LIGHT/TNFSF14 Protein, Mouse (sf9, His, solution)

Cat. No.:	HY-P73837A
Synonyms:	Tumor necrosis factor ligand superfamily member 14; TNFSF14; HVEM-L; LIGHT
Species:	Mouse
Source:	Sf9 insect cells
Accession:	Q9QYH9 (D72-V239)
Gene ID:	50930
Molecular Weight:	Approximately 20.5 kDa

PROPERTIES

Appearance	Solution.
Formulation	Supplied as a 0.2 μm filtered solution of 20 mM Tris, pH 8.0, 500 mM NaCl, 10 % gly.
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

LIGHT/TNFSF14 is a type II transmembrane protein produced by activated T cells, belongs to tumor necrosis factor (TNF) family. LIGHT/TNFSF14 is a TNFRSF14/HVEM (herpesvirus entry mediator) ligand, engages the receptor for the LTalphabeta heterotrimer but does not form complexes with either secreted lymphotoxin alpha (LTalpha) or LTbeta[1].

LIGHT/TNFSF14 is predominantly expressed in the spleen but also found in the brain. It is weakly expressed in peripheral lymphoid tissues and in heart, placenta, liver, lung, appendix, and kidney, and no expression seen in fetal tissues, endocrine glands, or nonhematopoietic tumor lines[1].

LIGHT/TNFSF14 has a transmemberane, thus it can be leaved into 2 chains: membrane form and soluble form. The soluble form of isoform 1 derives from the membrane form by proteolytic processing.

In tumor immunology, TNFSF14/LIGHT also serves as a novel immune checkpoint molecule for glioblastoma multiforme (GBM), as well as lung carcinoma, breast carcinoma, cervical cancer, and prostate cancer. TNFSF14/LIGHT can stimulate NK cells to produce IFNγ via nuclear factor-κB (NFκB) RelA/p50 signaling. TNFSF14/LIGHT sustains the function of $CD8^+$ effector T cells, trigger apoptosis of various tumor cells[2].

In cell signaling, TNFSF14/LIGHT binds to lymphotoxin-β receptor (LTβR) and HVEM for activating both of them, and disrupts the HVEM-BTLA complex in surface-bound form, and facilitates HVEM-BTLA complex formation in the soluble form[2].

TNFSF14/LIGHT promotes an inflammatory esophageal fibroblast in vitro via a LTβR-NIK-p52 NF-κB dominant pathway with promoting inflammatory gene expression and down-regulating homeostatic factors including WNTs, BMPs and type 3

semaphorins[3].
Beside that, TNFSF14/LIGHT protein is a costimulatory factor for the activation of lymphoid cells and as a deterrent to infection by herpesvirus. TNFSF14/LIGHT also prevents tumor necrosis factor alpha mediated apoptosis in primary hepatocyte[4][5].

REFERENCES

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