Cynomolgus Monkey LAG-3
Alexa Fluor® 350-conjugated Antibody

Recombinant Monoclonal Rabbit IgG Clone # 2561B
Catalog Number: FAB10395U
100 µg

DESCRIPTION

Species Reactivity	Cynomolgus Monkey
Specificity	Detects cynomolgus monkey LAG-3 in direct ELISAs.
Source	Recombinant Monoclonal Rabbit IgG Clone # 2561B
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Chinese Hamster Ovary cell line, CHO-derived cynomolgus monkey LAG-3 Val20-Leu450 Accession # XP_005570011.1
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 nm
Formulation	Supplied 0.2 mg/mL in a saline solution containing BSA and 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.*

	Recommended Concentration	Sample
Flow Cytometry	0.25-1 µg/10^6 cells	HEK293 Human Cell Line Transfected with Cynomolgus Monkey LAG-3 and eGFP

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

LAG-3 (Lymphocyte activation gene-3), designated CD223, is a type I transmembrane protein that is a member of the immunoglobulin superfamily (IgSF) (1, 2). LAG - 3 shares approximately 20% amino acid (aa) sequence homology with CD4, but has similar structure and binds to MHC class II with higher affinity, providing negative regulation of T cell receptor signaling (1, 2). The mature cynomolgus LAG-3 includes an extracellular domain (ECD) with four Ig-like domains, a transmembrane region and a highly charged cytoplasmic region. Within the ECD, cynomolgus LAG-3 shares 92%, 69% and 68% aa sequence identity with human, mouse and rat LAG-3, respectively. LAG-3 is expressed on activated CD4+ and $CD8^+$ T cells, NK cells, and plasmacytoid dendritic cells (pDC), but not on resting T cells (1-3). LAG-3 on activated $CD4^+CD25^+$ Treg cells plays a role in their suppressive activity (4). LAG-3 limits the expansion of activated T cells and pDC in response to selected stimuli (3-5). A soluble 54 kDa form, sLAG-3, can be shed by metalloproteinases ADAM10 and TACE/ADAM17 (6, 7). While monomeric sLAG-3 itself may be inactive, shedding allows for normal T cell activation by removing negative regulation (7). Binding of sLAG-3 to MHC class II molecules induces maturation of immature DC, and secretion of cytokines such as IFN-gamma and TNF-alpha by type 1 cytotoxic $CD8^+$ T cells and NK cells (8, 9). sLAG-3 has been used as a potential adjuvant to stimulate a cytotoxic anti-cancer immune response (9, 10). In mice, deletion of LAG-3 and another negative regulator, PD-1, facilitates anti-cancer response but also blocks self-tolerance and increases susceptibility to autoimmune diseases (11, 12). In humans, antibody-mediated down-regulation of LAG-3 and PD-1 allows more effective control of chronic malaria, while in NOD (non-obese diabetic) mice, deletion of LAG-3 alone accelerates diabetes (12-14). In addition, LAG-3 is an immune checkpoint protein that modulates T-cell activation and homeostasis and is a promising target for cancer immunotherapies (15, 16).

References:

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