Animal-Free FasL Protein, Human (His)

Cat. No.:	HY-P700052AF
Synonyms:	soluble Fas Ligand (sFasL); TNFSF6; CD95L; Apo I Ligand; APTL; APT1LG1; CD178
Species:	Human
Source:	E. coli
Accession:	P48023 (Q130-L281)
Gene ID:	356
Molecular Weight:	Approximately 17.31 kDa

PROPERTIES

AA Sequence	Q I G H P S P P P E K K E L R K V A H L T G K S N S R S M P L E W E D T Y G I V L L S G V K Y K K G G L V I N E T G L Y F V Y S K V Y F R G Q S C N N L P L S H K V Y M R N S K Y P Q D L V M M E G K M M S Y C T T G Q M W A R S S Y L G A V F N L T S A D H L Y V N V S E L S L V N F E E S Q T F F G L Y K L
Biological Activity	Measure by its ability to induce apoptosis in Jurkat cells. The ED_{50} for this effect is <1 ng/mL. The specific activity of recombinant human FasL is >1 x 10^6 IU/mg
Appearance	Lyophilized powder.
Formulation	Lyophilized from a solution containing 1X PBS, pH 8.0.
Endotoxin Level	<0.1 EU per 1 μg of the protein by the LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	Fas Ligand (FasL; FASLG; CD95L), is a ligand for TNFRSF6/FAS belonging to the tumor necrosis factor (TNF). FasL is a type II transmembrane protein, riggering apoptosis of lymphocytes[1]. FasL is expressed on a variety of cell types, including T cells, natural killer (NK) cells, monocytes, neutrophils, breast epithelial cells, and vascular endothelial cells[2]. FasL exerts different biological activity by cleaved into 4 isoforms including membrane form, soluble form, ADAM10-processed FasL form (APL) and SPPL2A-processed FasL form (SPA). Among them, the membrane-bound form and a soluble

form generated by proteolytic action of matrix metalloproteinases (MMP)[2].
FasL or soluble FasL binding to Fas results in receptor aggregation and in the interaction of a protein called Fas-associated death domain with the Fas cytoplasmic tail. The interaction triggers a cascade of intracellular events, including the activation of the IL-1-converting enzyme-like cysteine protease (caspase 8), that ultimately leads to nucleoprotein cleavage, DNA fragmentation, and cell apoptosis[5].
The loss of function due to mutations in murine FasL, murine Fas, human Fas, or human FasL leads to lymphoproliferation, lymphadenopathy, and autoimmune diseases[1][3].
Meanwhile, defective activation-induced cell death (AICD) results in spontaneous mutation of Fas and FasL genes in mice with lupus-like autoimmune disease[3].
Human Fas Ligand also involves in Jurkat cell apoptosis and binds TNFRSF6B/DcR3 to bolck apoptosis, which is a decoy receptor of apoptosis termination[2].
FasL is widely found in different animals, while the sequence in Human is different from Rat and Mouse with similarity of 77.26% and 78.06%, respectively.

REFERENCES

[1]. Schneider P, et al. Characterization of Fas (Apo-1, CD95)-Fas ligand interaction. J Biol Chem. 1997 Jul 25;272(30):18827-33.

[2]. Liu W, et al. Crystal Structure of the Complex of Human FasL and Its Decoy Receptor DcR3. Structure. 2016 Nov 1;24(11):2016-2023.

[3]. Martínez-Lorenzo MJ, et al. Release of preformed Fas ligand in soluble form is the major factor for activation-induced death of Jurkat T cells. Immunology. 1996 Dec;89(4):511-7.

[4]. Shudo K, et al. The membrane-bound but not the soluble form of human Fas ligand is responsible for its inflammatory activity. Eur J Immunol. 2001 Aug;31(8):2504-11.

[5]. Puppo F, et al. Fas, Fas ligand, and transfusion immunomodulation. Transfusion. 2001 Mar;41(3):416-8.

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