin-1) (1). In humans, the highest expression of CD302 transcripts was observed in the liver, followed by lungs, spleen, and myeloid PBMC populations including monocytes, granulocytes, and dendritic cells (DC) (2). Human CD302 is synthesized as a 232 amino acid (aa) protein that includes 22 aa signal peptide, a 146 aa extracellular domain (ECD), a 21 aa transmembrane segment, and a 43 aa cytoplasmic region. The extracellular domain is predicted to contain eight β strands and two α helices using NMR (3). Within the ECD, human CD302 shares 82% aa sequence identity with mouse and rat CD302. Unlike other classical C-type lectin receptors, CD302 is missing the known amino acid residues essential for calcium-dependent sugar binding, suggesting that CD302 may not have classic sugar binding capacity. However, CD302 did have the ability to behave as an endocytosis/phagocytosis receptor (1). In addition, CD302 was shown to colocalize with F-actin rich migratory structures, including filopodia, lamellipodia, and podosomes in macrophages, where CD302 may bind yet to be determined endothelial ligands involved in DC adhesion or migration (1, 2). Further evidence that CD302 is involved in regulating DC migration, includes that CD302 knockout mice had reduced frequency and numbers of migratory DC within the lymph nodes (LN) and reduced *in vivo* capacity to reach draining LN (2). CD302 was also found to exist as an intergenic splice variant able to form a fusion protein with DEC-205/CD205 in Hodgkin's lymphoma cell lines (4). The CD302/DEC-205 fusion protein was also found to be expressed by mature dendritic cells which altered endocytic capacity of DEC-205, although the wild-type single gene transcripts were the dominant isoforms expressed (5). Due to its selective expression in myeloid immune populations, CD302 has become a potential therapeutic target for acute myeloid leukemia (AML) (6).

References:

1. Kato, M. *et al.* (2007) J. Immunol. **179**:6052.
2. Lo, T-H. *et al.* (2016) J. Immunol. **197**:885.
3. Pospisilova, E. *et al.* (2016) Biomol. NMR. Assign. **10**:189.
4. Kato, M. *et al.* (2003) J Biol Chem. **278**:34035.
5. Butler, M. *et al.* (2017) J. Immunol. **120**:362.
6. Lo, T-H. *et al.* (2019) PLoS One. **14**:e0216368.

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