Fas/CD95 Protein, Mouse (HEK293, His)

Cat. No.:	HY-P70330
Synonyms:	rMuTumor necrosis factor receptor superfamily member 6//TNFRSF6, His; Tumor necrosis factor receptor superfamily member 6; Apo-1 antigen; Apoptosis-mediating surface antigen FAS; FASLG receptor; CD95; Fas; TNFRSF6
Species:	Mouse
Source:	HEK293
Accession:	P25446 (Q22-R169)
Gene ID:	14102
Molecular Weight:	25-35 kDa

PROPERTIES

AA Sequence	Q G T N S I S E S L K L R R R V R E T D K N C S E G L Y Q G G P F C C Q P C Q P G K K K V E D C K M N G G T P T C A P C T E G K E Y M D K N H Y A D K C R R C T L C D E E H G L E V E T N C T L T Q N T K C K C K P D F Y C D S P G C E H C V R C A S C E H G T L E P C T A T S N T N C R K Q S P R N R
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4.
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. For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).
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 receptor is the receptor for TNFSF6/FASLG, also known as apoptosis-mediating surface antigen FAS and Apo-1 antigen. It is a cell-surface protein that mediates apoptosis upon ligation with Fas ligand. Fas receptor belongs to tumor necrosis factor receptor superfamily, there are 7 isoforms produced by alternative splicing, some of which are candidates for nonsense-mediated mRNA decay (NMD). The Fas gene is expressed in several tissues in human and mouse, including thymus, spleen, ovary and heart, and on a number of cell types, including activated T- and B-lymphocytes. Isoform 1 and isoform 6 are expressed at equal levels in resting peripheral blood mononuclear cells. After activation there is an increase in isoform 1 and decrease in the levels of isoform 6[1]. Fas receptor contains a death domain. It has been shown to play a central role in the physiological regulation of programmed cell death, and has been implicated in the pathogenesis of

various malignancies and diseases of the immune system. It interacts with its ligand to allow the formation of a death-inducing signaling complex that includes Fas-associated death domain protein (FADD), caspase 8, and caspase 10[2]. To be specific, the autoproteolytic processing of the caspase in the complex triggers a downstream caspase cascade, including activation of the acidic sphingomyelinase, consumption of sphingomyelin, release of ceramide, and subsequent activation of JNK and p38-K. Thus, Fas receptor acts fucntion via caspase's regulation and leads to apoptosis[3]. Moreover, the signaling initiated from Fas is mediated by mitogen activated protein kinases (MAPKs) including extracellular-signal-regulated kinase (ERK) and c-Jun N-terminal kinase (JNK) which induce subsequent activation of NF-κB. Meanwhile, stimulation of Fas induced the expression of pro-inflammatory mediators such as matrix metalloproteinase (MMP)-9 and IL-8[4]. The amino acid sequence of human Fas protein has low homology with that of rat and mouse, and the similarity rate is 49.54% and 48.93%, respectively.

REFERENCES

[1]. Liu C, et al. Differential expression of human Fas mRNA species upon peripheral blood mononuclear cell activation. Biochem J. 1995 Sep 15;310 (Pt 3)(Pt 3):957-63.

[2]. Screaton RA, et al. Fas-associated death domain protein interacts with methyl-CpG binding domain protein 4: a potential link between genome surveillance and apoptosis. Proc Natl Acad Sci U S A. 2003 Apr 29;100(9):5211-6.

[3]. Brenner B, et al. Fas/CD95/Apo-I activates the acidic sphingomyelinase via caspases. Cell Death Differ. 1998 Jan;5(1):29-37.

[4]. Lee SM, et al. Stimulation of Fas (CD95) induces production of pro-inflammatory mediators through ERK/JNK-dependent activation of NF-κB in THP-1 cells. Cell Immunol. 2011;271(1):157-62.

[5]. Peng SL, et al. A tumor-suppressor function for Fas (CD95) revealed in T cell-deficient mice. J Exp Med. 1996 Sep 1;184(3):1149-54.

[6]. Mogil RJ, et al. Fas (CD95) participates in peripheral T cell deletion and associated apoptosis in vivo. Int Immunol. 1995 Sep;7(9):1451-8.

Caution: Product has not been fully validated for medical applications. For research use only.

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