Fas Ligand Protein, Human (HEK293, His)

Cat. No.:	HY-P72658
Synonyms:	Tumor necrosis factor ligand superfamily member 6; APTL; CD95-L; Fas ligand; FasL; CD178; TNFSF6
Species:	Human
Source:	HEK293
Accession:	P48023 (P134-L281)
Gene ID:	356
Molecular Weight:	20-30 kDa

PROPERTIES

AA Sequence	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	Loaded Human FAS-Fc on Protein A Biosensor, can bind Human Fas Ligand-His with an affinity constant of 2.82 nM as determined in BLI assay.
Appearance	Solution
Formulation	Supplied as a 0.2 μm filtered solution of PBS, pH 7.4.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	N/A.
Storage & Stability	Stored at -80°C for 1 year. It is stable at -20°C for 3 months after opening. It is recommended to freeze aliquots at -80°C for extended storage. Avoid repeated freeze-thaw cycles.
Shipping	Shipping with dry ice

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[3]. 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)[3].
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[6].
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[3].
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

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[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.

[6]. Ottonello L, et al. Soluble Fas ligand is chemotactic for human neutrophilic polymorphonuclear leukocytes. J Immunol. 1999 Mar 15;162(6):3601-6.

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