TNFSF3/Lymphotoxin Beta Protein, Cynomolgus (sf9, His)

Cat. No.:	HY-P76482
Synonyms:	Lymphotoxin-beta; LT-beta; Tumor necrosis factor C; TNF-C; LTB; TNFSF3
Species:	Cynomolgus
Source:	Sf9 insect cells
Accession:	XP_003897389 (Q49-G244)
Gene ID:	101013049
Molecular Weight:	Approximately 22.8 kDa.

PROPERTIES

Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of 20 mM Tris, 500 mM NaCl, 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	LT is expressed by T, B, natural killer (NK)- and lymphoid tissue-inducer cells. While, Lymphotoxin Beta is expressed on inflammatory cells, LPCs, and occasional small hepatocytes adjacent to fibrous septa[1][2]. The amino acid sequence of human Lymphotoxin Beta protein has low homology to mouse Lymphotoxin Beta protein. While human Lymphotoxin Beta shares 96.72% aa sequence identity with Rhesus macaque Lymphotoxin Beta protein. Lymphotoxin-β (LTβ) is exclusively anchored in the membrane as a type II transmembrane protein, binding LTα to form membrane-anchored heterotrimers (LTα1β2 and LTα2β1). LTα1β2 triggers LTβR, whereas LTα2β1 was reported to bind TNFR1 and TNFR2, LIGHT (TNFSF14) is an alternative ligand for the LTβR. Besides, LTαβ heterotrimers activate canonical/noncanonical nuclear factor-κB (NF-κB) signaling, a significant regulator of innate and adaptive immune responses, cell survival or apoptosis, cellular stress responses, development and maintenance of lymphoid organs[1]. Lymphotoxin-β (LTβ) is a proinflammatory cytokine of the tumor necrosis factor (TNF) family. Lymphotoxin-β activates NF-κB signaling and shows anti-inflammatory, anti-cancer activity[1]. Lymphotoxin-β increases the expression of IκBα mRNA and protein in hepatic stellate cells, and regulates hepatic stellate cell function and wound healing in a murine model of chronic liver injury[2]. Lymphotoxin-β promotes the mRNA expression of pro-inflammatory cytokines TNFα and IL-1β, enhances the mRNA expression of RelA, and may be a potential therapeutic target for bladder cancer[3].

REFERENCES

[1]. Wolf MJ, et al. The unexpected role of lymphotoxin beta receptor signaling in carcinogenesis: from lymphoid tissue formation to liver and prostate cancer development. Oncogene. 2010 Sep 9;29(36):5006-18.

[2]. Ruddell RG, et al. Lymphotoxin-beta receptor signaling regulates hepatic stellate cell function and wound healing in a murine model of chronic liver injury. Hepatology. 2009 Jan;49(1):227-39.

[3]. Shen M, et al. Lymphotoxin β receptor activation promotes mRNA expression of RelA and pro-inflammatory cytokines TNFα and IL-1β in bladder cancer cells. Mol Med Rep. 2017 Jul;16(1):937-942.

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