Recombinant Human Fas Ligand/TNFSF6

Catalog Number: 126-FL

DESCRIPTION

Source	Chinese Hamster Ovary cell line, CHO-derived human Fas Ligand/TNFSF6 protein Pro134-Leu281, with an N-terminal Met and 6-His tag Accession # Q53ZZ1
N-terminal Sequence Analysis	Met
Predicted Molecular Mass	18 kDa

SPECIFICATIONS

SDS-PAGE	19 - 32 kDa, reducing conditions
Activity	Measured by its ability to induce apoptosis of Jurkat human acute T cell leukemia cells. The ED_{50} for this effect is 0.3-1.5 ng/mL in the presence of 10 µg/mL of a cross-linking antibody Mouse Anti-polyHistidine Monoclonal Antibody (Catalog # MAB050).
Endotoxin Level	<1.0 EU per 1 µg of the protein by the LAL method.
Purity	>95%, 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 BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.
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.

BACKGROUND

Fas Ligand (FasL), also known as CD178, CD95L, or TNFSF6, is a 40 kDa type II transmembrane member of the TNF superfamily of proteins. Its ability to induce apoptosis in target cells plays an important role in the development, homeostasis, and function of the immune system (1). Mature human Fas Ligand consists of a 179 amino acid (aa) extracellular domain (ECD), a 22 aa transmembrane segment, and a 80 aa cytoplasmic domain (2). Within the ECD, human Fas Ligand shares 81% and 78% aa sequence identity with mouse and rat Fas Ligand, respectively. Both mouse and human Fas Ligand are active on mouse and human cells (2, 3). Fas Ligand is expressed on the cell surface as a nondisulfide-linked homotrimer on activated CD4+ Th1 cells, CD8+ cytotoxic T cells, and NK cells (1). Fas Ligand binding to Fas/CD95 on an adjacent cell triggers apoptosis in the Fas-expressing cell (2, 4). Fas Ligand also binds DcR3 which is a soluble decoy receptor that interferes with Fas Ligand-induced apoptosis (5). Fas Ligand can be released from the cell surface by metalloproteinases as a 26 kDa soluble molecule which remains trimeric (6, 7). Shed Fas Ligand retains the ability to bind Fas, although its ability to trigger apoptosis is dramatically reduced (6, 7). In the absence of TGF-β, however, Fas Ligand/Fas interactions instead promote neutrophil-mediated inflammatory responses (3, 8). Fas Ligand itself transmits reverse signals that costimulate the proliferation of freshly antigen-stimulated T cells (9). Fas Ligand-induced apoptosis plays a central role in the development of immune tolerance and the maintance of immune privileged sites (10). This function is exploited by tumor cells which evade immune surveillance by upregulating Fas Ligand to kill tumor infiltrating lymphocytes (8, 11). In gld mice, a Fas Ligand point mutation is the cause of severe lymphoproliferation and systemic autoimmunity (12, 13).

References:

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