TNF RII/TNFRSF1B Protein, Mouse (HEK293, Fc)

Cat. No.:	HY-P76108
Synonyms:	TNF-R2; CD120b; Tumor necrosis factor receptor superfamily member 1b; p75; Tnfr2
Species:	Mouse
Source:	HEK293
Accession:	P25119 (M1-G258)
Gene ID:	21938
Molecular Weight:	Approximately 65 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	TNFRII (TNFRSF1B) protein is a single-pass type I membrane protein belonging to the tumor necrosis factor (TNF) family. TNFRII is the major signaling receptor for TNF-α. TNFRII protein is highly regulated and typically found in immune system cells[1]. The amino acid sequence of mouse TNFRII protein has low homology between human and rhesus macaque TNFRII protein (less than 85%). TNFRII induces apoptosis. TNFRII does not directly engage the apoptotic program, but relies on the induction of endogenous, membrane-bound TNF, which subsequently activates TNFRI. TNFRII stimulates the action of the endogenously produced membrane-bound TNF on TNFRI is drastically enhanced. TNFRII competes with TNFRI for the recruitment of newly synthesized TRAF2-bound anti-apoptotic factors, thereby promoting the formation of a caspase-8-activating TNFRI complex. TNFRII competes with TNFRI for binding of TRAF2 and the TRAF2-associated anti-apoptotic cIAP1 and cIAP2 proteins. cIAP1-initiated degradation of TRAF2, which in turn enhances receptor competition for the remaining TRAF2, cIAP1 and cIAP2 molecules. cIAP1 would have an anti-apoptotic function upon recruitment into the TNFRI signalling complex, but would switch to a net proapoptotic function upon recruitment into the TNFRII signalling complex[1][2][3].

REFERENCES

[1]. Wajant H, et, al. Tumor necrosis factorsignaling. Cell Death Differ. 2003 Jan;10(1):45-65.

[2]. Fotin-Mleczek M, et, al. Apoptoticcrosstalk of TNF receptors: TNF-R2-induces depletion of TRAF2 and IAP proteinsand accelerates TNF-R1-dependent activation of caspase-8. J Cell Sci. 2002 Jul1;115(Pt 13):2757-70.

[3]. Masli S, et, al. Anti-inflammatory effectsof tumour necrosis factor (TNF)-alpha are mediated via TNF-R2 (p75) intolerogenic transforming growth factor-beta-treated antigen-presenting cells.Immunology. 2009 May;127(1):62-72.

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

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