

Recombinant Human ICOS Ligand/ICOSL Protein (His Tag)

Catalog Number: PKSH031085

Note: Centrifuge before opening to ensure complete recovery of vial contents.

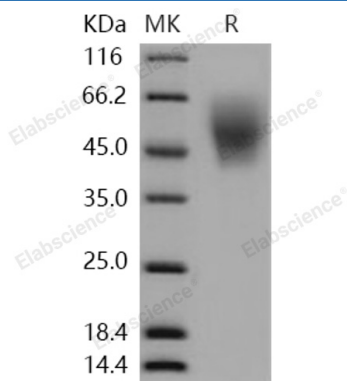
Description

Species	Human
Source	HEK293 Cells-derived Human ICOS Ligand/ICOSL protein Met 1-Ser 258, with an C-terminal His
Calculated MW	28.0 kDa
Observed MW	50-60 kDa
Accession	NP_056074.1
Bio-activity	Immobilized human B7-H2 at 1 µg/ml (100 µl/well) can bind human ICOS with a linear range of 1. 6-200 ng/ml.

Properties

Purity	> 98 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per µg of the protein as determined by the LAL method.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile PBS, pH 7.4 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 98 % as determined by reducing SDS-PAGE.

Background

Inducible co-stimulator ligand (ICOSL), also known as B7-H2, is a member of the B7 family of co-stimulatory molecules related to B7-1 and B7-2. It is a transmembrane glycoprotein with extracellular IgV and IgC domains, and binds to ICOS on activated T cells, thus delivers a positive costimulatory signal for optimal T cell function. The structural features of ICOSL are crucial for its costimulatory function. Present study shows that ICOSL displays a marked oligomerization potential, resembling more like B7-1 than B7-2. B7-H2-dependent signaling may play an active role in a proliferative response rather than in cytokine and chemokine production. The CD28/B7 and ICOS/B7-H2 pathways are both critical for costimulating T cell immune responses. Deficiency in either pathway results in defective T cell activation, cytokine production and germinal center formation.

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