General Information

Gene Name Synonym:
B7-h2; B7H2; B7RP-1; B7RP1; CD275; GL50; ICOS ligand; ICOS-L; ICOSL; ICOSLG; LICOS

Protein Construction:
A DNA sequence encoding the human ICOSLG (NP_056074.1) (Met1-Ser258) was expressed with a C-terminal polyhistidine tag. The purified protein was biotinylated in vitro.

Source: Human
Expression Host: HEK293

QC Testing

Purity: > 95 % as determined by SDS-PAGE.

Endotoxin:
< 1.0 EU per μg protein as determined by the LAL method.

Stability:
Samples are stable for up to twelve months from date of receipt at -70 °C

Predicted N terminal: Asp 19

Molecular Mass:
The recombinant human ICOSLG consists of 251 amino acids and predicts a molecular mass of 28.1 kDa.

Formulation:
Lyophilized from sterile PBS, pH 7.4.

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:
Store it under sterile conditions at -20 °C to -80 °C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:
Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:

Protein Description

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.

References