10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

# LIGHT Antibody [7B9E12]

Catalog # ASC12158

## Product Information

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| **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

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| **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 [7B9E12] is for research use only and not for use in diagnostic or therapeutic procedures. |

## Protein Information

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| **Name** | TNFSF14 |
| **Synonyms** | HVEML, LIGHT |
| **Function** | Cytokine that binds to TNFRSF3/LTB and TNFRSF14/HVEM and plays major roles in immune system regulation (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). Binds to the decoy receptor TNFRSF6B thereby disrupts the activation signal initiated by TNFSF14, resulting in reduced T-cell activation (PubMed:25087510). |
| **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.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.