

DESCRIPTION

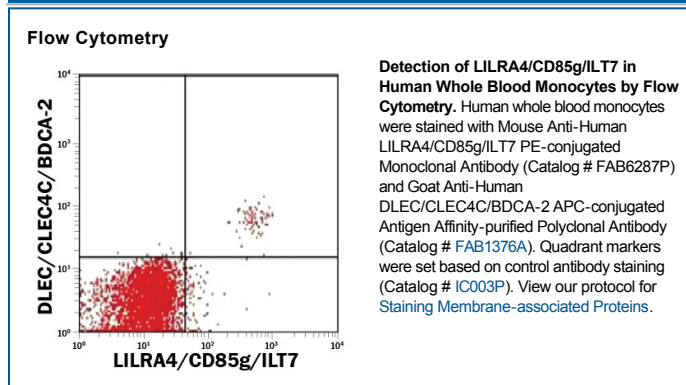
Species Reactivity	Human
Specificity	Detects human ILT7/CD85g in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant human (rh) LILRA5, ILT2, 3, 4, 5, or 6 is observed.
Source	Monoclonal Mouse IgG _{2A} Clone # 656688
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human ILT7/CD85g Glu24-Asn446 Accession # P59901
Conjugate	Phycoerythrin Excitation Wavelength: 488 nm Emission Wavelength: 565-605 nm
Formulation	Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *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.

	Recommended Concentration	Sample
Flow Cytometry	10 μ L/10 ⁶ cells	See Below

DATA



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. <ul style="list-style-type: none"> 12 months from date of receipt, 2 to 8 °C as supplied.

BACKGROUND

LILRA4 (Leukocyte Immunoglobulin-Like Receptor subfamily A member 4; also ILT-7 and CD85g) is a 35-38 kDa member of the Ig superfamily of molecules. Mature LILRA4 is a 476 amino acid (aa) type I transmembrane glycoprotein that possesses four C2-type Ig-like domains plus a nitrosylated tyrosine in its extracellular region (aa 24-446). There is one alternative start site at Met67 for human LILRA4, and rodent possesses no known gene counterpart for primate LILRA4. LILRA4 reveals a very restricted pattern of expression, being found only on/in microglia and immature plasmacytoid dendritic cells (pDC). On the cell surface, LILRA4 complexes with FcεRI, and appears to generally transmit downmodulating signals. Although controversial, one potential ligand for LILRA4 has been identified and found to be BST2/CD317. Initially, it was suggested that LILRA4 served as a brake on type I interferon (IFN-a/b) production. In theory, viral nucleic acid detection by TLR-7 or -9 in pDC would first induce IFN production, followed by IFN-stimulated upregulation of BST2 on fibroblasts and endothelium, and ending with BST2 binding to LILRA4 on pDC, downregulating the earlier virus-based stimulation of IFN secretion. While this may occur, an additional theory has been proposed for LILRA4 that is also based on TLR-7 and -9 signaling. In this case, LILRA4 is "activated" following TLR-7/-9 ligation and immature circulating pDC will differentiate into APCs. But in the absence of LILRA4 activation after TLR-7/-9 stimulation, pDC will differentiate into fully functional type I interferon producing cells.