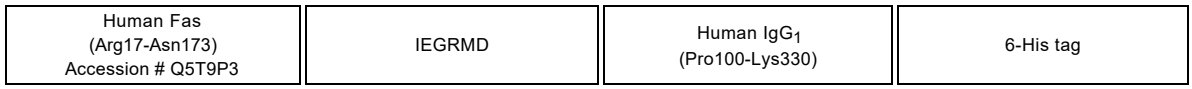
Recombinant Human Fas/TNFRSF6/CD95 Fc Chimera

Catalog Number: 326-FS

DESCRIPTION

Source

Mouse myeloma cell line, NS0-derived human Fas/TNFRSF6/CD95 protein



N-terminal Sequence Analysis	No results obtained: Arg17 predicted
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	45 kDa (monomer)

SPECIFICATIONS

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 0.01-0.04 µg/mL in the presence of 2 ng/mL recombinant human Fas Ligand.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>97%, by SDS-PAGE under reducing conditions and visualized by silver stain.
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, also known as APO-1 or CD95, belongs to the death receptor subfamily of the TNF receptor superfamily and is designated TNFRSF6 (1-3). The 335 amino acid (aa) human Fas includes a 25 aa signal peptide, a 148 aa extracellular domain (ECD) with three cysteine-rich TNFR repeats, a 17 aa transmembrane sequence, and a 145 aa cytoplasmic domain containing a death domain (DD), which is required for transducing apoptotic signals (4). Mature human Fas ECD shares 55%, 58%, 62%, 63%, and 64% aa sequence identity with mouse, rat, feline, bovine and porcine Fas, respectively. 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 appear to block the extrinsic apoptosis pathway induced by the Fas ligand (FasL, TNFSF6), a type II transmembrane protein of the TNF family that can be expressed on activated T-lymphocytes, NK cells and cells in immune privileged sites, or shed in soluble form (2, 6). Engagement of FAS induces oligomerization of preformed Fas trimers (1, 2). The activated receptor 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 cross-communicate with the BIM (mitochondrial/intrinsic) apoptosis pathway (1).

References:

1. Bouillet, P. and L.A. O'Reilly (2009) Nat. Rev. Immunol. **9**:514.
2. Strasser, A. *et al.* (2009) Immunity **30**:180.
3. Ashkenazi, A. and V. Dixit (1999) Curr. Opin. Cell Biol. **11**:255.
4. SwissProt accession P25445.
5. Liu, C. *et al.* (1995) Biochem. J. **310**:957.
6. Papoff, G. *et al.* (1996) J. Immunol. **156**:4622.
7. Thorburn, A. (2003) Cellular Signaling **16**:139.
8. Barreiro, R. *et al.* (2004) J. Immunol. **173**:1519.
9. Ferguson, T.A. and T.S Griffith (2006) Immunol. Rev. **213**:228.

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