

## Recombinant Human ICOSL Protein(His Tag)

Catalog Number: PDMH100324

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

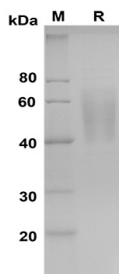
### Description

Species	Human
Source	Mammalian-derived Human ICOSL proteins Met1~Ser258, with an C-terminal His
Mol_Mass	28.3 kDa
Accession	O75144
Bio-activity	Not validated for activity

### Properties

Purity	> 90% as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU/mg 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 a 0.2 µm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

### Data



SDS-PAGE analysis of Human ICOSL proteins, 2 µg/lane of

Recombinant Human ICOSL proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 40-60 KD

### Background

### For Research Use Only

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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. The 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.