

DESCRIPTION

Source	Chinese Hamster Ovary cell line, CHO-derived human MICL/CLEC12A protein			
	MD	Human IgG ₁ (Pro100-Lys330)	IEGR	Human MICL/CLEC12A (Thr67-Ala265) Accession # AAI26292.1
	N-terminus		C-terminus	
	N-terminal Sequence Met			
	Analysis			
Structure / Form	Disulfide-linked homodimer			
Predicted Molecular Mass	50 kDa			

SPECIFICATIONS

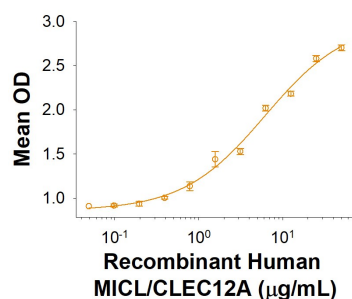
SDS-PAGE	70-85 kDa, under reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human Integrin alpha X beta 2 Protein (Catalog # 5755-AX) is immobilized at 5.00 µg/mL, 100 µL/well, the concentration of Recombinant Human MICL/CLEC12A Fc Chimera (Catalog# 10835-ML) that produces 50% of the optimal binding response is approximately 1.20-12.0 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 with Trehalose. 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. <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. • 3 months, -20 to -70 °C under sterile conditions after reconstitution.

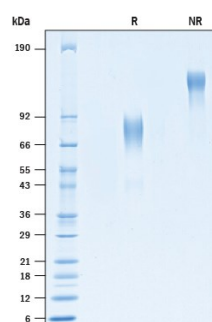
DATA

Binding Activity



Recombinant Human MICL/CLEC12A Fc Chimera Protein Binding Activity. When Recombinant Human Integrin alpha X beta 2 Protein (Catalog # 5755-AX) immobilized at 5.00 µg/mL, 100 µL/well, the concentration of Recombinant Human MICL/CLEC12A Fc Chimera (Catalog# 10835-ML) that produces 50% of the optimal binding response is approximately 1.20-12.0 µg/mL.

SDS-PAGE



Recombinant Human MICL/CLEC12A Fc Chimera Protein SDS-PAGE. 2 µg/lane of Recombinant Human MICL/CLEC12A Fc Chimera (Catalog # 10835-ML) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 70-85 kDa and 140-170 kDa, respectively.

BACKGROUND

C-type lectin domain family 12 member A (CLEC12A), also known as C-type lectin-like molecule 1 (CLL1), dendritic cell-associated lectin 2 (DCAL-2), myeloid inhibitory C-type lectin-like receptor (MICL), killer cell lectin like receptor-1 (KLRL1) and CD371, is a member of the C-type lectin receptor superfamily. Of the 17 groups in the superfamily, CLEC12A belongs to group V, which lacks the domain for calcium binding typically found in classical carbohydrate-binding CLECs (1). Mature CLEC12A consists of an extracellular domain (ECD) with a single C-type lectin-like domain, a type II transmembrane domain and a cytoplasmic tail containing an immunoreceptor tyrosine-based inhibition motif (ITIM) (3). Within the ECD, human CLEC12A shares 50% and 47% amino acid sequence identity with mouse and rat CLEC12A, respectively. Human CLEC12A differs from mouse CLEC12A in that it is heavily glycosylated and is found as a monomer rather than a dimer (2). CLEC12A is predominantly expressed in innate immune cells and plays a role in immunotherapy (1-6). CLEC12A preferentially associates with the protein tyrosine phosphatases SHP-1 and SHP-2 but not SHIP. Mechanistic studies with chimeric proteins have indicated that, similar to other ITIM-containing receptors, the cytoplasmic tail of CLEC12A can inhibit cellular activation upon ligand binding stimulation. The physiological functions of CLEC12A are poorly understood and the human ligand for CLEC12A is unknown. (7) Most CTL receptors require Ca²⁺ ions for binding (8). C-type lectins are the most diverse and prevalent lectin family in immunity. Particular interest has recently been attracted by the C-type lectin-like receptors on NK cells, which appear to regulate the activation/inhibitory balance of these cells, controlling cytotoxicity and cytokine production (9). CLEC12A receptor has emerged as a leukemia-associated and cancer stem cell marker in myeloid malignancies (10). CLEC12A is a myeloid lineage antigen that is highly expressed by AML cells and LSCs, but not expressed by normal hematopoietic stem cells (HSCs). The CLEC12A TriKE induced robust NK-cell specific proliferation, enhanced NK-cell activation, and killing of both AML cell lines and primary patient-derived AML blasts *in vitro* while sparing healthy HSCs. Additionally, the CLEC12A TriKE was able to reduce tumor burden in preclinical mouse models. These findings highlight the clinical potential of the CLEC12A TriKE for the effective treatment of AML (11).

References:

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