Fas Ligand Protein, Mouse (P.pastoris, His)

Cat. No.:	HY-P74157
Synonyms:	Tumor necrosis factor ligand superfamily member 6; APTL; CD95-L; Fas ligand; FasL; CD178; TNFSF6
Species:	Mouse
Source:	P. pastoris
Accession:	Q544E9 (P132-L279)
Gene ID:	14103
Molecular Weight:	Approximately 18.2 kDa

PROPERTIES

Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/μg, determined by 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[4].
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 Mouse is highly similar to Rat (91.37%), but very different from Human with similarity of 78.06%.

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

[5]. Miwa K, et al. Caspase 1-independent IL-1beta release and inflammation induced by the apoptosis inducer Fas ligand. Nat Med. 1998 Nov;4(11):1287-92.

[6]. 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.

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