Recombinant Cynomolgus Monkey Fas/TNFRSF6/CD95 Fc Chimera

Catalog Number: 10755-FS

DESCRIPTION

Source

Chinese Hamster Ovary cell line, CHO-derived cynomolgus monkey Fas/TNFRSF6/CD95 protein

Cynomolgus Monkey Fas/TNFRSF6/CD95 (Gln26-Asp173) Accession # Q9TSN4.1	IEGRMD	Human IgG_1 (Pro100-Lys330)

N-terminus C-terminus

N-terminal Sequence Analysis	Gln26, inferred from deblocking revealing Val27
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	43 kDa

SPECIFICATIONS

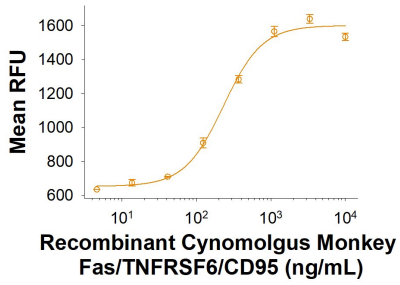
SDS-PAGE	55-70 kDa, under reducing conditions
Activity	Measured by its ability to inhibit Fas Ligand-induced apoptosis of Jurkat human acute T cell leukemia cells. Cheng, J. *et al.* (1994) Science **263**:1759. The ED_{50} for this effect is 40.0-240 ng/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein 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. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 500 µg/mL in PBS.
Shipping	The product is shipped with polar packs. 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.

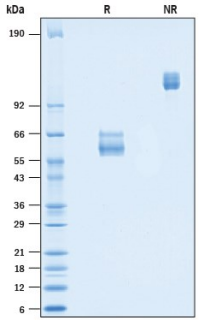
DATA

Bioactivity



Recombinant Cynomolgus Monkey Fas/TNFRSF6/CD95 Fc Chimera Bioactivity. Recombinant Cynomolgus Monkey Fas/TNFRSF6/CD95 Fc Chimera (Catalog # 10755-FS), inhibits Fas Ligand-induced apoptosis of Jurkat human acute T cell leukemia cells. The ED_{50} for this effect is 40.0-240 ng/mL.

SDS-PAGE



Recombinant Cynomolgus Monkey Fas/TNFRSF6/CD95 Fc Chimera SDS-PAGE. 2 µg/lane of Recombinant Cynomolgus Monkey Fas/TNFRSF6/CD95 Fc Chimera (Catalog # 10755-FS) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 55-70 kDa and 110-140 kDa, respectively.

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BACKGROUND

Fas (**fi**broblast **as**sociated; also known as APO-1 or CD95) is a member of the death receptor subfamily of the TNF receptor superfamily and is designated TNFRSF6 (1-3). The human Fas precursor is 335 amino acids (aa) in length, and contains a 25 aa signal peptide, a 148 aa extracellular domain (ECD), a 17 aa transmembrane sequence, and a 145 aa cytoplasmic region. The ECD possesses three cysteine-rich TNFR repeats, while the cytoplasmic region contains one death domain (DD) that is required for the transduction of apoptotic signals (4). Cynomolgus monkey Fas ECD shares 91% aa sequence identity with human Fas ECD. A human Fas isoform of 314 aa that lacks the transmembrane sequence is secreted by resting lymphocytes, while isoforms of 149, 132, 103 and 86 aa that also lack the DD and show substitutions for parts of the TNFR repeats are less prominently expressed (4-6). All five isoforms block the extrinsic apoptosis pathway induced by Fas ligand binding. Fas ligand (FasL; also TNFSF6) is a type II transmembrane protein that belongs to the TNF family and is expressed on activated T-cells, NK cells, and cells found in immune privileged sites. Alternatively, FasL is also shed as a soluble form (2, 6). Engagement of FasL induces oligomerization of preformed Fas trimers (1, 2). This activated receptor complex recruits the adaptor molecule FADD to form the Death-Inducing Signaling Complex (DISC). Upon activation, caspases in the DISC initiate the apoptotic signaling cascade (7). Fas is prominent in epithelial cells, hepatocytes, activated mature lymphocytes, virus-transformed lymphocytes and tumor cells. It is an essential mediator in the activation-induced death of T lymphocytes that terminates the immune reaction (1, 2, 8). In immune-privileged tissues, infiltrating Fas-bearing lymphocytes and inflammatory cells are killed by FasL engagement (9). Both humans and mice with genetic defects in Fas accumulate abnormal lymphocytes and develop systemic autoimmunity (1-3). The Fas pathway also appears to intersect with the BIM (mitochondrial/intrinsic) apoptosis pathway (1).

References:

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