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LIGHT Antibody [7B9F12]

Catalog # ASC12156

Product Information

Application	WB, IHC-P, IF, ICC, E
Primary Accession	O43557
Other Accession	NP_003798
Host	Mouse
Clonality	Monoclonal
Isotype	IgG2b
Clone Names	TNFSF14
Calculated MW	26350

Additional Information

Gene ID	8740
Alias Symbol	TNFSF14
Other Names	LIGHT Antibody: TNFSF14, LTg, CD258, HVEML, HVEM ligand
Reconstitution & Storage	LIGHT antibody can be stored at 4˚C for three months and -20˚C, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.
Precautions	LIGHT Antibody [7B9F12] is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	TNFSF14
Synonyms	HVEML, LIGHT
Function	Cytokine that binds to TNFRSF3/LTBR. Binding to the decoy receptor TNFRSF6B modulates its effects. Acts as a ligand for TNFRSF14/HVEM (PubMed:10754304, PubMed:9462508). Upon binding to TNFRSF14/HVEM, delivers costimulatory signals to T cells, leading to T cell proliferation and IFNG production (PubMed:10754304).
Cellular Location	[Tumor necrosis factor ligand superfamily member 14, membrane form]: Cell membrane; Single-pass type II membrane protein [Isoform 2]: Cytoplasm.
Tissue Location	Predominantly expressed in the spleen but also found in the brain. 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.

Background

LIGHT Antibody: LIGHT, also known as Tumor Necrosis Factor Superfamily member 14 (TNFSF14), is a co-stimulatory molecule that can regulate T-cell activation (1) and has recently been identified as an immune checkpoint protein. LIGHT binds to two different receptors, Herpes Virus Entry Mediator (HVEM) and Lymphotoxin beta Receptor (LTβR). While LIGHT binding to HVEM delivers a co-stimulatory signal to T cells (1), LIGHT binding to LTβR is critical for the formation of lymphoid structures which can stimulate T cell infiltration and activation of a tumor microenvironment, leading to rapid T cell-mediated tissue destruction (2). It has been shown that targeted delivery of LIGHT to tumors, thereby causing the T cell infiltration of the tumor, can enhance the response of the PD-1/PD-L1 checkpoint blockade anti-cancer therapy (3), suggesting that LIGHT may become a potent tool in anti-cancer treatment.

References

Wang Y, Zhu M, Miller M, et al. Immunoregulation by tumor necrosis factor superfamily member LIGHT. Immunological Reviews 2009; 229:232–43.Lee Y, Chin RK, Christiansen P, et al. Recruitment and activation of naive T cells in the islets by lymphotoxin beta receptor-dependent tertiary lymphoid structure. Immunity 2006; 25:499-509.Tang H, Wang Y, Chlewicki LK, et al. Facilitating T cell infiltration in tumor microenvironment overcomes resistance to PD-L1 blockade. Cancer Cell 2016; 29:285-296.