

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human LIGHT/TNFSF14 in direct ELISAs.
Source	Monoclonal Mouse IgG ₁ Clone # 115515R
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human LIGHT/TNFSF14 Asp74-Val240 Accession # O46557
Conjugate	Alexa Fluor 405 Excitation Wavelength: 405 nm Emission Wavelength: 421 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

ELISA Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Human LIGHT, also known as TNFSF14, is a type II membrane protein that is a member of the TNF superfamily. LIGHT is an acronym which stands for "is homologous to lymphotoxins, exhibits inducible expression, and competes with HSV glycoprotein D for HVEM, a receptor expressed by T lymphocytes". LIGHT has also been called HVEM-L and LT-γ. LIGHT is a 240 amino acid (aa) protein that contains a 37 aa cytoplasmic domain, a 22 aa transmembrane region, and a 181 aa extracellular domain. Similar to other TNF ligand family members, LIGHT is predicted to assemble as a homotrimer. LIGHT is produced by activated T cells and was first identified by its ability to compete with HSV glycoprotein D for HVEM binding. LIGHT has also been shown to bind to the lymphotoxin beta receptor (LTβR) and the decoy receptor (DcR3/TR6). LIGHT overexpression in tumor cells induces apoptosis, which can be enhanced by IFN-γ.

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