

## Product Data Sheet

## LIGHT/TNFSF14 Protein, Human (HEK293, His)

Cat. No.:	HY-P72522
Synonyms:	Tumor necrosis factor ligand superfamily member 14; TNFSF14; HVEM-L; LIGHT
Species:	Human
Source:	HEK293
Accession:	AAC39563.1 (L83-V240)
Gene ID:	8740
Molecular Weight:	18-24 kDa

PROPERTIES				
AA Sequence	LIOERRSHEV	ΝΡΑΑΗΙΤGΑΝ	SSITGSGGPI	
		LVVTKAGYYY	IYSKVQLGGV	GCPLGLASTI
	THGLYKRTPR	YPEELELLVS	QQSPCGRATS	SSRVWWDSSF
	LGGVVHLEAG	EKVVVRVLDE	RLVRLRDGTR	SYFGAFMV
Appearance	Lyophilized powder.			
Formulation	Lyophilized from a 0.2 μm	filtered solution of PBS, pH	7.4.	
Endotoxin Level	<1 EU/µg, determined by L	AL method.		
Reconsititution	It is not recommended to I	reconstitute to a concentra	tion less than 100 μg/mL in d	dH <sub>2</sub> O. For long term storage
	recommended to add a ca	rrier protein (0.1% BSA, 5%	HSA, 10% FBS or 5% Trehald	ose).
Storage & Stability	Stored at -20°C for 2 years	After reconstitution it is st	able at 4°C for 1 week or -20	C for longer (with carrier pro
Storage & Stability	recommended to freeze al	iquots at -20°C or -80°C for	extended storage.	e for tonger (with earlier pro
Shipping	Room temperature in cont	inental US; may vary elsew	here.	

## 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 <sup>[1]</sup> . 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 <sup>[1]</sup> .
	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<sup>+</sup> effector T cells, trigger apoptosis of various tumor cells<sup>[2]</sup>. In cell signaling, TNFSF14/LIGHT binds to lymphotoxin-β receptor (LTβR) and HVEM for activating both of them, and disrupts the MISTA PTI A second for the second for the MISTA PTI A second for the function of CM<sup>2</sup>.

the HVEM-BTLA complex in surface-bound form, and facilitates HVEM-BTLA complex formation in the soluble form<sup>[2]</sup>. 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<sup>[3]</sup>.

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<sup>[4][5]</sup>.

## REFERENCES

[1]. Mauri DN, et al. LIGHT, a new member of the TNF superfamily, and lymphotoxin alpha are ligands for herpesvirus entry mediator. Immunity. 1998 Jan;8(1):21-30.

[2]. Han M, et al. Comprehensive characterization of TNFSF14/LIGHT with implications in prognosis and immunotherapy of human gliomas. Front Immunol. 2022 Oct 20;13:1025286.

[3]. Manresa MC, et al. LIGHT controls distinct homeostatic and inflammatory gene expression profiles in esophageal fibroblasts via differential HVEM and LTßR-mediated mechanisms. Mucosal Immunol. 2022 Feb;15(2):327-337.

[4]. Hou Y, et al. Dual Roles of Tumor Necrosis Factor Superfamily 14 in Antiviral Immunity. Viral Immunol. 2022 Nov;35(9):579-585.

[5]. Miao X, et al. HES5-mediated repression of LIGHT transcription may contribute to apoptosis in hepatocytes. Cell Death Discov. 2021 Oct 23;7(1):308.

[6]. Zhang H, et al. TNFSF14, a novel target of miR-326, facilitates airway remodeling in airway smooth muscle cells via inducing extracellular matrix protein deposition and proliferation. Kaohsiung J Med Sci. 2020 Jul;36(7):508-514.

[7]. Holmes TD, et al. Licensed human natural killer cells aid dendritic cell maturation via TNFSF14/LIGHT. Proc Natl Acad Sci U S A. 2014 Dec 30;111(52):E5688-96.

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