Human LILRA4/CD85g/ILT7 Alexa Fluor® 647-conjugated Antibody

Monoclonal Mouse IgG_{2A} Clone # 656656

Catalog Number: FAB62871R

100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human LILRA4/CD85g/ILT7 in ELISA. No cross-reactivity with human LILRA1, LILRA2, LILRA3, LILRA5, LILRA6, LILRB2 and LILRB3 with ELISA was observed.
Source	Monoclonal Mouse IgG_{2A} Clone # 656656
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line, NS0derived human LILRA4/CD85g/ILT7 Glu24-Asn446 Accession # P59901
Conjugate	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 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.*

Neutralization	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

LILRA4, also known as ILT7 and CD85g, is an approximately 60-70 kDa variably glycosylated transmembrane protein that regulates immune cell activation (1). Mature human LILRA4 consists of a 423 amino acid (aa) extracellular domain (ECD) with four immunoglobulin-like domains, a 21 aa transmembrane segment, and a 32 aa cytoplasmic domain (2). Alternative splicing generates an additional isoform that lacks the signal peptide and a portion of the first Ig-like domain. LILRA4 is expressed on plasmacytoid dendritic cells (pDC) but is down-regulated in response to TLR9 signaling (3-5). Antibody mediated crosslinking of LILRA4 on pDC inhibits the production of type I interferons following TLR7 or TLR9 stimulation (3, 4, 6). It also blocks the up-regulation of CCR7 but enhances the up-regulation of Integrin β7 on TLR7/9-stimulated pDC (6). LILRA4 associates with the ITAM-containing adaptor protein Fcε RIγ (3, 4, 6), and this complex binds to cell surface BST2/Tetherin which is expressed on monocytes, plasmacytoid and myeloid dendritic cells, B cells, and activated $CD4^+$ and $CD8^+$ T cells (5, 8). This interaction inhibits the TLR-induced pDC production of type I interferons, IL-6, and TNF-α (8).

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