

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human LILRB4/CD85k/ILT3 in direct ELISA.
Source	Monoclonal Mouse IgG _{2A} Clone # 1057706
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line, NS0-derived recombinant human LILRB4/CD85k/ILT3 protein Pro17-His257 Accession # ABM86208
Conjugate	Alexa Fluor 532 Excitation Wavelength: 534 nm Emission Wavelength: 553 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.

Flow Cytometry	Optimal dilution of this antibody should be experimentally determined.
Immunocytochemistry	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

ILT3, also known as CD85k and LIR-5, is an approximately 60 kDa transmembrane glycoprotein that negatively regulates immune cell activation (1). Mature human ILT3 consists of a 238 amino acid (aa) extracellular domain with two Ig-like domains, a 21 aa transmembrane segment, and a 168 aa cytoplasmic domain with 3 immunoreceptor tyrosine-based inhibitory motifs (ITIM) (2). Alternative splicing of human ILT3 generates an isoform that lacks the first ITIM and a secreted isoform that circulates in the serum of cancer patients (3, 4). ILT3 is expressed on dendritic cells (DC), monocytes, macrophages, and vascular endothelial cells (EC) (2, 5, 6). Ligation of ILT3 triggers ITIM-mediated inhibition of cell-activating signaling, leading to enhanced immune tolerance and reduced allogeneic graft rejection (2, 4, 7, 8). Soluble ILT3 induces the differentiation of CD8⁺ T suppressor cells (Ts) that can inhibit the effector functions of CD4⁺ Th cells and CD8⁺ CTL (4, 7, 9). In turn, CD8⁺ Ts cells induce ILT3 up-regulation and a tolerogenic phenotype in monocytes, DC, and EC (5, 6, 8, 10, 11).

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